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A TREATISE

ON

DISEASES

OF THE

NERVOUS SYSTEM.

LEEDS SCHOOL OF MEDICINE

BY

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WITH FORTY-FIVE ILLUSTRATIONS.

[illegible]

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PREFACE.

In the following work I have endeavored to present a Treatise on Diseases of the Nervous System, which, without being superficial, would be concise and explicit, and which, while making no claim to being exhaustive, would nevertheless be sufficiently complete for the instruction and guidance of those who might be disposed to seek information from its pages. How far I have been successful will soon be determined by the judgment of those more competent than myself to form an unbiassed opinion.

One feature I may, however, with justice claim for this work, and that is, that it rests to a great extent on my own observation and experience, and is therefore no mere compilation. The reader will readily perceive that I have views of my own on every disease considered, and that I have not hesitated to express them.

The treatise embraces an introductory chapter, which relates to the instruments and apparatus employed in the diagnosis and treatment of diseases of the nervous system, and five sections. Of these, the first treats of diseases of the brain; the second, diseases of the spinal cord; the third, cerebro-spinal diseases; the fourth, diseases of nerve-cells; and the fifth, diseases of the peripheral nerves. Diseases of the sympathetic nerve are at present so little un-

derstood, if even one is recognized, that I have, for the present, deferred their consideration.

I have also omitted several affections which, by some authors, are classed with diseases of the nervous system. The chief of these are cerebro-spinal meningitis, chronic alcoholic intoxication, and Graves's disease. The first is an epidemic febrile affection, similar in general features to typhus fever, and in which the inflammation of the meninges of the brain and spinal cord is altogether a secondary phenomenon; chronic alcoholic intoxication affects the nervous system in conjunction with the organism generally, and is no more entitled to be considered in a work like the present than would be chronic poisoning from opium, hashish, mercury, or other substance; and Graves's disease—although there is reason to believe that the sympathetic and pneumogastric nerves are implicated—is probably essentially an affection the starting-point of which is in the blood.

My thanks are due to my friend Dr. R. L. Parsons, Superintendent of the New York City Lunatic Asylum, for the opportunity of selecting, from a large number of photographs of the patients in the institution under his charge, such as appeared most fully to illustrate my remarks on insanity.

The fourth volume of Prof. Austin Flint, Jr.'s, "Physiology of Man," which will be published during the coming season, will, with the present treatise, constitute a complete work on "The Physiology and Pathology of the Nervous System."

162 West 34th Street,
New York, *April* 20, 1871.

CONTENTS.

PAGE

INTRODUCTION, ... 9

The Instruments and Apparatus employed in the Diagnosis and Treatment of Diseases of the Nervous System, . 9

SECTION I.

DISEASES OF THE BRAIN.

CHAP. I.—CEREBRAL CONGESTION, ... 33

Active Cerebral Congestion.—Passive Cerebral Congestion.

II.—CEREBRAL ANÆMIA, ... 61

III.—CEREBRAL HÆMORRHAGE, ... 74

IV.—MENINGEAL HÆMORRHAGE, ... 114

Hæmatoma of the Dura Mater.

V.—PARTIAL CEREBRAL ANÆMIA FROM OBLITERATION OF CEREBRAL ARTERIES, ... 119

Thrombosis.—Embolism.

VI.—CEREBRAL SOFTENING, ... 137

VII.—APHASIA, ... 166

VIII.—ACUTE CEREBRAL MENINGITIS, ... 219

IX.—CHRONIC CEREBRAL MENINGITIS, ... 231

X.—TUBERCULAR CEREBRAL MENINGITIS, ... 234

XI.—SUPPURATIVE ENCEPHALITIS OR CEREBRITIS, . 246

XII.—DIFFUSED CEREBRAL SCLEROSIS, ... 260

XIII.—MULTIPLE CEREBRAL SCLEROSIS, ... 278

XIV.—TUMORS OF THE BRAIN, ... 301

XV.—INSANITY, ... 324

General Principles.—Perceptional Insanity.—Intellectual Insanity.—Emotional Insanity.—Volitional Insanity.—Mania.—General Paralysis.—Idiocy and Dementia.

SECTION II.

DISEASES OF THE SPINAL CORD.

PAGE

CHAP. I.—SPINAL CONGESTION, ... 385
II.—SPINAL ANÆMIA, ... 396
Anæmia of the Posterior Columns.—Anæmia of the Antero-Lateral Columns.
III.—SPINAL HÆMORRHAGE—SPINAL MENINGEAL HÆMORRHAGE, ... 440
IV.—SPINAL MENINGITIS, ... 444
Acute Spinal Meningitis.—Chronic Spinal Meningitis.
V.—ACUTE MYELITIS, ... 456
VI.—SPINAL SOFTENING, ... 463
VII.—SCLEROSIS OF THE ANTERO-LATERAL COLUMNS OF THE SPINAL CORD, ... 471
VIII.—SCLEROSIS OF THE POSTERIOR COLUMNS OF THE SPINAL CORD (LOCOMOTOR ATAXIA), ... 484
IX.—TUMORS OF THE SPINAL CORD, ... 517
X.—SECONDARY DEGENERATIONS OF THE SPINAL CORD, . 523
XI.—TETANUS, ... 529

SECTION III.

CEREBRO-SPINAL DISEASES.

I.—HYDROPHOBIA, ... 545
II.—EPILEPSY, ... 560
III.—CATALEPSY, ... 590
IV.—ECSTASY, ... 597
V.—CHOREA, ... 600
VI.—HYSTERIA, ... 619
VII.—MULTIPLE CEREBRO-SPINAL SCLEROSIS, ... 637
VIII.—ATHETOSIS, ... 654

SECTION IV.

DISEASES OF NERVE-CELLS.

I.—ATROPHY AND DISAPPEARANCE OF TROPHIC NERVE-CELLS (PROGRESSIVE MUSCULAR ATROPHY), . 663

PAGE

CHAP. II.—ATROPHY AND DISAPPEARANCE OF MOTOR NERVE-CELLS, ... 677
Glosso-Labio-Laryngeal Paralysis.

III.—ATROPHY AND DISAPPEARANCE OF MOTOR AND TROPHIC NERVE-CELLS, ... 689
Organic Infantile Paralysis.—Hypertrophy of Muscular Connective Tissue.

IV.—FUNCTIONAL DERANGEMENTS OF MOTOR NERVE-CELLS, 706
Paralysis Agitans.—Writer's Spasm.—Lead Paralysis.

SECTION V.

DISEASES OF PERIPHERAL NERVES.

I.—PERIPHERAL PARALYSIS, ... 722
Facial Paralysis.

II.—PERIPHERAL SPASM, ... 731
Facial Spasm.—Torticollis.

III.—PERIPHERAL ANÆSTHESIA, ... 735
Anæsthesia of Cutaneous Nerves.—Anæsthesia of the Fifth Pair.

IV.—PERIPHERAL HYPERÆSTHESIA (NEURALGIA), . . 739
Neuralgia of the Fifth Pair of Nerves.—Cervico-Occipital Neuralgia.—Cervico-Brachial Neuralgia.—Dorso-Intercostal Neuralgia.—Sciatic Neuralgia.

INDEX, ... 750

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LIST OF ILLUSTRATIONS.

Fig.			Page
1.	Æsthesiometer,	*Hammond*,	16
2.	Dynamometer,	*Mathieu*,	19
3.	Dynamograph,	"	20
4.	Duchenne's Trocar,	*Hammond*,	21
5.	Electro-Magnetic Machine,	"	23
6.	Cerebral Convolutions,	*Broca*,	195
7.	Agraphia,	*Hammond*,	213
8.	Dynamographic Tracing of Patient affected with Multiple Cerebral Sclerosis,	"	284
9.	Dynamographic Tracing of Patient affected with Multiple Cerebral Sclerosis,	"	284
10.	Malignant Tumor of Brain,	*Otis*,	307
11.	Cerebral Aneurism caused by Embolus,	*Prof. W. R. Smith*,	316
12.	Intellectual Insanity,	*Hammond*,	350
13.	Emotional Insanity,	"	356
14.	Acute Mania, with Mental Exaltation,	"	362
15.	" " " Depression,	"	364
16.	" " " "	"	365
17.	Dynamographic Tracing of Patient affected with General Paralysis,	"	370
18.	General Paralysis,	"	372
19.	Idiocy,	"	373
20.	Dementia,	"	375
21.	Chronic Spinal Meningitis,	"	453
22.	Dynamographic Tracing of Patient affected with Posterior Spinal Sclerosis,	"	491
23.	Dynamographic Tracing of Patient affected with Posterior Spinal Sclerosis,	"	492
24.	Transverse Section of Spinal Cord,	*Lockhart Clarke*,	506
25.	Longitudinal Section of "	"	506

Fig.			Page
26.	Tumor of Spinal Cord,	*Charcot*,	519
27.	" " "	"	519
28.	Writing of Patient affected with Multiple Cerebro-Spinal Sclerosis,	*Hammond*,	651
29.	Athetosis,	"	657
30.	" (after photograph from Hubbard),	"	661
31.	Progressive Muscular Atrophy,	"	667
32.	Glosso-Labio-Laryngeal Paralysis,	"	680
33.	Writing of Patient with Glosso-Labio-Laryngeal Paralysis,	"	683
34.	Glosso-Labio-Laryngeal Paralylis,	"	684
35.	Muscle of Patient affected with Organic Infantile Paralysis,	"	693
36.	Muscle of Patient affected with Organic Infantile Paralysis,	"	694
37.	Muscle of Patient affected with Organic Infantile Paralysis,	"	694
38.	Muscle of Patient affected wieh Organic Infantile Paralysis,	"	694
39.	Muscle of Patient affected with Organic Infantile Paralysis,	"	695
40.	Muscle of Patient affected with Organic Infantile Paralysis,	"	698
41.	Muscle of Patient affected with Organic Infantile Paralysis,	"	699
42.	Hypertrophy of Muscular Connective Tissue,	"	702
43.	" " " "	"	702
44.	Muscle of Patient affected with Hypertrophy of Muscular Connective Tissue,	"	704
45.	Writing of Patient affected with Writer's Spasm,	"	712

LEEDS SCHOOL OF MEDICINE

DISEASES OF THE NERVOUS SYSTEM.

INTRODUCTION.

THE INSTRUMENTS AND APPARATUS EMPLOYED IN THE DIAGNOSIS AND TREATMENT OF DISEASES OF THE NERVOUS SYSTEM.

Diseases of the nervous system, like those of the heart, lungs, and larynx, require special means of investigation and treatment. In no department of medical science has progress been more decided during the last decade than in that class of affections considered in this treatise, and undoubtedly a great deal of the advancement is due to the instruments and apparatus by which scientific research in this direction has become practicable.

In the present chapter I propose to describe the instruments and apparatus employed in the diagnosis and treatment of diseases of the nervous system, and to explain the methods by which they are used.

THE OPHTHALMOSCOPE.

The ophthalmoscope consists essentially of a concave mirror perforated in the centre, and of a double-convex lens. Several modifications of this arrangement are in use, but the simplest instrument is, in my opinion, the best, and this is Liebreich's.

It consists of a polished steel mirror about one and three-

quarter inches in diameter, concave, and perforated in the centre by a hole about the one-twelfth of an inch in diameter. The edges of this aperture are bevelled so as to afford as little obstacle as possible to the passage of the rays of light to the eye of the observer.

The mirror is set into a bronze ring with a handle, and there is attached also to this ring a clip for holding a concave ocular lens, which in some conditions of refraction, either in the eye of the patient or that of the observer, is necessary in order to produce the necessary divergence of the parallel rays emanating from the patient's eye, and thus render the image of the fundus distinct. A direct image is thus obtained. The lamp, which should furnish a steady flame, is placed on the side of the patient's head corresponding to the eye to be examined, and the eye of the observer very close to that of the patient. This process gives a very satisfactory view of the fundus with the optic disk and retinal vessels, but requires care, and is more difficult than that by which the inverted image is obtained.

In this case the observer illuminates the fundus with the ophthalmoscopic mirror, and then interposes between the mirror and the eye a double-convex lens which he holds lightly between the thumb and finger, resting the ring finger on the forehead of the patient, so as to make the hand steady, and the little-finger being disengaged so as to be employed in raising the eyelid if necessary.

The object-lens should have a focal distance of about two inches, and it should be held so as to bring the focus on the pupil. The lamp is placed behind and a little to one side of the eye to be examined. In order to see the optic disk, the patient is told to look at the ear of the observer on the side opposite to the eye being examined. In this way the axis of vision is directed inward, and the optic disk readily brought into view.

These examinations are made in a room lighted only by the lamp used in the processes. It is sometimes necessary

to dilate the pupil with atropia, in order to obtain a view of the disk, but experience and tact will generally enable the observer to dispense with this rather disagreeable procedure.

Ophthalmoscopic examinations require the observer to possess a very thorough acquaintance with the anatomy of the eye, and also with the science of optics. Unless these qualifications are enjoyed, it will be much better to send the patient to a competent ophthalmic surgeon for an examination, than to rush to hasty conclusions based on the most thorough ignorance. The real value of ophthalmoscopy in diseases of the nervous system is in danger of being disregarded through the sciolism of pert pretenders, who read papers and write memoirs without ever having seen the optic disk to recognize it.

Bouchut [1] gives the following list of abnormal conditions which are of importance in the diagnosis of diseases of the nervous system:

Papillary congestion; peri-papillary congestion; papillary anæmia, partial or general; phlebo-retinal flexuosities; venous pulsation in the retinal veins; dilatations of the retinal veins; retinal varices; phlebo-retinal hæmostases; phlebo-retinal thromboses; phlebo-retinal aneurism; hæmorrhages into the retina and choroid. The diseases in which he thinks ophthalmoscopy is valuable as a diagnostic means are—the several varieties of cerebral meningitis; cerebral hæmorrhage; chronic encephalitis; cerebral softening; meningeal hæmorrhage; chronic hydrocephalus; tumors of the brain; contusion, commotion, and compression of the brain; general paralysis; atrophy of the brain; chronic myelitis; locomotor ataxia; tetanus; epilepsy; essential convulsions; insanity, and several others of less importance.

To these may be added cerebral congestion, general and partial; cerebral anæmia; and the various forms of sclerosis affecting the brain and spinal cord.

[1] Du Diagnostic des Maladies du Système Nerveux, par l'Ophthalmoscopie. Paris, 1866, p. 15.

CEPHALOHÆMOMETER.

Although this instrument is intended for experiments on the lower animals, it enables us to arrive at very definite conclusions relative to the condition of the cerebral circulation. I first described it in a paper read before the New York Medical Journal Association in 1868, and shortly afterward published in the *New York Medical Gazette*.[1] It was devised in somewhat different form, independently of each other, by Dr. S. Weir Mitchell and myself. The instrument consists of a brass tube which is received into a round hole made in the skull with a trephine. Both ends of this tube are open, but into the upper end is secured another brass tube, the lower opening of which is closed by a piece of very thin sheet India-rubber, and the upper opening by a brass cap, into which is fastened a glass tube. This inner arrangement contains colored water. To this glass tube a scale is affixed.

This second brass tube is screwed into the first till the thin India-rubber presses upon the dura mater, and the level of the colored water stands at 0, which is in the middle of the scale. Now, when the quantity of blood in the brain is increased, the liquid rises in the tube, being pressed upward by the elevation of the thin rubber closing the lower opening; when the quantity of blood is lessened, the liquid falls by its own gravity.

It was by this instrument that I was enabled to demonstrate, in the most conclusive manner, that during sleep the amount of blood circulating in the cerebral vessels is much less than during wakefulness.[2]

ÆSTHESIOMETER.

The æsthesiometer is an instrument for the purpose of determining the degree of tactile sensibility possessed by the

[1] Also, Journal of Psychological Medicine, January, 1869, p. 47.

[2] Sleep and its Derangements. Philadelphia, 1869, p. 317.

patient. The instrument was devised in 1858 by Dr. Sieveking,[1] of London. Its value in cases of aberrations of sensibility depends upon the fact, ascertained by Dr. E. H. Weber, that the capability of distinguishing two impressions made upon the skin simultaneously, varies in different regions of the body according to the distance they are apart. In sensitive regions, as the end of the finger, the two points of a pair of dividers can be distinguished at about the twelfth of an inch apart, while in the middle of the back only one point is felt, though they are two inches apart. In accordance with this principle, the æsthesiometer is used to determine the sensibility of the skin in various diseases, it being well known that this is subject to variation.

Thus when the sensibility is intact, two points, touching the back of the hand at the same time, can be distinguished as two points when separated an inch. If, in examining a patient, we should find that, when the two points were two inches apart, the patient felt but a single impression, we should know that he had lost sensibility in the cutaneous nerves of that part of the body.

Dr. Sieveking's æsthesiometer is nothing more than a beam-compass. It consists of a rod of bell-metal four inches in length, graduated into inches and tenths of an inch. At one end is a fixed steel point; another steel point is made to slide upon the beam, and can be fixed at any distance from the first by a screw which works at the top of the slide.

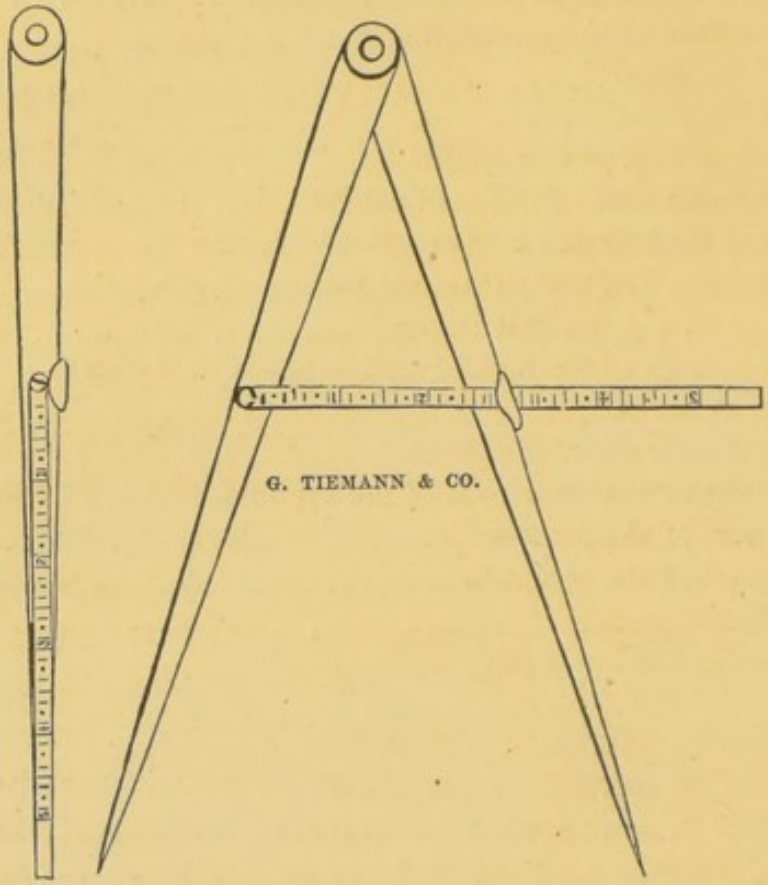
In 1861[2] I described an æsthesiometer which I believe was the first used in this country. It consisted of a pair of dividers, to one arm of which the arc of a circle, in brass, was affixed. This arc was divided so as to measure tenths of an inch. A short time since, I suggested to Mr. Stohlman, the instrument-maker, a modification of this instrument, which for convenience is, I think, superior to all

[1] Brit. and For. Med.-Chir. Rev., January, 1858, p. 281.

[2] A Clinical Lecture on Chronic Myelitis. Delivered in the Baltimore Infirmary, March 16, 1861. American Medical Times, January 8, 1861, p. 379.

others. This, as closed, for the pocket-case, and open, as in use, is seen in the accompanying woodcut (Fig. 1)[1] and need not be further described.

FIG. 1.



The minimum normal distances at which the two points of the æsthesiometer can be distinguished in different regions of the body are stated in the following table:[2]

Point of the tongue	½	a line.
Palmar surface of the third finger	1	"
Red surface of the lips	2	lines.
Palmar surface of second finger	2	"
Dorsal surface of third finger	3	"
Tip of the nose	3	"

[1] First described by me in the JOURNAL OF PSYCHOLOGICAL MEDICINE, October, 1868, p. 830.

[2] This table is quoted from Müller s Philosophy, translated by Baly. London, 1840, p. 752.

The palm over the heads of the metacarpal bones	3	lines.
Dorsum of tongue, one inch from the tip	4	"
Part of the lips covered by the skin	4	"
Border of the tongue, an inch from the tip	4	"
Metacarpal bone of the thumb	4	"
Extremity of the great-toe	5	"
Dorsal surface of the second finger	5	"
Palm of the hand	5	"
Skin of the cheek	5	"
External surface of the eyelids	5	"
Mucous membrane of the hard palate	6	"
Skin over the anterior surface of the zygoma	7	"
Plantar surface of the metatarsal surface of great-tòe	7	"
Dorsal surface of the first finger	7	"
On the dorsum of the hand over the heads of the metacarpal bones	8	"
Mucous membrane of the gums	9	"
Skin over the posterior part of the zygoma	10	"
Lower part of the forehead	10	"
Lower part of the occiput	12	"
Back of the hand	14	"
Neck under the lower jaw	15	"
Vertex	15	"
Skin over the patella	16	"
" " " sacrum	18	"
" " " acromion	18	"
The leg, near the knee and foot	18	"
Dorsum of the foot, near the toes	18	"
The skin over the sternum	20	"
" " " " five upper vertebræ	24	"
" " " " spine near the occiput	24	"
" " in the lumbar region	24	"
" " " " middle of the neck	30	"
" " over the middle of the back	30	"
The middle of the arm	30	"
" " " " thigh	30	"

THERMOMETER.

The thermometer is of use for the purpose of determining variations of temperature in different parts of the body. It should be graduated in tenths of a degree, and be held upon

the part subjected to examination so long as the mercury continues to rise or fall. Comparative determinations must be made under precisely similar conditions.

BECQUEREL'S DISKS.

By means of these little instruments very slight variations of temperature can be ascertained. They consist of an extremely thin plate of copper, about the size of a half-dime, soldered to a thin rod of bismuth. This latter is contained in a small tube of hard rubber furnished with a handle. The disks are two in number, and by means of delicate silk-covered wires are in communication with the poles of a galvanometer. If a lower extremity, for instance, is subjected to examination, one of the disks is placed upon it and the other upon the corresponding part of the other limb. If the temperature of both limbs be the same, the needle of the galvanometer remains quiet; if either be warmer than the other, the needle is deflected to the north or south according as one or the other limb has the higher temperature. By this apparatus very much less than the hundredth of a degree of temperature can be determined with absolute accuracy.[1]

THE DYNAMOMETER.

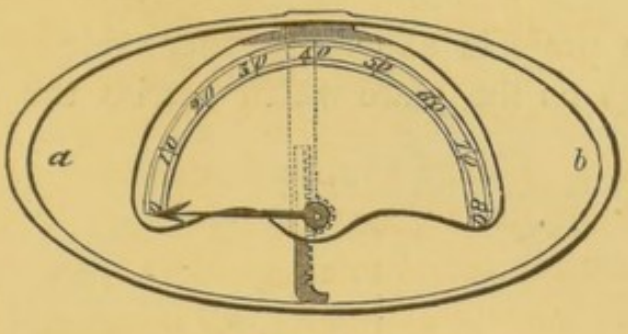
Several forms of an instrument for measuring the strength of patients have been devised. The best and most generally applicable is that of M. Mathieu, an instrument-maker of Paris. It is very simple, and for measuring the strength of the hands leaves nothing to be desired.

It consists, as is shown in the cut (Fig. 2), of an elliptical steel spring, to which is attached a semicircle of gilt brass, upon which a scale is marked. An indicator, terminating at one end in a cog-wheel, is capable of being moved freely around the arc of the circle by a steel arm, upon one side of which, cogs, fitting into those of the indicator, are cut. One

[1] See my memoir on the Pathology and Treatment of Organic Infantile Paralysis, in JOURNAL OF PSYCHOLOGICAL MEDICINE, No. 1, July, 1867, p. 53.

end of this arm (the lower) touches the elliptical spring, when the indicator points to the zero of the scale; a brass sheath upon the under side of the scale keeps this arm in place, at the same time allowing it to move freely.

FIG. 2.



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When the dynamometer is taken into the hand and pressed, the two sides of the spring are approximated and the steel arm, with the cogs being pushed by the lower side of the spring, turns the indicator. One great advantage of this instrument is that, when the pressure is taken off, the indicator does not return to the zero, but remains at the point to which it has been carried by the muscular power of the individual. We are thus enabled to see the extent of his strength, after he has made his effort, and do not have to watch him while he is using the instrument. It will also be seen that this dynamometer can be used to measure tactile force; for if two hooks with cords attached be fastened to the spring at the points *a* and *b*, traction on the cords will approximate the two sides of the ellipse, and thus push the steel arm so as to move the indicator as before.

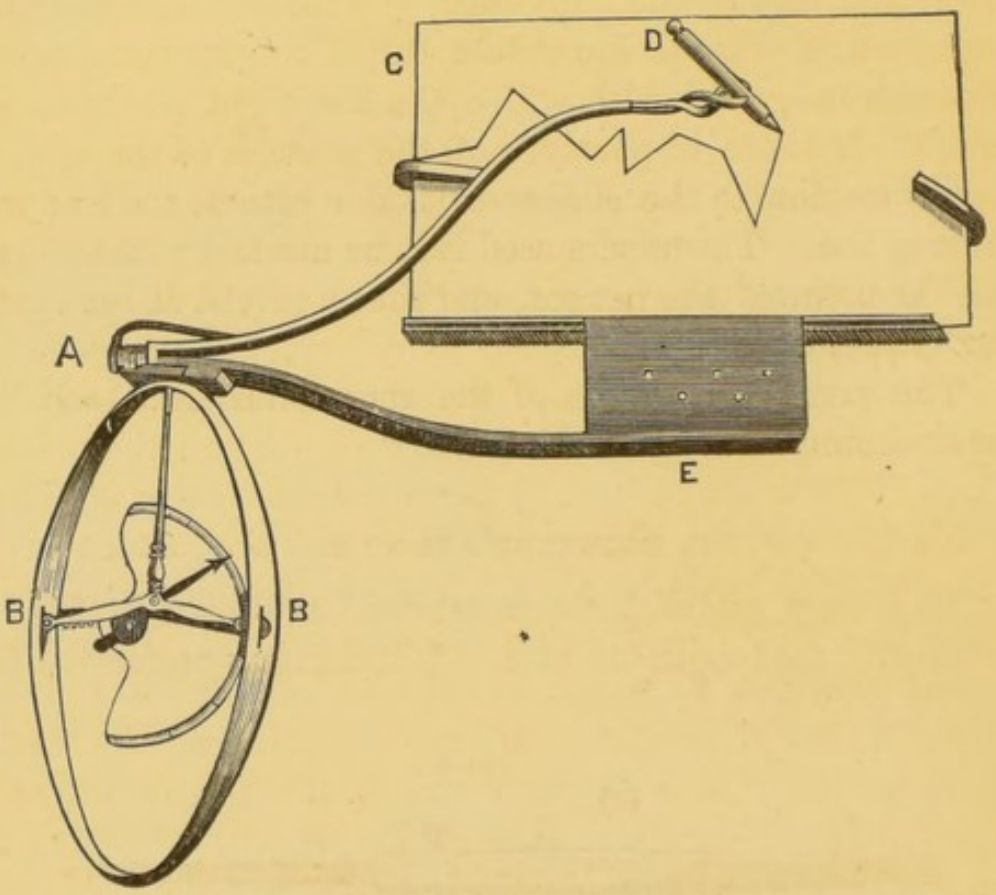
THE DYNAMOGRAPH.

This instrument, which is of great value in the diagnosis of diseases of the nervous system, is shown in Fig. 3.

It consists of the dynamometer B B, to which a toggle-joint, moving a steel rod, is attached. This steel rod plays through a hole in the end of the elliptical spring and moves the lever which raises the pencil D. At A is a screw which

varies the point at which the rod touches the lever, and thus increases or lessens the delicacy of the indications. C is a silvered plate upon which the paper is fastened by clips. To the lower part of this plate, a strip of gilt brass, with cogs cut in it, is attached. E is a gilt-brass box containing a watch movement like that of the sphygmograph. A cog-wheel which projects above the upper side of this box fits into the cogs on the plate which carries the paper. The

Fig. 3.



wheel for winding up the clock-work, and the wire for stopping it, or setting it in motion, are not seen in the figure, they being on the opposite side of the box.

To set the instrument in action, the sphygmograph movement is attached to the dynamometer at A. The clock-work is then wound up, and the plate holding the paper placed in the groove on top of the box E. The dynamometer is then grasped by the hand and squeezed firmly;

SCHOOL OF MEDICINE

the lever is thus moved, and the plate with the paper is carried along by the cog-wheel. As it moves, the pencil traces a line on the paper, the height and regularity of which depend upon the firmness and steadiness with which the dynamometer is pressed. As seen in the cut, the plate with the paper is in motion, and has about half completed its course. The patient should not look at the paper while using the instrument.

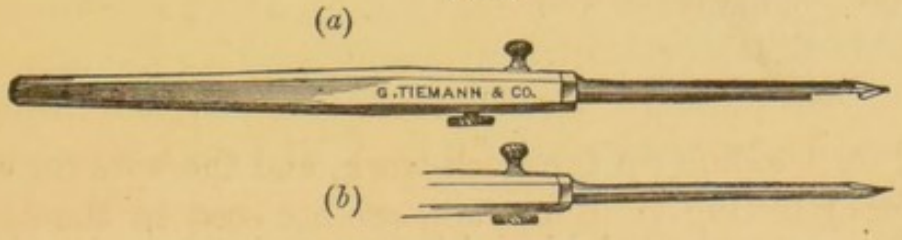
The dynamograph, therefore, writes down the muscular power and tone of the individual, and likewise indicates the perfection of what is sometimes called the muscular sense. A person in good health will make a straight line with the pencil. If there is paralysis of the muscles of the arm, or incoördination to the slightest possible extent, the line will be irregular. The papers used may be marked with the date and the name of the patient, and thus a record of his condition is preserved.

The pencil should be of the very softest lead, and the paper should be rough and unsized.[1]

DUCHENNE'S TROCAR.

This very useful little instrument is shown in Fig. 4. It is introduced open as at *a*. When it has perforated the

FIG. 4.



muscle under examination, the small button at the under part of the handle is pushed forward; this propels a half-cylinder of steel against the shoulder at the end of the tro-

[1] The first account of the use of the dynamograph was given by myself in the JOURNAL OF PSYCHOLOGICAL MEDICINE, January, 1868, p. 139.

car, and thus a small piece of muscle is detached and caught in the cavity. The lower figure *b* represents the instrument ready to be withdrawn. By drawing the button back, the bit of fibre can be taken out, and is then ready for microscopical examination.

ELECTRICAL APPARATUS.

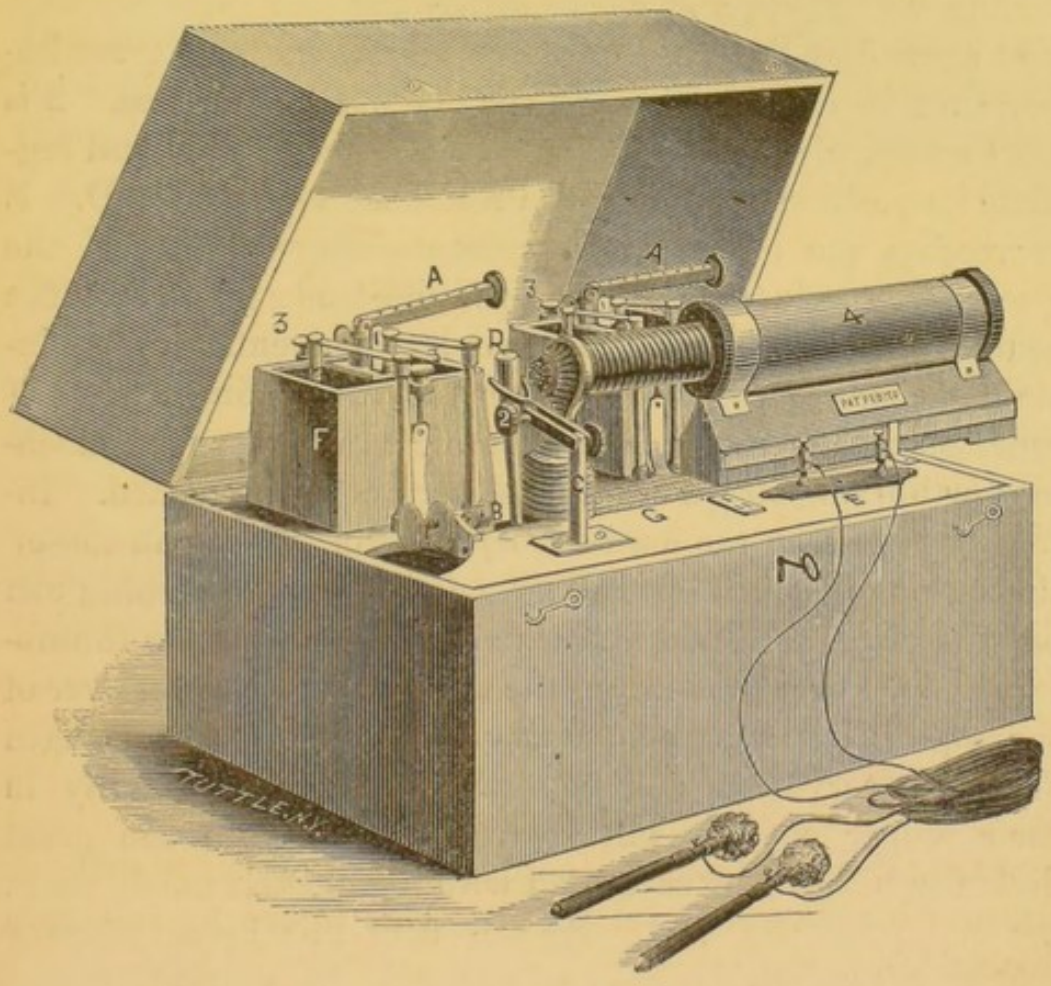
The electrical apparatus required in the diagnosis and treatment of diseases of the nervous system must be of two kinds: one for furnishing the primary or galvanic current, the other for yielding the induced or faradaic current. Among the machines of the first category are those of Stöhrer, which are now made very satisfactorily by the Galvano-Faradaic Manufacturing Company of New York, a combination of Smee's cells which is manufactured by Dr. Jerome Kidder, Electrical Mechanician to the New York State Hospital for Diseases of the Nervous System, and Daniell's batteries, which can be obtained of any electrical-instrument maker. My own preference is for Kidder's arrangement, which gives a very equable current and causes very little trouble.

Of induced-current batteries I have on several occasions before this commended those of Kidder, which are certainly very admirable instruments. Recently, however, I have used with great satisfaction an apparatus which is figured in the accompanying woodcut (Fig. 5), which represents the Portable Excelsior Electro-Magnetic Machine manufactured by the Galvano-Faradaic Manufacturing Company, with all the new improvements attached. This figure represents a double-cell battery:

A. The graduated hinged battery-rod, to the lower end of which the zinc plate is attached; the hinge enables the rod to be laid over horizontally when the battery is not required for action, thereby preventing an accidental immersion of the zinc plate into the battery-fluid. The graduated points on the rod exhibit the depth to which the zinc, when

the rod is raised vertically and lowered down, becomes immersed in the fluid, and indicates the battery-power obtained. It can be retained *in situ* at any desired depth by means of the binding screw. The brass spring 1 presses against the thumb-screw of the rod when this is fully down in the cell, and thereby maintains its conducting power, which becomes deteriorated if it is not kept free from acid, and perfectly clean.

FIG. 5.



B exhibits a combination of a movable platina disk, with that part of the retracting armature lever which plays against the adjusting-screw connecting it with the battery. After continued use this point of the disk becomes oxidized by the electric spark. When this happens, the shocks will be irregularly generated. The disk can be turned a very

little round on its pivot, whereby a fresh surface of its periphery will be brought to play against the point of the adjusting-screw.

C. The rheotome, or adjustable elastic fork. Its free end embraces the vibrating armature-spring or lever D between its prongs, so as to control the extent of the vibrations. When the rheotome is depressed, the upper end of the lever can vibrate freely. When it is raised, its prongs will, in proportion to the extent of its elevation, limit the space for the vibrations, and consequently increase their rapidity. The control of the velocity of the vibrations is of great importance in therapeutics and the diagnosis of disease. 2 is a set screw, placed to work against the elastic fork, and regulate its position laterally, with reference to the lever D. E represents the rheotrope or current-changer, between the electro-magnetic machine and the electrodes, by which the course of the primary and secondary currents can be instantly changed without moving the conducting wires. F represents the hydrostat or metallic capping, with stanchions or binding-straps and thumb-screws 3 3 attached. Interposed between the hard-rubber covering plate and top of the battery-cell is a soft-rubber packing. This capping can be tightened to the desired extent, by means of the thumb-screws 3 3 ; thus preventing the spilling or splashing over of the liquid, impeding evaporation, maintaining the strength of the battery-fluid, and maintaining the cells steadily in their proper places. The hydrostat overcomes the great difficulty hitherto experienced with all electric machines in which liquids are used. By means of this invention they are now rendered portable, and can be carried around charged and ready for use, without danger of spilling the battery-fluid. G represents an indicator or scale, affixed beneath the movable coil 4, in English and French measurement, graduated both ways, to enable the practitioner to make exact record of the intensity of the electricity applied to his patients at each *séance;* so determines the different

degrees of susceptibility evinced on each occasion. As the strength of the primary current is increased by drawing out the movable coil 4, we count from left to right, when we apply that current. The intensity of the secondary current is increased by pushing inward the movable coil 4; in this case, we made our record from right to left. These machines are fitted up in black-walnut cases, with handles and locks attached.

Nothing can exceed the efficiency and convenience of this instrument. It possesses the great advantages of supplying both the inducing and induced currents, and enabling the physician to regulate the interruptions so as to give the shocks very slowly, a matter of great importance in the treatment of paralytic disorders.

SECTION I.

DISEASES OF THE BRAIN.

CHAPTER I.

CEREBRAL CONGESTION.

Cerebral congestion is of two kinds, which differ as regards their mode of origin and symptoms. In the *active* form, there is an increase in the amount of arterial blood circulating in the vessels of the brain; in the *passive*, the quantity of venous blood is augmented. Occasionally the two conditions coexist.

ACTIVE CEREBRAL CONGESTION.

This is much the more common form. Of six hundred and twenty-two cases recorded in my note-book, as occurring in my private practice, five hundred and seven were of this description.

Andral recognized eight varieties, all of which may, with advantage, be comprehended in three, which are appropriately designated from the chief feature characterizing the attack, namely, the *apoplectic*, the *epileptic*, and the *maniacal*. Either of these may occur with scarcely a moment's warning. Generally, however, there is a premonitory or first stage, the symptoms of which, though well marked, are

not peculiar exclusively to any one of the fully-established conditions mentioned. It is therefore impossible to predict with accuracy, from the symptoms of this prodromatic stage, whether the apoplectic, the epileptic, or the maniacal form, will be developed. An attentive study of this stage should always be made, and active measures taken for the relief of the patient at a time when success can generally be obtained.

Symptoms. First Stage.—Among the earliest symptoms of active cerebral congestion, wakefulness is especially noticeable, and may be for a time the only evidence of disorder which attracts the attention of the patient. He goes to bed feeling weary, and as if sleep would very quickly overtake him, but he is disappointed, for he obtains but an hour or two of unquiet slumber, which is generally broken by unpleasant dreams. During the remainder of the night he tosses restlessly from side to side of the bed, his mind either occupied by the thoughts which have occurred to him through the day, or else filled with the most preposterous ideas. He consequently rises unrefreshed, feverish, and ill-prepared for either mental or physical exertion.[1] So far as the mind is concerned, there is an inability to give the attention to any subject requiring much thought, and at times an absolute want of power to get correct ideas of even simple matters. This is especially seen in those who have arithmetical questions to solve, or long columns of figures to add up. Indeed, mental labor of all descriptions is not only difficult, but is irksome in the extreme.

Before long the evidences of intellectual derangement become more evident. The ideas are confused and without logical arrangement; the memory begins to fail, especially in regard to recent occurrences; and there seems to be a special proclivity to forget words, and to substitute others having a similar sound when pronounced, or appearance when written.

[1] For a more complete account of wakefulness in all its relations, see the author's treatise on "Sleep and its Derangements." J. B. Lippincott & Co., Philadelphia, 1870.

The names of persons and places are particularly difficult to recollect. The judgment is weak and vacillating; the most strongly-expressed determination is changed apparently without reason, and again there may be an impossibility of arriving at a decision in cases where ordinarily but little reflection would be necessary. Any effort toward continuous or severe thought increases the difficulties of the mind, and augments the pain or uneasiness which generally exists in the head. Illusions, hallucinations, or delusions, may be present, but are not usually fixed; and the patient will often laugh at the absurd images he has seen, or ideas he has entertained not five minutes before. Persons thus affected will frequently reason clearly in regard to apparitions or voices, of the unreality of which they are fully sensible.

The emotional system participates in the general mental disturbance, and the passions are thus easily roused into activity by slight exciting causes. Trifling circumstances produce great annoyance, and the little every-day troubles of life appear of vast importance. The disposition accordingly becomes suspicious, peevish, and fretful.

In conjunction with these mental phenomena, there are certain physical symptoms of disordered cerebral action. Thus there are pain, heat, a feeling of fulness or distention in the head, or the sensation as if a tight band encircled it. Vertigo is very generally complained of, and may be so severe as to prevent the patient moving about. In some cases headache constitutes the chief feature of the disorder, and is almost constantly present. There are noises, such as roaring, rumbling, and ringing, in the ears, and occasionally loud reports such as those produced by the discharge of fire-arms. Sometimes there are bright flashes of light from over-excitation of the retinæ, and at others dark spots—*muscæ volitantes*—render the vision indistinct. Ophthalmoscopic examination, which should never be omitted, shows the vessels of the retina to be increased in number, diameter, and tortuosity, and occasionally the optic disk is found more or less con-

gested. The conjunctivæ are suffused, the pupils are contracted, there is intolerance of light, and motion of the eyeballs is painful. Loud noises are likewise disagreeable. The face is flushed, the carotids and temporals throb with more than ordinary force, and there may be involuntary twitching of one or more of the facial or other muscles. Bleeding from the nose is not infrequent.

Sensation and the power of motion are usually affected, and generally, though not always, on one side of the body only. Thus the arm or the leg feels heavy, and a sensation as of ants crawling over it, pins and needles sticking in it, or as if the limb is "asleep," is experienced. These abnormal sensations may be restricted to the face or the trunk. Examination with the æsthesiometer shows that the ability to distinguish the two points of the instrument at the normal distance apart is less on the affected side than on the other, and that thus to get the sensation of two points they must be more widely separated when applied to the diseased side than is necessary for the corresponding parts of the sound side. The muscular strength is also lessened generally, but sometimes the difficulty is especially noticed in particular muscles, such as the tibialis anticus or the deltoid, which, losing a portion of their contractile power, cause the patient to experience an awkwardness in raising the foot, or elevating the arm from the side. The face, however, is rarely affected, even when the muscular power is diminished on all the rest of one side of the body, and the tongue, when protruded, comes out straight. Careful observation will generally detect some difficulty, perhaps slight, about the speech. Words are not pronounced with as much distinctness as before, especially when the patient is fatigued or has been speaking for some time. The linguals and labials among letters are particularly troublesome, as well as all words which require the nice management of the end of the tongue for their enunciation. The articulation is thick, and sometimes whole syllables are slurred over in a slovenly way.

The other organs of the body are more or less deranged. The pulse is unusually slow and full, the appetite capricious, the digestion imperfect, the bowels costive, and the urine scanty and high colored.

The foregoing constitute the ordinary assemblage of symptoms which are first met with in congestion of the brain. Some of them may be absent, others so slightly manifested as to escape ordinary observation, and others again so strongly exhibited as to excite the grave apprehensions of the patient and his friends, and to require him to keep his bed. Generally, however, they are not so severe as to prevent him attending in a measure to his ordinary avocations, and they may altogether disappear either spontaneously or in consequence of appropriate medical treatment.

A spontaneous cure is, however, rare, and without proper management on the part of the patient or his medical attendant the symptoms pass sooner or later into one of the fully-developed forms mentioned. Thus, of the five hundred and seven cases already cited, the disease was arrested at the first stage in four hundred and seventy-eight by appropriate treatment, while there was not a single instance of spontaneous cure.

Second Stage. *a. The Apoplectic Form.*—Occasionally this variety of cerebral congestion is initial, but ordinarily it is preceded by the group of symptoms just detailed. In either event the onset is generally sudden. The patient is perhaps walking in the street, when he staggers, loses consciousness, and falls. The loss of intelligence and sensibility is, however, rarely complete, and may last but a few minutes or even seconds, though sometimes continuing for several hours.

Paralysis to a greater or less extent is always present for a time. One limb only may be affected or those of one side, or all four members. It is never complete, the patient being able to perform some movements, though not to exert his full strength. The face is rarely involved, and the patient,

though answering briefly when addressed in a loud voice, speaks indistinctly and with difficulty.

The respiration is loud, slow, but rarely stertorous, and it is not often that there is puffing of the lips and cheeks.

The pulse is slow, hard, and full. Sometimes the face is flushed, and sometimes it is unusually pale. The sphincters generally retain their power.

The senses, though weakened, are often capable of being exercised by tolerably strong excitations. A bright light causes uneasiness and closure of the eyelids. A loud noise is productive of discomfort, and a limb, when pinched, is withdrawn.

The power of the mind is greatly lessened, and some faculties are altogether abolished. Answers more or less direct are given to simple questions put in a loud tone, but even moderate intellectual action seems to be impossible.

Gradually the attack passes off, leaving the patient in a state of mental and physical depression, which may last for several days. The paralysis usually entirely disappears, but occasionally it does not, one or more limbs or muscles remaining permanently, or for a long time, disabled.

It sometimes happens, however, that the termination is not so favorable. The vessels may remain congested, serum may be effused, and death may result without there being any vascular lesion. Two cases have come under my notice, in which death ensued from this cause in first attacks.

A person who has once had a paroxysm, such as has been described, is thereby rendered more liable to subsequent seizures, each one of which still further permanently impairs his mental and physical powers. In one case, occurring in my practice, there have been eleven attacks in five years; and in another, fourteen in four years. In both of these, and in several similar instances I have witnessed, there was paralysis, which had become more profound with each accession. It is therefore inexact to say, as do some writers, that the paralysis of cerebral congestion always disappears in a short time.

Of twenty-nine cases of fully-developed, active cerebral congestion of which I have notes, sixteen were of the apoplectic form.

b. The Epileptic Form.—This, like the variety just described, may come on suddenly, or may be preceded by premonitory symptoms. The phenomena of the attack do not differ from those attendant on an ordinary epileptic paroxysm, except that there is never an aura, and no peculiar cry, such as is so often met with in pure epilepsy. There is the same tonic spasm, followed by clonic convulsions, which may or may not be confined to one side of the body, and which may or may not be followed by temporary or long-continued paralysis. Stupor likewise supervenes, but is neither of so long a duration nor so profound as in true epilepsy.

This form of cerebral congestion never occurs during sleep, for then the brain contains less blood than when the individual is awake. It may occur during stupor induced by certain drugs, constriction of the neck, or the dependent position of the head; but stupor is not sleep, although the two conditions are frequently confounded. Epilepsy occurring during ordinary sleep is never the result of congestion. This point will be more fully considered under the head of epilepsy.

After the stupor the patient may feel comparatively well, or there may be delirium continuing for several hours. As in the apoplectic form, there may be a succession of attacks, and the mind and physical power of the patient are thereby greatly weakened.

The variety under consideration is, perhaps, more liable to occur in individuals past the age of forty, though I have witnessed several cases in quite young persons. It is not often met with in old age, and, when it is, is generally fatal, probably from secondary lesion. Nine of the fully-developed cases, of which I have record, were epileptic in character.

c. The Maniacal Form.—This variety, though not so common as either of the others, is yet not infrequent. It is

characterized by an accession of mental derangement not materially different from that indicative of acute mania. The delirium is of a very active character, the eyes are suffused, the face is red, the head hot, the motility active, and the whole manner, character, disposition, and mental processes are changed. During the paroxysm the patient may commit some crime of violence, and it almost always happens that his combative proclivities are aroused. He may likewise attempt to injure himself.

The attack may come on with great suddenness. In the case of a gentleman recently under my charge, it was the result of eating a hearty meal in a great hurry at a railway station. A few minutes after his return to the train he was attacked with furious delirium, during which he attempted to injure himself and all within his reach. He was seized and held, but continued, as far as he was able, to bite, scratch, and kick at those who were near him. The paroxysm lasted about two hours. He then fell into a heavy stupor, from which he did not arouse for two hours longer. For several days his mind was weak, and there was numbness in various parts of his body. Gradually, however, he regained his former powers, but he suffered from occasional confusion of thought and difficulty of speech, with headache and wakefulness for several weeks.

Paralysis, as in the other two forms, may be one of the phenomena of this variety of cerebral congestion.

Death may take place during the attack, or from secondary lesions afterward.[1] Of the twenty-nine fully-developed cases, four were of the maniacal form.

What is called temporary insanity, mania ephemera, or impulsive insanity, generally depends upon cerebral congestion. The subject, therefore, is of vast importance in its medico-legal relations.[2]

[1] The whole subject of cerebral congestion has been well considered by Calmeil in his "Traité des Maladies Inflammatoires du Cerveau." Paris, 1859.

[2] See a memoir by the author, entitled "A Medico-Legal Study of the Case of

SCHOOL OF MEDICINE

THIRD STAGE.—This period may be considered as beginning after the immediate effects of the paroxysm, whether it has been of the apoplectic, epileptic, or maniacal form, have passed off. It is characterized by feebleness of body and mind, by gastric or intestinal derangement, by pain in the head with transient attacks of vertigo, and occasionally by numbness and slight paralysis of one or more of the limbs. Many of the symptoms met with in the first stage are again found in this.

But the principal phenomena are those connected with secondary lesions, such as inflammation, abscess, softening, and adventitious growths of various kinds. These will be considered under their proper heads.

It must not be forgotten that one circumstance always exists, and that is, the proclivity to other paroxysms of some one of the fully-developed forms.

PASSIVE CEREBRAL CONGESTION.

This condition is the result of causes which increase the amount of venous blood in the brain. It is more commonly met with in old persons. One hundred and fifteen cases out of six hundred and twenty-two, occurring in my practice, were of this form.

Symptoms. FIRST STAGE.—As in active cerebral congestion, there is a premonitory stage, the symptoms of which are similar to those previously described. There is, however, a tendency to stupor, and the other phenomena are, in the main, less strongly marked. Vertigo, pain, illusions, hallucinations, and delusions, are nevertheless generally present at one time or another. But the stupor or tendency to somnolence is the most prominent feature, and the sleep, even when comparatively natural, is attended with dreams unpleasant or even frightful in character.

Daniel McFarland," in the JOURNAL OF PSYCHOLOGICAL MEDICINE for July, 1870. Also published separately by D. Appleton & Co. New York, 1870.

The degree of congestion may be suddenly increased, or, what is a more probable sequence, there may be effusion of serum, and then the second stage exhibiting itself either in the apoplectic, the epileptic, or the maniacal form results.

The proportion of cases of passive cerebral congestion which pass to the second stage is greater than in the active form of the affection. Thus, of the one hundred and fifteen cases cited, thirty-one went on unchecked to the second stage.

Second Stage. *a. The Apoplectic Form.*—Though this variety may be developed suddenly as in active cerebral congestion, it usually is more slowly evolved. In this latter case a general numbness is commonly the first symptom, and the drowsiness gradually increases. At first it is easy to rouse the patient from this stupor, but eventually it is more difficult, and at times impossible. The faculties of the mind may likewise, at the beginning, be excited into a moderate degree of activity, but with the advancing coma they are no longer manifested. The cutaneous sensibility becomes less and less; the urine dribbles, from paralysis of the bladder and its sphincter; and the bowels, if not obstinately constipated, allow their contents to pass involuntarily.

This condition may last for several weeks, and, though recovery may take place, this is never complete. It generally ends in death.

Nine cases of the thirty-one were of this character.

b. The Epileptic Form.—This may not differ materially from the epileptic form of active congestion except as regards increased length of the fit and prolonged stupor. Generally, however, there is a repetition of the convulsive seizures, and I am led to believe from my experience that there is a greater tendency to biting the tongue. Paralysis is a more common sequence, and is of longer duration.

Nineteen cases of the thirty-one were epileptic in form.

c. The Maniacal Form is not often met with in passive cerebral congestion, and, when it is, the delirium, so far from

being of a furious type, is low. The patient mutters to himself incoherently and exhibits great muscular restlessness, but never attempts to do violence to himself or others. Coma often occurs as a sequence. Three cases were of this type.

Causes.—The causes of cerebral congestion are: of the active form, those influences which are capable of increasing the quantity of arterial blood in the brain; of the passive, those which produce a similar effect upon the amount of venous blood circulating in the vessels within the cranium. The causes of the first category induce activity of circulation, those of the second torpidity.

The causes of active cerebral congestion may either by their gradual operation initiate the premonitory stage, or they may suddenly induce the development of this stage into one or other of the varieties already described as constituting the second stage. Among them are temperature either very high or very low. Thus the disease is more frequent in hot climates than in those of more temperate character, and in the summer months than in the spring or autumn. It is, however, more common in very cold than in warm weather. Thus Andral, of one hundred and fourteen cases, found that twenty-six occurred in summer and fifty in winter. My own experience is to the same effect, as will be seen from the following table, which embraces the cases in my private practice in the city of New York during a period of five years, beginning January, 1865, and ending December, 1870:

January	66	July	68
February	64	August	74
March	50	September	27
April	39	October	31
May	42	November	52
June	37	December	72
		Total	622

An examination of this table shows that one hundred and ten cases occurred in the autumn months, one hundred and

thirty-one in the spring, one hundred and seventy-nine in summer, and two hundred and two in winter.

Passive cerebral congestion is very much more frequent in cold than in warm weather.

The direct rays of the sun are capable of producing sudden attacks (insolatio), of which congestion is a prominent feature, but which require separate consideration, and it is not uncommon for artisans, whose heads are exposed to heat from furnaces, to suffer in a similar manner.

Some authors contend that certain winds increase the liability to cerebral congestion. Leuret, quoted by Mosmant,[1] could attribute an epidemic of cerebral congestion which appeared at Charenton to nothing but a long-continued wind from the northwest. The supposition that atmospheric electricity is a causative influence rests upon nothing but hypothesis.

The ingestion of a large quantity of food into the stomach may occasion passive congestion by the pressure which the distended organ makes upon the large veins of the abdomen. Rapid eating, even though the quantity of food be moderate, may cause the active form of the affection by some influence exerted through the sympathetic system.

Sudden and violent physical exertion, especially if made in the stooping posture, is very liable to induce cerebral congestion. Child-birth is an instance in point, and I have known several cases to be caused by severe straining in the water-closet. The constipation of the bowels rendering such efforts at defecation necessary, is itself productive of the disease.

A dependent position of the head and constriction of the neck from the dress are also, by impeding the return of blood from the head, liable to induce congestion of the passive form.

Certain articles of food and medicine, such as spices, alcoholic liquors, opium, belladonna, quinine, etc., act either by augmenting the action of the heart, or by their influence

[1] Essai sur la Congestion Cérébrale. Paris, 1858.

on the sympathetic, paralyzing the vaso-motor nerves, and thus increasing the calibre of the cerebral blood-vessels.

Tumors in the neck, or in other parts of the body where the return of blood from the head may be impeded by their pressure, likewise cause congestion. Other causes are to be found in certain diseases, as fevers of various kinds, erysipelas, disorders of menstruation, the suppression of hæmorrhagic or other discharges; local affections of the brain, as embolus, thrombosis, tubercle or apoplectic clots, and sympathetically by worms in the intestinal canal, or irritation existing in other portions of the system.

But the most influential and common causes of cerebral congestion are to be found in long-continued intellectual exertion, mental anxiety, or sudden, violent, or prolonged emotional disturbance. It is from the action of such factors that the premonitory symptoms are generally induced, though they may, especially those embraced in the last-named category, immediately develop a fully-formed attack. The fact that cerebral exercise increases the amount of blood in the head is made evident to all of us at times by the distention of the superficial vessels, the suffusion of the eyes, the heat and pain which we feel when we have overtasked our brains. Cerebral action is always attended with hyperæmia, just as is the activity of the liver, the kidneys, or other organs. Active cerebral congestion is thus induced, and is within certain limits perfectly normal. But these limits are liable to be exceeded, and in this active period of the world's history often are, and then the condition described as the first stage of congestion is established. The vessels, from continued overdistention, lose their contractility, just as does the india-rubber band, used to keep a bundle of letters together, when the package is too large or it has been kept stretched for a long time. An additional disturbing force, heat, cold, an overloaded stomach, increased mental labor, emotional excitement, or any of the causes mentioned, may suddenly evolve a fully-developed paroxysm.

Emotion acts in a similar manner, though, as has been said, often with more suddenness. The emotions of shame, of anger, and others, cause the face to become red from dilatation of the blood-vessels, and a like effect is produced in the vessels within the cranium. If the emotion is very strong or lasting, a correspondingly-increased hyperæmia results.

There are certain circumstances which render the action of the causes specified more effectual or powerful. These are inherent in the individual, and may be classed as predisposing causes. Among them are sex, the disease being more common in males; age, it being more frequently met with in middle-aged or old persons; hypertrophy of the left ventricle of the heart, by which the flow of blood to the head is directly increased; dilatation of the right ventricle, by which its power is diminished, and the return of blood from the head impeded; insufficiency of the auriculo-ventricular valves, or constriction at the auricular or ventricular orifices on the same side, by which a similar result is produced, and perhaps, though this point is by no means established, shortness of the neck.

Diagnosis.—Cerebral congestion may be confounded with cerebral hæmorrhage, meningeal hæmorrhage, embolism, thrombosis, softening, epilepsy, urinæmia, stomachal vertigo, and with the very opposite condition, cerebral anæmia. From each of these affections it is, however, distinguished by well-marked characteristics.

The premonitory symptoms are not liable to be mistaken for cerebral hæmorrhage, but this error may be made as regards the second stage. The apoplectic form is, however, distinguished from apoplexy due to extravasation, by the fact that in it the loss of intelligence is rarely complete, and that, when it is so, the mind is dormant but for a few moments; that sensibility and the power of motion are never altogether abolished; that coma, when present, is rarely profound; that the paralysis, when it exists, is seldom

limited to one side of the body; by the general absence of stertor, and puffing of the lips and cheeks in breathing; and by the short duration of the symptoms.

From meningeal hæmorrhage it is discriminated by the comparative lightness of the symptoms, and by the fact that they do not progressively augment in severity or intermit in violence.

Cerebral congestion and embolism present some features in common, and it is therefore occasionally difficult to distinguish them. In the former, however, the pulse is slow and the respiration regular and deep, in the latter the pulse is more rapid, is often irregular, as is also the respiration; in the former there is increased heat of the head, in the latter the temperature of this part of the body is unchanged; in cerebral congestion the symptoms are transient, in embolism they are more lasting; in the former there is often a distinct premonitory stage, in the latter the attack always takes place without a moment's warning. In the former, though there may be cardiac difficulties, they are different from those predisposing to embolism, which are consecutive to endo-carditis—generally rheumatic—and which implicate the semi-lunar or mitral valves, and in the fact that recovery from an attack of cerebral congestion is generally complete, which is rarely the case in embolism.

From thrombosis cerebral congestion is diagnosticated by the circumstances that in the former the progress of the disease is slow, that there is usually well-marked paralysis from the beginning, that the phenomena indicating mental difficulty are more strongly pronounced, that the articulation and memory for words are more decidedly affected, and, notwithstanding occasional remissions, by the persistency and gradual advance of the symptoms.

In softening there is often a sudden loss of consciousness, persistent hemiplegia, and death in a few days. Again, there is delirium without paralysis or convulsions, and again there is a gradual accession of the symptoms. This latter is the

only form liable to be mistaken for cerebral congestion. It is attended with headache, feebleness of intellect, and a gradually-advancing paralysis generally, beginning in one of the lower extremities, and extending to the whole of one side of the body. The speech is always seriously impaired, and the mental disorder is of a far graver character than that due to cerebral congestion. The gradual advance of the affection to a fatal termination is also a characteristic circumstance.

With urinæmia cerebral congestion may be confounded, if only the more obvious head symptoms be taken into consideration. The history of the case and full inquiry will always, however, enable the proper discrimination to be made. Thus in urinæmia the existence of kidney-disease as evidencd by a chemical and microscopical examination of the urine, the anasarca of the face or limbs, and the repeated attacks of convulsions and coma, will be sufficient diagnostic marks.

From epilepsy cerebral congestion is distinguished by the fact that the former is not preceded by the group of symptoms constituting the first stage of congestion, that the period of greatest congestion of the vessels of the face and neck is at the beginning of the attack, that an aura is often present, that there may be a peculiar cry, that the patient does not stagger and fall slowly to the ground, but drops as if knocked down by a severe blow, and that the tongue is frequently bitten. The reverse is the case as regards all these phenomena in cerebral congestion. Nevertheless so accurate and experienced an observer as Trousseau, in his clinical lecture on *Apoplectiform Cerebral Congestion in its Relations to Epilepsy and Eclampsia*,[1] confounds the two conditions. Trousseau's views on this subject do not, however, appear to be accepted by any large number of medical authorities. Epileptic vertigo is, as will be shown at the proper place, a very different affection from any form of cerebral congestion, and

[1] Clinique Médicale. Tome ii., p. 56. Also Bazire's Translation. London, 1866, p. 19.

is not likely to be confounded with it. Epileptic mania has, likewise, very few points in common with the disease under consideration.

In stomachal vertigo the attacks of dizziness are often severe, but they are clearly associated with gastric derangement, and only occur while the stomach is digesting its contents. Other symptoms of dyspepsia will also be noticed, while the mental and physical disturbances which constitute so prominent a feature of cerebral congestion are absent. The distinction, however, is not always made.

From cerebral anæmia the first stage of congestion is frequently not clearly distinguished, and I have seen several cases in which patients had been treated for the one condition when the other was indubitably present. In both there are headache, sense of constriction, vertigo, noises in the ears, numbness, mental confusion, loss of memory, inaptitude for labor of any kind, and at times loss of consciousness. But in anæmia the face is not flushed, the carotid and temporal arteries do not throb with violence, the pulse is quick, feeble, and irregular, the respiration is hurried, the pupils are dilated, there are bellows murmurs at the base of the heart and in the veins of the neck, and the general aspect of the patient is not of that rugged appearance so generally associated with cerebral congestion. In the syncope of cerebral anæmia the paleness of the face, coldness of the skin, and feebleness of the heart's action, will serve to draw the line between it and the apoplectic form of congestion. The ophthalmoscope will at all stages prove of great value in the diagnosis.

Prognosis.—The prognosis is materially modified, according to the stage of the disease present when the patient is seen, and the form of attack from which he may be suffering. Active cerebral congestion is a more favorable type than the passive. If the affection has not gone beyond the first stage, a fortunate issue may safely be predicted under the use of suitable medical treatment. Of the six hundred and twenty-two cases under my care, but sixty passed to the

second stage, and several of these had already suffered from previous seizures; no death took place in any patient during the premonitory stage. The apoplectic form is the most grave, and the prognosis is rendered more unfavorable with each attack. The epileptic form is ordinarily not dangerous to life, nor is the maniacal, except in old persons. Occasionally, however, even in young and robust patients, death ensues during the paroxysms of these forms.

The liability to secondary lesions, such as softening, cerebritis, hæmorrhage, aneurisms, general paralysis, etc., must be taken into account when forming a prognosis. The more frequent the paroxysms of any form, the greater the risk of some such finality.

The habits of the patient are also important elements in forming an opinion in regard to the ultimate result. If these are bad, and are persisted in, the probability is that no treatment will be of much avail in preventing a recurrence. Moreover, by such a condition of the brain as the excessive use of alcohol, inordinate mental exertion, or continual emotional excitement induce, the chance of escaping some secondary morbid process is very much lessened.

Of the sixty fully-developed cases which have been under my observation during the past five years, there were twelve deaths; four from the apoplectic form, all after repeated attacks; three from the maniacal, one of which was that of a young man, about thirty years of age; and five from secondary lesions. Of these latter, two were from softening, one from cerebritis, one from hæmorrhage, and one from general paralysis.

Morbid Anatomy.—There are certain appearances seen in the brains of those who have died of cerebral congestion which are characteristic, although it must be confessed that some or all of them are occasionally absent. These are:

An increased size of the capillaries and large blood-vessels, both of the brain and the pia mater. It thus happens that, when a section of the brain is made, the red points ordinari-

ly seen are larger and more numerous than usual, and that the pia mater presents in spots, or throughout its extent, a red or rose-colored appearance.

The white matter of the brain is increased in density, and the gray matter is red, or even violet in hue.

There is sometimes a large quantity of subarachnoidean effusion, and the ventricles may contain an excessive amount of fluid.

If there have been repeated attacks of cerebral congestion, it is not unusual to find, by microscopical examination, little granules of hæmatin in contact with the blood-vessels. The same means of exploration show the minuter capillaries to be more than naturally tortuous, and to have little aneurismal swellings. These may or may not involve the whole circumference of the vessel. Their presence and import were first pointed out by Laborde.[1]

On making a transverse section of the hemisphere, a cribriform appearance is seen, if the patient has repeatedly suffered from attacks of cerebral congestion, and especially if he be advanced in years. This is due to the presence of numerous little holes with sharply-defined margins. The brain-tissue bounding these is generally without material change, either in color or consistence. This condition, called by Durand-Fardel, to whom the credit of first describing it is usually given, "l'état criblé,"[2] is supposed to be due to the fact that the vessels have been so distended during life as to press with increased force upon the perivascular tissue, and that, shrinking after death, they no longer fill their former space, which remains empty. Calmeil[3] was the first to notice this condition. He has very often found in maniacs the white substance, rendered cribriform by vessels distended with blood, sometimes empty, but always greatly

[1] La Ramollissement et Congestion du Cerveau principalement considérés chez de Vieillard. Paris, 1866.

[2] Traité Pratique des Maladies des Vieillards. Paris, 1854.

[3] De la Paralysie considerée chez les Aliénés, etc. Paris, 1826.

dilated. This state, although frequently met with in congestion, is not uncommon in other pathological conditions, such as the several forms of softening, of which, however, congestion is often the first stage.

Pathology.—It is almost useless at this day to discuss the question of the possibility of the quantity of blood in the brain being subject to variation. Still it may be interesting to recall briefly the facts which establish the affirmative in the matter.

In the cases of infants in whom the interior fontanelle is still open, the scalp is seen to be elevated above the level of the skull when the head is dependent, and depressed when the head is elevated.

The same fact is observed in persons who have suffered injury of the skull, involving the loss of a portion of its substance. During strong emotional excitement, or the action of any cause capable of increasing the force of the circulation, the scalp is elevated. From the action of opposite causes it is depressed. Both in infants and in persons who have received injuries such as those cited, the scalp is seen to be depressed during sleep, and to rise as soon as the individual awakes.

A dependent position of the head causes a sensation of fulness, or even pain, and blood may flow from the nostrils. The eyes are observed to be "bloodshot," and the countenance indicates congestion. A tumor, a ligature, or any other cause capable of exerting pressure on the jugular veins, will produce like effects. Ophthalmoscopic examination under such circumstances shows the veins of the retina to be enlarged, indicating that an obstruction exists to the return of blood through the sinuses and veins within the cranium. Post-mortem examination of persons dying, who, during life, have suffered interruption to the perfect return of blood from the head, reveals the existence of intracranial congestion. Animals subjected to experiments calculated to act in the manner stated, are after death found to have congested brains.

In animals bled to death the brain is found anæmic to an extreme degree.

Direct experiment still more positively establishes the fact under consideration. If a portion of the skull of an animal be removed, and the aperture be then securely closed with a watch-glass, the vessels will be seen to enlarge and contract according to the cause brought into action, and the brain will be correspondingly elevated or depressed.

By means of an instrument, devised, independently of each other, by Dr. S. Weir Mitchell and myself, the degree of pressure within the cranium can be accurately measured. It is thus seen that the quantity of blood circulating in the brain undergoes material variation.[1]

The anatomical arrangement of the blood-vessels of the cerebral tissue is such as to admit of an enlargement of their calibre without necessarily subjecting the perivascular substance to pressure. Robin[2] discovered the existence of sheaths around these vessels, and his observations were subsequently confirmed by His,[3] who ascertained that the same arrangement exists in the spinal cord. According to His, "Fine transverse sections of a hardened brain, having its vessels injected or otherwise, show that all the blood-vessels, arteries, veins, and even capillaries, are surrounded by a clear space, broadest in the case of the larger vessels, but in all cases quite sharply defined externally. In transverse sections the vessels are seen to be surrounded by a ring-like space, and in parallel sections the space is seen on each side of the trunk of the vessel, and follows it in all its ramifications."

[1] For a more complete argument on the subject, and for a statement in detail of the experiments of Mr. Durham and myself on this point, the reader is referred to the author's monograph, "Sleep and its Derangements." Philadelphia: J. B. Lippincott & Co., 1870. The cephalo-hæmometer referred to in the text is described in that work (Appendix), and also in the introduction to this treatise.

[2] Journal de la Physiologie de l'Homme et des Animaux, 1859, p. 527.

[3] "Zeitschrift für Wissenschaftliche Zoologie," 1865, B. xv., quoted in the Journal of Anatomy and Physiology. Translation by Dr. Bastian.

These perivascular canals are lined by a hyaline membrane, and are capable of being injected, and, in cases of chronic congestion, may become permanently enlarged, so as to cause the appearance referred to under the heading of morbid anatomy.

The pathology of the subject receives further elucidation from a consideration of the causes capable of giving rise to cerebral congestion, and which have been already mentioned in detail.

Treatment.—Recollecting the two grand forms of cerebral congestion, the principles which should guide us in treatment will be clearly apparent. In the active type of the disease the force of the cerebral circulation and the quantity of blood in the blood-vessels of the brain are to be lessened; in the passive variety the force of the circulation is to be increased, and at the same time the accumulation of blood in the veins to be diminished. In the active form of this affection the abstraction of blood from the arm was formerly very generally practised, but is now rarely performed. I have never seen a case in which it was required. Local bleeding is more generally applicable, and a few cups to the nape of the neck will often afford marked relief. Leeches to the temples are also useful, though they are preferably applied just inside the nostrils. I have many times witnessed the most satisfactory results from a couple of leeches thus used, and from accidental nasal hæmorrhage.

Cold is another very useful agent in the treatment. It may be applied to the nape of the neck, or directly to the cranium, either as very cold water or in the form of ice.

The advantages of position should also be brought to bear. The head should be kept elevated, especially during sleep, and no severe muscular exertion should be taken while stooping.

The clothing should be kept loose about the neck. As derivatives, a mustard-plaster applied to the epigastrium is often of service, and the same may be said of warm or even

hot water to the feet. Blisters I rarely employ, though I have occasionally done so with advantage.

The constant galvanic current possesses the power of contracting the cerebral blood-vessels, when so used as to stimulate the sympathetic nerve. For this purpose one pole, the positive, should be placed over this nerve in the neck; and the other, the negative, on the neck, as low down as the seventh cervical vertebra. The current from about fifteen Smee's cells is sufficient, and it should not be allowed to act for more than two minutes. If extreme vertigo be produced, the number of cells should be lessened. This property of the primary current was first pointed out by Bernard, Waller, and Budge, but its demonstration by the ophthalmoscope was first made by myself.[1] Observation with this instrument, while the current is acting, shows that the vessels of the retina contract, and hence there can be no doubt that the result is produced upon those of the brain. A similar effect is caused by passing the current directly through the brain, the poles being applied to the mastoid processes. A slight feeling of vertigo follows both when the circuit is closed and opened. The good effects of this practice are well marked, a few applications being often sufficient to abolish the vertigo and unpleasant feelings in the head, and to restore mental and physical activity.

Of internal remedies the number is not large, and those which it is advisable to employ are generally effectual, with or without the external measures mentioned, in entirely relieving the patient.

First among these must be placed the bromide of potassium. Over five years ago I pointed out the value of this

[1] See a memoir entitled "Spinal Irritation," read before the Medical Society of the County of New York, January 17, 1870, and published in the Journal of Psychological Medicine for April of the same year. Also another, "On some of the Effects of Excessive Intellectual Exertion," in the Bellevue and Charity Hospital Reports for 1870. In both these papers, and in my lectures to the class of the Bellevue Hospital Medical College, I have made distinct mention of this fact.

medicine, and explained the *rationale* of its action. As others have since claimed the discovery as their own, I hope I may be excused for quoting the following passage from a memoir upon an analogous subject,[1] in which the action of the bromide is clearly indicated :

" Bromide of potassium can almost always be used with advantage to diminish the amount of blood in the brain, and to allay any excitement of the nervous system that may be present in the sthenic form of insomnia. That the first-named of these effects follows its use, I have recently ascertained by experiments upon living animals, the details of which will be given hereafter. Suffice it now to say that I have administered it to dogs whose brains have been exposed to view by trephining the skull, and that I have invariably found it to lessen the quantity of blood circulating within the cranium, and to produce a shrinking of the brain from this cause. Moreover, we have only to observe its effects upon the human subject, to be convinced that this is one of the most important results of its employment. The flushed face, the throbbing of the carotids and temporals, the suffusion of the eyes, the feeling of fulness in the head, all disappear as if by magic under its use. It may be given in doses of from ten to thirty grains, the latter quantity being seldom required, but may be taken with perfect safety in severe cases."

Since then, experiments with the cephalo-hæmometer and ophthalmoscope have abundantly confirmed these views, and more extensive experience in the treatment of cerebral congestion has placed the matter beyond the possibility of a doubt. Other observers have also confirmed the opinions here expressed.

The prescription which I usually employ consists of bromide of potassium, ℥j; water, ℥iv; of this a teaspoonful is taken three times a day in a little water. Occasionally the

[1] On Sleep and Insomnia. New York Medical Journal, June, 1865, p. 203.

bromide is increased to ℥iss, and sometimes a saturated solution—which contains grs. xxx to ʒj—is used. I continue the medicine till drowsiness, a slight feeling of weakness in the legs, and contraction of the blood-vessels of the retina—detected by the ophthalmoscope—are produced. The more prominent head-symptoms generally disappear in four or five days, and the results above-mentioned ensue in about ten days.

Latterly I have used the bromide of sodium in corresponding doses instead of the bromide of potassium. It is more pleasant to the taste, and does not cause so much constitutional disturbance as sometimes follows the administration of the bromide of potassium in large doses.

In conjunction with one or other of the bromides mentioned, I very generally employ the oxide of zinc, which experience has taught me is a powerful agent in relieving cerebral congestion, and giving tone to the nervous system. It should be given in doses of grs. ij, three times a day, either in the form of a pill or powder, and to avoid any nausea should be taken after meals. At the end of about ten days it will generally be found that all symptoms of congestion—subjective and objective—have disappeared, leaving a little debility and mental depression. It then becomes expedient to give tonics and restoratives, and those which have a special action on the nervous system are to be preferred. Among them, strychnia, phosphorus, and cod-liver oil, stand first.

Strychnia may be advantageously administered in conjunction with iron and quinine dissolved in dilute phosphoric acid, as in the following formula: strychniæ sul. gr. j; ferri pyrophosphatis, quiniæ sul., āā, ʒj; acid. phosp. dil., zingiberis syrupus, āā, ℥ij. M. ft. mist. Dose, a teaspoonful three times a day in a little water. I prefer this extemporaneous prescription to any of the syrups or elixirs with like ingredients. If for any reason the iron and quinine are not indicated, the strychnia can be given alone with the dilute phosphoric acid.

LEEDS SCHOOL OF MEDICINE

Phosphorus almost always acts well in such cases as those under consideration. It may be given in the form of the phosphorated oil, as in the following formula: ℞. Olei phosphorat., ℥ss; mucil. acaciæ, ℥j; olei bergamii, gtts. xl. M. ft. emulsion. Dose, gtts. xv three times a day. A very elegant preparation of phosphorus is the phosphide of zinc, which was imported at my request about two years ago, by Mr. Neergaard, the eminent pharmaceutist of this city, and which I was certainly the first to use in this country. M. Moutard Martin, M. Dujardin-Beaumetz, my friend M. Gueneau de Mussy, and other Parisian physicians, had previously employed it. My experience with this medicine has been very extensive. I have never known it to produce the least unpleasant effect, and have rarely been disappointed in obtaining the full results to be expected from phosphorus in corresponding doses. I am, therefore, not in accord with Dr. M. Clymer[1] on this point.

The chemical formula of the phosphide of zinc is P_2Zn_3, and consequently a grain represents a little more than one-seventh of a grain of phosphorus. The proper dose, therefore, is about the tenth of a grain. I usually prescribe it in cerebral congestion, according to the following prescription: ℞. Zinci phosphidi, grs. iij; rosar. conserv., q. s. M. ft. in pill, No. xxx. Dose, one three times a day. Instead of the conserve of roses, grs. x of the extract of nux-vomica may be substituted if strychnia is not being administered in some other form.

Such is the treatment I have found to be most advantageous in active cerebral congestion, and I rarely have occasion to supplement it with other measures, unless some special indication is to be fulfilled. Thus, if the bowels are constipated, a mild purgative may be given, or preferably an enema of warm water or olive-oil; or, if the urine is scanty and high colored, saline diuretics are useful.

In the passive form of the disease it is sometimes advis-

[1] New York Medical Journal, vol. x., 1870, p. 476.

able to give stimulants, which may be done from the first in conjunction with the bromide of potassium or sodium. Alcohol in some form is to be preferred when it is well borne, though carborate of ammonia is sometimes a useful substitute. In several cases of passive cerebral congestion in old people, and in one notable instance occurring in the person of a very prominent, elderly gentleman of this city, I derived the most satisfactory results from sulphuric ether inhaled from a handkerchief to the extent of a teaspoonful, several times a day. The pain, constriction, vertigo, numbness, wakefulness, and inability to exert the mind, were lessened with every dose, and finally entirely disappeared. Ether may likewise be given by the stomach—gtts. xv several times daily—in case the inhalation is contraindicated from any cause.

Of course, any influence capable of interfering with the due return of blood from the head should be counteracted at once.

Hygienic treatment should in both types of the disease be persistently carried out. The food should be nutritious, digestible, and ample, though not excessive, in quantity. Alcohol and tobacco, if used habitually by the patient, should be restricted to moderate limits; I have never seen the latter do harm unless used to excess. Tea and coffee may safely be left to the patient's own inclinations and experience. I believe more harm is done by suddenly breaking off a habit even though it be somewhat injurious, than by tolerating it within due bounds. Exercise in the open air—walking, horseback-riding, or driving—is always beneficial. The same cannot be said of gymnastic contortions, which, to make them worse, are usually performed in hot rooms. Bathing daily and subsequent friction with a tape towel are exceedingly useful in determining blood to the surface of the body. The Turkish bath cannot be too highly commended.

But, above all, those persons who have brought on the disorder by inordinate mental exertion or anxiety must con-

sent to use their brains in a rational manner if they wish to recover or to avoid future attacks. They have received a warning, and, if they do not heed it, sooner or later other diseases, more difficult if not impossible of cure, will make their appearance.

The cause, whatever it be, must, if practicable, be removed, and it must continue removed.

LEEDS SCHOOL OF MEDICINE

CHAPTER II.

CEREBRAL ANÆMIA.

In cerebral anæmia the quantity of blood in the brain is either reduced below the normal standard, or the quality of the circulating fluid is impoverished. The first-named condition is due either to direct loss of blood, to deficient action of the heart, to impaired nutrition, or to some cause preventing the due access of blood to the brain; the second to disease of some organ concerned in hæmatosis or to a general cachexia.

The two states very often coexist, and they may properly be considered together.

Symptoms.—In cerebral anæmia, suddenly induced from profuse hæmorrhage, the most prominent symptom is syncope. Vertigo is generally an attendant, and there are paleness of the features and coldness of the extremities. The pulse is frequent, thread-like, and weak. The respiration feeble and accelerated.

But, when the accession is more gradual, headache is very generally present. It may be, and usually is, confined to a limited portion of the head, sometimes to a spot not larger than the point of the finger. A feeling of constriction, especially across the brows, is complained of, and the vertigo, notably increased on rising from the recumbent posture, is as troublesome a feature as in the worst attacks of cerebral congestion. There is ringing in the ears, and loud noises are not only painful but are exceedingly irritating to the nervous system. The pupils are largely dilated, and are slug-

gish, contracting slowly and but little on exposure to a strong light. These phenomena may be restricted to one eye, a circumstance which generally occasions needless alarm on the part of the patient. The retinæ are extremely sensitive, and hence ophthalmoscopic examination is painful. When employed, the vessels at the fundus of the eye are seen to be small and straight, and the choroid is paler than is normal.

The complexion is pale, the lips almost colorless, or else redder than in health. The skin is cold and clammy.

Nausea and vomiting are present in extreme cases, and convulsions of an epileptic character may occur. In the rapidly-developed form of the disease caused by sudden and great loss of blood they are always present, and in the milder and more gradual variety they are occasionally seen. Feebleness of muscular power is always met with, and there may be general or partial paralysis with the usual derangements of sensibility indicative of anæsthesia, such as coldness, formication, and "pins and needles."

The mind, of course, participates in the general disorder. In extreme cases, due to active hæmorrhage, the patient is completely insensible. In less severe forms there may be all the gradations from low delirium to great mental irritability, or a condition of intellectual lassitude approaching dementia.

Hallucinations and illusions are common in the slowly-developed forms of cerebral anæmia, and may affect any one or all of the senses. Those of sight and hearing are, however, more prominent. In the case of a young lady now under my care, and whose only marked disorder is that under consideration, the hallucination that she sees a black man is almost constantly present. At times she converses with this imaginary being, tells him not to trouble her, that she no longers fear him, etc. She believes firmly in his presence, and hence has a delusion.

In all cases of cerebral anæmia there is more or less drowsiness, from the profound syncope of the rapid form to the

rather agreeable languor present in slight cases. In instances of medium severity the patient readily falls asleep in the sitting posture, but recumbency induces wakefulness from the fact that the quantity of blood in the brain is thereby suddenly increased above the habitual standard, and a state of comparative hyperæmia is thus induced. I have, in another place,[1] called attention to this form of insomnia, and adduced several cases in illustration.

Examination of the heart by auscultation reveals the existence of bellows murmurs both systolic and diastolic. They are heard more loudly at the base of the heart. There are also very generally venous murmurs which are heard most distinctly in the jugular veins, especially when the head is turned toward the opposite side. Arterial murmurs may also occasionally be perceived.

These sounds are sometimes heard by the patient, and are then exceedingly annoying. I have now under my charge a gentleman suffering from cerebral anæmia, who constantly hears a sound originating apparently in the head, and which, as he describes it, resembles that caused by a large shell placed to the ear. That these murmurs are anæmic is shown by the fact that they disappear under appropriate treatment.

A form of cerebral anæmia met with in young children is of great importance, from the fact of its liability to be confounded with another far more dangerous affection, almost its opposite. This was first clearly described by Dr. Gooch,[2] although previously noticed by other observers. In children suffering from this affection, the symptoms, so far as they are noticeable, are similar to those present in the anæmia of adults. The drowsiness is well marked, the head is cool, the pulse is small and weak, the features are pinched, the pupils large and insensible to light, and the fontanelle, if still open,

[1] "Sleep and its Derangements."

[2] On Some of the most Important Diseases peculiar to Women; with Other Papers. New Sydenham Society Publication. London, 1859, pp. 179.

has the scalp covering it depressed. After death, the vessels of the brain are found to be almost empty, and the ventricles distended with fluid. From its resemblance in some respects to hydrocephalus or tubercular meningitis, this affection was called by Dr. Marshall Hall hydrocephaloid. The distinction, however, is so well defined that none but the most ignorant or superficial observers would fail to recognize it.

Causes.—Hæmorrhage or other exhausting discharge ranks first among the causes of cerebral anæmia. I have known several severe cases induced by epistaxis, and one by the continued loss of blood from leech-bites. Hæmorrhoidal bleeding has also caused it in my experience. No influence of the kind is, however, more common than uterine bleeding, such as occurs before, during, or after labor, from miscarriages and abortions, especially if they are frequently repeated, and from excessive menstrual discharge.

Chronic dysentery and diarrhœa, malarial and other fevers, the rheumatic, strumous, and cancerous diatheses, diseases of the bones and joints, and long-continued purulent discharges, are likewise causes of cerebral anæmia.

I have several times seen the affection apparently caused by congestion of internal organs. Niemeyer, referring to this possibility, cites the fact that it may follow the use of Jounod's boot. At the present time, when this appliance is variously modified and extended beyond its legitimate use by itinerant quacks, it is well to call special attention to this liability. Several cases in point have come under my observation, and in one, a young lady suffering from epilepsy with cerebral anæmia, whom I saw in consultation with my friend Dr. J. Marion Sims, severe paroxysms were induced by each application of the "exhauster." In this case the operator placed the whole body, with the exception of the head, in a vacuum. In another instance exhaustion from the leg alone caused syncope every time the operation was performed.

Pressure or obliteration upon the arteries supplying the brain is another cause. A lady was recently under my no-

tice in whom both carotid arteries had been tied, for cirsoid aneurism of the scalp, by the late Dr. Kearney Rodgers and my friend Prof. W. H. Van Buren. When I saw her, several years after the operation, there was well-marked cerebral anæmia, the most striking symptoms of which were vertigo and drowsiness. Tumors of various kinds may act in a similar manner. Feebleness of the heart's action, such as results from fatty degeneration, may also occasion cerebral anæmia.

As we have seen, excessive mental exertion is a common cause of cerebral congestion. Strange as it may appear, I have had several cases of cerebral anæmia under my care in which the disease was clearly the result of a like cause, and these were instances in which the brain had been overtasked to an extreme degree. A little reflection will, I think, show that such cases are strictly in accordance with what takes place in other parts of the body. Thus we see the moderate use of a muscle or set of muscles increase their size and strength. Inordinate exercise induces hypertrophy, but, if the power of the muscles be still more severely tried, atrophy results. One of the worst cases of progressive muscular atrophy I ever saw occurred in the person of a ballet-dancer, whose gastrocnemii muscles were the apparent starting-points of the disease. Excessive cerebral action produces exhaustion and exhaustion causes anæmia, as surely as anæmia causes exhaustion.

The action of mental emotions is more obvious. We know that some emotions increase the amount of blood in the brain. Others diminish it, and sometimes with such suddenness as to cause syncope. Fear is one of these, and we have all seen the face become pale under its influence.

Certain medicines are causes of cerebral anæmia, both by their action on the vaso-motor nerves and in diminishing the power of the heart. Tobacco, tartarized antimony, calomel, oxide of zinc, and the bromides of potassium, sodium, and lithium, are among the chief of these. I was the

first to point out this influence of the bromides, and in a recently-published memoir [1] have given several cases in illustration of its action. The drowsiness, vertigo, nausea, fainting, weakness of the muscular system, numbness, failure of memory, mental aberration, pallor of the countenance, and anæmia of the retina, all go to show that the quantity of blood in the brain is diminished. Recent investigations not yet published have convinced me that the oxide of zinc acts in a similar manner.

Insufficient nutrition, either from deficient or improper food or disease of the digestive or assimilative organs, is a very common cause. Through its influence not only is the absolute amount of blood lessened, but its quality is deteriorated. The quantity sent to the brain is hence diminished, and that which is supplied is lacking in its proper proportion of red corpuscles. Many of the cases of cerebral anæmia occurring in large cities originate from such influences, and likewise from the vitiated air of narrow and crowded streets, from cold and from deprivation of light.

Diagnosis.—The principal affection with which cerebral anæmia is liable to be confounded is cerebral congestion. Indeed, there is no other which can be mistaken for it, if even ordinary perception and judgment be exercised.

From this it may be diagnosticated by the history of the case, and a careful inquiry into the etiology, by the fact that drowsiness, not wakefulness, is a prominent symptom; that the pupils are dilated instead of being contracted; that the pain is more apt to be fixed in a limited part of the head instead of being general, that it and the vertigo are increased by the assumption of the erect position, and diminished by lying down; that the ophthalmoscope shows retinal anæmia;

[1] On Some of the Effects of the Bromide of Potassium when administered in Large Doses. Quarterly Journal of Psychological Medicine, January, 1869, p. 46. In this paper I stated that one of the most constant phenomena was contraction of the pupils. Very greatly increased experience has convinced me that this is an occasional circumstance, which occurs during the early period of administration only.

that the face is pale and the skin cold; that the pulse is weak and frequent, and that bellows murmurs are heard at the base of the heart and in the veins of the neck. The effect of stimulants and tonics in mitigating these symptoms, and the fact that they are increased by exertion, and debilitating influences, are also important points to be considered in forming a diagnosis. Attentive consideration of these differential phenomena will prevent a mistake which may be fatal to the patient.

Prognosis.—The prospect of recovery in cases of cerebral anæmia depends mainly upon the removal of the cause, and the adoption of suitable treatment. In those cases which are the result of sudden and profuse loss of blood, the prognosis is grave, and this is especially so if the patient is pulseless and convulsions have occurred. In such instances, even though the hæmorrhage has been arrested, it may be impossible to save the patient.

In the gradually-developed form the prognosis is generally favorable.

Morbid Anatomy.—The vessels of the brain and its membranes are observed upon post-mortem examination to contain less than the normal amount of blood. The tissue of the brain is pale, and section shows a diminished number of the red points in the white substance. Sometimes there is an increased amount of serous effusion in the subarachnoid space, but the ventricles are generally empty.

Pathology.—The questions to be discussed under this head are similar to those connected with the same point in cerebral congestion. That the quantity of blood within the cranium can be diminished as well as increased admits of no doubt, and the fact that the symptoms grouped together as indicating the existence of cerebral anæmia are really the result of deficient blood-supply to the brain is equally certain. The experiments of Kussmaul and Tenner,[1] as well as those of other physiologists, are perfectly convincing.

[1] Untersuchungen über Ursprung und Wesen der fallsuchtartigen Zuckungen,

To observe in man the effects of even temporarily cutting off the supply of blood to the brain, it is only necessary to compress the carotid arteries for a few moments. I have repeatedly done this in rabbits to the extent of producing insensibility and convulsions. Jacobi[1] relates the following symptoms as generally observed in the human subject: Dimness of sight, dizziness, stupor, weakness in the legs, staggering, swooning, loss of consciousness, and sudden apoplectic falling down.

Dr. Alexander Fleming[2] tried the effect of compressing the carotid arteries. "There is felt a soft humming in the ears, a sense of tingling steals over the body, and in a few seconds complete unconsciousness and insensibility supervene and continue as long as the pressure is maintained. I have recently performed this experiment several times, with the effect of producing similar phenomena, together with pallor of the countenance, dilatation of the pupils, and temporary headache.

In many cases of cerebral anæmia, the cause, as we have seen, resides in the blood-producing functions, and is such as to cause the formation of blood which does not contain its due supply of red corpuscles. Here, although there may be no diminution in the actual volume of this fluid circulating in the cerebral vessels, the effect is the same so far as the nutrition of the organ is concerned, and hence the symptoms of anæmia are slowly evolved.

Again, it cannot be doubted that spasm of the blood-vessels produced through the sympathetic and vaso-motor nerves explains the origin and continuance of many cases of cerebral anæmia. It is in this way that mental emotions

Frankfurt, 1857. Also, On the Nature and Origin of Epileptiform Convulsions, caused by Profuse Bleeding, etc. New Sydenham Society Translation, 1859.

[2] Quoted by Kussmaul and Tenner.

[1] British and Foreign Medico-Chirurgical Review, April, 1855, p. 529, in a paper entitled "Note on the Induction of Sleep and Anæsthesia by Compression of the Carotids."

act, and sometimes with such rapidity as to cause instant death. This spasm may be kept up for a very considerable period, with the effect of developing the ordinary symptoms of cerebral anæmia, even after the emotion which originated it has long since disappeared.

Treatment.—The first indication to be fulfilled in the treatment of cerebral anæmia is to get rid of the cause. It often happens that this is still in active operation when patients come under our care, and there is no hope of permanent success till it is removed. Thus, if there is hæmorrhage from a divided vessel, from the uterus, the bowels, the lungs, or other part of the body, it must be arrested; if there is exhausting discharge from the air-passages, the intestines or the genital organs, it must be stopped; if the digestive or assimilative organs do not perfectly perform their offices, they must be put in good condition, if a tumor or other obstruction to the due course of the blood to the brain exist, it must be removed; and if the hygienic conditions surrounding the patient be bad, or the food inadequate in quantity or quality, they must be improved.

No medicine exercises so powerful an effect in cerebral anæmia as alcohol in some form or other. Perhaps, all things considered, the spirituous liquors, such as whiskey, brandy, and rum, are more generally applicable. For the influence is more rapidly felt, and there is not the same risk of exciting or aggravating gastric disorder, as when vinous or malt liquors are used. The quantity must be regulated according to the circumstances of each case, and should always be large enough to materially increase the force of the heart.

But if this were the only effect of alcohol, its benefits in cerebral anæmia would be but temporary, and would certainly be followed by a period of depression. Aside, however, from its stimulating action in the heart, its tendency is to improve the appetite and digestive power, and to relax any spasm of the blood-vessels that may be present.

Occasionally it happens that alcohol is badly borne by

anæmic patients. The brain has for so long a time been deprived of a due amount of its natural stimulus—blood—that time is required to enable it to tolerate and be improved in tone by the increased supply. Thus the physician will find that in some cases the patients will be apparently rendered worse by the remedy which of all others is calculated to do them most good. The headache and vertigo are increased, and the general feeling of debility and *malaise* greatly augmented, and the complaint made that the liquor has "gone to the head."

Now, it must be recollected that the brains of anæmic persons are in very much the same condition as the eyes of those who have for a long time been shut out from their natural stimulus—light. When the full blaze of day is allowed to fall upon their retinæ, pain is produced, the pupils are contracted, and the lids close involuntarily. The light must be admitted in a diffused form, and gradually, till the eye becomes accustomed to the excitation. So it is with the use of alcohol in some cases of cerebral anæmia. The quantity must be small at first, and it must be administered in a highly-diluted form, though it may be frequently repeated. Cases in which this intolerance of stimulants is exhibited are almost invariably of long duration, and are as those in which from a like cause wakefulness is produced by the recumbent posture.

The carbonate of ammonia, or the aromatic spirits of ammonia, may be given if there are any special reasons why alcohol should not be used, but they are not to be compared to it in efficacy.

In very extreme cases ether is preferable for the time being to any other remedy, on account of its diffusive nature; and transfusion may be necessary to save life.

As adjuncts to alcohol, the bitter tonics, such as quinine, gentian, colombo, and quassia, are useful. Iron is almost always required, though there are patients who do not tolerate it. In such cases manganese may be substituted with advantage. I have frequently used the sulphate, in doses of

five grains, with excellent results. When iron is borne, I know of no better combination than that given on page 57. Cod-liver oil is also a valuable agent in the disease under consideration.

It must not be forgotten that food is the most important factor in relieving chronic cerebral anæmia. The main permanent influence of stimulants and tonics is exerted upon the appetite and digestion, and the blood and tissue forming functions mainly as an excitant. The real strength must come from the food. This should, therefore, be of good quality; animal food, such as milk, eggs, and meats of various kinds, forming its chief portion.

The influence of position should always be taken advantage of to facilitate the flow of blood to the head, and the erect posture avoided as far as possible, especially during the early stages of the treatment. Thus the patient should be encouraged to pass a good portion of the day in a recumbent position, and should be instructed to assume it at once on the occurrence of any aggravation of the symptoms.

The opposite course is fraught with danger. Physicians are often anxious that their patients should take physical exercise, but it must be recollected that those who suffer from cerebral anæmia have very little vital energy and a diminished amount of blood circulating through the organ from which the greater part of their nervous power comes. Muscular exercise lessens the energy, and still farther reduces the quantity of blood in the brain, for the muscles require an increased supply while in a state of activity. To be sure, after the strength of the system is in a measure improved, the blood increased in quantity and quality, and the brain supplied with something like its proper proportion, moderate physical exercise is of the greatest service.

I have several times witnessed severe consequences from the assumption of the sitting or erect position too soon after a profuse hæmorrhage, and in one case death resulted.

As regards mental labor, there is not much need of cau-

tion, for the reason that it is impossible for the patient to undertake it to any dangerous extent. But, as he improves in strength, the desire to make use of his increased power may be manifested. It is, therefore, well at this time to prohibit any such exertion as will probably be followed by marked depression. Moderate mental exercise is, however, far from being prejudicial, for it tends to increase the amount of blood in the brain.

Emotional disturbance should also, as a rule, be avoided, although at times it may be productive of great benefit, especially if it be possible to bring into action an emotion contrary to that which may have produced the disease. Thus a lady became subject to cerebral anæmia, directly the result of painful emotions due to domestic trouble. The difficulty was very suddenly removed, or rather the knowledge of its removal was suddenly communicated to her. The reaction was very great; she was thrown into a state of joyous excitement, attended with considerable febrile disturbance, and I was apprehensive for a time that her mind might become permanently deranged, for there were hallucinations and delusions of various kinds, and many symptoms of cerebral congestion. But in the course of a few days, during which she was kept in entire seclusion, and as far as possible from all mental and physical agitation, she entirely recovered both from the secondary and primary disorders.

One word in regard to what not to do. From what has already been said in this and the previous chapter, the reader will have perceived that it would be exceedingly injudicious to administer any of the bromides in the treatment of cerebral anæmia. I should not, therefore, deem it necessary to say any thing further in regard to this point, but for the fact that I am very sure, from my experience, that wrong ideas prevail among some physicians relative to this subject. I see many patients affected with the disease under consideration, who have been treated with the bromide of potassium, and invariably with the effect of aggravating the difficulty.

Care in making a diagnosis and a knowledge of the fact that the bromides lessen the amount of blood in the brain are points which it is necessary to insist upon, even at the risk of being tiresome by repeating what has already been said.

CHAPTER III.

CEREBRAL HÆMORRHAGE.

Under the designation of cerebral hæmorrhage I propose to consider that disease which is often known as apoplexy, hemiplegia, or a paralytic stroke, and which is due to the rupture of a blood-vessel, and the consequent extravasation of blood either into the substance of the brain or into its ventricles.

Two forms of the affection, differing essentially only in the extent or seat of the lesion, but presenting different symptoms, are to be distinguished; these are the *apoplectic* and *paralytic*. In the first there is loss of consciousness; in the second the mind, though perhaps impaired, is not suspended in its action.

Symptoms.—Before the full development of the attack there often is, for several days, a group of symptoms present which indicate cerebral disorder. These are very much of the same character as those denoting the first stage of cerebral congestion, but, though generally not so numerous, are far more striking.

Among the more obvious is a sudden difficulty of speech arising from slight paralysis of the tongue and other muscles concerned in articulation. Words are not pronounced with the usual distinctness; the tongue seems to occupy more space in the mouth than it should, and is not moved with the requisite degree of promptness and rapidity.

The other muscles on one side of the face may be affected, and hence there is a little distortion, lasting, perhaps, but for a few hours.

MEDICINE

Defects of sight may occur, usually characterized by the presence of dark spots in the axis of vision. Such difficulties are due to minute extravasations in the retinæ, and are always of most serious importance. I have known retinal clots to precede by more than a year the occurrence of a more severe lesion.

Bleeding from the nose is a common precursor, and when occurring without being increased by severe muscular exertion, blows, a dependent position of the head, or other obvious cause in a person over the age of forty, is always to be regarded as a symptom of moment.

Numbness limited to one side of the body is of itself sufficient to excite apprehension. I have known several cases in which this symptom was the only premonitory sign. It may be present several days before, or may precede the attack by only a few minutes.

In addition, there may be headache, vertigo, slight confusion of mind, a tendency to stupor, and vomiting.

None of the premonitory symptoms may be present, and then the attack, if of the apoplectic form, occurs with great suddenness. Even if they have been noticed, there is more or less of abruptness in the onset.

Thus the individual is perhaps standing engaged in conversation, when he is instantaneously struck with unconsciousness, and falls to the ground as if shot; sensibility and the power of motion are abolished, and no signs of vitality are apparent to the ordinary observer, with the exception of the slow and labored action of the heart and respiratory muscles. The breathing is stertorous, the lips and cheeks are puffed out with each expiration, and the pupils are generally largely dilated and insensible to light.

Reflex movements are abolished at first, but after a few moments they reappear, and are even more readily excited than in health, owing to the fact that the controlling influence of the brain is removed.

The voluntary power of swallowing is lost, but it is usu-

ally not difficult to cause contraction of the muscles of deglutition by excitation of the pharynx. When these cannot be produced, the prognosis is, if possible, increased in gravity, for the reason that the extravasation is probably in the medulla oblongata, or so situated as to compress it.

The urine and fæces are often evacuated involuntarily.

An apoplectic attack of this character usually terminates in death without the patient recovering his intellect in the slightest degree. If life should be prolonged for thirty-six hours, the probability of a fatal termination is materially lessened. I have never seen a case of cerebral hæmorrhage that was instantaneously fatal, and, although from anatomical and physiological considerations I admit the possibility of such instances, I am persuaded that they must be rare. Jaccoud [1] expresses the opinion that death is immediate in those cases in which the hæmorrhage is in the medulla oblongata, or in those which occur in both hemispheres. Dr. Hughlings Jackson,[2] on the contrary, though conceding from theoretical grounds that hæmorrhage into or near the medulla oblongata might cause instant death, has never witnessed such a termination; and Dr. Wilks [3] says that apoplexy is very rarely, if ever, a suddenly fatal disease, no matter what part of the brain may be the seat of the effusion. Among the reports of several thousand post-mortem examinations at Guy's Hospital, there was but one in which death was asserted to have been instantaneous, and that was a case of meningeal hæmorrhage. Even this was doubtful, for the patient had fallen, some distance from the hospital, and was brought in dead.

I have several times had cases under my observation in which, it was said, death had been as sudden as though the individual had been struck by lightning; but careful inquiry

[1] Traité de Pathologie Interne. Paris, 1870. Tome premier, p. 166.

[2] On Apoplexy and Cerebral Hæmorrhage. Reynolds's System of Medicine. London, 1868. Vol. ii., p. 520.

[3] Guy's Hospital Reports, 1866, p. 178.

and post-mortem examination have either shown that the observers were deceived, or that there had been no extravasation at all, death being the result of heart-disease.

In the majority of cases attended with complete loss of consciousness, the course of the disease is not so rapid or hopeless as in the form just described. The patient falls, is comatose, breathes stertorously, and presents a similar general appearance, but after a time consciousness begins to return, and it is possible to partially rouse him from the condition of insensibility. He turns over in the bed, though with difficulty, and may attempt to speak. Articulation is, however, indistinct, for the muscles of one side of the face are paralyzed, and the tongue, from a like cause, is restricted in its movements. The paralysis is found to exist in the limbs of the same side, and involves the loss of sensibility, as well as of motion, though rarely to the same extent. In some exceedingly rare cases, perhaps not clearly understood, the paralysis of the limbs is on the opposite side to that of the face. A man thus affected was present at my clinic, in October, 1870, at the Bellevue Hospital Medical College. He was a patient under my charge at the New York State Hospital for Diseases of the Nervous System, and had been attacked several years previously. His history, as elicited with great care by my clinical assistant and resident physician of the hospital, Dr. Cross, was perfectly clear on this point.

The facial paralysis presents several points of great interest in a diagnostic point of view. The affected side is incapable of expression, but, so long as the patient does not attempt any facial movements, scarcely any distortion is perceived. Should he endeavor to open his mouth to spit or to puff out his cheeks, the paralysis is at once perceived. Owing to the fact that the antagonism of the muscles is destroyed, the face is drawn toward the sound side, the angle of the mouth being slightly depressed. It is remarkable, however—and the fact is of importance as a diagnostic mark

between the facial paralysis of cerebral hæmorrhage with hemiplegia and the simple facial paralysis from injury or disease of the eighth pair—that the patient does not lose the ability to close the eye of the affected side.

If the fifth pair of nerves is involved in the lesion, sensibility is impaired, which is never the case in simple facial paralysis, and the masseter and pterygoid muscles, which receive their motor influence from this nerve, will consequently be paralyzed. The ability to masticate on the affected side is therefore lost, and the cheek hangs lower than on the sound side.

The tongue is also only paralyzed upon one side. When, therefore, it is protruded from the mouth, the point deviates toward the paralyzed side, owing to the uncompensated action of the sound genio-hyoglossus.

All these paralyses occur on that side of the body opposite to the seat of the lesion. The muscles are relaxed ordinarily, though sometimes they are in a state of tonic rigidity. Generally, however, rigidity, when it exists, is in the muscles of the non-paralyzed side.

The temperature of the affected side, as determined by the thermometer placed in the axillæ, is at first higher than on the sound side, but at a subsequent period it becomes lower.

It is rarely the case that the third nerve is affected. When it is, there is external strabismus from paralysis of the internal rectus muscle, and ptosis from paralysis of the elevator of the upper eyelid. The pupil is dilated, and is insensible to light.

Another phenomenon is sometimes observed, and that is the rotation of both eyes toward the sound side. This is accompanied by a like movement in the head, so that, if the patient is paralyzed on the left side, the eyes and head are turned to the right, and consequently, as the patient lies in bed, the right side of the face rests on the pillow. I have observed these symptoms in about one-third of the cases of

cerebral hæmorrhage which have come under my observation. They were present from the very beginning, and disappeared in a few days.

Slight convulsive or involuntary movements are occasionally noticed. The most frequent of these is yawning, a symptom which Dr. Todd[1] regards as troublesome, and even unfavorable, but which, in my experience, is not very annoying or dangerous. The other convulsive actions may be on the whole of either side of the body, or on both sides, or may be restricted to a single limb or even a group of muscles.

Reflex movements can always be excited, especially in the lower extremity, by tickling the sole of the foot. Deglutition, though imperfect, can generally be made to take place by reflex action, unless, as previously stated, the hæmorrhage is in, or in the vicinity of, the medulla oblongata.

In the less severe apoplectic form of cerebral hæmorrhage now under consideration, the urine and fæces are sometimes passed involuntarily from paralysis of the sphincters, and are at times obstinately retained from paralysis of the bladder and abdominal muscles.

The mental symptoms are at first scarcely distinguishable from those which are present in the severest form of the disease. The coma and insensibility are complete, but after a time, which varies in duration with the extent of the lesion, consciousness begins to return. The patient opens his eyes, and gives a little attention when loudly spoken to; and is perhaps able to express, to some extent, his wishes by signs and gestures. Gradually the mental power increases; he attempts to speak, but his words are misplaced or forgotten, and his articulation, owing, as already stated, to the paralysis of the face and tongue, is thick and indistinct. Those words which are enunciated by the movements of the lips and tongue are especially troublesome, while those formed in the throat are not difficult to pronounce.

The mental characteristics of the patient will be found

[1] Clinical Lectures. Second edition. London, 1861, p. 708.

to have undergone a radical change. He is irritable, unreasonable, and fretful. His sense of the proprieties of life, which may in health have been very delicate, becomes obtuse; his memory is notably impaired, and his reasoning power greatly diminished. The greatest change, however, is perceived in the emotional faculties. He laughs at the veriest trifles, and sheds tears profusely at the least circumstances calculated to annoy him. Even for years afterward this peculiarity is noticed.

Such is the first stage of an attack of cerebral hæmorrhage marked by apoplexy and paralysis, as ordinarily observed when amendment takes place. It is often the case, however, that this stage is not fully developed, owing to the continuance of the hæmorrhage. In such an event the coma becomes more profound, the breathing more irregular and less frequent, the pulse intermits and loses in force, the face becomes purple from imperfect aëration of the blood, and death ensues. In other cases a certain degree of improvement may be attained, and then the hæmorrhage may recur, and the patient dies comatose. Taking, however, a case in which the improvement has been progressive up to the point of partial resumption of the mental faculties, we find that a second stage characterized by different symptoms often supervenes. This is the period of inflammation.

It may begin at a variable time after the occurrence of the extravasation, usually not later than the eighth day. It is marked by febrile excitement and pain in the head, the latter being often very severe. There is gastric derangement, as evidenced by nausea and vomiting; and convulsive movements of the limbs, with contractions of the flexors of the paralyzed side, are generally present. Delirium is also a prominent feature. Sometimes there is obstinate wakefulness, and at others a strong tendency to coma. This stage may last three or four days, or at most five or six, when it either causes death by extension of the inflammation from the immediate vicinity of the lesion to other parts of the

brain, terminates in the formation of an abscess, or gradually ends in resolution, with abatement of the symptoms.

Disregarding for the present the first two of these results, we proceed with the consideration of the phenomena of a case in which resolution takes place.

With the cessation of the inflammatory action, the improvement of the patient becomes very marked. His speech is every day more distinct, his mind more active, his paralyzed limbs more capable of motion. Usually the leg recovers power with much greater rapidity than the arm, and thus the patient is able to walk tolerably well before he can raise his arm from his side, bend the elbow, or extend the fingers. The paralysis in the leg is most marked in those muscles whose office it is to elevate the foot, and this necessitates a peculiar gait in order to avoid dragging the toes along the ground. The abductors are rarely affected to any great extent. The patient in walking, therefore, throws the leg out from the body, and then, swinging it around, clears the ground in this manner.

In the upper extremity there is almost invariably a disposition toward contraction of the pectoralis major and minor muscles, by which the arm is drawn across the front of the thorax. At the same time the latissimus dorsi, the trapezius, the rhomboidei, the teres major and minor, are generally in a state of relaxation, and eventually tend to atrophy. The elbow is slightly flexed, the wrist bent upon the forearm, and the fingers drawn in toward the palm of the hand. These actions may, in a great measure, be prevented by appropriate treatment, and they may vary in extent according to the gravity of the attack. It is a curious fact that the muscles of respiration are never paralyzed in cerebral hæmorrhage unless the medulla oblongate be insolved.

Trousseau [1] has insisted, with great force, on the fact that, when the arm regains power before the leg, the termination

[1] Lectures on Clinical Medicine. Bazire's Translation. Part I. London, 1866, p. 16.

is always fatal. That this is the general result, I am very sure from my own experience, but it is not invariable, for there are now in the New York State Hospital for Diseases of the Nervous System two patients affected with cerebral hæmorrhage whose arms have improved to a very great extent, while the legs are still as much paralyzed as ever.

Now, with all these troubles of motility, sensibility may likewise be affected to a greater or less extent. When this is the case, the limbs of the affected side at first feel heavy as if made of lead, and after a while numbness, as exhibited by a feeling as if ants were crawling over the skin, or water trickling over it, as if pins and needles were sticking in it, or as if that part of the body were "asleep," is noticed. Sometimes the sense of touch is greatly lessened, while the ability to feel pain is scarcely impaired, and indeed is often considerably increased. Again, there may be hyperæsthesia of the skin of the affected regions, and pain along the course of the nerves.

The circulation is inactive in the paralyzed limbs, and this, together with the deficient nervous power, tends to cause a permanent reduction of temperature. The difference may amount to as much as five or six degrees, and, as the ability to resist cold is diminished, the patient is obliged to use additional covering on the paralyzed members.

From continued disuse, atrophy of the paralyzed muscles always takes place unless suitable treatment be begun at an early period.

Thus far we have only considered those attacks of cerebral hæmorrhage which are accompanied with unconsciousness. One of these forms kills, without the patient so far recovering as to show whether he is paralyzed or not, though of course he is so to a profound degree; the other allows of more delay; the brain can still act to some extent, and, if death does not ensue from continuance of the hæmorrhage, the patient is found to be paralyzed on the side of the body opposite to the seat of the brain-lesion. One other form

requires notice, and it is, perhaps, the one most frequently met with. It differs from the attacks just described, in the important fact that it is unattended with unconsciousness.

Like the others, this species of cerebral hæmorrhage may take place very suddenly, without premonitory symptoms, or it may, like them, happen while the patient is said to be asleep. Generally, however, though there may be no long prodromatic stage, there are symptoms occurring immediately before the attack which indicate both mental and physical disturbance. These are headache, vertigo, numbness, vomiting, irritability of temper, and, perhaps, slight difficulties of speech.

When the attack comes, the individual, if standing, falls, from the immediate paralysis of one leg. He is fully sensible of his condition, although there is generally more or less mental change. The arm and face are affected, and the speech is rendered impossible or is indistinct.

If the patient be sitting or lying, he is aware that something has happened, but does not discover its exact character till he attempts to rise. A distinguished general officer of the army, after a fatiguing day of ceremony, entered his carriage with his wife, to be driven to his hotel. As he passed along Fifth Avenue, he felt an indescribable sensation, and immediately afterward noticed that he could only see the half of objects. He made no effort to speak, though he is confident he did not for a moment lose his consciousness. When he attempted to get out of the carriage, he found, to his surprise, that he was paralyzed on the right side, and that his speech was so much impaired that he could not be understood.

Another gentleman was reading an amusing book, at which he laughed heartily. He felt suddenly a feeling of vertigo, and the book dropped from his hand. He attempted to pick it up, but found he had lost power in the arm, and, on trying to call to his wife, who was in the same room, discovered that he could not speak. At this time he could

walk, but in a moment or two afterward he fell, from paralysis of his leg. So far as the paralysis is concerned, I have rarely seen a more severe case than this.

Another went to bed, perfectly well, to all appearance, having enjoyed uninterrupted good health for several years. In the morning he arose, but felt a little pain in his head. As he stood before his glass, he thought his face was slightly twisted, and he noticed as he was shaving himself that he did not feel the razor on one side. While he was testing his facial mobility and sensibility, he experienced a trace of numbness in his left hand. This gradually increased, and in addition the limb lost power. In a few minutes he could not move it at all. By the time I saw him—two hours afterward—the paralysis had extended to the leg. At no period was there insensibility or mental confusion.

A gentleman retired at night in good health. On attempting to get out of bed he discovered that he was paralyzed in the leg. Neither the arm nor the face was affected.

Several cases have been under my care in which only the face or the tongue was paralyzed; others in which the arm alone was involved; and others, like the one just mentioned, in which the symptoms were confined entirely to the leg. Sometimes there was a momentary feeling of vertigo, sometimes a vacant stare, something like that of the *petit mal* of epilepsy, sometimes a slight degree of intellectual confusion, sometimes headache, and, again, no head-symptoms whatever. The subsequent progress of such attacks requires no special consideration beyond that already given to the more severe forms.

Now, no matter how light the attack may have been, nor how rapid the improvement, the patient who has had cerebral hæmorrhage is never mentally or physically the same as he was before. If the seizure has been severe, he may advance so far toward a complete cure as to evince very little disorder of his mind or body. But close observation shows

that he is not entirely restored, and, though he may do very well for light intellectual and physical exertion, severe labor of either kind is beyond his powers—and no one is more sensible of this fact than himself. Even after years his emotions are abnormally excitable. A patient now in the New York State Hospital for Diseases of the Nervous System informs me that he sheds tears every time a funeral passes him, and that even hearing of any one's death, or reading the obituary column in a newspaper, causes his feelings to get the better of him. In the lightest forms of the attack, this easily-aroused emotional disturbance is a marked feature for years subsequently, if it ever entirely disappears. And as regards the muscles which have been paralyzed, it is very certain that, though they may be made strong enough for all practical purposes, they never can be restored to their former sound condition.

The character and general mental type of the individual usually undergo some change; and this may be to the extent of reversing his ordinary traits.

Causes.—Advanced age is one of the most influential circumstances which predispose to an attack of cerebral hæmorrhage, and this fact has long been known. Thus Hippocrates [1] states that apoplexy is most common between the ages of forty and sixty, and modern investigation establishes the truth of the proposition as regards the actual number of cases. It is probable, however, that the liability increases, as Dr. Flint [2] says, from the age of twenty upward, and that there are not so many cases occurring in persons over sixty as below, for the reason that the number of individuals alive of that age is less.

Of two hundred and twenty-nine cases of cerebral hæmorrhage which have been under my professional care during the last five years, two hundred and four occurred in persons

[1] Aphorisms, chapter vi., aph. 57.

[2] "A Treatise on the Principles and Practice of Medicine." Third edition. Philadelphia, 1868, page 582.

LEEDS SCHOOL OF MEDICINE

over forty years of age. Of these, one hundred and seventy-two were between forty and sixty, twenty-four between sixty and seventy, five between seventy and eighty, and three over eighty.

Of the twenty-five cases in persons under forty, seventeen were between forty and thirty, seven between thirty and twenty, and one under twenty. This latter was a boy of seventeen, whom I exhibited at my clinic at the Bellevue Hospital Medical College in the autumn of 1870.

The disease is certainly more common among men than women, though some authors have asserted the contrary. Falret ascertained that, of twenty-two hundred and ninety-seven cases, sixteen hundred and sixty occurred in males and only six hundred and thirty-seven in females. In my own experience, of two hundred and twenty-nine cases, one hundred and fifty-three were in males and seventy-six in females.

Temperament and organization are supposed to have an influence in predisposing to cerebral hæmorrhage. It was formerly thought that those of sanguine temperament and plethoric habit who had stout bodies, large heads, florid complexions, and short, thick necks, were especially liable; but more exact and thorough investigation would appear to show that such is not the case, and that thin and pale individuals show fully as great a proclivity. Dr. Flint[1] expresses the opinion that there is no special apoplectic constitution, and my own experience is decidedly to the same effect.

That the tendency to cerebral hæmorrhage is often hereditary, appears to be very certainly established. Within my own knowledge, I am aware of several striking instances which support this opinion. A gentleman consulted me for hemiplegia, the result of cerebral hæmorrhage, whose grandfather, father, two uncles, two brothers, and one sister, had died of this disease, and whose son, thirty-six years of

[1] Op. cit., p. 583.

age, had been attacked. In another case a lady had her father, two brothers, and one sister, die of the disease; and, in a third very remarkable case, the great-grandfather, grandmother, father, four uncles and aunts, and two brothers, all in a direct line, died of cerebral hæmorrhage.

Piorry[1] cites the case of a woman, herself paralytic, whose three children had died of convulsions, and whose mother, uncle, and brothers and sisters, to the number of twelve, had died of cerebral hæmorrhage or convulsions. It has very often happened in my experience that the father or mother of a hemiplegic patient, whose condition resulted from cerebral hæmorrhage, had been affected in a similar manner.

As regards the influence of diseases of the heart, Legallois, Brichteau, Rostan, Andral, and Bouillaud,[2] adduce instances in support of the existence of a definite relation. While others, among whom Rochoux, Walshe, and Flint are to be placed, deny the existence of any causative influence. As tending to produce active or passive cerebral congestion, disease of the left or right side of the heart would reasonably seem to be conducive to the occurrence of cerebral hæmorrhage. The tension of the blood in the vessels of the brain is increased thereby, and the liability to the rupture of a diseased vessel rendered greater.

The condition of life has also been supposed to exert an effect in predisposing to cerebral hæmorrhage, it being asserted by some authors that the affection is much more common with the rich, and those living in ease, luxury, and refinement, than in the poor and laboring classes.

It is difficult to arrive at any very definite conclusion on this point, owing to very obvious reasons, but I am inclined to think the theory to be not well founded. It is only necessary to visit our large hospitals, to see how many of the inmates, drawn as they generally are from the laboring

[1] De l'Hérédité dans les Maladies, p. 107.

[2] Traité de Clinique des Maladies du Cœur, 2d edi., t. ii., p. 580

classes, are suffering from cerebral hæmorrhage or its effects.

Thus far we have only considered the more important, intrinsic, predisposing causes; there are, however, others which may be called extrinsic.

Season is one of the chief of these. The disease is much more common in winter than in the other seasons, although some statistics would seem to show more cases during summer. A careful examination of such, however, shows that under the head of apoplexy are included not only cerebral hæmorrhage, but congestion, sunstroke, embolus, and in fact nearly every other affection attended with sudden loss of consciousness. My own researches have been very exact on this point, and as their results I find that, of the two hundred and twenty-nine cases of which I have notes, eighty-five occurred in winter, forty-one in spring, fifty-six in summer, and forty-seven in autumn. It has been noticed, too, that sudden variations of temperature, especially from mild to cold weather, increase the number of cases of cerebral hæmorrhage.

Of the exciting causes, a long list can readily be made. Among them are the excessive use of alcoholic liquors and other stimulating substances, the use of opium in excess; the ingestion of large quantities of food, especially such as is stimulating and indigestible; excessive physical or mental exertion, strong emotional disturbance, such as anxiety, extreme joy, anger or terror; the act of coition, especially in old people; straining at stool; enlarged prostate, or paralysis of the bladder, requiring strong muscular efforts for the evacuation of the urine; childbirth; tight clothing about the neck, chest, or abdomen; certain occupations which require the head to be depressed; vomiting, sneezing, coughing, and laughing; exposure to the direct rays of the sun or other sources of great heat; the sudden arrest of a customary flux, such as hæmorrhoidal bleeding; the sudden application of cold water to the body; long-continued bathing in very

warm water; the circumstance that the patient has had a previous attack, and certain diseases, as gout and syphilis.

In regard to some of these causes, I may state that several very interesting cases have occurred in my own practice. In one, a lady was attacked on hearing that her cook had left her, in another the emotion excited by the fall of a picture from the wall caused a seizure. Four cases produced by straining at stool have come under my observation. In one of them a gentleman well known in public life retained sufficient consciousness and intelligence to take a large key out of his pocket with the non-paralyzed hand, and to rap on the floor for assistance.

Two cases occurred during sexual intercourse, one in a man, the other in a woman. In one of these there was, subsequently, a great increase of venereal desire. In one case, the seizure was induced by stooping over to tie the shoe. This was in the boy, seventeen years of age, already mentioned. It must be confessed, however, that very frequently, perhaps in the majority of cases, no immediate cause can be reasonably alleged. Of the two hundred and twenty-nine cases noted by myself, no cause was noted in one hundred and fifty-three.

Relative to the influence of sleep, I am by no means in accord with those authors who regard it as a powerful, exciting cause. During sleep the quantity of blood circulating in the cerebral blood-vessels is diminished, and hence there is less tension upon their walls than during wakefulness. I doubt very much whether cerebral hæmorrhage ever occurs during healthy, undisturbed sleep.

But there is a condition which supervenes upon sleep, and which, to ordinary observers, presents the usual phenomena of sleep, but which is really a very different state, both as regards the brain and the symptoms—and that is stupor due to venous congestion. In this affection there is an increase of the pressure upon the brain, produced by the over-distended vessels; and hence coma, to some extent, ensues.

This state is characterized by difficulty of awaking the individual, by turgescence of the larger veins of the neck, by a more or less purple hue of the face, by snoring, and by the puffing out of the lips and cheeks in breathing. Both of these latter phenomena are due to paralysis.

In this condition it is not unusual for cerebral hæmorrhage to occur, but the existing state is not sleep.

So far as my own experience extends, I have not found a majority of the cases, where I have examined into this point, to have taken place either during sleep or the stupor to which I have referred. I have made it a rule, not only in those cases of cerebral hæmorrhage which have been under my own care, but all others, in which I could do so, to inquire particularly with reference to the matter in question, and have found that, in two hundred and fifty-five out of three hundred and forty-two cases, the individuals were awake at the time of the attack.

Doubtless much of the confusion has arisen, not only from the non-discrimination of sleep with stupor, but also from treating of apoplexy as a disease instead of regarding it as a symptom due to several very different pathological conditions of which cerebral hæmorrhage is only one, and of which embolism, thrombosis, congestion, meningeal hæmorrhage, and epilepsy, are others.

Finally, it may be said of the etiology, that whatever tends to increase the flow of blood to the head, or to retard its exit, is capable of acting as an immediate cause of cerebral hæmorrhage.

Diagnosis.—The diagnosis of cerebral hæmorrhage is ordinarily not difficult, but it must be confessed that one or two affections are very liable to be confounded with it, and the attendant circumstances surrounding a patient in a condition of insensibility may be such as to materially increase the obstacles to the formation of a correct opinion.

Thus, supposing an individual to be found in a state of profound insensibility, the condition may be due to compres-

sion from injury of the skull, to concussion from a fall or blow, to congestion, to asphyxia, to syncope, to a recent epileptic fit, to uræmic intoxication, to hysteria, to narcotism, or to drunkenness.

A mistake of either of these states for cerebral hæmorrhage would be, in the end, embarrassing to the physician, and, perhaps, injurious to the patient.

The coma might also be the result of embolism, of thrombosis, of tumor, of abscess, or of meningeal hæmorrhage; but, as regards these conditions, no opprobrium could be attached to the physician, or harm come to the patient, by any error of diagnosis, although a regard for scientific exactness should always prompt us to be as specific as possible in our inquiries and examinations.

From asphyxia, cerebral hæmorrhage is distinguished by the fact that in the former the respiration is suspended. The cause is also often apparent. A careful examination of the cranium, and a survey of the surrounding circumstances, will enable the physician to ascertain the existence or non-existence of compression from traumatic cause. This cause may either be depression of bone, the rupture of an internal blood-vessel, or the entrance of some foreign body, as a bullet, into the interior of the skull. So far as symptoms are concerned, there might be considerable difficulty in diagnosticating either of these accidents from cerebral hæmorrhage, but the history would render a mistake impossible.

Concussion presents more difficulties, because the comatose person may be found in such a situation as to warrant the opinion that he has fallen from a height, or otherwise received a blow on the head, when in fact he is suffering from cerebral hæmorrhage. But if he has fallen from a height or been struck, there will probably be more severe bruises about his person than if he is affected with cerebral hæmorrhage, and there may be bleeding from the ears or nose—symptoms of cranial injury not met with in the latter condition.

If, however, the individual has fallen from a height, he may have done so in consequence of an extravasation of blood in his brain, and he may present all the marks of suffering simply from the concussion, or he may have fractured skull with compression. It is, therefore, impossible to make a correct diagnosis in all cases, or to lay down any certain rules which will constitute infallible guides. It is perfectly possible to meet with cases such as those referred to, in regard to which no human judgment can be certainly correct. Such instances are of course rare, and accordingly, in the great majority, the circumstances and the presumption will generally lead to a correct opinion.

From congestion of the apoplectiform variety cerebral hæmorrhage can generally be distinguished without much difficulty. The absence of stertorous breathing, the short duration of the coma, the transient character of the paralysis, the contraction of the pupils, the fact that the loss of sensibility and the power of motion are not generally confined to one side of the body, and the longer continuance of premonitory symptoms, will be sufficient indications of the existence of congestion. Syncope is distinguished by the circumstances that the respiration and circulation are both diminished in power if not suspended, that there is no hemiplegia, that the face is pale, the skin cold, and that these phenomena are all transitory in character. The history of the case will also assist us in arriving at a correct judgment.

Epilepsy, if seen from the beginning of the paroxysm, cannot be mistaken for cerebral hæmorrhage, nor this latter for epilepsy, if the onset of the attack has been witnessed. But a person found in a comatose condition, with no previous history to guide us, may be supposed to be either in the comatose stage of an epileptic paroxysm, or to be laboring under a seizure due to extravasation of blood. In such a case, if the fit has been epileptic, foam will be found around the mouth, and perhaps blood from injury of the tongue or cheek. Moreover, the stupor of epilepsy is not

usually of long duration, and is not generally characterized by stertorous breathing.

In uræmia, the coma of which is very similar to that resulting from cerebral hæmorrhage, the history of the case is our chief reliance for a correct diagnosis, though the absence of hemiplegia and the general presence of anasarca are of course of great value. Moreover, in very doubtful cases the urine may be drawn off by the catheter, and examined for albumen and tube-casts. If these are present, the probability of the stupor being due to Bright's disease and uræmic intoxication is very much increased.

Coma is sometimes a manifestation of hysteria, but a very little acquaintance with the phenomena of this condition will suffice to prevent mistakes. In some cases of hysterical coma there is well-marked hemiplegia; but even when this complication is present, the facts that the hysterical diathesis exists, that there have probably been other manifestations of hysteria, that the pulse is small, weak, and frequent, and that the breathing is free from stertor, will enable a correct diagnosis to be formed.

In narcotism the condition often bears a close resemblance to that due to cerebral hæmorrhage. But in the former there is no hemiplegia, the pupils are generally contracted, the respiration is not stertorous, and the coma comes on gradually.

Drunkenness and cerebral hæmorrhage are often confounded. I have known some sad mistakes of the kind to be made, both by professional and non-professional persons, many of which were unavoidable, for it must be confessed that there are great difficulties connected with the subject. The habit of drinking alcoholic liquors is so general that no reliance can be placed upon the test of smelling the breath. A person may have just taken a glass of wine or of brandy, and be seized with extravasation of blood in his brain immediately afterward, and when not in the least intoxicated. And, even if dead-drunk, he may at the same time have

cerebral hæmorrhage. In such a case as the latter, discrimination would be impossible. In ordinary cases of alcoholic intoxication the patient can generally be roused to some extent; the pupils are dilated, but this latter is often the case in hæmorrhage; the breathing is usually free from stertor, but some drunkards always snore; the pulse is small and weak, and there is no hemiplegia. When all these symptoms are in accord, there will be little difficulty; when they are not, the physician must be guarded in his expressions of opinion, and diligently inquire into the personal characteristics of the patient and all matters bearing on the history of the case.

From the centric diseases previously mentioned, the diagnosis of cerebral hæmorrhage is easy as regards some, and difficult as to others. Thus, from embolism it cannot in many cases be distinguished in the first stage. But when all the phenomena are taken into consideration the chance of error is very much diminished. Embolism is generally accompanied with disease of the left side of the heart, and there is often a history of rheumatism; there are never any premonitory head-symptoms; it occurs in young persons as well as old; for reasons which will be explained when the subject of partial cerebral anæmia from embolism is considered, the resulting hemiplegia is generally on the right side; the paralysis generally disappears in a few hours after the attack; if it does not, there is no gradual improvement, as in cerebral hæmorrhage; there are no contractions or partial convulsions,[1] and there is more frequently delirium.

The gradual development of the symptoms in thrombosis, tumor, or abscess, and the frequency with which convulsions ensue in the latter diseases, together with the associated symptoms, will prevent the coma which sometimes exists being mistaken for the stupor of cerebral hæmorrhage.

[1] Jaccoud (op. cit., p. 141) so asserts, though I have seen one case in which post-mortem examination revealed the presence of an embolus in the middle cerebral artery, and in which there had been convulsions.

During the subsequent stages of cerebral hæmorrhage, when the mental condition and the hemiplegia are the most prominent features, inquiry into the antecedent history will bring out the foregoing points, and assist us in arriving at a correct idea of the cause. Even, however, should we be baffled in this respect, no great inconvenience could result either to the patient or physician.

Prognosis.—The prognosis depends upon the extent of the hæmorrhage, and refers to the probability of saving life during the period of attack and immediately afterward, and of curing or mitigating the subsequent paralysis.

In the severe apoplectic form, death is almost inevitable; so far as my experience goes, it is the invariable result. It generally takes place within a few hours. If, however, life be prolonged till the fourth day, there is some hope. Irregularity of pulse, or one very rapid, impossibility of swallowing, involuntary evacuation of the fæces, and cold sweats, render, if possible, the prognosis still more unfavorable.

In the apoplectic form attended with paralysis, the gradual increase of the coma and hemiplegia indicate the continuance of the hæmorrhage, and are consequently of grave importance. About one-third of those attacked with this form die. The prognosis is bad in accordance with the strength and age of the patient, and the circumstances under which the attack has occurred. Thus, if it has supervened in a person who has had no obvious exciting cause, the probability is that there is serious disease of the blood-vessels, whereas, coming on in a young person as the result of severe muscular exercise, or mental strain, the prognosis is more favorable. A second attack is more apt to prove fatal than a first, and a third than a second, and so on.

In the mild form characterized by paralysis, but no loss of consciousness, the prognosis is generally favorable. It must be recollected, however, that the risk of inflammation is quite great, both in this and the apoplectic form with

paralysis, and that the patient is not safe from it till after the eighth day.

And in both forms, if the temperature rise above 100° Fahr., if the respiration be chiefly abdominal, and rattling of mucus is heard in the throat, the prospect of recovery is bad. The same may be said of pain in the head and contractions of the paralyzed muscles.

As regards the probability of recovery from the paralysis, much depends upon the opportunities the patient may have for receiving proper medical treatment. The tendency is generally toward amendment even in the worst cases. Gradually the speech improves, the breathing becomes better, and the arm acquires more strength; but the improvement often stops now, and never goes on unaided to complete recovery. The longer the paralysis has lasted, the less prospect there is of great progress under any treatment, and, if strong contractions producing distortions have taken place, the prognosis is unfavorable.

Certain muscles recover better than others. The extensors of the foot and hand are especially intractable, but, as a rule, those of the lower extremity improve more rapidly than those of the upper.

The mind ordinarily improves, *pari passu* with the physical symptoms, though not always. I have witnessed several exceptions to the rule. Even in slight cases the intellect may suffer to a great extent, and in no case is it ever in all respects as good as before the attack. Among the unfavorable signs are persistent iritability of temper, failure of memory, and the existence of delusions. Difficulties of speech, whether as regards the memory of words, or the ability to coördinate the muscles of speech so as to pronounce them properly, are often very persistent. I have now under my care a gentleman who was attacked with cerebral hæmorrhage two years ago, whose physical powers are quite good, and whose mind is not seriously impaired, but who cannot yet remember sufficient words to

carry on an ordinary conversation. When the difficulty is simply due to paralysis of the tongue and facial muscles, the prognosis is more favorable.

Morbid Anatomy.—The seat of the extravasation from cerebral hæmorrhage may be in the substance of the cerebral tissue, or in the ventricles. The former is much the more common.

Now, the blood which is poured out from a ruptured vessel into the substance of the brain must, of course, occupy its place by separating or lacerating the fibres. It thus forms for itself a cavity which enlarges as the hæmorrhage goes on, until at last the resistance to further separation or laceration may be so great as to overcome the tension of the blood, and thus put a stop to the bleeding.

The shape of the cavity varies according to the manner by which it has been produced. When it is formed by the separation of the cerebral fibres, it is generally elongated; whereas, when produced by laceration, it is oval, round, or irregular in form. The situation of the hæmorrhage modifies the form of the cavity. In the hemisphere it is usually round, in the motor tract irregular or oval. The variations as regards size are great. I have seen clots no larger than a pea, and again as large as an orange. When hæmorrhage occurs in the motor tract, the clot is almost invariably small; whereas, in the hemispheres, in the cerebellum, or in the ventricles, it is large.

A clot does not always consist of blood alone. Brain-tissue is very often mixed with it, and this is especially the case when the extravasation has been in the hemispheres.

The ordinary seat of cerebral hæmorrhage is the motor tract, and especially the corpus striatum and optic thalamus. The crus cerebri may also be the seat of the extravasation, though not often primarily.

When the hæmorrhage occurs in the mesial line of the pons varolii, the paralysis is, of course, on both sides of the body. Two cases of the kind have occurred in my practice,

and both were from severe blows on the skull. One side of the pons is more frequently the seat.

The medulla oblongata is not often involved, though a few cases are on record.

The hemispheres and the lateral ventricles are occasionally the seats of extravasation, the cerebellum rarely.

Extravasated blood undergoes certain changes. Instead of separating into two parts, the clot and the serum, as does blood when exposed to the atmosphere, it remains for a time homogeneous and gelatiniform. About the fifth or sixth day it separates into two parts: the one, the serum, is absorbed by the surrounding tissue; the other, consisting mainly of the fibrine and the red corpuscles, contracts and becomes hard. By the fifteenth day it has become fibrinous in texture, and is changed from its former black hue to a yellow color. Microscopic examination, made at any period during these changes, reveals the presence of red corpuscles, crystals of hematoidin and sometimes of cholestrin. It never entirely disappears.

In the early period of the extravasation, the walls of the cavity are rough, and discolored with blood. But, as the changes are going on in the clot, the walls likewise alter in appearance; the inequalities and irregularities disappear, and a new formation of connective tissue lines the cavity. Blood-vessels appear in it, and aid in the absorption of the fluid portion of the extravasated blood. As the process of separation and absorption goes on, the cavity contracts upon its contents, and eventually forms a cicatrix which encloses the remains of the clot. This cicatrix is generally of a yellow color, and firm in texture.

Sometimes, however, absorption does not take place. The contraction of the walls of the cavity does not therefore ensue, and it remains distended with more or less altered blood. This may be the starting-point of secondary lesions, or a new hæmorrhage may occur into the same cavity, or an abscess may result.

MEDICINE

Pathology.—The theory of cerebral hæmorrhage brings us to the consideration of several important points. One of the first questions to be solved is, Can the rupture of a vessel of the brain take place—not including traumatic causes—unless the vessel is in a diseased condition? Both sides of this proposition have their adherents. On the one part, it is urged that cerebral hæmorrhage never takes place spontaneously unless the walls of the bleeding vessel have been so injured by disease as to destroy their strength and elasticity; on the other, that it is perfectly possible for a blood-vessel to give way, owing to increased tension of the blood or disease of the perivascular tissue, without the walls of the vessel itself being in the least diseased. While admitting that, in the majority of cases, the structure of the yielding vessel will be found to be impaired, I am satisfied that either of the other two causes may produce a rupture. The reasons for this opinion will be apparent in the course of the following remarks.

The most common disease to which the cerebral arteries are liable is chronic endarteritis, a condition which has been well described by Virchow,[1] and which is particularly apt to be met with in those who, from age or other debilitating influence, have had their nutrition impaired. As the consequence of this state, the vessels lose their elasticity, become brittle, and are therefore often unable to bear the ordinary tension of the blood, much less any severe strain. This disease may terminate in fatty degeneration of the arterial walls, or this last condition may be the primary affection. Fatty degeneration, like chronic endarteritis, is most commonly met with in badly-nourished persons, but who are at the same time cachectic. The inner coat is the point of origin, and hence it sometimes happens that this and the middle coat give way, leaving the external coat entire, and

[1] Ueber die Erweiterung kleinerer Gefasse. Archiv für Path. Anat. und Physiol., B. III., 1848, and Cellular Pathology, London, 1860, Lecture XVI.

thus forming an aneurism. But Bouchard,[1] who has examined into this matter with great minuteness, denies that such aneurisms are ever found, and asserts that the so-called aneurismal sac consists of the lymphatic membrane, lining the cavity in the perivascular tissue, through which the vessel passes, and that the blood, in such cases, has already ruptured the vessel. In reality, however, there is no hæmorrhage into the cerebral tissue till this membrane gives way.

In a subsequent memoir, by MM. Charcot and Bouchard,[2] this point is still more thoroughly considered, and the opinion expressed that cerebral hæmorrhage is almost invariably due to these so-called miliary aneurisms, which are the result of arteritis, and which are not necessarily preceded by atheroma.

In sixty-nine cases of cerebral hæmorrhage in which post-mortem examinations were made, atheroma was found but in fifteen, or twenty-two per cent., while these miliary aneurisms were met with in every case. They appear as little globular masses in the small intracranial vessels, and are in size from one-tenth of a millimetre to one millimetre. If they contain liquid blood, they are red; but, if the blood be coagulated, the color is dark, almost black in some cases. In the order of frequency, they are found in the optic thalami, the corpora striata, the convolutions, the tuber annulare, the cerebellum, the centrum ovale, the crura cerebri, and the medulla oblongata.

The condition of the perivascular tissue, or the brain-substance, has much to do with the occurrence of hæmorrhage. One reason why extravasation more frequently occurs in the brain than in the liver, for instance, is that its tissue is softer, and therefore not capable of giving as much

[1] Études sur quelques Pointes de la Pathogenie des Hemorrhagies cérébrales. Paris, 1866.

[2] Nouvelles Recherches sur la Pathogenie de l'Hemorrhage cérébrale. Archives de Physiologie Normale et Pathologique, 1868, pp. 110–643.

support to the blood-vessels as is the latter organ. Now, when the cerebral substance is softened by disease in any part, the natural support of the vessels of that part is still further lessened, and the tendency to hæmorrhage increased. Again, in the condition sometimes met with in old people, in which the brain becomes atrophied, the vessels may undergo dilatation and subsequent rupture. This view is opposed by Jaccoud,[1] but in one case of cerebral hæmorrhage, terminating in death, and in which I had the opportunity of making a post-mortem examination, the right hemisphere, the seat of the extravasation, was very considerably atrophied, and weighed three ounces and a quarter less than the left. The possibility of the existence of this cause may, therefore, be admitted, although it cannot be considered as definitely established. The researches of Cotard[2] would appear to show that cerebral hæmorrhage is not infrequently a cause of partial atrophy of the brain.

In the next place, the state of the blood, as regards quality and tension, must be considered. There can be no doubt that certain diseases affecting the general system may so deteriorate the blood as to render it unfit to properly nourish the blood-vessels, and hence their tissue is more readily broken down. Among these conditions are typhus, scurvy, chlorosis, and syphilis.

The tension of the blood in the vessels is subject to constant variation from the operation of many physical and mental causes, and may, through their action, be so increased as to overcome the resistance afforded by the vascular walls. These influences have been sufficiently considered in the section on causes, and need not, therefore, be dwelt upon here at any length. My own opinion of their sufficiency, without preëxisting disease of the blood-vessels, to produce rupture and extravasation, has been formed after much observation and reflection. Analogous phenomena

[1] Op. cit., p. 155.

[2] Étude sur l'Atrophie partielle du Cerveau, Paris, 1868.

take place every day, and are not supposed to be due, in any extent, to vascular disease. Thus nasal hæmorrhage occurs from strong muscular exertion of such a character as to retard the flow of blood from the brain, from emotional or other kind of mental excitement, and from hypertrophy of the left side of the heart, by which the amount of blood in the cerebral vessels is increased. All these causes augment the tension, and it would be singular if at times a healthy intracranial vessel did not give way through their influence as well as one outside of the skull.

A point of very great importance remains to be considered as a part of the pathology, and that is whether it is possible or not to determine during life in what part of the brain an extravasation has taken place? While I am afraid we cannot be as explicit in this matter as is desirable, I am very sure we can often, from a careful study of the symptoms, arrive at conclusions more or less accurate, and can sometimes determine the question with absolute certainty. The great difficulty is, that we are not yet sufficiently acquainted with the physiology of the several parts of the brain, and hence are not able to ascribe, with as much sureness as is desirable, variations from healthy action to derangement of the proper anatomical part of the cerebral mass.

As we have seen, hæmorrhage is more liable to take place within the ganglia constituting the motor tract than any other part of the brain. This is mainly due to the fact that this is the most vascular part of the cerebral substance.

When the lesion is limited to the corpus striatum of one side, there is loss of the power of voluntary motion on the opposite side, but no abolition of sensibility, except, perhaps, for a few hours. Cases in illustration of this fact have been given by Andral[1] and Luys,[2] and one instance in my own experience was established by post-mortem examina-

[1] Clinique Médicale, t. v., pp. 319–321, 442.

[2] Recherches sur le Système Nerveux Cérébro-Spinal, etc., p. 545.

tion. The patient, a man sixty-two years of age, had been hemiplegic for eleven years, and died suddenly, in April, 1851. Post-mortem examination showed the cause of death to have been fatty degeneration of the heart. On examining the brain, a cicatrix was discovered in the right corpus striatum. The hemiplegia was on the left side, and had never been accompanied with any loss of sensibility. There was no other lesion of the brain, so far as could be ascertained.

The optic thalamus is another common seat of extravasation. In such a case the observed symptoms are especially connected with the organs of the special senses. Thus there are double vision, dilatation or convulsive movements of the pupil, blindness, and anæsthesia or hyperæsthesia of the paralyzed parts of the body. As in lesion of the corpus striatum, the paralysis of motion, when it exists, is on the opposite side of the body. The hearing and smell may also be affected. Luys[1] has collected a large number of cases in support of the view here enunciated.

It generally happens that an extravasation, originating in either the corpus striatum or optic thalamus, involves both these ganglia. Hence we have, as the most common symptoms of cerebral hæmorrhage, loss or impairment of the power of motion, disturbance of sensibility, dilatation or irregular movements of the pupil, aberrations of vision, etc.

When the extravasation beginning in the left optic thalamus or corpus striatum extends to the fissure of Sylvius so as to involve the posterior part of the third frontal convolution, the island of Reil, or other part supplied by the middle cerebral artery, or when it originates in this region, aberrations of speech occur. These are independent of paralysis of the tongue, and are such as are embraced under the term aphasia. This subject will be hereafter more fully considered.

Hæmorrhage into the crus cerebri produces hemiplegia

[1] Op. cit., p. 534, et seq.

of the opposite side, more or less extensive, according to the size of the clot, with loss of sensibility. The third pair arises in part from the crus, and hence may be paralyzed, producing ptosis and external strabismus on the side corresponding to the seat of the lesion, and consequently opposite to the hemiplegia.

When the pons varolii is affected, the crossed paralysis is still more marked. The limbs are paralyzed on the opposite side, and the face in whole or in part on the same side as that in which the hæmorrhage takes place. If the extravasation is in the mesial line, both sides of the body are paralyzed. According to Trousseau,[1] however, crossed paralysis is not always due to a lesion of the pons, as asserted by Gubler,[2] and as supported by additional cases collected by Luys.[3] Trousseau rests his opinion on one case in which after death very extensive lesions of the brain were found, but none involving the pons.

The principal symptoms indicating the medulla oblongata as the seat of extravasation are, loss of the power of swallowing, from paralysis of the glosso-pharyngeal, difficulty of protruding the tongue, from paralysis of the hypoglossal, and huskiness of the voice, tumultuous action of the heart, dyspnœa and gastric derangements, from paralysis of the pneumogastric nerve.

In lesions strictly limited to the ganglia cited, there is no aberration of the intellectual faculties. It is only when the gray substance of the cerebrum or cerebellum is involved that the mind participates. In those cases of cerebral hæmorrhage, therefore, attended with coma or other mental phenomena, we may be very sure that the gray substance of the organs mentioned is affected, and this may be either from the hæmorrhage which has originated in the motor tract

[1] Lectures on Clinical Medicine, Bazire's Translation, part ii., p. 333.

[2] Sur l'Hémiplégie Alterne, Gaz. Hebd., October, 1856, and Mémoire sur les Paralysies Alternes, etc., Gaz. Hebd., 1859.

[3] Op. cit., p. 529, et seq.

being so extensive as to compress the cerebrum or cerebellum, or from an extravasation beginning in the first place in these ganglia. The researches I have made[1] relative to the functions of the cerebellum would seem to show that its office is not materially different from that of the cerebrum. Still, I think there are some indications which, although not perhaps giving us the right to form a definite conclusion, are yet sufficiently well marked to enable us to arrive at a probable diagnosis between hæmorrhagic lesion of the cerebrum and that of the cerebellum. Thus, vertigo is almost an invariable accompaniment of the cerebellar extravasation; vomiting is much more generally met with than when the cerebrum is affected; hemiplegia is not so common; the sensibility is never disturbed; and the pain is in the back of the head.

Besides a number of cases, some of which are referred to in the memoirs cited, one has occurred in my experience, in which I had the opportunity of making a post-mortem examination.[2]

A man had suffered from vertigo, occasional convulsions, attacks of nausea and vomiting, and a constant and violent pain affecting the back of the head. The symptoms had ensued in consequence of a severe blow which he had received on the back of the head by raising himself too soon while the horse he was riding was passing under a low archway.

When this man attempted to walk he reeled and staggered as if he were drunk. The upper extremities and the organs of speech were not affected; he had the entire control of his legs when lying down, and there was no diminution of sensibility anywhere. At last he became paraplegic, and shortly afterward died in a convulsion. The post-mortem examination showed the existence of an abscess which

[1] The Physiology and Pathology of the Cerebellum. Quarterly Journal of Psychological Medicine, April, 1869.

[2] Op. cit., p. 209.

had obliterated nearly the whole of the left lobe of the cerebellum. The other parts of the brain were, so far as could be perceived, perfectly healthy.

Extensive hæmorrhage may take place in the white substance of the cerebrum, and little disturbance of either motion or sensibility result. It usually happens, however, that the extravasation makes its way to the motor tract, and then the symptoms due to lesion of this part of the brain make their appearance. Besides the occurrence of local secondary lesions, the immediate result of the presence of a foreign body in the cerebral tissue, there are others, which are due to the interruption of the normal brain-functions, which hæmorrhage so generally induces. Thus, atrophy of the cerebral structure may result, as has been pointed out by Cotard[1] and others, or the degeneration may extend to the spinal cord, as so well shown by Bouchard.[2] In this latter event the process does not begin till about the end of the fourth or fifth month.

Treatment.—The means of treatment in cerebral hæmorrhage are, first, those which are applicable to the prodromatic stage, with a view of preventing any lesion; second, those proper during the seizure; and, third, those which are to be directed against the consequences of an attack.

It often happens that an attack may be prevented, even where the threatenings are very decided. The condition of the brain is such that the indications are to lessen the tension of the blood as much as possible. As I have already remarked, under the head of cerebral congestion, the bromides of potassium and sodium are peculiarly efficacious in accomplishing this end. Lately, in consequence of the investigations of Dr. S. Weir Mitchell, of Philadelphia, I have made much use of the bromide of lithium in cerebral congestion with or without a tendency to hæmorrhage, and

[1] Étude sur l'Atrophie Partielle du Cerveau, Paris, 1868.

[2] Des Dégenérations Secondaires de la Moelle Épinière, Archiv. Gén. de Médecine, 1866. Also, Hun's translation, American Journal of Insanity, 1869.

have had reason to prefer it to either the potassium or sodium salt. One feature of its action, which renders it especially useful in such cases as those now under notice, is the short interval which elapses between its administration and the effect. I am very sure I have given it successfully in several cases in which the other bromides would not have acted so happily. In one of these, a gentleman from the South, who had already had an attack, and who was in consequence hemiplegic, was relieved of his vertigo, headache, numbness, and thickness of speech, by one dose of thirty grains, in less than half an hour. The oxide of zinc may also be given with advantage.

The bowels, if costive, should be opened by a brisk purgative; the stomach, if overloaded, should be emptied by an emetic, during the action of which warm water should be freely drunk so as to avoid, as far as possible, all straining; muscular exertion should be avoided, the head should be kept cool and well elevated, and the mind in a state of the utmost tranquillity.

During an attack, and throughout the whole period of reparation of damages, the less that is done in the vast majority of cases the better. The question of the propriety of bloodletting will generally even yet arise, but should in nearly every case be decided in the negative. I say nearly, for I know of but one possible form of attack in which it can by any possibility not only not be useful, but fail to do harm; and that is in a strong, plethoric person, with a full, bounding pulse, in whom, from the gradual development of the symptoms, we have reason to suspect that the hæmorrhage is still going on. In such a case, six or eight ounces of blood may be taken from the arm. But in a case of cerebral hæmorrhage attended by coma and the ordinary symptoms of the apoplectic condition, there is nothing to be done in the way of medication, which can afford the slightest prospect of relief. It is true, a patient thus situated may recover if his attack is not of the severest kind, but it is not

through any medicines we give him. Correct views relative to this point are far from being prevalent, and can only be established by regard being paid to the morbid anatomy and pathology of the subject.

A clot in the brain is, to all intents and purposes, a foreign body, and both it and the walls of the cavity must undergo certain fixed and definite changes. In order that these changes may go on with the utmost possible regularity and certainty, all the powers of the system are requisite. The processes are not morbid; on the contrary, they are in the highest degree conservative. To take blood from a body which is striving by all its agencies to repair an injury, is to deprive it of a portion of its strength without in the slightest degree accelerating the actions at the seat of the lesion. As Trousseau [1] remarks, no physician ever thinks of bleeding for an extravasation of blood under the skin, for he knows how perfectly absurd such a practice would be; and yet, except as regards location, there is no difference between it and the cerebral clot. A prize-fighter, for instance, receives a blow in the face which ruptures a blood-vessel and gives him a "black eye." He has an extravasation of blood into the cellular tissue. What would be thought of the physician who would recommend bloodletting from the arm with a view of causing the absorption of the clot? The prize-fighter has found out by experience that he can open the skin with a knife and let the blood out. The practice is excellent, and would be admirable for the brain also, were this organ of no more vital importance than the skin of the face. I have never bled a patient for cerebral hæmorrhage since 1849, and I am very sure that I have had no reason to regret the abandonment of the practice.

It is a common practice for purgatives to be given, and even so conservative a practitioner as Dr. J. Hughlings Jackson [2] puts "two drops of croton oil on the tongue," why, he

[1] Lectures on Clinical Medicine, Bazire's Translation, Part I., p. 10.

[2] Reynolds's System of Medicine, vol. ii., Article Apoplexy and Cerebral Hæmorrhage, p. 541.

does not state, and certainly the practice is in direct antagonism not only with his assertion that "the chief thing is to keep the patient quiet," but with the general tenor of his theory of treatment. I have seen great annoyance and an aggravation of the symptoms from the indiscriminate administration of croton oil. It is only, in my opinion, admissible when there is obstinate constipation, and when after three or four days the bowels have not been moved.

And then as regards iodide of potassium. There seems to be an idea prevalent that this substance exerts a powerful influence in causing the more rapid absorption of the extravasated blood, and hence it is frequently administered in large and frequently-repeated doses. I have often seen patients, at as early a period as possible, while still in a state of profound coma, dosed with the iodide of potassium to the extent of five grains every hour, with the object of causing the immediate absorption of the extravasated blood. That such a result is impossible no one acquainted with the morbid anatomy and the pathology of the subject will deny.

In fact, there is nothing to be done beyond keeping the patient perfectly quiet, with the head well elevated, and in a room, when possible, with a temperature of about 60° and thoroughly ventilated. Indications should be met as they arise. The bowels, if not moved naturally every day, may be emptied by an enema of warm water; the urine, if not passed by the patient, should be drawn off with the catheter; the strength, if feeble, as indicated by the pulse, should be kept up by the cautious use of stimulants; and, if the patient is restless and does not sleep well, some one of the bromides should be administered.

The food should be of the most nutritious character, so as to be small in quantity, and should be taken frequently, day and night. Beef-tea, or the extract of beef, made according to Liebig's formula, supplies every indication.

If symptoms of inflammation make their appearance, cold applications may be made to the scalp, or a blister

may be applied to the nape of the neck. Blisters or mustard plasters to the wrists or ankles are absurd.

Nothing should be done for the relief of the paralysis till all signs of irritation of the brain have disappeared, and the patient begins to feel the restraint of confinement, and to make efforts to move his paralyzed limbs. These evidences of improvement generally begin soon after the eighth day. In about two weeks, therefore, it will be proper, in the majority of cases, to take active measures to restore the power of motion, and to prevent those contractions which tend to make a restoration much more difficult. The agents to be employed are passive motion, strychnia, phosphorus, and electricity. The first is accomplished by flexing and extending the joints of the affected limbs, by friction, and by kneading the muscles with the fingers. These movements should be performed every day for five or ten minutes at a time. The patient should likewise be encouraged to move the limbs by his own volition as often as possible short of causing fatigue. Strychnia should be given in doses of the one-twenty-fourth of a grain three times a day, or, preferably, by subcutaneous injection, in somewhat smaller doses, once a day. In old cases of hemiplegia, the effects of strychnia thus administered are often well marked, and are exhibited when administration by the stomach has failed to produce a beneficial result. This is seen in the following brief abstract of thirteen cases which will serve as types of numerous others which have occurred in my private practice.

Case I.—H. A., aged fifty; male; right hemiplegia. Came under treatment January, 1865; strychnia ineffectual by the stomach; thirteen injections, of from one-thirty-second to one-twenty-fourth grain; much improved.

Case II.—J. S.; forty-two; male; left hemiplegia. February, 1865; thirteen injections; much improved.

Case III.—S. T.; sixty; female; right hemiplegia. February, 1865; strychnia ineffectual by the stomach; nine injections; much improved.

Case IV.—I. S.; sixty; female; right hemiplegia. April, 1865; five injections; much improved.

Case V.—M. T.; fifty-two; male; right hemiplegia. April, 1865; strychnia ineffectual by the stomach; eleven injections; cured.

Case VI.—O. S.; sixty-three; female; left hemiplegia. April 30, 1865; secondary contractions; twenty-two injections; no improvement.

Case VII.—B. R.; forty-seven; male; left hemiplegia. June 11, 1865; strychnia ineffectual by the stomach; seven injections; much improved.

Case VIII.—R. F.; fifty; male; left hemiplegia. June, 17, 1865; strychnia ineffectual by the stomach; eight injections; cured.

Case IX.—T. W.; forty-eight; male; left hemiplegia. September 5, 1865; eight injections; much improved.

Case X.—T. S.; forty-nine; male; left hemiplegia. September 7, 1865; secondary contractions; five injections; no improvement.

Case XI.—J. J.; fifty-seven; male; left hemiplegia. September 11, 1865; secondary contractions; no improvement.

Case XII.—J. W.; fifty-two; male; right hemiplegia, affecting arm only, at the time treatment was begun. September 27, 1865; strychnia ineffectual internally; six injections; cured.

Case XIII.—W. M.; forty-five; male; left hemiplegia. October 19, 1865; strychnia ineffectual internally; seven injections; cured.

Case XIV.—S. M.; forty-one; male; right hemiplegia. June 17, 1867; arm alone affected; strychnia ineffectual by the stomach; twenty injections; cured.

Case XV.—M. C.; forty-four; male; right hemiplegia, affecting tongue and face only. July 1, 1867; ten injections; so much improved as to be able to talk with fluency.

Case XVI.—C. C.; fifty; male; right hemiplegia. May

4, 1869; strychnia ineffectual by the stomach; thirty-five injections; much improved.

Dr. Charles Hunter[1] has called attention to the advantages to be derived from the hypodermic use of strychnia in hemiplegia; and my late clinical assistant, Dr. R. A. Vance,[2] has adduced several cases to the same effect. Instances in support of the views above set forth occur daily in my private practice, and at the New York State Hospital for Diseases of the Nervous System. I have every reason, therefore, to be convinced of the good results to be derived from the practice.

Phosphorus administered in the form of phosphide of zinc, separately or in combination with the extract of nux-vomica, according to the formula given on page 58, is also a useful remedy.

But no agent is so valuable in hemiplegia as electricity, and amendment almost invariably follows its use, even in old cases, in which there are tonic contractions. If the case is seen soon after the seizure, the induced current will generally be sufficient to produce contractions of the paralyzed muscles. The poles, terminated by wet sponges, should be applied to the skin covering the muscles, or in some cases to the nerves. The current should be strong enough to cause slight pain, or, if sensibility is lessened, to produce contraction. In old cases attended with atrophy of the muscles, and diminished or abolished electro-contractility, the primary current may be necessary. It should be applied in such a manner as to be interrupted, for contractions are only caused when the circuit is closed and opened. As the muscles improve in size and irritability, the induced current should be used. Care should be taken not to fatigue the patient, or to cause excessive pain by employing a current of too great intensity.

[1] British and Foreign Medico-Chirurgical Review, April, 1868.

[2] Journal of Psychological Medicine, April, 1870. The first thirteen cases cited in this work were published in Dr. Vance's paper.

As regards the restoration of sensibility, it will generally be found to be less difficult than the removal of the motor paralysis. The anæsthesia very often disappears or becomes much less spontaneously, and it does so from the centre to the periphery; that is, if there be anæsthesia of the leg, the sensibility returns in the upper part first, and subsequently in the lower part. The treatment consists mainly in the use of the electric wire-brush, which should be passed gently over the skin previously made dry. The other pole consists of a wet sponge. Either the induced or primary current may be used. If the latter, however, be employed, the wire-brush should constitute the positive pole.

I have frequently succeeded in curing almost complete anæsthesia from cerebral hæmorrhage by this treatment alone. In recent cases it will almost invariably prove effectual. Hyperæsthesia, if present, may be similarly managed.[1]

[1] The subject of the employment of electricity in medicine is too extensive to receive more than slight notice in a work like the present. For full details in regard to it, the reader is referred to the author's translation of Meyer's "Electricity in its Relations to Practical Medicine." New York: D. Appleton & Co., 1870.

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CHAPTER IV.

MENINGEAL HÆMORRHAGE.

By the term meningeal hæmorrhage is meant an extravasation of blood into the subarachnoid space, or between the dura mater and arachnoid.

Symptoms.—The most prominent symptom of meningeal hæmorrhage is coma, which may appear suddenly, or be preceded by premonitory symptoms, such as headache, vertigo, and general convulsions. The stupor is usually profound, and does not differ from that observed in the severe forms of cerebral hæmorrhage. The power of motion is generally lost throughout the body, and consequently there is no hemiplegia. The reason for this is, that the hæmorrhage is so extensive as to press upon both hemispheres. Reflex and automatic movements remain, except when the medulla oblongata is involved, when some of them are abolished. In this latter situation death soon takes place from cessation of respiratory actions.

In ordinary cases the patient may pass out of the comatose condition from the fact of the brain becoming accustomed to the pressure, and he then may be able to speak, and to move his limbs, but his mental and physical faculties are greatly enfeebled, and a renewal of the hæmorrhage again plunges him into a state of coma, from which he may again emerge. This sequence may be repeated several times, until death at last takes place. Before this termination there are vomiting, incontinence of urine and fæces, insensibility, and occasionally general convulsions.

It has sometimes happened that meningeal hæmorrhage, resulting from an injury of the cranium, has not caused any very prominent symptoms for a considerable period afterward. A teamster was struck on the head by a club in the hands of another man, was stunned for a few minutes, then recovered, and went about his business without complaining of his head. In about twelve hours afterward coma supervened, and he died without being aroused. A case is reported by Dr. Gibson,[1] in which a still longer period intervened. A man, of about sixty years of age, was found one morning, about eight o'clock, seated as if asleep at a desk, his arms crossed before him and his head resting on them. It was discovered that he was profoundly insensible. He was sent to the hospital, where he lay comatose, breathing stertorously, and paralyzed on the whole of one side. At the end of two days he died. On post-mortem examination there was found fracture of the left side of the cranium, with rupture of the dura mater and middle meningeal artery, from which latter, extensive hæmorrhage had taken place. It was ascertained that, five days before, he had fallen down a stone staircase, was stunned for a few minutes, but had soon recovered his senses. Doubtless during the whole of the intervening period the bleeding from the ruptured vessel had been going on.

Causes.—Meningeal hæmorrhage is often produced by injuries of the skull, and results from sudden rupture of a healthy artery or vein. It may follow blows on the head, falls, or injuries with instruments which perforate the cranium, and may or may not be associated with fractures of the bones.

The larger vessels, or the capillaries, may give way from being diseased, and consequently unable to resist the tension of the blood. Such a condition may be the result of the long-continued excessive use of alcoholic liquors, or may be due to hepatic disease.

[1] Edinburgh Medical Journal, September, 1870, p. 199.

In new-born children, there is sometimes meningeal hæmorrhage from the pressure made by the instruments used to effect delivery.

The **Prognosis** in meningeal hæmorrhage is always unfavorable, death occurring either during the state of coma which first appears, or in some one of the subsequent accessions.

Diagnosis.—Meningeal hæmorrhage is readily distinguishable from cerebral hæmorrhage by the facts that the stupor comes on gradually, that there is no hemiplegia, and by the remissions which take place when the patient does not die at first.

The **Morbid Anatomy** and the **Pathology** call for no additional remarks beyond those already made; and the **Treatment** does not differ from that proper in cerebral hæmorrhage.

HÆMATOMA OF THE DURA MATER.

A peculiar form of meningeal hæmorrhage, called hæmatoma, is met with under the dura mater. The blood is not diffused, but is collected in sacs which are formed of false membranes, the result of chronic inflammation. These capsules are flattened ovals in shape, are three or four inches in diameter, and half an inch thick. They are usually situated at the vertex, and involve both hemispheres. When this is the case, the paralysis which results is bilateral.

Symptoms.—The initial symptoms of hæmatoma of the dura mater are the results of chronic inflammation, and are slow in their progress. In many respects they resemble those indicative of softening, and consist of weakness of intellect, vertigo, a dull, circumscribed, persistent pain, and more or less tendency to stupor. The power of motion is generally diminished on both sides of the body, though occasionally there is hemiparesis. Paralysis is scarcely ever complete. Gradually, through a period extending over several months, the stupor increases, and finally the patient

becomes apoplectic. During the whole course of the disease the pupils are strongly contracted. The patient dies comatose and frequently convulsed.

Causes.—Early and old age are both predisposing causes, the disease being met with mainly in children and very old persons. It is frequently seen in the insane, and may probably result from rheumatism, the excessive use of alcoholic liquors, and fevers. The cause is sometimes to be found in wounds or injuries of the skull.

Diagnosis.—It is doubtful if hæmatoma of the dura mater can be definitely recognized either in the stage of inflammation or that of hæmorrhage. Legendre[1] states that, in children, the most important diagnostic mark is the permanent contraction of the hands and feet, which is so generally present; but this symptom is certainly met with in other cerebral disorders, and may even result from reflex irritations. The diagnosis is rendered still more difficult by the fact that the disease under consideration is often associated with other cerebral disorders which mask or modify its symptoms. The absence of fever, the contraction of the pupils, the slowness and irregularity of the pulse, the facts that there are no vomitings and no general convulsions, that the nerves distributed to the several parts of the face are not paralyzed, that there are constant and very severe headache, and a gradually increasing tendency to stupor, are, according to Jaccoud,[2] sufficient to indicate the presence of hæmatoma of the dura mater. I am of the opinion that they only enable us to give a guess which has some basis in probability, for I have several times witnessed exactly such a condition as that described, and after death found other morbid conditions than hæmatoma.

The **Prognosis** is unfavorable, death resulting sooner or later, according to the extent of the disease and the natural powers of the patient.

[1] Recherches sur quelques Maladies de l'Enfance, Paris, 1846.

[2] Traité de Pathologie Interne, tome i., Paris, 1870.

Morbid Anatomy and Pathology.—The first stage of hæmatoma of the dura mater is characterized by the formation of the false membranes, to which allusion has already been made. These membranes are found on the internal surface of the dura mater, and are reticulated, presenting somewhat the appearance of spiders' webs. They generally have their seat near the sagittal suture, and extend to both hemispheres, being only separated from them by the arachnoid and pia mater. Virchow, who has studied their formation with greater care than any other observer, has found more than twenty layers of them, one on top of the other, and traversed by numerous blood-vessels.

Owing to this great vascularity, to the extreme tenuity of the vessels, and to the absence of any perivascular support, hæmorrhage is liable to occur, and the several lamellæ thus constitute a sac into which the blood may be poured. This, pressing upon the cerebrum below, and constantly being enlarged by subsequent hæmorrhages, gives rise to the symptoms observed during life. The vessels may be more liable to rupture from the existence of atheromatous degeneration of their coats.

Treatment.—This requires no amplification at my hands, as I do not believe in the efficacy of any means in curing the affection. All that can be done is to palliate the more violent symptoms, such as the headache and feebleness of mind and body, by anodynes and stimulants, and of these, morphia administered hypodermically, and alcohol in some one or other of its numerous forms, are to be preferred. Bloodletting and blistering are worse than useless.

CHAPTER V.

PARTIAL CEREBRAL ANÆMIA FROM OBLITERATION OF CEREBRAL ARTERIES.

One or more arteries of the brain may be obliterated and anæmia of those parts supplied by it produced through the action either of thrombosis or embolism.

THROMBOSIS.

By thrombosis is understood a condition in which a blood-vessel undergoes narrowing of its calibre by the deposition of fibrine from the blood on its internal surface. The clot thus formed is called a thrombus.

Symptoms.—The phenomena observed in consequence of the formation of a thrombus in a cerebral artery are gradual in their development, and are often interrupted by stages of apparent improvement. Headache, as in so many other affections of the brain, is a prominent symptom and is almost constantly present. It is rarely diffused over the whole head, but occupies a place having a close relation in situation with the seat of the disease. It is rarely of a very aggravated character, and is remarkable rather for its persistency than its severity. In several cases which have come under my notice, the pupil of the eye of the affected side was dilated from the first, and there were ptosis and strabismus, showing that the third nerve was involved.

At a very early period in the progress of the disease it is not uncommon to meet with marked difficulties in the faculty of speech, and these not only relate to the articulation,

but to the memory of words. As regards the first-mentioned form, there may be restraint in the movements of the tongue, the lips, or both, or there may be a loss of coördinating power in the muscles concerned in speech without any actual paralysis. Special inconvenience is, therefore, experienced when attempts are made to pronounce words in which the labial and lingual letters are prominent. The gutturals in such cases are enunciated without difficulty. In the other form in which the memory of words is impaired, the patient is constantly at a loss for language with which to express his ideas; and, though the proper words may be supplied to him, he almost immediately forgets them again. The full consideration of this interesting subject will be found under the head of aphasia.

Vertigo, though generally present, is not usually severe, at least in the early stages.

The incipient symptoms of paralysis soon make their appearance in the majority of cases, and, though there is a gradual advance in the loss of power, there are periods of almost entire remission. Thus the leg, or the arm, or the face, may be the original seat of the paralysis, and eventually the whole of one side be involved. In a case of thrombosis in a gentleman now under my charge, the paralysis was at first limited to the muscles supplied by the ulnar nerve and those concerned in deglutition. For one period of five days after I first saw him, there was an entire remission of his symptoms, and he could move his hand and swallow as well as ever, but gradually the power was again lost, and other muscles became involved. At the present time (December 1, 1870), he is almost entirely hemiplegic.

Sensibility is also generally abolished or impaired on the paralyzed side, and thus the various forms of numbness, such as tingling, formication, etc., are present.

The mental symptoms are usually apparent from the first, but may be altogether absent or else so slightly shown as not to attract attention. The memory is impaired, not only as

regards words to which reference has already been made, but also events and circumstances, especially those of recent date. The names of persons and things are likewise readily forgotten. In the case of a gentleman whom I saw in consultation, and in whom I diagnosticated thrombosis, there was left hemiplegia involving both arm and leg, but not the foot, which had begun in the fingers and gradually extended. There was no special difficulty of speech except as regarded the recollection of words, but the memory was wonderfully impaired in every other respect. I entered his room upon one occasion just as the servant was carrying out a tray with the remains of his breakfast. Not three minutes had elapsed since he had eaten, and yet he assured me he had tasted nothing since the day before. The loss of memory was the first symptom observed in this case. Soon afterward he began to improve, and he is now, after fifteen months, free from paralysis, and with his memory almost as good as ever. The loss of memory in such cases seems to be due in the main to the fact that the power of concentrating the attention upon any subject is very much diminished. There is likewise an indisposition to exert the powers of the mind or body, and thus the patient tends to pass into a condition of apathy. Somnolence is a frequent symptom.

During the first stage of thrombosis, before the artery is entirely closed, amendment, and even complete recovery, may take place. The remissions in the symptoms already referred to are due to the establishment of the collateral circulation, and this may become so complete as to eventuate in cure. It must be confessed, however, that the condition of anæmia to which the foregoing symptoms are due, in the great majority of cases ends in softening—a subject which will presently be considered as one of the consequences of thrombosis and other morbid states.

Causes.—Thrombosis may result from atheroma of the artery, by reason of which its elasticity is diminished and the smoothness of its lining membrane destroyed. Both

these conditions retard the course of the blood, and favor the deposition of fibrine on the internal periphery. The walls of the vessels may be healthy, and a thrombus may then be formed through a weak action of the heart—the result of fatty degeneration or other cause impairing its strength.

The predisposing causes are, age—the disease being rare in persons under fifty years—the excessive use of alcoholic liquors or of fatty or starchy articles of food, with insufficient exercise, and perhaps inordinate mental exertion, which, by impairing the tone of the arteries, in consequence of their constant overdistention, diminishes their elasticity, and may consequently lead to the formation of thrombi.

Diagnosis.—Thrombosis is distinguished from cerebral congestion by the facts that the mental and other symptoms are more profound in character, and that the patient has generally passed the prime of life. The existence of paralysis among the early symptoms will likewise tend to the formation of a correct opinion. From cerebral hæmorrhage it is diagnosticated by the circumstance of its gradual development; from encephalitis by the absence of fever and the more chronic nature of the disease; and from embolism by its slow progress and the impossibility of defining the exact period of its beginning.

Prognosis.—The prognosis in cerebral thrombosis is unfavorable, for the reason that, although the morbid process may advance slowly, and may even be spontaneously arrested in its course before the artery is closed, the tendency to complete obliteration is always great, and the chance of sufficient circulation being carried on by the collateral vessels is very remote. The disposition to softening, therefore, always exists, and generally cannot be overcome. The inadequacy of any medical treatment to control the action going on within the artery, or to aid to any great extent in the development of the collateral circulation, is also an element in forming an opinion as to the ultimate result.

Morbid Anatomy and Pathology.—Although Virchow[1] was the first to write distinctly in regard to the nature of thrombosis, the condition was recognized long before his researches were made, and cases of clots plugging up the vessels are to be found detailed by many of the older medical authors, among whom Abercrombie, Carswell, and Cruveilhier, may be mentioned. Since Virchow began his observations in this direction, many instances have been recorded and a large number of memoirs have been issued upon the subject. An interesting case was related by Dr. Packard,[2] of Philadelphia, at a meeting of the Pathological Society of that city held in December, 1859. The patient, who had been under the care of Dr. Heller, was a bachelor, fifty-one years of age. At six o'clock in the morning, at the beginning of February, he was seized with paralysis of the left arm and leg. He was a man of very regular habits, and of fanatical love for every thing instructive, and an accomplished scholar in botany, geography, and languages. The paralysis was soon relieved, and he was able, four weeks afterward, to go out again and to use his arm tolerably well. About the middle of March, in consequence of a fatiguing walk the previous evening, and an attack of diarrhœa during the night, complete paralysis returned. From this he never recovered, but yet did not die till the December following. Previous to this termination he had confusion of ideas and delirium. Upon post-mortem examination, among other morbid changes, a cavity in the right corpus striatum was found, and this was surrounded by a spot of softening of the cerebral substance as large as an egg. The basilar artery was completely blocked up with clots, as was also the right carotid. These vessels were atheromatous, and the basilar artery was aneurismally dilated. The clots had all the appearance of being old.

Dr. Dickinson[3] has brought forward five cases of occlu-

[1] Froriep's Neue Notizen, 1846. Heft xxxvii.

[2] North American Medico-Chirurgical Review, vol. iv., 1860, p. 306.

[3] On the Formation of Coagulæ in the Cerebral Arteries. St. George's Hospital Reports, vol. i., 1866, p. 257.

sion of arteries, several of which I am disposed to think were of embolism, instead of thrombosis, as he considers them to be. Dr. Dickinson nowhere alludes to Virchow's investigations, but gives the whole credit of the discovery of the relation between emboli and the formation of concretions in the heart to Dr. Kirkes. The conclusions which he draws from his cases are by no means original, although he evidently so regards them.

The questions to be considered in connection with the morbid anatomy of thrombosis relate to the condition of the artery, the nature of the clot, and the changes which take place in those parts of the brain which are deprived of their due supply of blood.

The affections of the artery, being similar to those which render it liable to rupture, need not be dwelt upon at any length here, as they have already been noticed under the head of the morbid anatomy of cerebral hæmorrhage. Suffice it, therefore, to say that endarteritis and atheromatous degeneration are the diseased states generally met with.

The clot which closes the vessel is, in the beginning, coagulated blood, and hence consists of fibrine and white and red blood-corpuscles. The elements, with the exception of the fibrine, are gradually disintegrated and washed away by the current of blood which continues to flow through the vessel before it is entirely closed, and therefore the layers nearest the arterial wall consist almost entirely of fibrine and the one nearest the centre of the vessel, which is the latest formed, of fibrine and corpuscles. An examination of such a clot with the microscope shows that the above-mentioned morphological elements are found in its centre, more or less changed, however, according to the age of the formation. A thrombus may undergo purulent softening and disintegration to such an extent as to result in its breaking up into fragments, which may lodge in the vessel or its branches farther on, and thus constitute emboli.

The region of the brain to which the artery undergoing

occlusion is distributed is, of course, deprived to some extent of its blood, and hence presents at first an appearance of anæmia. And this is not prevented by the increase of the collateral circulation, which is never sufficiently vigorous to compensate entirely for the loss by the primary vessel.

Microscopic examination shows the capillaries to be smaller and less numerous than in the normal condition, though there is not any palpable softening.

But after the artery is entirely closed a change ensues. The anæmic portion of the brain becomes red or pink, and this color is deepest on the borders, owing to the collateral circulation which is now fully established. This stage has been called red softening, but I am disposed to think the designation erroneous, and that it is liable to convey false ideas of the pathology. For it is perfectly possible at this time for the anæmic portion of the brain to be restored through the activity of the collateral circulation, with the effect of causing a cessation of the symptoms. If, however, this should be insufficient to provide for the due nutrition of the affected region, softening takes place, and a cure becomes almost impossible.

Obliteration of a cerebral artery does not always produce notable symptoms. For these to follow, the morbid process must be set up in a vessel with but few and small collateral branches. Thus, if the internal carotid be obstructed, the circulation is carried on through the circle of Willis by the supply of blood derived from the vertebrals. The basilar artery might also be occluded at any limited region between a pair of transverse arteries, and the circulation still kept up by the carotids on the one side, and the vertebrals on the other. But any closure so as to involve one or more of the transverse arteries must lead to anæmia, and subsequent softening of the pons Varolii. Thus, in a case reported by Bennett,[1] in which there had been vertigo and other head-symp-

[1] Clinical Lectures on the Principles and Practice of Medicine, third edition, Edinburgh, 1850, p. 370

toms for several years, and in which paralysis of the left arm, without loss of consciousness, had suddenly supervened, the basilar artery was found entirely obliterated throughout its entire extent, all the transverse arteries were of course closed, and the supply of blood to the pons cut off on both sides of the mesial line.

A very interesting memoir by Hayem [1] alleges occlusion of the basilar artery by thrombus to be a cause of sudden death. In all of his cases, four in number, the artery was closed throughout a great part of its extent, as the result of extensive arteritis and the formation of dense clots. In the fourth case there was also thrombosis of the left middle cerebral artery, with difficulty of speech.

The vessels the closure of which produces the greatest disturbance of function are the anterior, middle, and posterior cerebral which supply the hemispheres, the corpus striatum, optic thalamus, and other important ganglia. Besides the effect due directly to the anæmia, more or less disturbance results from the congestion posterior to the clot, and the consequent effusion of serum.

Treatment.—A knowledge of the morbid anatomy and pathology of cerebral thrombosis must satisfy us of the insufficiency of any medical treatment to cause the absorption of the clot obliterating the channel of the artery. Yet I have several times heard it gravely proposed to administer the iodide of potassium, with the view of accomplishing this object. As regards facilitating the establishment of the collateral circulation, nature will generally take care of this, and may even so far overdo it as to cause hæmorrhage from the rupture of vessels not accustomed to the increased tension of the blood. It may therefore be necessary, in this latter condition of excessive action, to give the bromide of potassium in large doses. Should the circulation be feeble, the skin cold, and the patient disposed to somnolence, we

[1] Sur la Thrombose par Artérite du Tronc Basilaire, comme cause du mort rapide, Archives de Physiologie Normale et Pathologique, 1868, p. 270.

have reason to suppose that the collateral circulation is not being formed with sufficient rapidity, and therefore the patient should be kept with the head low, brandy or other spirituous liquors administered, and the body wrapped up in warm blankets.

For some time after the successful establishment of the collateral circulation there is more or less feebleness of mind and body. For this condition strychnia and phosphorus are especially applicable, and may be administered according to the formulas recommended under the heads of cerebral congestion and cerebral hæmorrhage. Electricity is almost always useful.

EMBOLISM.

Embolism is the term applied by Virchow to the closure of an artery by an embolus, which is a clot formed in some other part of the body and transported by the current of the blood to the vessel which it occludes. It therefore differs from thrombosis in the facts that it is not associated with previous disease of the artery, and that the closure of the vessel is sudden.

Symptoms.—There are no premonitory symptoms. As in cerebral hæmorrhage, the patient may be sitting perfectly quiet when he suddenly loses consciousness and falls to the ground, comatose. As the stupor passes off, he finds that he is paralyzed upon the side of the body opposite to the seat of the lesion.

Or there may be no coma, but merely slight confusion of ideas for a moment or two with sudden accession of paralysis on a limited portion of one side, involving only the arm or leg. Or, again, the face or the tongue may be the only parts paralyzed. Or there may be no paralysis anywhere, and no mental symptoms except as regards the faculty of language, which is entirely or partially lost.

Sometimes there are ocular troubles, such as ptosis, strabismus, or blindness.

Experience shows that the embolus, for reasons which will be given hereafter, generally lodges in the left middle cerebral artery, and that with the right hemiplegia—if there is paralysis at all—there is often aberration of the faculty of speech.

The symptoms of mental derangement, with the exception of the coma of severe attacks, are not ordinarily prominent. I have, however, witnessed several cases in which they formed a very striking feature of the case. In one of these, in which the clinical history of the patient disclosed the preëxistence of several attacks of acute articular rheumatism, with subsequent endocarditis and mitral and aortic valvular lesions, there were hallucinations and delusions in addition to the complete paralysis of the left side. All these phenomena entirely disappeared within thirty-six hours. This case is one of the few in my experience in which the embolus had occluded an artery on the right side of the brain.

In another, likewise with valvular disease of the left side of the heart, there was delirium from the first, and this disappeared as the collateral circulation was established.

Erlenmeyer has written very excellently of cerebral embolism, but is, I think, incorrect in some points of his symptomatology. He states the ordinary phenomena of an attack to be as follows:

There are no prodromata; sudden loss of consciousness, with paralysis of several parts of the body. The facial, the hypoglossal, and the nerves of the extremities, are always more or less affected. Sensibility is abolished in the conjunctiva, but is retained in the cornea. The pupils remain sensitive, and are neither contracted nor dilated, neither are there symptoms of concussion or compression. There are no vomitings and no contractions. The pulse is weak and small, and the temperature rather below the normal standard. Occasionally there are epileptiform convulsions. Psychical troubles do not ordinarily appear till the collateral

circulation becomes active, and local hyperæmia is thus induced.

The principal exception I have to make to the foregoing sequence of symptoms is the too absolute assertion of the paralysis of the facial, hypoglossal, and other nerves. I have seen several cases in which there was no paralysis to be detected in any part of the body by the most careful examination, and several others are on record. In one very interesting instance, occurring in a lady who had had repeated attacks of acute rheumatism, and who had at the time marked aortic insufficiency, headache and vertigo suddenly occurred while she was conversing with a friend, and her speech was cut short with as much suddenness as though she had been shot. There was no paralysis of the tongue, but all idea of language was abolished. Within forty-eight hours she recovered entirely the faculty of speech. In another, that of a gentleman with a similar clinical history, headache, vertigo, confusion of ideas, and amnesic aphasia, suddenly supervened. That both these were cases of embolism can scarcely, I think, be doubted.

And then, as regards the state of the pupils, my experience does not coincide with that of Erlenmeyer, for I have frequently found either dilatation or contraction of both pupils, or dilatation of one and contraction of the other.

In examining a case of recent embolism, the ophthalmoscope should always be used to view the fundus of the eye, and even in old cases valuable signs will often be obtained. The middle cerebral artery, the ordinary seat of embolus, arises from the internal carotid after the anterior cerebral and ophthalmic have been given off. Occlusion of its channel must, of course, throw an increased amount of blood into these last-named arteries, and, as the arteria centralis retinæ is derived from the ophthalmic, it and its branches become enlarged. The ophthalmoscope will enable us to discover the congestion thus produced, and will often be the means of helping us to determine, in the absence of paralysis,

which side of the brain is the seat of the lesion. In older cases we will frequently find retinal congestion.

The following case I quote not only as being the first of which I have any knowledge in which the ophthalmoscope was used in a case of cerebral embolism, but as being interesting from the fact that the embolus was on the right side. It is reported as

Cerebral Embolism following Valvular Disease of the Heart.—John Turnbull, aged seventeen, was admitted into the Hull General Infirmary, on April 25, 1867. He was tall, much wasted, and had a suffering expression, and converging strabismus of the left eye, the mouth being drawn very slightly toward the left side. Pulse 70, very thrilling in character, and a large coarse systolic murmur near the left nipple. He was perfectly sensible, complained of severe frontal headache, with confusion of vision, and stated that he had been in much the same condition for seven weeks, his illness beginning spontaneously with headache and vomiting, unaccompanied by loss of consciousness or convulsions. He had had an attack of acute rheumatism in the previous summer. He was ordered gr. iij of blue-pill and gr. ij of extract of henbane in a pill, and a draught of acetate of ammonia, three times a day, and spirit-lotion to the head. "No marked alteration in his condition, except progressive debility, took place till May 2d, when he complained of increased headache and dimness of vision, and being unable to expectorate, from excessive weakness, death from bronchial obstruction threatened. With the aid of some champagne, he rallied in about twenty-four hours, and at the end of a week was much improved, having a clean tongue and good appetite, but the headache, strabismus, and deviation of the tongue to the left, remained. On May 16th it was noticed that these symptoms had passed off, with the exception of the last mentioned. He was ordered a mineral-acid mixture.

"A week later, as he still complained of some dimness

of sight, he was examined with the ophthalmoscope. The retinal vessels were found much enlarged, and the veins very tortuous; the optic nerve-entrance of an intense red color, not being distinguishable from the surrounding parts except by the entrance of the vessels, the redness being chiefly due to a number of very fine vessels radiating from the centre. There was no morbid effusion in any part. He could spell easily from No. 15 of Jaeger's test-types (being unable to read and write). He was again examined at the end of another week, when the optic nerve-entrance was observed to be paler in color, so that its circumference could be distinguished, but still much injected, and the vessels nearly as large and tortuous as before; sight was apparently perfect. He was discharged convalescent.

"The peculiar form of paralysis in this case denoted some morbid condition within the cranium, which appeared to have its most easy and natural explanation in cerebral embolism, an opinion further supported by the perfect recovery of the patient. The case received much additional interest from the information afforded by the ophthalmoscope, for one may fairly believe that the intense congestion of the retinæ denoted a similar condition of the brain, perhaps a state of reaction after the circulation had been re-established through collateral channels."[1]

Causes.—The most common first step in the causation of cerebral embolism is acute articular rheumatism, which, by inducing acute endocarditis, leads to the formation of emboli on the valves of the heart and other parts of the endangium. Aneurisms of the aorta or other large artery, resulting in the coagulation of the blood in the aneurismal sacs, may likewise induce it, by a portion of the clot being washed off by the current. Esmarch[2] details a case in which, while an examination was being made of an aneu-

[1] British Medical Journal, 1867, also QUARTERLY JOURNAL OF PSYCHOLOGICAL MEDICINE, January, 1868, p. 178.

[2] Archiv für Pathol. Anatomie und Physiologie, B. xi., Heft. 5, 1857.

rism of the carotid, the patient suddenly fell back in an apoplectic stupor. The whole right side was at once paralyzed, the facial muscles on the left were convulsed, and four days afterward death ensued. Post-mortem examination showed that the left internal carotid, the middle cerebral, and the ophthalmic, were completely closed by coagula, which were identical in structure and appearance with the clot in the aneurismal sac.

Emboli may also originate in the lungs, and, entering the left auricle through the pulmonary veins, finally lodge in a cerebral artery.

Age appears to exercise no influence over the formation of emboli, but men are much more commonly the subjects than women, for the reason, undoubtedly, that they are more liable to attacks of rheumatism.

Of thirty-seven cases under my care, either alone or in consultation, in which I had reason to diagnosticate cerebral embolism, there was organic disease of the heart in all but one. Three of the cases were over sixty years of age; four between fifty and sixty; seven between forty and fifty; thirteen between thirty and forty; and ten under thirty. Twenty-five were males and twelve were females.

Diagnosis.—From cerebral hæmorrhage, embolism may be distinguished by the following signs. It occurs without relation to age, while hæmorrhage is much more frequent in persons over forty; there are no prodromata: the resultant paralysis is generally on the right side, while in hæmorrhage there is no such predisposition; and it is in the great majority of cases associated with organic disease of the left side of the heart. Care, however, must be taken not to overestimate the value of this diagnostic mark, valuable as it is. In one case under my charge, in which the symptoms pointed strongly to the existence of a cerebral embolus, and in which, after death, the left middle cerebral artery was found occluded, the heart was perfectly healthy; and in one other, in which cerebral embolus was diagnosticated, and in

which there was mitral regurgitation, extravasation into the corpus striatum was discovered to be the cause of death. A case has recently been reported by Dr. J. Hughlings Jackson,[1] in which there was cerebral hæmorrhage with hemiplegia, together with extensive valvular disease of the heart.

A patient now in the New York State Hospital for Diseases of the Nervous System has left hemiplegia, involving face, arm, and leg. It has already lasted seven months, although greatly improved. The hand and arm are much contracted. The attack was apparently induced by strong muscular exertion being made while in a stooping and constrained position. Most physicians would be disposed to agree with my diagnosis, that the case is one of cerebral hæmorrhage, for the obvious cause of the paroxysm, the lesion being on the right side of the brain, the steady improvement and the muscular contractions, all point to extrasavation of blood instead of embolus. Yet he is under twenty years of age, and, before the seizure, had an attack of acute rheumatism, with heart-difficulty. He now has aortic and mitral regurgitation. Such cases as the above are very instructive, and they show us how necessary it is to weigh all the facts, and how great is the possibility of making a mistake after all. For, although I am inclined to the view of hæmorrhage, no definite opinion can be given without a post-mortem examination.

Still in a case of partial or complete hemiplegia, with or without apoplexy, in which the patient was below the age of forty, with the hemiplegia involving the right side, no muscular contractions and organic disease of the left side of the heart, with or without previous attacks of acute articular rheumatism, cerebral embolus may safely be said to be the cause of the symptoms. Moreover, the paralysis from embolism, if it does not disappear within seventy-two hours after the seizure, does not gradually fade away as it so frequently does to a great extent in hæmorrhage.

[1] British Medical Journal, October 29, 1870, p. 459.

The suddenness with which embolism takes place, to say nothing of the other points in the clinical history, will suffice for the discrimination from thrombosis.

Prognosis.—The prognosis in cerebral embolism is grave, for the reason that the tendency to softening of the anæmic cerebral tissue always exists. But, if the patient passes over the first four or five days without any aggravation of his symptoms, and especially if they be mitigated in violence, there is considerable hope of a favorable result. Still, a guarded opinion should always be given till all head-symptoms have disappeared.

Morbid Anatomy and Pathology.—The first rational explanation of embolism was made by Virchow,[1] in 1847, who, in his paper on acute inflammation of the arteries, distinctly explained the manner in which the vessels were occluded by clots transported in the blood from distant parts of the body, and who associated these coagula with valvular disease of the heart. In two of the cases cited by him in which arteries were found closed by such clots, the valves of the heart were discovered to have others still attached to them, and exhibited traces of the separation of those which were found in the vessels.

Subsequently (in 1852), Dr. Senhouse Kirkes[2] called special attention to the plugging up of the middle cerebral artery as a cause of softening of the brain. Three cases, in which death followed, are adduced, in each of which the condition of non-inflammatory softening was found to exist in the brain. Dr. Kirkes's observations appear to have been made without any knowledge of Virchow's prior researches. He states that the paralysis met with in young persons may

[1] Ueber die akute Entzundung der Arterien. Archiv für Pathol. Anatomie, B. i., 1847, p. 272. In a paper on Occlusion of the Pulmonary Artery, published in Froriep's Neue Notizen in 1846, he enunciated a similar theory.

[2] On some of the Principal Effects resulting from the Detachment of Fibrinous Deposits from the Interior of the Heart, and their Mixture with the Circulating Fluid. Medico-Chirurgical Transactions, vol. xxxv., 1852.

be due to the interruption of a due supply of nutriment to the brain by the occlusion of an artery by a plug derived from the left side of the heart.

Schutzenberger,[1] among others, has written with great fulness on this subject. Among other conclusions not specially applicable to the particular point now under consideration, he states that fibrinous concretions may form in the heart, or large vessels may subsequently be detached and carried by the blood to the cerebral arteries, where they produce symptoms not essentially different from those noticed in cerebral hæmorrhage or acute softening.

The only essential points of difference under this head between thrombosis and embolism, are the suddenness of the attack, the part of the brain most liable to be affected, the origin of the clot, and the state of the blood-vessel which is obliterated.

Relative to the first, the abrupt closure of a vessel as in embolism will, of course, produce more violent symptoms than if the occlusion has taken place gradually, and thus time have been afforded for the establishment of the collateral circulation. In the first case, not only is the blood at once shut off from a portion of the brain, but the vessels behind the clot receive a greater quantity than they normally do, and hence the regions they supply are immediately congested. In examination of the brain of a person who has died during the first stage of cerebral embolism, we find those parts of the brain ordinarily supplied by the obliterated vessel paler than natural, with a zone of congested tissue, and perhaps numerous small extravasations of blood on the periphery.

The place where emboli are most frequently found is, as has already been stated, the left middle cerebral artery. The left common carotid arises from the arch of the aorta in a line almost exactly coinciding with the course of the blood-current. It therefore happens that an embolus which has

[1] Gazette des Hôpitaux, No. 80, 1857.

formed on the lining membrane of the heart, and which has passed into the aorta after having been detached, enters this vessel instead of the innominata. From the common carotid it passes into the internal carotid and thence with the stronger and more direct current into the middle cerebral artery, which is lodged in the fissure of Sylvius. Of forty-two cases of cerebral embolism collected by Meissner, in thirty-four the left hemisphere was the seat. Of thirty-seven cases occurring in my own practice, and to which reference has been made, thirty-one were accompanied with right hemiplegia, and were consequently on the left side of the brain. Post-mortem examinations were made in seven of these cases, and in all the embolus occupied the left middle cerebral artery.

The pathology of the genesis of the clot has already been sufficiently dwelt upon in other connections, and the fact that the artery in which it is found is not diseased has been mentioned.

The further consequences of embolism belong to cerebral softening and will be considered under that head.

Treatment.—It is not necessary to make any remarks on this point in addition to those made in regard to the treatment of thrombosis. There is very little to be done besides meeting indications as they arise, and attempting to relieve the paralysis and other vestigia, for which ends my views have been sufficiently expressed in the preceding chapters.

CHAPTER VI.

CEREBRAL SOFTENING.

As a consequence of several of the conditions described in the foregoing pages, and especially as resulting from thrombosis and embolism, cerebral softening naturally comes next in order for consideration. Most authors treat of it in direct connection with obliteration of the cerebral arteries, but, although frequently due to this cause, it may be produced by others, and occlusion is not always followed by softening. For these reasons I have preferred to consider it as it really is, a distinct pathological condition—as much so as sclerosis or any other morbid anatomical state.

Symptoms.—When softening is the result of hæmorrhage, thrombosis, or embolism, the symptoms peculiar to those affections are first met with. Thus there are troubles of the intelligence, the sensibility, and the power of motion, such as have already been described under the heads mentioned, and, if the morbid process goes on within the cranium, there are peculiar aggravations and the development of new symptoms. The condition of softening is not set up after either hæmorrhage, thrombosis, or embolism, till about the tenth day, and the symptoms now to be mentioned are those which are coincident with what some pathologists have designated the "second stage;" the "yellow softening" of others.

In addition to the continued paralysis of motion and the loss of sensibility which exist on one side of the body, the mental symptoms become more strongly marked. There

may be delirium with the occurrence of hallucinations and delusions, though these are generally evanescent. Occasionally a fixed idea obtains possession of the patient's mind, and for a while influences him in his conduct, but his mental tenacity is not strong enough to enable him to retain it for any length of time, so it soon yields to another.

The intelligence is notably diminished, so that the patient is unable to conceive an exact idea of his situation, or to obtain a moderately complete notion of quite simple matters which may be submitted for his mental action. Thus he refuses to credit the assertion that he is ill, declares that his health, both in mind and body, is excellent, and that he is fully capable of transacting his business or of performing any intellectual operation.

The memory is invariably impaired, and things of the greatest familiarity are forgotten. Thus a patient laboring under cerebral softening, the result of embolism, could not tell his wife's name, nor by what means he came to my office. Another, sent to me by Dr. Michel, of St. Louis, in whom thrombosis was the probable cause, could not tell me where he came from, nor the names of his children. He insisted with great vehemence that he was perfectly able to attend to his ordinary business, and yet was unable to add three numerals together.

In another case, likewise having the clinical history of thrombosis, which I saw in consultation with my friend Dr. J. W. Ranney, of this city, the patient, a gentleman of about sixty years old, could not tell his age; declared that Dr. Ranney, whom he had known for many years, was a grocer, "who lived around the corner;" and held to the delusion that his sons had made several forcible attempts to rob him.

The power of giving the attention to subjects is very greatly lessened. The patient may seem to be listening to what is said, or observing what is passing about him, but, if he be questioned he at once, shows that he really has not

been heeding; even when things are forcibly brought to his mind, and he is told to mark them, he is incapable of doing so to any considerable extent.

The speech is almost invariably affected either in the form constituting aphasia, or from paralysis of the tongue and other muscles concerned in articulation. There is a disposition to misplace words, or to clip them by cutting off the last syllable. Thus a patient reading the title of a book in my library called it the "Unit. Stat. Dispenst." for United States Dispensatory; another was the "Philosoph. as Absol. Scien." for Philosophy as Absolute Science; and he told me he was "a lawy. by professi.," when he meant to say he was a lawyer by profession. The same fault is shown in reading from a printed page, and in writing. Only a few days ago I received a letter from a gentleman, in which the final letter of nearly every word was omitted. The emotions, especially those of a sorrowful character, are very easily excited, and therefore the least untoward event causes the exhibition of feeling. Sometimes the patient sheds tears without being able to assign any cause, or may get into uncontrollable fits of weeping; occasionally of laughing.

All these symptoms indicate failure of the mental power, but it is, nevertheless, true that softening of the cerebral tissue may exist without the manifestation of the least degree of imbecility. It not unfrequently happens that, while there is a general loss of intelligence, some one or two faculties of the mind are notably increased in vigor.

I have a patient now under my charge whose intellectual force is greatly reduced, who cannot pronounce the simplest sentence correctly, who is paralyzed throughout the whole of one side, and who has so lost the sense of propriety that if he feels the desire to urinate he yields to it at once, no matter where he may be or who are present, but whose volitional power is even greater than before the accession of his disease. Thus he will read volume after volume, turning

over the pages regularly, and scarcely, except by oversight, skipping a word, although it is very certain he does not comprehend a tenth part of what he reads, and that what he does for the moment understand is immediately forgotten. The strength of his will is also shown in the impossibility of inducing him to do any thing which either caprice or habit prompts him not to do. His appreciation of harmony has become so sensitive that a discord of sounds made on the piano causes him real mental sufferring, whereas when he was in health his musical taste and discrimination of the pitch and quality of sounds were below mediocrity.

Drowsiness is very generally present; at first, perhaps, to a slight extent, but sooner or later as a prominent feature. Headache is very common, and is usually dull and circumscribed. The forehead is its most common seat. Other sensations in the head, such as vertigo, fulness, weight, and constriction, are scarcely ever absent.

Gradually, the condition of the patient, mentally and physically, becomes weaker and weaker, and death ensues, immediately preceded by coma, convulsions, delirium, or a combination of these phenomena.

Not unfrequently, softening of the brain is not preceded by hæmorrhage, thrombosis, embolism, or other evident affection, but begins obscurely, and advances very gradually. In this form the symptoms previously described make their appearance in succession; but the paralysis, instead of being present from the inception, comes on very slowly, commencing as a slight weakness, conjoined with numbness, in one or more of the extremities, or in the face. Ordinarily, the first evidence of paresis is discovered in the leg, which is not lifted clear of the ground. The toe consequently strikes against the inequalities of the pavement, and the patient is apt to fall. Sometimes the weakness is shown by the leg suddenly giving way at the knee. I have had several patients with cerebral softening, in whom this accident was of common occurrence, and who had thereby received severe

injuries. Or, when the arm is the paretic member, the grasp, as shown by the dynamometer, is materially lessened in strength, and things held in the hand are dropped. I have now a patient in charge in whom the affection is in its very earliest stages, and of which the only manifestations are, clipping of the words in speech and paresis of one arm.

This inability of the muscles to maintain a continuous contraction for a short time, though met with in several other affections, is to some extent characteristic of cerebral softening, and, in conjunction with the other phenomena, is a valuable indication. Even before it has become so far developed as to attract the attention of the patient or those about him, its existence may be ascertained by means of the dynamograph described in the preliminary chapter of this treatise. It will often be found that a straight line cannot be made, but that the pencil pursues a zigzag course, or else one descending with more or less regularity.

The paralysis usually goes on to complete loss of power, though its progress is often very slow, and is marked occasionally by periods of decided improvement. At these times the patient's friends imagine that he is about to recover, and if, as is sometimes the case, the mental symptoms are likewise mitigated, their hopes are still further exalted. It is necessary that the physician should not be deceived. In a case which I saw in consultation with Dr. Chamberlain, of this city, I diagnosticated chronic softening. At the time, there were feebleness of memory, paresis of one side of the body, and difficulties of speech. I gave an unfavorable prognosis, but soon afterward amendment began, and the patient, who was an insurance agent or appraiser, resumed his business to some extent. I nevertheless adhered to my opinion, for I had seen too many cases of similar character to be deceived in so clear a one as this. I never saw the patient again, and am therefore unacquainted with the subsequent phenomena, except that about a year afterward I was invited by Dr. Chamberlain to be present at the post-

mortem examination. His brain contained a *foyer* of softened tissue as large as a walnut, apparently the result of obliteration of the posterior branch of the left middle cerebral artery, and involving a portion of the middle lobe of the left hemisphere.

In another case, which I had very thorough opportunity for studying, the patient, a gentleman thirty-five years of age, was the subject of chronic softening, without any history of previous lesions. The disease had come on very insidiously, first showing itself by a slight impediment of speech and impairment of memory. Gradually he lost power in both arms and both legs, though the right side was more affected than the left. His gait became titubating, and although he never lost the ability to walk, yet he did so with great and increasing difficulty. But his stages of apparent improvement were at first numerous and well marked. His memory at such times was stronger, his countenance brighter, his articulation distinct, his emotions more under command, his power of attention increased, his intelligence equal to all ordinary occasions, and his walk free from any sign of debility. Then all these steps would be suddenly lost, and he would again become imbecile and weak. Finally, a severe convulsion, more evident on the right side than the left, supervened one evening after dinner, as he was quietly smoking a cigar. Between seven and twelve o'clock that night he had over a hundred fits. He died at the latter hour. The post-mortem examination revealed the existence of a large centre of softening, involving the middle lobe of the left hemisphere.

Sometimes the course of the disease is still more irregular. No evidence of cerebral disorder is perceived beyond aphasia, and the patient remains in the full possession of his intellect, and without paralysis, up to a short time before death. Durand-Fardel[1] cites the case of a man, thirty years of age, who entered the Hôtel Dieu, presenting all the signs

[1] Traité du Ramollissement Cérébral, Paris, 1843.

of pulmonary phthisis. In a few days afterward he experienced difficulty of articulation, in thirty hours he became comatose, and, in twenty more, died. The post-mortem examination revealed the existence of softening of the inferior surface of the left middle lobe of the cerebrum. Although it is not so stated—Durand-Fardel having written previously to Virchow's observations—there is little doubt that the cause of the softening was an old embolus in the left middle cerebral artery.

Lallemand,[1] in his first letter, cites several cases in which the disease was marked by singular symptoms, such as convulsions, contractions, and delirium.

In a case which I saw in consultation with Prof. C. A. Budd and Dr. J. T. Taylor, occurring in a gentleman about thirty-five years of age, there were coma and violent hemiconvulsions, evidently due to softening from embolism, of which there had been two attacks, the last several weeks previously. Death ensued, but no post-mortem examination was, I believe, obtained.

A gentleman is now under my charge who has valvular disease on the left side of the heart, the consequence of rheumatic endocarditis, and who, six months since, had an apoplectic attack conjoined with aphasia and right hemiplegia. He soon became able to speak pretty well, and regained power and sensibility to a great extent in the paralyzed limbs. During the past two weeks, however, he has exhibited symptoms of mental derangement, as shown by the existence of hallucinations and delusions, and is gradually losing the power of motion and of sensation on the right side. His speech is as perfect as it ever was, and there is yet no sign of dementia.

It has happened that individuals have died who, on post-mortem examination, were found to have softening of the brain, but who, during life, had exhibited no symptoms of

[1] Recherches Anatomico-Pathologiques sur l'Encéphale et ses Dépendances, Paris, 1824.

LEEDS SCHOOL OF MEDICINE

this or any other cerebral disorder. Rostan, who was the first to write systematically on the disease, refers to such cases, and Durand-Fardel is still more explicit. The latter says:

"We meet with softening of the brain in persons who, up to the time of death, had presented no appreciable derangement of the cerebral functions, and in whom softening has been developed without having given any evidence whatever of its existence." In such instances the white matter of the hemisphere can alone be involved.

One such case verified by post-mortem examination has occurred within my own experience. The patient, a soldier of the Second United States Infantry, died at Fort Riley, in Kansas, of which post I was the medical officer, of chronic dysentery, the result of exposure. There were no mental symptoms, no difficulty of speech, no paralysis; nothing, in fact, indicating the existence of brain-disease. He died in full possession of his intellectual faculties. The post-mortem examination revealed the existence of ulceration of the small intestines, and, as the cause of death was very evident, the brain was not examined. I reserved it, however, for purposes of study, and, on making a section of the right hemisphere an hour afterward, discovered an encysted centre of softening including more than two-thirds of the posterior lobe. The right posterior cerebral artery was entirely obliterated by thrombosis. The man had been at the fort for several months, and had never made complaint of any illness till he was attacked with dysentery six weeks before.

The duration of cerebral softening is very variable. Rostan found it to range from a few days to several years. Andral, from an analysis of one hundred and five cases, found that the period was from twelve days to three years. The most rapid case occurring in my experience terminated in death at the end of eighty hours. Some confusion on this point has arisen from the fact that some authors regard embolism and thrombosis as essentially identical with soften-

ing, a doctrine which is clearly erroneous, as, in many cases of these affections, recovery or death may take place without the stage of softening being reached. In the case above referred to, post-mortem examination showed that the condition known as yellow softening was just making its appearance. As I have already stated, I cannot regard the alteration called by some pathologists red softening any thing more than the congestion due to the active collateral circulation.

The case of longest duration, of which I have any personal knowledge, was that of an eminent scientific gentleman, who had suffered from the symptoms of softening of the brain for nearly four years, when he died. There was no post-mortem examination, but the history of the case was that of thrombosis of the left middle cerebral artery, and the course of the disease left no room for doubt as to its nature.

When death results, it may be directly due either to the disease itself, or to some intercurrent affection. Thus the patient may die from pure exhaustion or from slow asphyxia caused by the imperfect action of the respiratory function, or he may choke to death either by being unable to swallow food which he has taken into his mouth, or by the regurgitation of the contents of the stomach during a convulsion, or a severe convulsive seizure may cause immediate asphyxia, or a series of convulsions may produce a more gradual asphyxia, or he may die in a state of profound coma.

The intercurrent affections may be either meningitis or hypostatic congestion of the lungs from long confinement to the recumbent posture, or diarrhœa, or a fresh attack of thrombosis or embolism.

Causes.—The etiology of cerebral softening has already been considered to some extent under the heads of cerebral hæmorrhage, thrombosis, and embolism, of which conditions it is so often a sequence; but, as it frequently occurs without having been preceded by either of these or other

noticeable affections, a few additional observations are necessary.

Age is certainly a strong predisposing, if not an actual existing cause, although the disease is observed at all periods of life. Rostan, whose cases were collected at the Salpêtrière, a hospital containing only old women, found that there were ten cases in persons between the ages of sixty and sixty-nine, twenty between seventy and seventy-nine, and ten between eighty and eighty-seven. Andral, excluding cases occurring in infants, found that, of one hundred and fifty-three cases, there were between the ages of

15	and	20	10
20	"	30	18
30	"	40	11
40	"	50	19
50	"	60	27
60	"	70	34
70	"	80	30
80	"	89	4

Durand-Fardel, from an analysis of fifty-five cases, found between the ages of

30	and	40	3
40	"	50	8
50	"	55	2
60	"	70	14
70	"	80	23
80	"	87	5

The period of life, therefore, at which softening is most apt to occur, is from the age of fifty to eighty.

During the past six years twenty-eight cases of cerebral softening, not the result either of hæmorrhage, thrombosis, or embolism, have been under my care or been seen by me in consultation. Of these one was under twenty years of age, three were between twenty and thirty years; four between thirty and forty; four between forty and fifty; six

between fifty and sixty; eight between sixty and seventy; and two between seventy and eighty. The general results, therefore, go to show the greater proclivity which advanced age gives to the occurrence of the disease. In one of those between seventy and eighty, the mind was scarcely impaired till about two months before death, though there had been paresis, headache, and aphasia, for two years.

No definite statistics have been collected relative to the influence of sex, although the opinion appears to prevail that the affection is more liable to occur in females than in males. Of the twenty-eight cases just cited, twenty were males and eight females.

The season of the year does not appear to exercise much influence. Durand-Fardel, from sixty-three cases, found that seventeen occurred in winter, thirteen in spring, twenty in summer, and thirteen in autumn. I have found it difficult in many cases, from the insidious or latent character of the early symptoms, to fix the period of beginning with accuracy.

Intense and long-continued intellectual exertion is one of the most common causes of cerebral softening. Eleven of the cases occurring in my experience were clearly the result of this cause. Severe and protracted emotional disturbance was apparently the cause in four cases.

Rostan, among the causes, cites insolation, the action of intense cold upon the head, blows, and excessive use of alcoholic liquors.

The influence of thrombosis and embolism in producing partial cerebral anæmia, and hence as leading to the supervention of softening, has already been dwelt upon at sufficient length.

Diagnosis.—The history of hæmorrhage, thrombosis, or embolism, when these affections have either of them given rise to softening, will aid in the diagnosis. The signs which serve to distinguish these affections from others have already been sufficiently considered.

When there is no such previous clinical history, softening of the brain may be confounded with chronic meningitis, meningeal hæmorrhage, or tumors. From chronic meningitis it is to be distinguished in many cases by the facts that in the former the headache is generally diffused, while in softening it is fixed, that the paralysis is more limited, that there are frequent spasms of the limbs, that there are well-marked febrile exacerbations, and that there is not the progressive enfeeblement of the intellect so characteristic of the vast majority of cases of cerebral softening. At the same time it must be admitted that the diagnosis sometimes cannot be clearly made out.

In meningeal hæmorrhage coma occurs as an early symptom, gradually increasing in intensity, whereas in softening it comes on at a late period. Hæmatoma of the dura mater, however, may readily be confounded with softening. The history of the case will aid in the formation of a correct diagnosis.

In tumors the most prominent symptoms are pain and convulsions, while the intellect usually remains unaffected. The pain is exceedingly intense, while in softening it is dull. The speech in tumors is generally unaffected.

Prognosis.—Cerebral softening in general ends in death. Nevertheless, it is not altogether hopeless. If the patient be young, of good constitution, and of temperate habits; if the centre of softening be small, and not involving the more important parts of the brain, there is some encouragement to expect a favorable termination. Some of the cases cited in this chapter go to show that recovery is possible, and I have certainly seen others with the ordinary initial symptoms of cerebral softening recover with appropriate medication. Such patients, however, were all under the age of forty, and were of good constitution and habits. In softening due to embolism, and occurring after rheumatism and endocarditis, the liability to future attacks must not be overlooked. I have seen as many as six attacks of embolism oc-

curring in the same patient, and yet no morbid condition beyond that of anæmia set up, and again cases in which a single embolus has caused softening and death.

Morbid Anatomy.—In the softening of the brain which results from thrombosis or embolism, the first stage after that of congestion from the excessive action of the collateral circulation is what is called yellow softening. This is not, as some authors have supposed, produced by the infiltration of pus into the cerebral substance, but is caused by regressive metamorphosis of the brain-cells into fat, the granules of which are mixed with the coloring matter of the blood which gives rise to the peculiar yellow color. The white corpuscles of the blood also undergo degeneration into fat.

These altered white corpuscles were described by Gluge[1] as inflammation corpuscles, under the idea that softening was always the result of inflammation. Laborde,[2] who has studied this subject with great success, shows, however, very conclusively that the transformation is a true degeneration, a part of the fat-corpuscles being derived, as stated above, from the nervous fibres, the cylinders of which disappear, the contents being extravasated, and with the myeline being converted into fat; and another part consisting of altered white blood-corpuscles. At this time the cerebral tissue is pulpy, constituting a centre of softening or a *foyer*, the consistence of which is greater at the circumference than at the centre. The blood-vessels passing through the disorganized portion are easily separated from the perivascular tissue and are covered with oil-globules.

The second stage is designated white softening, and in it the brain-substance loses altogether its morphological characteristics, and appears as a white, cream-like matter so soft that a weak stream of water, allowed to impinge upon it, washes it away. In this semi-liquid matter, whitish flakes of denser tissue are suspended. Microscopical examination

[1] Atlas of Pathological Histology. Translated by Leidy. Philadelphia, 1853.

[2] Op. cit.

shows that all traces of nervous structure have disappeared, and that no anatomical elements remain except oil-globules and organic corpuscles somewhat resembling leucocytes.

When the morbid process involves the cortical substance of the cerebrum, the convolutions undergo a peculiar kind of transformation first pointed out by Cruveilhier, and then by Durand-Fardel[1] as occurring in the senile form of softening.

This is characterized by the formation of yellow plates, irregular in form, soft to the touch, but yet sufficiently dense to resist the action of a thin stream of water. Microscopically they are seen to consist of nucleated fibres, fat-corpuscles, fat-globules, and degenerated capillaries, with blood-crystals and granular matter. Essentially, therefore, they are formed of connective tissue.

The degenerated nerve-tissues, constituting a focus of softening, may undergo absorption. In such a case, a cicatrix, similar in general characteristics to that resulting from the curative process of hæmorrhage, remains.

In the softening resulting from inflammation, a somewhat different set of morbid appearances exists. Thrombosis and embolism produce a true death of the parts previously supplied by the occluded vessels, a necrobiosis, as it has been called by Virchow. The process is accompanied, as we have seen, by degeneration of the nervous tissue, but in the softening due to inflammation new formations result. Sometimes the two coexist, but the latter is occasionally an entirely independent action.

When such is the case, connective tissue is generated, and the nervous substance is rapidly broken down. An exudation of an albuminous fluid containing fine granules, the disintegrating nervous substance and numerous flakes of coagulated fibrine, takes place, and with blood-corpuscles causes the centre of softening to present the appearance of a red-

[1] Maladies des Vieillards. Paris, 1854, p. 72.

dish pultaceous mass, easily washed away by the action of a weak stream of water. With age the color of this softened tissue becomes brown or yellow. Sometimes, when the inflammation has extended to the deeper parts of the cerebrum, the contents of the cyst are penetrated by the new connective tissue. The pulpy mass undergoes partial absorption, and is replaced by a white turbid liquid, called by Cruveilhier and Dechambre "milk of lime" (lait de chaux). Durand-Fardel designates this form of softening "cellular infiltration."

The softening resulting from occlusion of the capillaries, a condition not recognizable during life, does not differ essentially, except in its situation, from that which follows thrombosis or embolism. The centres of the process are, however, smaller, and are generally numerous.

When disease of the capillaries has been the cause of the softening, these may be ruptured, and we meet with minute extravasations of blood in the disintegrated perivascular tissue constituting the "capillary hæmorrhage" of Cruveilhier.

Pathology.—The first definite accounts of cerebral softening were given by Lallemand [1] and Rostan,[2] both of whom published their works in the same year, 1820.

In the very beginning of his first letter, Lallemand awards to MM. Récamier, Bayle, and Cayot, the credit of describing the condition under consideration, and of giving it the designation by which it is so generally known, even out of France, of ramollissement. Lallemand then proceeds to define the term by saying that, by ramollissement of the brain, he understands a kind of liquefaction of a part of its substance, the remainder preserving its ordinary consistence. He then quotes cases from Morgagni and Abercrombie, and cites others from his own experience; and then concludes by declaring that he

[1] Recherches Anatomico-Pathologiques sur l'Encéphale. Paris, 1820.

[2] Recherches sur le Ramollissement du Cerveau. Paris, 1820. My references to Rostan's work are to the second edition, of 1823.

does not hesitate to range cerebral softening among the inflammations, in which opinion he is supported by Abercrombie.[1] Rostan[2] regarded the disease as sometimes being due to inflammation, and sometimes to degeneration of the blood-vessels. Bouillaud[3] viewed it as an anatomical feature of inflammation. Cruveilhier[4] considered what he called red softening as resulting from the capillary hæmorrhage previously mentioned, and that other forms were certainly due to inflammation.

Andral[5] recognized the fact that softening might result from inflammation or capillary hæmorrhage, but he also insisted that it might be due to special alterations of nutrition, caused by different morbid influences, such as obliteration of the arteries supplying the brain, or impoverishment of the blood.

MM. de la Berge and Monneret[6] adopted in part the views of Rostan relative to degeneration of the cerebral vessels as a cause of softening. Carswell[7] regarded softening occurring during life as being effected by these circumstances—inflammation, obliteration of arteries, and modification of nutrition.

Fuchs[8] appears to think that inflammation is not a necessary antecedent, but that congestion is. He also admits obstruction of the arteries at the base of the brain to be a cause.

The studies of Durand-Fardel[9] have been very thorough, and have contributed greatly to our knowledge of cerebral softening. According to him, the affection is an inflamma-

[1] Op. cit., p. 205. [2] Op. cit., Chapter VII.

[3] Traité de l'Encéphalite. Paris, 1825.

[4] Art. Apoplexie, in Dictionnaire de Médecine et de Chirurgie Pratique.

[5] Clinique Médicale.

[6] Compendium de Médecine Pratique.

[7] Art. Softening of Organs, in Cyclopædia of Practical Medicine, vol. iv., p. 176, American edition.

[8] Beobachtungen und Bemerkungen über Gehirnerweichung. Leipzig, 1838.

[9] Traité du Ramollissement du Cerveau, Paris, 1843.

tion which does not differ essentially from other inflammations occurring in the young or old. White softening he regards as the chronic form of the disease.

Other pathologists published the results of their observations and generally to the same effect as those which have been quoted, viz., that cerebral softening was an inflammatory process, and sometimes one resulting from obliteration or disease of the arteries. A few, however, held to the view of Lallemand and Durand-Fardel, that inflammation was always the starting-point.

In 1847 Virchow published his observations relative to embolism, and the partial cerebral anæmia produced by occlusion of an artery thus became a recognized fact. In reality, it came to be regarded as the only cause capable of giving rise to softening, and many pathologists of the present day entertain such an opinion. But I think this is carrying the theory further than facts will warrant. I cannot altogether disregard the researches of Durand-Fardel,[1] Calmeil,[2] Rokitansky,[3] Wedl,[4] and others, and although I cannot agree that all cerebral softening is a consequence of inflammation, I am very sure it has this and other causes besides thrombosis and embolism. Calmeil's work is a monument of careful observations and scientific deductions, and his fifth chapter (t. ii.), entitled "*Du Ramollissement cérébral local aigu, ou de l'Encéphalite locale aiguë sans caillots sanguins siégeant sous la forme d'un foyer ou des plusieurs foyers circonscrits, soit à la surface, soit dans la profondeur de la masse encéphalique*," contains cases which are amply sufficient to establish the point for which he contends. He shows, too, in other chapters of his treatise, that softening results about the periphery of clots due to cerebral hæmorrhage.

[1] Maladies des Vieillards, Paris, 1854.

[2] Traité des Maladies Inflammatoires du Cerveau, Paris, 1859.

[3] Pathological Anatomy, Sydenham Society, translation, 1850.

[4] Rudiments of Pathological Histology, Sydenham Society, translation, 1855.

The weak feature of Calmeil's otherwise very complete work is, that he altogether ignores Virchow, and those after him, who have confirmed his facts and theories.

Soulier,[1] on the other hand, can see in softening nothing of the nature of inflammation. For him it is always a necrobiosis, produced by the cessation of the physiological action of the blood, obliteration by embolus or thrombus, by diminution of the calibre of the vessels, or occlusion resulting from atheroma or obstruction of a vein or sinus. He admits that the obliteration of an artery may cause congestion behind the point of obstruction, by which the coagulation and capillary hæmorrhage of acute softening—the capillary apoplexy of Cruveilhier—are to be explained. This red ramollissement has, however, nothing of the nature of inflammation about it.

The only points in which I differ with Soulier are, that I cannot regard softening as being solely due to occlusion of blood-vessels, and that I am very sure the congestion which follows thrombosis or embolism is not necessarily the first stage of softening. There is no more reason why partial cerebral anæmia should always result in softening, than that ligation of the femoral artery should always lead to gangrene of the parts below.

Obstruction of veins and sinuses in the brain may be followed by softening. The clot is usually the result of injuries or disease of the cranial bones or cerebral membranes, especially the dura mater. It may also be caused by certain cachectic conditions in which the blood is deteriorated in quality, such as typhus and typhoid fevers and cholera.

Four cases, in which this latter affection was followed by thrombosis of the superior longitudinal sinuses, with consective softening, have come under my observation. In two of them there were also thrombi in both femoral veins. The upper surfaces of both hemispheres were the seats of the softening, which involved the gray matter only.

[1] Journal de Médecine de Lyon, Février, 1867.

Thrombosis of the veins or sinuses may also in general terms be produced by whatever cause is capable of retarding the current of blood. Mr. Toynbee,[1] in his chapter on diseases of the mastoid cells, has brought forward several cases in which the lateral sinus was occluded by coagula, and in which there was cerebral softening.

Cerebral softening may also result from the formation of adventitious growths, or from the presence of foreign bodies in the brain. In such cases the process begins with inflammation, and is similar to the action which sometimes goes on around an extravasation of blood.

Acute cerebritis or meningitis may likewise result in softening. This fact is admitted by Drs. Russell Reynolds and Bastian, in their admirable essays on cerebritis and softening of the brain, though with evident reluctance.

We see, therefore, that cerebral softening may be caused either by anæmia or inflammation, and that it is of two kinds, inflammatory and non-inflammatory. The seat of the softening may be in any part of the brain, although some regions are more liable than others. When due to thrombosis, there appears to be no predilection for any particular location, but, as embolism is generally found on the left side in the middle cerebral artery, the parts of the brain supplied by this vessel are more liable than the corresponding parts of the right side.

Durand-Fardel, however, did not arrive at this conclusion. Of one hundred and sixty-nine cases of softening, he found the left hemisphere the seat in sixty-nine, the right in seventy-one, both in twenty-six, and the middle line in three.

The gray matter is generally supposed to be more frequently the seat of softening than the white. It is true that, of thirty-three cases of acute softening observed by Durand-

[1] The Diseases of the Ear, their Nature, Diagnosis, and Treatment: London, 1860.

Fardel,[1] the convolutions were involved in thirty-one, but in nine only were they the sole part affected.

In fifty-three cases which the same author collected from the writings of Rostan, Lallemand, and others, the centres of softening were found to be as stated in the following table. Occasionally more than one region was involved.

Convolutions and white substance	22
Convolutions alone	6
White substance alone	5
Corpus striatum and optic thalamus	6
Corpus striatum alone	11
Optic thalamus alone	4
Pons Varolii	3
Crux cerebri	1
Corpus callosum	1
Walls of the ventricles (septum)	1
Fornix	1
Cerebellum	1

Bastian,[2] has therefore erred in citing these statistics as showing the greater liability of the convolutions; for in six cases only were the convolutions the sole seat of the disease.

Rostan, on the other hand, found the corpora striata, around the optic thalami, to be the parts most frequently affected, and after these the central part of the hemispheres. He met with but few cases involving the median line.

As regards the frequency with which the convolutions with the white substance were involved, as compared with the motor tract, he found that, of one hundred and seventy-seven cases of acute and chronic softening, the convolutions were affected in one hundred and nineteen, and the corpora striata and optic thalami in fifty-eight.

The middle lobe is more liable than any other, as is seen in the following statement of Durand-Fardel, based upon an analysis of ninety-five cases :

[1] Traité du Ramollissement du cerveau, Paris, 1843. [2] Op. cit.

Posterior lobe	18
Middle	51
Anterior	13
Posterior and middle	7
Posterior and anterior	2
Middle and anterior	2
Whole convexity of hemisphere	1
Middle line	1

In more than one-half of the cases, therefore, the middle lobe was the seat of the disease.

A question connected with the pathology of cerebral softening, as with hæmorrhage, is, Can we determine, from a consideration of the symptoms, what part of the brain is the seat of the lesion? The answer must be the same. We can do so with some approach to accuracy, but, till we are better acquainted with the physiology of the different ganglia composing the brain, we cannot expect to do so with absolute certainty. Indeed, owing to the greater extent of tissue involved, compared to that affected in hæmorrhage, we have a more complicated set of phenomena to deal with. I have nothing further to add to the remarks made on a similar point, under the head of cerebral hæmorrhage.

Treatment.—The treatment proper for cerebral softening should depend very much upon the cause from which it has arisen, and must more or less be directed against the symptoms which are manifested. Thus, if there is reason to suspect the existence of thrombosis or embolism, and a consequent anæmic condition of a portion of the brain, the judicious use of stimulants and tonics is advisable, while the body should be kept warm by additional clothing, or the application of artificial heat—at the same time the recumbent posture should be assumed, and the head supported on a low pillow. Mental exertion should, of course, be absolutely interdicted. If there be much headache, it is probably due to too great an activity of the collateral circulation, and in such a case some one of the bromides may be given

in large doses, repeated as often as may be necessary. I have frequently seen great relief follow their administration.

Delirium is often due to a like cause and may be similarly treated. Dr. Reynolds [1] speaks highly of the Indian hemp in doses of a quarter to half a grain of the extract; but, I have found the bromide of potassium, in doses of thirty grains every three or four hours, more efficacious.

It is also the most beneficial remedy in the convulsions which frequently precede a fatal termination.

In that form of softening which is obscure in its origin and gradual in its progress, there is a little more hope of a favorable result, though even here it must be confessed that treatment is not often effectual. Still, as I have said, when speaking of the prognosis, there are undoubtedly cases in which recovery has taken place, and I am very sure that I have several times succeeded in curing individuals who, so far as I have been able to judge, were affected with cerebral softening. As these cases are interesting in themselves, and as the histories will show the means of treatment employed, I do not hesitate to transcribe the following typical ones from my case-book:

I.—Mr. R., a gentleman, twenty-four years of age, awoke one morning about the middle of March, 1870, with a sensation of numbness extending through the whole of the left arm and leg, and with a feeling of vertigo which was insupportable when he arose from the bed. He sat down in a chair, and while in this position was conscious of a buzzing sound in the right ear. In the course of half an hour the vertigo passed off, but the numbness and sound in the ear remained, and he occasionally saw double. In a few days afterward he noticed a slight difficulty of articulation, owing to apparent thickness of the tongue, and about the same time observed that in the morning the pillow was wet with the saliva which had run from his mouth during sleep. His uncle, a wealthy gentleman of this city, sent him off travel-

[1] Article, Softening of the Brain, in System of Medicine, vol. ii.

ling, but he returned in a few weeks with loss of power in the left arm and leg, which had begun to be manifested to a slight extent before his departure. He came under my charge May 15, 1870.

At this time the paralysis, of both motion and sensation, was well marked on the left side, as shown by the æsthesiometer and dynamometer. The line made by the dynamograph with the right hand was perfectly straight, while that made by the left was at an angle of forty-five degrees with the other. In his conversation he clipped his words, and sometimes left out the smaller ones. His memory he stated was materially impaired. There was almost constant headache over the whole frontal region, and attacks of vertigo were frequent. There was no marked paralysis of the face, though the muscles of both sides were paretic, and he often had double vision. The right pupil was largely dilated and was insensible to light.

Ophthalmoscopic examination showed the left eye to be perfectly normal, but the retinal vessels of the right were smaller and straight, and the choroid was paler than natural.

Upon inquiry I ascertained that he had given extraordinary attention to his business for a period of several months before the attack of numbness, frequently being up making calculations till three o'clock in the morning, and thus depriving himself of the necessary amount of sleep.

My opinion was, that he was suffering from incipient softening of the brain due to disease of the capillaries, which, in its turn, resulted from cerebral congestion and exhaustion. I was further of the opinion that the lesion involved the right hemisphere and motor tract.

I prescribed the phosphide of zinc in the dose of the tenth of a grain, with half a grain of extract of nux-vomica in pill three times a day, with the constant galvanic current three times a week, the latter to be derived from fifteen of Smee's cells, and to be passed from forehead to occiput for

three or four minutes at a time. At the end of ten days he had lost his diplopia, the pupil of the left eye had regained its natural diameter and irritability, and the vertigo and headache had notably diminished. The treatment was continued, and at the end of a month he had recovered the sensibility and power on the paralyzed side to such an extent, and had improved so much in other respects, that I advised him to take a short journey. He was absent two weeks, during which period he continued to take the pills as before, and on his return was, to all appearance, well. He has since remained in excellent health.

II.—Mr. R. W., a merchant of this city, consulted me in April, 1868, under the following circumstances:

After a long period of great domestic anxiety, during which he had been engaged in some heavy commercial transactions, and had suffered from wakefulness, he experienced one afternoon, while riding in the park in his carriage, a slight quivering motion at the apex of the tongue. It continued until he reached home; and then, upon looking in a mirror, he could see the fibrillary movement very distinctly. He was not alarmed, and went to bed at his usual hour. In the morning he noticed a little thickness of speech, but the movement had ceased. That afternoon he had a violent headache, attended with vertigo and nausea. Becoming alarmed, he sent for his family physician, who ascribed the symptoms to indigestion, and administered a mild cathartic. The following day, on attempting to rise from the bed to go to the water-closet, he was attacked with such a severe vertigo that he was obliged to lie down again; and, though he did not for a moment lose consciousness, his fæces escaped from him involuntarily. From this time he gradually lost strength in both arms and legs, and his speech became very defective. His memory suffered to such an extent that he forgot the names of his children. There was very little headache, the vertigo had ceased, there was no disturbance of vision, and no loss of power over the sphinc-

ters. About six weeks after the occurrence of the first symptom noticed, he came under my care.

At this time there was anæsthesia of both sides of the body, both legs and both arms had lost power; he clipped his words, and frequently substituted others of similar sound or meaning for those he ought to have used. His memory was much weakened, and there was a strong tendency to stupor. There were no troubles of the special senses—ophthalmoscopic examination revealed nothing abnormal—there was no facial paralysis. I diagnosticated softening of the brain from general cerebral anæmia consequent upon congestion and cerebral exhaustion, and I prescribed a liberal allowance of wine, a full and nutritious diet, carriage exercise, and amusements of various kinds. This was the very reverse of the treatment to which he had been subjected. In addition, I recommended the constant galvanic current, to be applied as in the previous case, and gave the following prescription: ℞. Oleii phosphorat. ʒss; mucil. acaciæ, ʒj; ol. bergamot gtts. xv. M. ft. emulsion. Dose, gtts. xv. ter die.

The treatment was carried out with the result of obtaining a gradual and permanent improvement, so that at the end of about six months the patient was well. He then went to Europe, where he now is, with as good health as he has ever enjoyed.

Three other cases, similar in their general features, have been under my care with a like result in each, and several others have been very decidedly improved and relieved of the more prominent symptoms of the disease without, however, regaining full health. The means of treatment thus far consist in the use of tonics, stimulants, and especially phosphorus and strychnine, the avoidance of all severe mental exertion, and all excessive emotion, open-air exercise, and the use of the constant galvanic current.

The beneficial effects of maintaining the physical strength were several years since pointed out by Mr. F.

LEEDS SCHOOL OF MEDICINE

Skey[1] in a clinical lecture delivered at St. Bartholomew's Hospital, but it must be confessed that the opposite plan of treatment has been very generally followed.

Softening from the effects of thrombosis or embolism is, as I have said, not much under the control of the physician. Patients recover from it, however, when they are of good constitution, and when the focus of softening has not been extensive. The mind and body may, and in such cases generally do, remain false, and we are therefore consulted for the relief of the condition. In such cases tonics, and among them phosphorus, strychnine, and wine, occupy a prominent place; the constant galvanic current to the head, and the induced to the paralyzed muscles, will rarely fail to be of service.

III.—Thus a gentleman, who had been a distinguished officer of the army, suffered from loss of memory, defective articulation, ptosis, double vision, and right hemiplegia, probably the result of embolism. Several years before he came under my charge, he had been treated by my friend Dr. J. T. Metcalfe, for heart-disease, the result of acute rheumatism. I gave the phosphide of zinc and extract of nux-vomica according to the formula previously mentioned, advised a liberal use of wine and beefsteaks, applied the primary current to the brain, and the induced to his paralyzed arm and leg, and in a few weeks had the satisfaction of seeing such a degree of improvement as almost to constitute a cure. The ocular troubles had disappeared, his memory had improved, he talked as well as ever, and the numbness and loss of strength were no longer remarked unless he overexerted himself, which, owing to his general feeling of *bien aise*, he was very apt to do. He remained in this condition for over a year, when he had several other attacks of embolism, each of which left him more weak, mentally and physically, than before, and of which he eventually died.

[1] On the Value of Tonic Treatment in some Diseases of the Brain, more especially cases of Ramollissement. Dublin Hospital Gazette, November, 1858.

There were some interesting features connected with this case, which will be referred to at greater length hereafter.

IV.—In another case, in which there was reason to think a *foyer* of softening had been absorbed, a marked relief from the sequelæ was obtained. The patient, a literary gentleman of distinction, had, several years previously to my seeing him, suffered from an attack of acute rheumatism with endocarditis. About a month after his recovery, as he was sitting in his library before the fire, he felt a sensation as if one side of his face had suddenly become much heavier than the other. Almost immediately afterward he lost consciousness, and fell to the floor. He could not have been in this condition longer than five minutes when he came to himself, to find that he was paralyzed in the right arm and leg. Attempting to call for assistance, he found he could not articulate. His wife soon afterward entered the room, and medical aid was obtained. He was bled to the extent of sixteen ounces, and purged with croton-oil.

The following day he was much better; could move his arm and leg, and articulate with some degree of distinctness, but toward evening headache ensued, he became delirious, and the paralysis increased. Of the condition immediately following, he could give no very clear account. He only knew that he was confined to his bed for several weeks, was delirious part of the time, and that, after the acute attack passed off, he was left with an enfeebled mind, imperfect articulation, and paralysis of the arm and leg on the right side. He went to Europe, travelled extensively, and returned at the end of a year very much improved, but still with some degree of mental weakness, defective speech, and paralysis, remaining.

When he came under my observation, the following were the principal symptoms observed: The strength of the right arm, as measured with the dynamometer, was not one-third that of the left; the extensors of the leg and foot were almost entirely paralyzed, so that in walking he ab-

ducted the leg so as to cause the foot to clear the ground · electro-muscular contractility was much weakened, though the induced current caused feeble contractions. His speech was affected mainly as regarded the memory of words. He spoke with a good deal of volubility, but constantly used the wrong expressions. Thus, when he wished to tell me that he had visited Europe for the benefit of his health, he said: "I went to elope for the bequest of my hedge," and then went on—continually making other mistakes—to tell me a long story which I could scarcely understand. His emotions were easily disturbed: he cried because he had to wait a few minutes in my reception-room before seeing me.

Ophthalmoscopic examination showed pale choroids and straight and attenuated retinal vessels. Auscultation revealed the existence of both mitral and aortic regurgitation.

Taking into consideration the history of the case and the present condition of the patient, I diagnosticated embolism of the left middle cerebral artery, subsequent softening and eventual absorption of the diseased part of the brain. My idea was that the brain, as a whole, was anæmic, and that, with improved nutrition of it and the paralyzed limbs, amelioration of the symptoms was possible.

I therefore prescribed the phosphide of zinc and nux-vomica pills as before mentioned, directed the use of wine to the extent of half a bottle of champagne daily, and advised that animal food should form the principal portion of each meal. Since his illness he had, by direction of his physician, left off the use of coffee. I directed it to be resumed, and to be taken strong. The primary galvanic current was passed through the head in the manner previously indicated in this chapter, and the induced current was applied for half an hour three times a week to the arm and leg, each paralyzed muscle receiving a full share of attention.

It was not long before signs of amendment were noticed. His strength became greater in the arm, and he was able to extend the leg and to raise the foot after half a dozen elec-

trical applications. His speech next gave evidence of improvement, and his mind became stronger. The treatment was continued for about four months, with only an intermission of a week. At the end of that time his gait was almost natural, though he still swung the foot a very little, his arm was nearly as strong as the other, his mind was not perceptibly weaker than that of other persons of his age (fifty-five), and his speech was excellent except when he was excited and very anxious to express himself correctly and fluently.

There is one point in regard to which a few words are perhaps necessary, and that is to enter a protest against the use of counter-irritation of any kind, and to discountenance, as far as I can, the employment of the actual cautery. I have never seen the least advantage follow the application of croton-oil to the shaven scalp, nor can I conceive how such a measure can be recommended on rational grounds. I have several times witnessed its action, and have invariably seen it aggravate the symptoms. In the case of a gentleman from St. Louis, affected with cerebral softening, the effect was to make his speech still more imperfect and his mind weaker. A lady, who was affected with all the more prominent symptoms of softening of the brain, had all the phenomena increased in violence after the application of the actual cautery to the nape of the neck. I could easily adduce other examples to the same effect, were it necessary.

CHAPTER VII.

APHASIA.

The subject of aphasia is of such interest, and so much attention has recently been given to it by physiologists and pathologists, that, although it is only a symptom common to several morbid conditions, a treatise on diseases of the nervous system would scarcely be regarded as complete without its being fully considered.

By aphasia is understood a condition produced by an affection of the brain by which the idea of language, or of its expression, is impaired. The word is derived from the Greek —*a*, privative, and *φασις*, speech—and, as stated by Trousseau, was proposed by M. Chrysaphis, a distinguished Greek scholar, as a substitute for *alalia*, used by Lordat, and *aphemia*, employed by Broca, to designate the same condition.

In the definition which I have given of aphasia, the term is limited to impairment of the idea of language or of its expression. It does not, therefore, include those cases in which the individuals are able to speak, but will not; such as are met with among the insane. The idea of language is as perfect as ever, and is doubtless entertained, but the person does not speak because he does not will to do so, and this failure may arise either from a lack of the necessary power, or from a stubborn determination not to speak. A lady was a short time since under my charge who had been treated by a homœopathic physician as a case of aphasia. A very slight examination was sufficient to convince me that the case was one of hysteria. She had not spoken for several months, but

upon one occasion she came to my office with her maid, whom she required to repeat the alphabet, and when the right letter was reached she signified the fact by raising her hand. She thus spelled out the words she wished to use. Subsequently she procured a card with all the letters on it, such as are used for children learning their alphabet, and she composed her words from this. Of course all these facts showed that her idea of language was intact, but she still might have lost the power of coördinating the muscles concerned in articulation so as to express herself in spoken words. Although I was sure this was not the case, I failed to make her speak, until one morning, she became very much interested in something I was saying, and, finding her alphabet too slow a means of expression, dropped it and began to speak with great fluency. After talking with energy for a quarter of an hour, she suddenly recollected herself and took up her card of letters again, but the charm was broken, and by degrees she resumed her speech. At one time this lady was under the care of my friend Prof. Flint, for some chest or throat difficulty, and on one occasion spoke very well.

Neither does aphasia embrace cases of inability to speak from paralysis of the tongue or other muscles of articulation. Defective speech from this cause is frequently met with in hemiplegia, in glosso-laryngeal paralysis, and some other affections. In such instances the idea of language remains, but the patient does not speak because he is unable to put the organs of articulation in motion. A few days ago a gentleman, a prominent merchant of the city, was sent to me as a case of aphasia. As he entered my consulting-room, I saw that he was hemiplegic on the left side, and, on telling him to put out his tongue, found that he could not get it beyond the teeth, or touch the roof of his mouth with it. The history of the case was that of ordinary cerebral hæmorrhage, and he regained the power of speaking after several applications of the primary and induced gal-

vanic currents had been made to the tongue and muscles of the face.

The distinction between aphonia and aphasia must also be made. In the one the idea of speech is undisturbed, and articulation is not interfered with except as regards phonation. Aphonic patients can whisper, but are unable to speak in full voice, owing to some laryngeal affection impairing the tone of the vocal cords.

The fact that the faculty of speech may be deranged independently either of the will, paralysis, or loss of voice, appears to have been noticed at a very early period in the progress of science. Thus Isaiah[1] says, "For with stammering lips and another tongue will he speak to this people;" and again,[2] "Thou shall not see a fierce people, a people of a deeper speech than thou canst perceive; of a stammering tongue that thou canst not understand."

Thucydides mentions that many, who suffered from the plague which raged at Athens, found on recovering that they had not only forgotten the names of their friends and relations, but also their own names.

Pliny,[3] in the chapter entitled *Memoriæ Exempla*, says, in speaking of this faculty: "For nothing is so weak in man; disease, falls, injuries, even a fright, may impair it partially, or destroy it altogether. A blow from a stone has abolished the memory of the alphabet. A fall from a high roof has caused a man to cease to recognize his mother and neighbors, another even forgot his slaves, and Messala Corvinus, the orator, could not recall his own name."[4]

Suetonius[5] relates that Claudius so far lost his memory that he forgot the names of persons to whom he desired to speak, and could not even recollect the words he wished to use.

[1] Chapter xxviii. 11. [2] Chapter xxxiii. 19. [3] Lib. vii., cap. xxiv.

[4] Trousseau has translated this passage somewhat differently. I quote from an illuminated copy printed at Tarvisium (Treviso), in October, 1479.

[5] C. Suetonii Tranquilli, xii., Cæsares.

Passing over several authors of later times who have recognized the existence of the difficulty in question, we come to Crichton,[1] who remarks as follows: "There is a very singular defect of memory, of which I have myself seen two remarkable instances. It ought rather to be considered as a defect of that principle by which ideas and their proper expressions are associated, than of memory, for it consists in this, that the person, although he has a distinct notion of what he means to say, cannot pronounce the words which ought to characterize his thoughts. The first case of this kind which occurred to me in practice was that of an attorney much respected for his integrity and talents, but who had many sad failings to which our physical nature too often subjects us. Although nearly in his seventieth year, and married to an amiable lady much younger than himself, he kept a mistress, whom he was in the habit of visiting every evening. The arms of Venus are not wielded with impunity at the age of seventy. He was suddenly seized with great prostration of strength, giddiness, forgetfulness, insensibility to all concerns of life, and every symptom of approaching fatuity. His forgetfulness was of the kind alluded to. When he wished to ask for any thing, he constantly made use of some inappropriate term. Instead of asking for a piece of bread, he would probably ask for his boots; but, if these were brought, he knew they did not correspond with the idea he had of the thing he wished to have, and was therefore angry. Yet he would still demand some of his boots and shoes, meaning bread. If he wanted a tumbler to drink out of, it was a thousand to one he did not call for a certain chamber utensil, and, if it was the said utensil he wanted, he would call it a tumbler or a dish. He evidently was conscious that he pronounced wrong words, for, when the proper

[1] An Inquiry into the Nature and Origin of Mental Derangement, comprehending a Concise System of the Physiology and Pathology of the Human Mind, and a History of the Passions and their Effects. London, 1798, vol. i., p. 371.

expressions were spoken by another person, and he was asked if it were not such a thing he wanted, he always seemed aware of his mistake, and corrected himself by adopting the appropriate expression. This gentleman was cured of the complaint by large doses of valerian and other proper medicines."

Dr. Crichton subsequently met with another case similar to the foregoing, and he quotes the following from Prof. Gruner, of Jena, in vol. vii. of the *Psychological Magazine*. The patient, a learned gentleman, after his recovery from an acute fever, suffered a loss of memory for words. Among the first things he desired to have was coffee (*kaffee*), but, instead of pronouncing the letter *f*, he substituted in its place a *z*, and therefore asked for a cat (*kazze*). In every word which had an *f* he committed a similar mistake, substituting a *z* for it.

He also cites, from Van Goens, the case of Madame Hennert, wife of the professor of mathematics at Utrecht, who suffered a similar defect of memory. When she wished to ask for a chair she asked for a table, and when she wanted a book she demanded a glass. But, what was singular in her case was, that when the proper expression of her thought was mentioned to her, she could not pronounce it.

She was angry if people brought her the thing she had named instead of the thing she desired. Sometimes she herself discovered that she had given a wrong name to her thoughts. This complaint continued several months, after which she gradually recovered the right use of her recollection. It was only in this particular point that her memory seemed to be defective, for M. Van Goens assures us that she conducted her household affairs with as much regularity as she ever had done, and that she used to show her husband the situation of the heavens on a map with as much accuracy as when she was in perfect health.

The following case, in Gesner's *Endeckungen der Neues-*

ten Zeit in der Arzneigelehrheit, is likewise quoted by Crichton:

"A man, aged seventy, was seized, about the beginning of January, with a kind of cramp in the muscles of the mouth, accompanied with a sense of tickling all over the surface of the body, as if ants were creeping over it. On the 20th of the same month, after having experienced an attack of giddiness and confusion of ideas, a remarkable alteration of his speech was observed to have taken place. He articulated easily and fluently, but made use of strange words, which nobody understood. The number of these does not at present seem to be great, but they are frequently repeated. Some of them he seems to forget entirely, and then new ones are formed. When he speaks quick he sometimes pronounces numbers, and now and then he employs common words in their proper sense. He is conscious that he speaks nonsense. What he writes is equally faulty with what he speaks. He cannot write his name. The words he writes are those he speaks, and they are always written conformably to his manner of pronouncing them. He cannot read, and yet many external objects seem to awaken in him the idea of their presence."

Dr. Rush,[1] in the work the title of which is cited below, in chapter xii., which treats *of Derangement in the Memory*, refers so specifically to affections of the speech that I quote his language with some degree of fulness, and I do so with the less hesitation as his observations appear to have escaped notice, both in this country and in Europe. He says:

"1. There is an oblivion of names and vocables of all kinds.

"2. There is an oblivion of names and vocables, and a substitution of a word no ways related to them. Thus, I knew a gentleman afflicted with this disease, who, in calling for a knife, asked for a bushel of wheat.

[1] Medical Inquiries and Observations upon Diseases of the Mind. Fourth edition. Philadelphia, 1830, p. 274. The first edition was published in 1812.

"3. There is an oblivion of the names of substances in a vernacular language, and a facility of calling them by their proper names in a dead or foreign language. Of this, Wepfer relates three instances. They were all Germans, and yet they called the objects around them only by Latin names. Dr. Johnson, when dying, forgot the words of the Lord's prayer in English, but attempted to repeat them in Latin. Delirious persons, from this disease of the memory, often addresstheir physicians in Latin or in a foreign tongue,

"4. There is an oblivion of all foreign and acquired languages, and a recollection only of vernacular language. Dr. Scandella, an ingenious Italian, who visited this country a few years ago, was master of the Italian, French, and English languages. In the beginning of the yellow fever which terminated his life in the city of New York in the autumn of 1798, he spoke English only; in the middle of his disease he spoke French only; but on the day of his death he spoke only in the language of his native country.

"5. There is an oblivion of the *sound* of words, but not of the letters which compose them. I have heard of a clergyman in Newburyport, who, in conversing with his neighbors, made it a practice to spell every word that he employed to convey his ideas to them.

"6. There is an oblivion of the mode of spelling the most familiar words. I once met with it as a premonitory symptom of palsy. It occurs in old people, and extends to an inability, in some instances, to remember any more of their names than their initial letters. I once saw a will subscribed in this way by a man in the eightieth year of his age, who during his life always wrote a neat and legible hand.

. . . . . .

"9. There is an oblivion of names and ideas, but not of numbers. We had a citizen of Philadelphia many years ago, who, in consequence of a slight paralytic disease, forgot the names of all his friends, but could designate them cor-

rectly by mentioning their ages, with which he had previously made himself acquainted."

Dr. Rush remarks of these cases, that "there appears to be something like a palsy of the mind, *quoad* these specific objects."

Thus far there had been no attempt to define with precision the seat of the faculty of language, or even to establish its existence; but, in the early part of the nineteenth century, Dr. Gall, a German physician, announced that such a faculty did exist, and that it was seated in those convolutions of the brain which rest upon the posterior part of the supra-orbital plate, and that a large development of the organ was indicated by prominence and depression of the eyes. He was first led to believe in the existence of such an organ by observing that some of the scholars with whom, as a young man, he had to compete, excelled him in the ability to learn by heart, and he noticed that those thus endowed with great memory for words possessed prominent eyes. From these circumstances, he was gradually carried on to the foundation of his phrenological system.

In reality, however, Gall considered that there were two organs of language in each hemisphere—the one originating the idea of words, the other the talent for philology, and for acquiring the spirit of languages. The former organ he describes as lying on the posterior half of the supra-orbital plate, as before mentioned. It gives a talent for learning and recollecting words, and persons possessing it large recite long passages by heart after reading them once or twice. The other is placed on the middle of the supra-orbital plate, and when it is large the eyeball is not only rendered prominent but is depressed, causing the lower eyelid to assume the appearance of a bag or fold. Persons having this organ large have not only an excellent memory for words, but a particular talent for the study of languages, for criticism, and in general terms for all that has reference to literature.

Dr. Spurzheim, however, admits but one organ, lying transversely on the posterior portion of the supra-orbital plate, and this view is accepted by Combe and other distinguished phrenological authorities.[1]

In support of his theory that there is such an organ, Gall cites the case of a notary reported by Pinel.[2] The latter, in speaking of apoplexy, says this affection may be limited in its action to the words which are used to express ideas. In the case mentioned, the patient forgot, after an attack of apoplexy, his own name, that of his wife, those of his children and friends, although there was not the least paralysis of his tongue. He no longer knew how to read or write, and yet his memory as regarded other things was unimpaired.

Dr. Gall[3] refers also to the case of a soldier, sent to him by Baron Larrey, who was affected in a manner similar to that of the notary. It was not his tongue which was involved, for he was able to move it about in all directions, and to pronounce words, but he had lost the memory for words, although he recollected other things as well as ever.

I shall presently have occasion to refer to a still more interesting case, reported by Larrey, and one which appears to have escaped the notice of all writers on the subject of aphasia.

Spurzheim mentions the case of one Lereard, of Marseilles, who, having received a blow from a foil on the eyebrow (which one is not stated), lost the memory of proper names entirely. He sometimes even forgot the names of his intimate friends, and even of his father.

Gall, therefore, located the organ of language in a limited

[1] For a full account of the subject, the reader is referred to a System of Phrenology, by George Combe, Boston, 1834, or to Phrenology, etc., by J. S. Spurzheim, Boston, 1833.

[2] Traité Médico-Philosophique, sur l'aliénation mentale. Second edition. Paris, 1809, p. 90.

[3] Physiologie du cerveau, vol. iv., p. 84.

part of the anterior lobe of each hemisphere; but he adduced very little evidence to support his opinion, and hence his views did not meet with any thing like general acceptance. A number of cases, however, reported by Lallemand, Rostan, and others, support it, while several adduced by the same authors are opposed to it.

In 1825 Bouillaud,[1] who had collected a great number of cases of affections of the brain, was surprised to find how frequently the loss of speech coexisted with disease or injury of the anterior lobes. He also confirmed, what others before him had noticed, that the loss of the power of expressing ideas in articulate language was often the only evidence of a brain-affection.

He made one very important step in advance, and his views on this particular point are adopted—and often without credit—by the majority of the present writers on aphasia: he divided the faculty of speech into two distinct categories of phenomena:

1. The faculty of creating words as representatives of our ideas, and of recollecting them—internal speech.

2. The power of coördinating the movements necessary for the articulation of these words—external speech.

This classification forms the basis of the division of aphasia into the two varieties, the amnesic and the ataxic.

The cases which Bouillaud adduced in support of his theory were many of them in patients who exhibited no other symptoms than the loss of the power of articulate language. They preserved their intelligence, comprehended perfectly questions put to them, and knew the value of words; but, although there was no paralysis of either the tongue or the lips, they were unable to utter a word. At the post-mortem examination, the lesion was always found in the anterior lobes. Sixty-four cases formed the basis of

[1] Traité de l'encéphalite, Paris, 1825; and also, Recherches cliniques, propres à démontrer que la perte de la parole correspond à la lésion des lobules antérieurs du cerveau, Archives de Méd., 1825.

his conclusions. A part were direct, and went to show that lesion of the anterior lobes was accompanied by derangement in the faculty of speech; the other part were indirect, and established the fact that, when the anterior lobes were not affected, the lesion being in some other region of the brain, the faculty of speech remained intact.

Cruveilhier opposed Bouillaud's views, and, in a paper read at the Athénée de Médecine in the same year, brought forward seven cases of persons, some of whom had lost the faculty of speech, but who, on post-mortem examination, were found to have no disease of the anterior lobes; and others who had spoken, but in whom there were more or less profound changes in these parts.

Subsequently Andral[1] reported the results of the analysis of thirty-seven cases of lesion of one or both anterior lobes. Of these, speech was abolished twenty-one times, and preserved sixteen times. Lallemand[2] also opposed Bouillaud with several cases; but the latter rejoined[3] with a fresh array of thirteen cases in support of his doctrine, and with many arguments against the validity of those brought against him. Longet[4] declares that Bouillaud appears to have refuted many of the objections of his adversaries, and to have demonstrated that some of their cases were badly interpreted. At the same time, while admitting that it is possible that different parts of the brain preside over different voluntary movements, he affirms that there is nothing positively established as regards the localization of the active principles of these movements.

Subsequently, in other memoirs, Bouillaud brought forward additional cases in support of his theory, making a

[1] Clinique Médicale, t. ii., p. 135.

[2] Op. cit., lettres 6, 7, 8.

[3] Exposition de nouveaux faits à l'appui de l'opinion qui localise dans les lobes antérieurs du cerveau le principe législateur de la parole. Bulletin de l'Académie de Médecine, 1839, tom. iv., p. 282.

[4] Traité de la Physiologie, t. ii., p. 438.

total of one hundred and three, and offered a prize of five hundred francs to any one who would adduce an instance of profound lesion of the anterior lobes without troubles of speech. Many years subsequently Velpeau announced that he should claim this prize, for that, in March, 1843, he had related the case, and presented the brain, of a wig-maker who had come under his care for prostatic disease. This man was in full possession of his reasoning faculties, and, moreover, was noted for his unconquerable loquacity. He died a few days subsequently, and on post-mortem examination a scirrhous tumor was found to have entirely taken the place of the two anterior lobes of the brain. Very little faith seems to have been put by physiologists or pathologists in the history of this case. If it proves any thing, it is that the anterior lobes are useless appendages to the rest of the cerebral system.

But Bouillaud was not content with the deductions to be drawn from pathology. In a series of experiments, he endeavored to establish the truth of his idea, and thus bring the science of physiology to his support. These experiments were detailed in a paper[1] read before the Academy of Sciences, in September, 1827, which was subsequently (1830) published in the tenth volume of Magendie's *Journal de Physiologie*, from which I quote.

The experiments relative to the anterior lobes were made on dogs. Only one was entirely successful—the animals in the others dying too soon after to admit of satisfactory deductions being made. But the twentieth experiment was more satisfactory.

On the 28th of June, 1826, he passed a gimlet through the anterior part of the brain of an active, docile, and intelligent dog. Immediately afterward the animal was convulsed, and could not rise from the ground. Sight and hearing remained. Symptoms of compression soon came on;

[1] Recherches expérimentales sur les fonctions du cerveau (lobes cerebraux) en général et sur celles de sa portion antérieure en particulier.

the result, probably, of the hæmorrhage. Eventually, the animal recovered, but it was found to have lost much of its intelligence and agility. The faculty of memory seemed to have been entirely abolished, and there was a decided expression of imbecility in its countenance. It could no longer ascend or descend a staircase; the fore-legs were lifted very high in walking, and its movements were all badly coördinated. When struck or made to walk, it uttered sharp cries, but it had lost entirely the ability to bark. As Bouillaud remarks, "it no longer barked, either to show its affection, or to drive away strangers who came to the house." Once only, on the 18th of July, it tried to bark at a passer-by, but failed in the attempt.

This is the only experiment I have been able to find which has any bearing upon the question of the localization of the faculty of language. And I do not quote it as proving much on the subject. The difficulties in the way of experimentation are almost insuperable, to say nothing of the fact that it is doubtful if any of the sounds made by animals can be compared with human speech.

But unintentional experiments have been performed upon the human subject, which tend to show that, though the faculty of language may be located in one or both anterior lobes, either may be seriously injured without the faculty of language suffering to any appreciable extent. Two of them have happened in this country, and, although referred to in connection with aphasia by Seguin and Harris, I take great satisfaction in bringing them forward on account of their great importance to the question under consideration.

The first is related by Dr. Harlow,[1] of Vermont:

The subject was a strong, healthy man, twenty-five years of age, and was engaged in ramming down a charge of

[1] Boston Medical and Surgical Journal, December, 1849, vol. xxxix., p. 389. Also Descriptive Catalogue of the Warren Anatomical Museum. Boston, 1870, p. 145.

powder in a rock to be blasted, when an explosion took place, and the tamping-iron was driven clear through his head.

In a few minutes he recovered his consciousness, was put into a cart and carried three-quarters of a mile to his residence, where he got out and walked into the house. Two hours afterward he was seen by Dr. Harlow. He was then quite conscious and collected in his mind, but exhausted by extensive hæmorrhage from the hole in the top of his head. Blood, pus, and particles of brain, continued to be discharged for several days, but by January 1, 1849, the wound was quite closed and his recovery complete. There was no pain in the head, but a queer feeling, which he could not describe. As regarded his mind, he was fitful and vacillating, though obstinate, as he had always been. He became very profane, never having been so before the accident. He lived till May 21, 1861, twelve and a half years subsequent to the accident, when he died, after having had several convulsions. His cranium was obtained, and, with the bar, is now preserved in the Warren Anatomical Museum at Boston. Dr. J. B. S. Jackson[1] thus describes the skull:

"The whole of the small wing of the sphenoid bone upon the left side is gone, with a large portion of the large wing, and a large portion of the orbital process of the frontal bone, leaving an opening in the base of the skull two inches in length, one inch in width posteriorly, and tapering gradually and irregularly to a point anteriorly. This opening extends from the sphenoidal fissure to the situation of the frontal sinus, and its centre is an inch from the median line. The optic foramen and the foramen rotundum are intact. Below the base of the skull the whole posterior portion of the upper maxillary bone is gone. The malar bone is uninjured; but it has been very perceptibly forced outward, and the external surface inclines somewhat outward from above downward. The lower jaw is also uninjured. The

[1] Descriptive Catalogue of Warren Anatomical Museum, loc. cit.

opening in the base, above described, is continuous with a line of old and united fracture that extends through the supra-orbitary ridge in the situation of the foramen, inclines toward and then from the median line, and terminates in an extensive fracture that was caused by the bar as it came out through the top of the head. This fracture is situated in the left half of the frontal bone, but inferiorly it extends somewhat over the median line. In form it is about quadrilateral; but it measures two and a half by one and three-quarter inches. Two large pieces of bone are seen to have been detached and upraised, the upper one having been separated at the coronal suture from the parietal bone, and being so closely united that the fracture does not show upon the outer surface. The lower piece shows the line of fracture all around. Owing to the loss of bone, two openings are left in the skull; one that separates the two fragments has nearly a triangular form, extends rather across the median line, and is four inches in circumference; the other, situated between the lower fragment and the left half of the frontal bone, is long and irregularly narrow, and is two and five-eighths inches in circumference. The edges of the fractured bones are smooth, and there is nowhere any new deposit."

From this account it will be seen that the left anterior lobe of the brain suffered severely by this terrible injury, and yet it is not stated that the subject had ever shown any difficulties of speech. If the faculty of language resides in the whole of the lobe, such an immunity could scarcely have existed. It must be noted, however, and the photograph of the cranium establishes the fact, that the third frontal convolution and the island of Reil escaped all injury. Another interesting circumstance is the addiction to profanity after the accident. A like phenomenon has been noticed in cases of aphasia.

The second instance is almost as extraordinary. I quote the history of the case, 952, from Dr. Jackson:[1]

[1] Op. cit., p. 149

"Cast of the head of a man who was transfixed through the head by an iron gas-pipe, and who, to a very considerable extent, recovered from the accident.

"The patient, a healthy and intelligent man, about twenty-seven years of age, was blasting coal when the charge exploded unexpectedly, and the pipe was driven through his head, entering at the junction of the middle and outer thirds of the right supra-orbitary ridge, and emerging near the junction of the left parietal, occipital, and temporal bones. One of his fellow-miners saw him upon his hands and knees, and struggling as if to rise; and, going to his assistance, he placed his knee upon his chest, supported his head with one hand and with the other withdrew the pipe. This last projected about equally from the front and back of the head, and much force was required for its withdrawal."

Brain escaped from the anterior opening, and coma and collapse supervened. "In seven weeks he sat up, and in one more walked about. The right hand he used somewhat, but less well than the left. For about ten months after the accident his memory for some things was nearly lost, but during the next two months there was a considerable improvement."

The accident happened on May 14, 1867, and in June, 1868, the patient, with the gas-pipe, was exhibited to the Massachusetts Medical Society. "The man appeared to be in a good state of general health; and, though his mental powers were considerably impaired, there was nothing unusual in his expression, nor would there be noticed, in a few minutes' conversation with him, any marked deficiency of intellect."

It is very evident that in this case the right anterior lobe was seriously injured—the left escaping—and yet there does not appear to have been any aberration of speech. It is to be regretted, however, that the history is not more specific as to the things in regard to which the memory was deficient.

There are other cases which militate against Bouillaud's doctrine. Thus, M. Peter[1] states that a drunken cavalry-soldier fell from his horse on the back of his head, and fractured his skull. Stupor set in at once, followed by the most violent delirium. The man kept constantly shouting the worst possible oaths, and held connected conversations with imaginary persons. He died at the end of thirty-six hours, without having recovered his reason. On dissection, a fracture of the roof and base of the skull was found in all its length. The posterior lobes of the brain were found, on post-mortem examination, to have sustained no injury, but both anterior lobes were in a pulpy condition, through a most violent contusion, caused by their being knocked against the anterior wall of the cranium. The whole thickness of the lobes was disorganized. As Trousseau remarks, this case shows that the two frontal lobes may be destroyed in their anterior portion without causing a loss of the faculty of speech. Trousseau also cites the case of two officers, who, after a quarrel, fought a duel. One of them fired first, and the ball entered his adversary's head at one temple, passed through the brain, and then raised the temporal bone on the opposite side. The ball was extracted, and the patient immediately made a sign with his hands, and expressed his thanks in a very low voice. He recovered, for the time being, and, during five months thereafter, could speak perfectly well, and was remarkable for the wit and fluency of his conversation and writing. He subsequently died of softening; and it was found, on post-mortem examination, that the ball had passed through the two frontal lobes in their middle portion. A still more striking case is referred to by Dr. Bazire, in a note to Trousseau's lecture on aphasia, in the work cited. It was reported in 1843 by M. Aug. Bérard, to the Anatomical Society of Paris. The patient, a miner, was knocked down and severely injured by

[1] Quoted by Trousseau, Lectures on Clinical Medicine. Translated by Bazire, vol. i., p. 256.

an explosion in a mine. He did not lose consciousness, but managed to creep out of his hole and to call to his help some men who were working a short distance off. He begged them to fetch a cart and to take him to M. Bérard's house. He was there examined. The whole frontal region was laid open, the integuments hung in shreds, the bones were splintered and in detached fragments, and the brain was exposed. Both anterior cerebral lobes were completely destroyed, and in their stead was a mixture of blood, of bony splinters, and brain-substance. In spite of this frightful injury the man could relate in all its details how the accident had occurred. He died the next day.

Whether or not we accept this case in all the import claimed for it, there can be no doubt that Bouillaud is wrong in claiming that injury of the anterior lobes is necessarily followed by some derangement in the faculty of speech. It is only fair, however, to state that latterly he has admitted that the organ of language may occupy the posterior part of either lobe.

Dr. M. Dax, in 1836, read a paper before the medical congress which met that year at Montpellier, in which he came to the conclusion that the faculty of language "was seated, not as Gall and Bouillaud had contended, in both anterior lobes of the brain, but that it occupied only the left anterior lobe." He based this opinion on one hundred and forty cases of aphasia attended with paralysis, and in which the loss of power was on the right side; showing, therefore, that the lesion which produced the aberration of speech also caused the hemiplegia, and that this lesion must have been on the left side. This paper at the time attracted very little attention, and was forgotten till the year 1861 witnessed the reopening of the discussion.[1]

It would be very easy to quote a large number of cases confirmatory of Dr. Dax's doctrine, but a few will suffice to

[1] Dr. Marc Dax's Memoir was republished in the Gazette Hebdomadaire, No. 17, April, 1865.

show the general bearing of a great many others. The following case seems to have escaped notice. It is not the one referred to by Gall as being sent to him by Larrey. In that case the left anterior lobe was injured and there was aphasia, but the lesion was caused by a sword.

Baron Larrey[1] presented to the Academy the cranium of a subject, with the following history:

Toward the end of the year 1815 an officer of dragoons came to the hospital with a wound from a ball which he had received at Waterloo. The missile had entered the left side of the cranium at a point about six or eight millimetres from the eyebrow and near the temporal ridge. At first he had suffered loss of consciousness and profuse hæmorrhage, but had recovered, with but slight loss of motor power. So far as his mind was concerned, there was no derangement except as regarded the faculty of speech; he had lost the memory of substantives. For this reason he was unable to drill his company, and, though able to distinguish his men by their size, their form, their complexion, or their voice, he could not call them by name. He refused to allow the operation of trephining to be performed, and in 1827 died of phthisis.

A post-mortem examination was made. The ball was found embedded in the thickness of the bone, having elevated and fractured the internal table. The dura mater was strongly adherent to the whole of the left anterior cranial fossa; it was also thicker and denser than in the natural state. A spheroidal excavation, five centimetres in its horizontal and seven or eight in its vertical diameter, was discovered at the summit and on the temporal side of the left anterior lobe of the brain.

Mr. Thomas Hood[2] reported the history of a patient, a

[1] Blessure du Cerveau avec perte de Mémoire des Noms Substantives. Journal de Physiologie de Majendie, t. viii., 1828, p. 1.

[2] Phrenological Transactions. Quoted by George Combe in his System of Phrenology, Boston, 1834, p. 429.

sober, intelligent man, sixty years of age, who, on the evening of September 2, 1822, suddenly began to speak incoherently, and became quite unintelligible to those around him. It was discovered that he had forgotten the name of every object in Nature. His recollection of things seemed to be unimpaired, but the names by which men and things were known were entirely obliterated from his mind, or rather he had lost the faculty by which they were called up at the control of the will. He was by no means inattentive, however, to what was going on, and he recognized friends and acquaintances perhaps as quickly as on any former occasion; but their names, or even his own or his wife's name, or the names of any of his domestics, appeared to have no place in his recollection.

"On the morning of the 4th of September," says Mr. Hood, "much against the wishes of his family, he put on his clothes and went out to the workshop, and when I made my visit he gave me to understand, by a variety of signs, that he was perfectly well in every respect, with the exception of some slight sensations referable to the eyes and eyebrows. I prevailed on him with some difficulty to submit to the reapplication of leeches, and to allow a blister to be placed over the left temple. He was now so well in bodily health that he would not be confined to the house, and his judgment, in so far as I could form an estimate of it, was unimpaired, but his memory of words was so much a blank, that the monosyllables of affirmation and negation seemed to be the only two words in the language the use and signification of which he never entirely forgot. He comprehended distinctly every word which was spoken or addressed to him; and, though he had ideas adequate to form a full reply, the words by which these ideas are expressed seemed to have been entirely obliterated from his mind. By way of experiment I would sometimes mention to him the name of a person or thing, his own name for example, or the name of some one of his domestics, when he would repeat it

after me distinctly once or twice; but generally before he could do so a third time the word was gone from him as completely as if he had never heard it pronounced. When any person read to him from a book, he had no difficulty in perceiving the meaning of the passage, but he could not himself then read, and the reason seemed to be that he had forgotten the elements of written language, viz., the names of the letters of the alphabet. In the course of a short time he became very expert in the use of signs, and his convalescence was marked by his imperceptibly acquiring some general terms which were with him, at first, of very extensive and varied application. In the progress of his recovery, time and space came both under the general application of time. All future events and objects before him were, as he expressed it, '*next time*;' but past events and objects behind him were designated '*last time*.' One day, being asked his age, he made me to understand that he could not tell; but, pointing to his wife, uttered the words, '*many times*,' repeatedly, as much as to say that he had often told her his age. When she answered sixty, he answered in the affirmative."

On the 10th of January he suddenly became paralytic on the left side [this is evidently a typographical error for right side]. On the 17th of August he had an attack of apoplexy, and on the 21st he expired. In the *Phrenological Journal*, vol. iii., p. 28, Mr. Hood has reported the dissection of his brain: "In the left hemisphere, lesion of the parts was found, which terminated at half an inch from the surface of the brain, where it rests on the middle of the supra-orbital plate." Two small depressions or cysts were found in the substance of the brain, "and the cavity considered as a whole expanded from the anterior part of the brain till it opened into the ventricle in the form of a trumpet. The right hemisphere did not present any remarkable appearance."

Dr. Thomas Hun,[1] of Albany, in detailing a case of amnesia in which there were no symptoms of paralysis, and in which there was no post-mortem examination, cites the case of a lady who died of cancer of the brain, occupying, at the time of her death, the greater portion of the left anterior lobe. In the early stages of her disease she was often unable to call the most familiar objects by name, and had to express herself by signs or by pointing at the object. When the word she wanted was pronounced before her, she recognized it, and was able to repeat it.

Other cases, and especially several which have occurred in my own experience, are reserved for future consideration.

Up to this period we have the organ of articulate language limited to the left anterior lobe of the brain, but in 1861 its location was still further restricted. In that year M. Gratiolet, in discussing before the Anthropological Society of Paris a question relative to the comparative development of the brain and mind among different races, brought up the subject of cerebral localization, to which he announced himself as being strongly opposed. M. Auburtin, on the contrary, contended that the localization of the faculty of speech at least was definitely established, through the researches of Bouillaud, in the anterior lobes. In support of this view, he adduced cases which had already been brought forward, and cited others in addition, which went to show that loss of speech was the consequence of traumatic lesion of these parts of the brain. His adversaries cited other cases in which persons had preserved the faculty of language notwithstanding extensive lesions of the anterior lobes. M. Auburtin responded that, if such profound and extensive injuries had not interfered with speech, it was because that part of the lobes in which the organ is situated was not involved. And he then cited the case of a patient in the Hospital for Incurables, who for many years had been deprived of the power of speech, and he declared that he would re-

[1] American Journal of Insanity, vol. vii., 1850–'51, p. 359.

nounce the doctrine of Bouillaud if the autopsy of this patient did not reveal disease of the anterior lobes. The patient in question was under the charge of M. Broca, and the latter, a decided opponent, accepted the challenge of M. Auburtin, and declared that, when the man died, the examination should be made.

Some time afterward the patient died, the post-mortem examination was made, and the lesion was found to occupy the left anterior lobe.[1]

From this time forward, M. Broca, who had been a most determined opponent of Bouillaud's views of localization, became converted, and carried them to a still more extreme point than even M. Marc Dax had done. Taking, as his principal case, the one to which M. Auburtin had pinned his faith, he read, in 1861, before the Anatomical Society of Paris, a memoir,[2] in which he discusses the question of the location of the faculty in question with all his perspicuity and directness. As the two cases cited by him are of historical interest, I give the chief details of them:

A man named Le Borgne, who had been an inmate of another department of Bicêtre for over twenty years, was transferred to one of the wards under M. Broca's care, to be treated for a severe attack of phlegmonous erysipelas. The man was a confirmed epileptic, and had not spoken, since his entrance into the hospital, more than a few words, which he employed for the expression of all his ideas. It is stated that in other respects his intelligence was good. Le Borgne was known in the hospital by the name of "Tan," a word which he habitually used, and which, with the oath, "*Sacré nom de Dieu*," constituted his entire vocabulary. "Tan," owing to the constancy with which he used it, was the name by which he was known in the hospital; and, when

[1] See Étude sur la localisation de la Faculté du Langage Articulé. Thèse de Paris de M. Carrier, 1867.

[2] Sur le siége de la faculté de langage articulé avec deux observations d'aphemie. Bull. de la Soc. Anatomique, t. iv., 1861.

he could not make himself understood by his signs, he employed the oath, and gave other manifestations of anger.

For several years he had remained in the hospital with no other lesion than that of speech, with an occasional epileptic paroxysm; but, after a few years, his right arm became paralyzed, and four years subsequently the leg of the same side was involved; his sight was likewise enfeebled, and for the past seven years he had been entirely confined to his bed.

Notwithstanding the fact that he was almost in a dying condition when M. Broca first saw him, some important points in his cerebral difficulty were noted. To any question put to him, he replied, as usual, "*Tan*," but at the same time endeavored to make himself understood by signs. Thus he raised six fingers to indicate that six days had elapsed since the inception of his erysipelas, and by opening and shutting his hand four times and then raising one finger signified that he had been twenty-one years in Bicêtre.

Sensibility was lessened on the affected side; there was no deviation of the tongue, which could be moved freely in all directions, and no paralysis of the face beyond a slight weakness shown by the swelling of the left side when he breathed; there was a little difficulty of swallowing, from the fact that the muscles of the pharynx were gradually becoming implicated.

After a few days the man died.

As I have said, the autopsy showed that the lesion was situated in the left anterior lobe. More exactly, however, it should now be stated that it involved the inferior marginal convolution of the temporo-sphenoidal lobe, the convolutions of the island of Reil, and in the frontal lobe, the frontal transverse convolution, and the posterior half of the second and third frontal convolutions. The left corpus striatum was also affected. According to Broca, the disease had in all probability begun in the third frontal convolution, and had gradually extended to the other parts; the paraly-

sis marking the implication of the island of Reil and the corpus striatum.

The other case was that of a man named Le Long, aged eighty-four years, who had entered the hospital for a fracture of the neck of the femur. Eighteen months before, he had been treated in the medical service for a temporary apoplexy, which had deprived him of the faculty of speech, but had caused no paralysis. Le Long, whose intelligence, facial expression, and ability to gesticulate, were very striking, made himself perfectly well understood, although able to pronounce indistinctly a very few words, but which were nevertheless properly applied. These words were "*oui*," "*non*, *toujours*, *tois*" for *trois*, and *Lelo* for *Le Long*. Thus, when asked, "Can you write?" he answered, "Oui." "Have you any children?" "Oui." "How many? "Tois," but at the same time, as if aware that he was not answering correctly, he raised four fingers. "How many boys?" "Tois," raising two fingers. "How many girls?" "Tois," holding up two fingers. "What time is it by this watch?" "Tois," at the same time raising ten fingers to signify that it was ten o'clock. "How old are you?" To this question he replied by two gestures; the one consisting of raising eight fingers, the other of four fingers, by which he meant that he was eighty-four years old.

Aside from this application of the word *tois* to all numbers, his answers were perfectly correct. The tongue was neither paralyzed nor thickened; on one side the larynx was mobile, and his limbs possessed their normal power for his age. It was therefore a case of pure aphasia, or, as Broca then designated the affection, aphemia.

Twelve days after the accident, the patient died. The post-mortem examination revealed the existence of lesions, almost identical in situation with those of the former case. The posterior part of the third left frontal convolution, and the contiguous part of the second, had been absorbed and replaced by a serous fluid. Two cases can scarcely decide

any point in pathology; but, without venturing to assert positively that the organ of language resides exclusively in the posterior part of the third frontal convolution, M. Broca expressed the opinion that the integrity of this convolution, and perhaps of the second, is indispensable to the normal operation of the function of speech.

Many cases were adduced by Charcot,[1] by Falret,[2] by Perroud[3] of Lyons, by Trousseau,[4] and others, in support of the localization of the faculty of articulate language in the left side of the brain. Most of these cases were accompanied by right hemiplegia, and, in several, post-mortem examinations showed the lesion to exist in the parts designated by Broca.

In the early part of 1863, M. G. Dax, son of the M. Dax who had placed the organ of language in the left hemisphere, presented, through M. Lelut, a memoir to the Academy, in which he claimed with his father that aphasia was always the result of lesion of the left hemisphere, but he assigned a still more restricted position, by limiting it to the anterior and exterior part of the middle lobe. He cited forty cases of loss of the power of speech, coincident with lesion of the left hemisphere.

Now, besides these direct cases, there are others which bear with almost as much effect on the affirmative of the doctrine in question. Thus M. Fernet, in 1863, presented a case to the Société de Biologie, in which there was left hemiplegia, but no aphasia. After death, softening of the right hemisphere, from thrombosis of the right middle cerebral artery, was found to exist. M. Parrot[5] adduced another case in which there was complete atrophy of the island of Reil, and of the third convolution of the right side, but in which there was no trouble of speech. These cases go to

[1] Gazette Hebdom., 1863, pp. 473, 525.

[2] Archives de Méd., t. iv., Mars et Mai, 1864.

[3] Journal de Méd. de Lyon. Jan. et Fév., 1864.

[4] Clinique Médicale.

[5] Gazette Hebdom., 1863, p. 506.

show that the organ of articulate language is not situated in the right hemisphere.

M. Lesur [1] has reported a case which is of very great interest. A child was kicked on the head by a horse, and a fracture of the frontal bone was thus produced. The operation of trephining was performed at a point about an inch and a quarter above the left eye. After the operation and during the progress of the case, it was observed that, whenever pressure was made upon the brain through the hole in the cranium, the child lost the power of speech, and that when this pressure was removed she regained it. A similar case occured several years ago in my own practice.

Among British writers, Dr. Hughlings Jackson [2] has given the histories of thirty-four cases of loss of speech coinciding with right hemiplegia. He is entitled to the credit of making a beautiful application of anatomy and physiology to the pathology of the subject under consideration. The part of the brain designated by Broca as the seat of the organ of articulate language is nourished by the left middle cerebral artery. An obstruction of this artery would of course interfere with the perfect action of that region, and thus aberrations of speech would be produced. But the same artery also supplies blood to the corpus striatum of the same side. Hence the frequency with which aphasia is associated with right hemiplegia. The cause of the obstruction is generally, according to Dr. Jackson, embolism, for in twenty of his cases the heart was more or less affected, and in thirteen of them there was valvular disease.

Among other British writers, some of whom will be more fully referred to hereafter, must be mentioned, Dr. Sanders,[3] Dr. Moxon,[4] Dr. Ogle,[5] Dr. Bateman,[6] and Dr. Bastian.[7]

[1] Gazette des Hôpitaux. [2] London Hospital Reports, vol. i.

[3] Edinburgh Medical Journal, August, 1866.

[4] British and Foreign Medico-Chirurgical Review, April, 1866.

[5] St. George's Hospital Reports, vol. ii., 1867.

[6] Journal of Mental Science, January, 1868, and subsequent numbers.

[7] British and Foreign Medico-Chirurgical Review, January and April, 1869.

The matter does not appear to have attracted much attention from German physiologists and pathologists, since the discussion in the French Academy in 1861. Previous to that period several excellent memoirs upon the physiology of speech were published by Germans, among which that of Dr. Bergman[1] is preëminent. A memoir by Nasse[2] is also interesting.

In 1865 Von Benedict, and Braunwart[3] published a very thorough paper on the subject, and other observers have reported cases.

In this country there have been several very excellent memoirs upon aphasia, and, as we have already seen, the subject early attracted attention, and the fact that such a condition could exist without other manifest symptoms was fully recognized. Thus, Prof. A. Flint[4] detailed the histories of six cases, in one of which post-mortem examination showed extensive disease of the left anterior lobe, and in four, in which the situation of the hemiplegia was noted, the right was the affected side.

Dr. H. B. Wilbur,[5] in a memoir on aphasia, treats of the aberrations of the faculty of language as they existed in certain idiots under his observation. His cases, though interesting, are scarcely in point, as the difficulties of speech were clearly the result of mental deficiencies.

A very important memoir is that of Dr. E. C. Seguin,[6] in which a very excellent history of the subject is given, with the citation of forty-eight cases from the records of the New York Hospital, in which there were difficulties of speech coexisting with hemiplegia, and two in which there was no hemiplegia. In several of these cases, however,

[1] Einige Bemerkungen über Störungen des Gedächtniss und der Sprache. Allgemeine Zeitschrift für Psychiatrie, 1849, s. 657.

[2] All. Zeitschrift, u. s. w., 1853, s. 523.

[3] Canstatt's Jahresbericht, 1865, s. 31.

[4] Medical Record (New York), March 1, 1866.

[5] American Journal of Insanity, July, 1867.

[6] Quarterly Journal of Psychological Medicine, etc., January, 1868.

as Dr. Seguin states, the loss of the faculty of speech was due to paralysis of the tongue and other muscles concerned in articulation.

Another excellent paper is by Dr. T. W. Fisher,[1] of Boston. Dr. Fisher has studied the subject very philosophically, and records thirty-eight cases in which post-mortem examinations were made with definite results. Cases have also been published by Bartholow[2] and others.

With this outline statement of the history of the subject of aphasia, we are in a position to inquire more fully into the evidence which locates the organ of language in a particular region of the brain.

A clear idea of the anatomy of the parts fixed upon latterly as the seat of the faculty will aid in the understanding of the subject.

The following account is condensed by Dr. Bateman[3] from Broca's description in his essay "Sur le Siége de la Faculté du Langage articulé:"

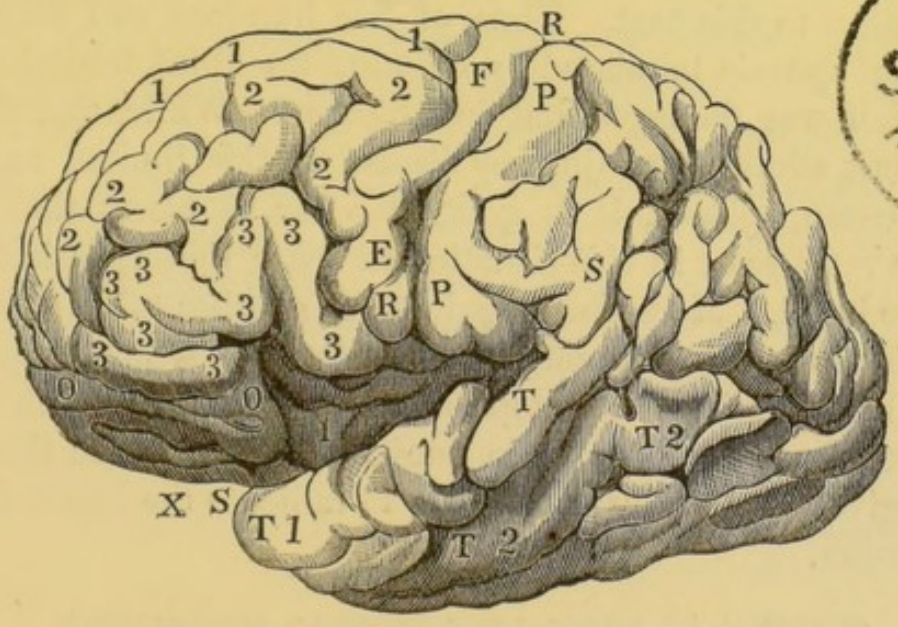
"The anterior lobe of the brain comprises all that part of the hemisphere situated above the fissure of Sylvius, which separates it from the temporo-sphenoidal lobe and in front of the furrow of Rolando which divides it from the parietal lobe. The furrow of Rolando separates the frontal from the parietal lobe; it traverses from above downward all the external surface of the cerebral hemisphere, starting from the inter-hemispheric median fissure, and ending at the fissure of Sylvius. In front, this furrow is bounded by the transverse frontal convolution, and behind by the transverse parietal convolution. The anterior lobe is composed of two stories or divisions—one inferior or orbital, the other superior—situated beneath the frontal and under the most anterior part of the parietal. This superior division of the anterior lobe is composed of four fundamental convolutions;

[1] Boston Medical and Surgical Journal, September 1, 1870, and subsequent numbers.

[2] Medical Repertory, Cincinnati, January, 1869. [3] Op. cit., p. 522.

one posterior, the others anterior. The posterior is that which has been described as the *transverse frontal*, and which forms the anterior border of the furrow of Rolando; the three other convolutions have all an antero-posterior direction, and are distinguished by the names of *superior or first frontal*, *middle or second*, *and inferior or third frontal* convolutions. This last, by its posterior half, forms the superior border of the fissure of Sylvius, the inferior border being formed by the superior convolution of the temporo-sphenoidal lobe. In drawing asunder these two convolutions which bound the fissure of Sylvius, the lobe of the insula (the island of Reil) is exposed, which covers

FIG. 6.



LEEDS SCHOOL OF MEDICINE

FROM BROCA, AS MODIFIED BY DR. HUGHLINGS JACKSON.

1. First Frontal Convolution; 2. Second Frontal Convolution; 3. Third Frontal Convolution; O. Orbital Convolutions; E F. Transverse Frontal Convolution; P. Parietal Lobe; T S. Temporo-sphenoidal Lobe; T 1. First Temporo-sphenoidal Convolution; T 2. Second Temporo-sphenoidal Convolution; I. Island of Reil; R R. Furrow of Rolando; S. Fissure of Sylvius.

the extra ventricular nucleus of the corpus striatum. The result of these relations is that a lesion, which is propagated from the frontal to the temporo-sphenoidal lobe, or *vice versa*, will pass almost necessarily by the lobe of the insula, and thence, in all probability, it will extend to the extra

ventricular nucleus of the corpus striatum, seeing that the proper substance of the insula, which separates the nucleus from the surface of the brain, is composed only of a very thin layer."

The lobe of the insula, or the island of Reil, is found in no other mammal than man and the monkey. In the latter, however, it is very slightly developed, and has no trace of convolutions. In aberrations of speech this part is very often involved in the lesion.

Now, although there are several cases on record in which post-mortem examination would appear to show that lesion of the third left frontal convolution is sufficient to produce derangement of the faculty of articulate language, the weight of evidence is decidedly against limiting the seat of the organ to this part. Thus, of five hundred and fifty-six cases of aphasia tabulated by Seguin,[1] the third frontal convolution was damaged but in nineteen. While, therefore, we must admit that injury or disease of this limited region will cause aphasia, it is going too far to assert that the lesion must exist in this situation in order that aphasia may be produced. Moreover, Seguin gives another table of cases which must definitely settle the matter, and which I quote in full. It relates to autopsies which were made with special reference to the point in question, and in which the details given were sufficient clearly to indicate the location of the lesion.

QUESTION OF THIRD LEFT FRONTAL CONVOLUTION.

AUTHORITIES.	For.	Against.
Trousseau, 1865 (in Acad. de Méd.)	14	18
Peter, Legrand, Béclard, Delpech, Bérard, Farge, Jackson, Bigelow	..	8
Jackson, Richardson, Russel	3	..
New York Hospital, 1830–1867	1	7
Bellevue Hospital, October, 1867	..	1
Total	18	34

[1] Op. cit., p. 97.

Other cases might readily be adduced, but the above are amply sufficient to decide the question against Broca's doctrine. One case of aphasia occurring without lesion of the third frontal convolution would of course invalidate his claim that this part is the exclusive seat of the organ of language, and no number of cases showing coexistence of aphasia with disease or injury of the third left frontal convolution would be sufficient to establish the point affirmatively with the results of our present experience disproved. Nevertheless, as showing further that disease of this part will cause aphasia, I subjoin the following case from Dr. W. Ogle's[1] very interesting memoir:

"Joel B., October 18, 1866. Had rheumatic fever and endocarditis twenty-five years ago, but since that has had good health. While at work, October 15th, fell down suddenly without losing consciousness, and found that he was speechless, and hemiplegic on the right side.

"On admission he was found to have extensive heart-disease, with the pulse characteristics of aortic regurgitation. There was complete lax palsy of the right arm and leg, with unimpaired sensibility. There was at first some difficulty in deglutition and in protruding the tongue, but this latter symptom passed away in a few days. There was slight pain in the left side of the head.

"His speech was limited to the two words 'yes' and 'no.' These he used correctly. After he had been in the hospital some time, he recovered the power of saying some few words, chiefly monosyllables.

"He could write with his left hand, with sufficient distinctness, words which he could not pronounce when asked to do so. In his writing there was often a tendency to reduplication of letters. For instance, he wrote 'Testatament' for 'Testament.' But I cannot say whether this was more than the result of deficient education.

[1] Aphasia and Agraphia. St. George's Hospital Reports, vol. ii., 1867, p. 105.

"His mind seemed quite clear. He understood all that was said to him; took interest in all that was going on about him; listened to conversation with an animated, lively look, laughing at any little joke, and expressing himself frequently by suitable pantomime. In December he was attacked by œdema of the lungs, and died on the 20th.

Post-mortem; œdematous lungs, extensive aortic and mitral disease. "Much semigelatinous fluid in subarachnoid space. Surface of brain healthy, excepting at one limited spot. This was the posterior part of the third frontal convolution on the left side. Here was a softened, almost diffluent patch about three-quarters of an inch in breadth, reaching from the highest point of the third convolution backward and downward to the fissure of Sylvius. The softened patch was not actually the most posterior part of the convolution, for there was a narrow unsoftened strip between it and the transverse frontal convolution. In cutting into the brain, a second small patch of softening was seen in the centre of the left hemisphere, external to and rather above the corpus striatum, and extending toward the posterior termination of the fissure of Sylvius. All the rest of the brain was apparently healthy.

"The left middle cerebral artery was firm in its main trunk, but in one of its secondary branches at a bifurcation was a hard shotty bit of fibrine completely obstructing the passage, so that when water was injected into the vessel it could not pass, though considerable force was used. There were also fibrinous blocks in the spleen."

The theory of M. Marc Dax locates the faculty of speech in the left hemisphere. He based this opinion upon the fact that aphasia is associated almost, if not invariably, with right hemiplegia, when there is any paralysis at all. That this is really the case is beyond question. Without, however, referring again to the cases cited by M. Dax, I quote the following table from Dr. Seguin's paper:

APHASIA WITH HEMIPLEGIA.

AUTHORITIES.	Right Hemiplegia.	Left Hemiplegia.
Trousseau, 1865 (Acad. de Méd.)	125	10
Baillarger, later in 1865 (Salpêtrière)	30	1
Jackson, loc. cit.	34	3
Robertson, loc. cit.	3	..
Medical Times and Gazette, September 9, 1865	2	..
Archives Gén. de Méd., 1866	2	..
Flint, New York Medical Record, vol. i.	4	..
New York Hospital, 1830–'67	43	3
Total	243	17

From this table we learn that, of two hundred and sixty cases of aphasia associated with paralysis, the left hemisphere —as determined by the situation of the hemiplegia—was the seat of the lesion in two hundred and forty-three cases, and the right in only seventeen.

I also quote the following table from Dr. Seguin:

QUESTION OF LEFT ANTERIOR LOBE.

AUTOPSIES BY	For.	Against.
Marc Dax, in 1861, and G. Dax (Acad. de Méd., 1863)	370	..
Bouillaud, 1848	85	..
" 1865	31	..
Trousseau (Acad. de Méd.)	18	16
Vulpian (Leçons de Phys.)	5	..
New York Hospital, 1830–'67	2	6
Jackson, Richardson, A. Clark, 1866, 1867	3	..
Peter, Legrand, Béclard, Delpech, Bérard, one each	..	5
Farge, Bigelow, Detmold, and Stokes, one each	..	4
Total	514	31

This table is based on autopsies, and may be considered conclusive as to the relative frequency with which aphasia is connected with disease of the left anterior lobe.

From various sources I have obtained the following additional cases, in which the seat of the lesion was determined

either by post-mortem examination or by the situation of the hemiplegia:

AUTHORITIES.	Left Hemisphere.	Right Hemisphere.
Larrey	1	..
Falret	2	..
Perroud	6	..
Magnan	30	..
Carrier	15	..
W. Ogle	25	..
Bartholow	1	..
Bateman	..	1
W. Wadham	..	1
Total	80	2

The immense preponderance of disease of the left hemisphere, and especially of its anterior lobe, as a concomitant of aphasia, is therefore placed beyond a doubt. Indeed, so far as I am aware, the fact is not questioned. How, now, is it to be explained?

We cannot claim, even with all the disparity of cases, that the organ of language is located in the left anterior lobe, or even in the left hemisphere, to the exclusion of the other. Broca has attempted to account for the assumed restriction, on the ground that the left hemisphere receives a larger supply of blood, and is earlier developed than the right. This is doubtless correct, but still the fact remains that lesion of the right hemisphere is sometimes followed by aberrations of speech; the left remaining perfectly healthy. One such case—and there are several on record in which the autopsy confirmed the deductions drawn from the symptoms—is sufficient to overturn the theory which restricts the situation to one side of the brain; and one such as that reported by Dr. Simpson,[1] in which there was extensive lesion of the third left frontal convolution in its posterior part, and no epilepsy, paralysis, or aberra-

[1] Medical Times and Gazette, December 21, 1867.

tion of speech, is of course utterly destructive of Broca's views.

The fact that aphasia is more frequently conjoined with right hemiplegia is undoubtedly due mainly to the fact previously insisted upon in my remarks on cerebral embolism, that the left middle cerebral artery is much more liable to be plugged by an embolus than the right; and it is by embolism that aphasia is generally caused. Dr. Hughlings Jackson[1] has very satisfactorily worked out the relation, and my own experience, presently to be related, abundantly confirms the fact.

At the same time it appears to be clearly shown that the left anterior lobe, or rather, in accordance with Dr. Jackson's views, those parts of the brain nourished by the left middle cerebral artery, are more intimately connected with the faculty of articulate language than any other region of the encephalic mass. It is probably true, as originally advanced by Dr. Moxon,[2] and since urged by Dr. William Ogle,[3] that the organ of speech is to be found in both hemispheres, and that one side is more generally employed than the other, just as we ordinarily give a preference to one eye or one ear or one hand, and that this side is the left. Gratiolet's facts, adopted by Broca to support his view of exclusiveness, will certainly lend force to the argument in favor of preference. This careful anatomist found that the left hemisphere is developed before the right, and that it is better nourished. Both of these circumstances are owing to the greater supply of blood which it receives.

Undoubtedly many of the cases which have been brought forward as militating against the doctrine of localization of the organ of speech are not cases of aphasia at all, but simply instances of inability to speak, from paralysis of the

[1] London Hospital Reports, vol. i., l. c.

[2] On the Connection between Loss of Speech and Paralysis of the Right Side. British and Foreign Medico-Chirurgical Review, April, 1866, p. 481.

[3] Aphasia and Agraphia. St. George's Hospital Reports, vol. ii., p. 83.

muscles concerned in speech. This is certainly true of the greater number of Seguin's cases, and also, as Bartholow[1] has stated, of those adduced by Ladame.

Again, in very many instances the post-mortem examination has not been properly made, and lesions involving one or the other anterior lobe have been overlooked. It is now a well-recognized fact that the cerebral tissue may be materially diseased, and the lesion not be detected without microscopical examination.

Giving a very full consideration, therefore, to the facts and arguments which have been urged on all sides of the question, I am constrained, while rejecting the restricted location of MM. Dax, and the still more limited situation contended for by Broca, to believe:

1. That the organ of language is situated in both hemispheres, and in that part which is nourished by the middle cerebral artery.

2. That while the more frequent occurrence of right hemiplegia, in connection with aphasia, is in great part the result of the anatomical arrangement of the arteries which favors embolism on that side, there is strong evidence to show that the left side of the brain is more intimately connected with the faculty of speech than the right.

These views are further supported by several interesting cases, the histories of which I now propose to relate:

Case I.—In the summer of 1857, while I was on duty, as medical officer of the army, with a body of troops and topographical engineers, making a road from Fort Riley to Bridger's Pass, in the Rocky Mountains, a quarrel occurred between two of the laborers, which resulted in one of them striking the other a violent blow on the head with a club. The injured man fell to the ground stunned, and remained in a state of coma for several hours. Upon examining him a few minutes after the affair took place, I ascertained that

[1] On Aphasia. Journal of Psychological Medicine, etc., vol. ii., p. 341, *et seq.*

there was no stertor and no indication of paralysis. He was unconscious and breathing quietly, with a pulse of about 80. He had received a blow on the left temple, which, though laying open the scalp, had not fractured the skull. Gradually he regained consciousness so as to be able to comprehend what was passing about him, but he had entirely lost the memory of words, though not the faculty of articulation. Thus he was unable to speak unless the words were first repeated to him, and then he could do so without any defect of articulation, provided too many words were not given to him at once.

Thus, when I said to him in Spanish—he was a Mexican, and could not speak English—"Como sientes ahora?" "How do you feel now?" he repeated, "Como sien. sien. sien.," and then, looking at me in apparent despair, burst into tears. And this was repeated time and again during the hour I spent with him.

The next morning, at about seven o'clock, as he attempted to rise from his bed, he fell, and was found a few minutes afterward by the hospital attendant, lying on the ground in a state of complete coma. I saw him almost immediately; he was breathing stertorously, blowing out his lips and cheeks at each expiration, and exhibiting a general resolution of all his limbs. He died at about eleven o'clock A. M. that day.

That afternoon I made a post-mortem examination. On removing the calvarium, the first thing that attracted my attention was an ecchymosed spot about the size of a half-dollar-piece involving the left anterior lobe at its lateral and posterior margin. There was no extensive hæmorrhage at this point. But, on the opposite side, there had been a rupture of the middle meningeal artery, and an immense extravasation of blood which had infiltrated between the lobes of the right hemisphere and collected in the base of the skull. My theory of the case was that the hæmorrhage from the artery had been suddenly stopped during the condition of

primary insensibility before any considerable quantity of blood had been effused, and that during the night his heart had recovered its power; and this, with the muscular effort he made in attempting to get out of bed, had dislodged the coagulum, and allowed the hæmorrhage to take place. At that time I attached no especial importance to the injury of the left anterior lobe; but, since the debate in the French Academy in 1861, I have had no doubt that to it the amnesic aphasia was entirely due.

It will be observed that there was no defect of articulation in this case, either from paralysis or incoördination, but that the difficulty was solely as regarded the memory of words.

Case II.—J. H., a captain of a coasting-vessel, consulted me in November, 1864, for a difficulty of speech with which he had been affected for several months. Upon inquiry, I ascertained that one morning early he had been called from his bed upon some duty connected with his vessel; that he had risen rather hastily and gone on deck; that while giving an order he suddenly became very dizzy, and fell, unconscious. He soon regained his senses, but found that he was paralyzed on the right side, and had lost the ability to speak. He soon afterward reached port, and remained at home for three months, during which period the paralysis disappeared almost entirely, and he reacquired the ability to speak. The aphasia was of both the amnesic and ataxic forms. He could neither speak nor write.

He then went to sea again as a passenger to Cuba, and while in Havana had another attack similar to the first, but without paralysis of motion, though there was loss of sensibility on the right side. The memory for words was entirely destroyed, though he could pronounce distinctly any word he was told to say, if he did not allow too long a period to elapse between the direction and the response. About four months after his last seizure he consulted me.

At this time he could say a few words, and he employed

them to express all his ideas, assisting himself with very energetic gestures, which, however, were rarely expressive of his thoughts. The words he thus constantly used were "sifi," which signified both yes and no, and "time of day," which he employed when he had any other answer than simple affirmative or negative to give. Besides these expressions, he had an oath, "Hell to pay!" which he ejaculated whenever he did not succeed in making himself understood, and sometimes without any such exciting cause. These were the only expressions he could originate, but he could pronounce distinctly any word he was told to say, and even as many as three short successive words. When told to write, he took the pen, and, on my telling him to give me his name and address, wrote "Time of day," and then, seeing that that was not the correct answer, immediately followed it with "Hell to pay!" On my remarking to him that he had given me wrong information, he immediately wrote "sifi." Any word, however, which I told him to write, he did without any difficulty, and thus I obtained several long sentences from him.

From his brother, who came with him, I obtained the facts in his history I have mentioned. Examining his heart, I found that he had a strong systolic murmur, and was told by his brother that he had had, fifteen years ago, a first attack of acute articular rheumatism, which had been followed by several other attacks.

The main point of interest about this case—and it is one of those I shall again draw attention to—is, the occurrence of ataxic aphasia with hemiplegia as concomitants of the first attack, while the second was characterized by purely amnesic aphasia and no paralysis.

Case III.—During the winter of 1868–'69, a man came to my clinique, at the Bellevue Hospital Medical College, who was aphasic, and from whose friends, his own gestures, and the few words he could speak, I obtained the following history: Some months previously he had been working in a stone-quarry, and was struck by some piece of machinery on

the left side of the head, at about the junction of the frontal with the temporal bone. For a short time he was unconscious, recovering, however, without paralysis, but with loss of the memory of words. When he came under my observation, he was very intelligent, comprehended every word said to him, and made repeated and persistent efforts to talk, but he could not utter a word spontaneously beyond "yes" and "no," which he always used correctly. Thus, when I asked him where he was born, he became much excited, gesticulated violently, and apparently made every effort to tell me. The perspiration stood out in large drops on his forehead, but no sound came from his lips. Then the following conversation took place:

"Were you born in Prussia?" "No."

"In Bavaria?" "No."

"In Austria?" "No."

"In Switzerland?" "Yes, yes, yes, Switzerland, Switzerland," at the same time laughing, and moving his hands actively in all directions. He could pronounce words well, but could not write.

I took occasion to speak at length on the subject of aphasia, and gave it as my opinion that there had been a fracture of the internal table of the skull, and that a fragment of bone was pressing on the posterior and lateral part of the anterior lobe. My friend Prof. Sayre was present, and I advised him to trephine the patient, with the view of elevating any depressed piece of bone, and restoring the normal function of that part of the brain. The operation was performed a few days afterward, the patient being placed under the influence of ether. The internal table was found to be fractured, and a splinter was pressing on the posterior frontal convolution. It was removed, and, as soon as the patient emerged from the anæsthetic condition, he spoke perfectly well.

This, as will be seen, was also a case of amnesic aphasia unaccompanied by paralysis.

Case IV.—A. E., formerly a bookseller, consulted me in the autumn of 1869 for what was considered by his friends to be, and what probably was, softening of the brain. Before any symptom of disease appeared, he had been noted for his remarkable memory, but was now exceedingly forgetful, especially as regarded words. Thus he had forgotten his first name, and could not tell me the names of his children. His conversation was marked by great hesitancy, from his not remembering the words he wished to use, and there was, besides, marked difficulty of articulation, and some words he could not pronounce at all. There was right hemiplegia, which had gradually been getting worse, and which, when I saw him, was extensive enough to interfere materially with the movements of his arm and leg. The left side was not affected, and the tongue and face were apparently not paralyzed. He was subsequently lost at sea in the steamer City of Boston.

This case, therefore, exhibited both the amnesic and ataxic forms of aphasia, and was accompanied by right hemiplegia. I regard the condition as being due to thrombosis, probably of the left middle cerebral artery.

Case V.—W. W., aged forty-one, entered the New York State Hospital for Diseases of the Nervous System, August 22, 1870, hemiplegic on the right side, and affected with ataxic aphasia. In the month of March, 1868, as ascertained by Dr. Cross, the resident-physician of the hospital, he was seized with a dull pain in the right knee, accompanied with numbness, formication, and pricking sensations, limited to the right foot, while general numbness of the whole side soon supervened. These, with loss of power, gradually extended and increased till at the end of two weeks the patient was entirely hemiplegic. There was at no time any loss of consciousness nor any mental aberration. On the 11th of May following, the patient suddenly lost the power of speech, but his mind remained perfectly clear, and, though he could not utter a word, he understood well every thing that was

said to him. He remained nearly completely aphasic for four months, being only able during that time to utter a few sounds, which could not be interpreted into intelligible words.

About September, 1868, he began to enunciate a few words, at first very slowly and indistinctly, and gradually acquired more facility. When I presented him before the class at the Bellevue Hospital Medical College, in November, 1870, although he could talk, his power of coördination was very imperfect, and many words were articulated with great difficulty. This trouble was chiefly manifested in regard to labials and linguals, such words as "truly rural," "Peter Piper," "baker," and others of the kind, causing him to make repeated efforts before he could even imperfectly pronounce them. There was no paralysis of the tongue, no deviation when it was protruded, and but very slight if any paresis of the orbicularis oris or other facial muscles. The arm and leg on the right side were profoundly paralyzed.

In this case there was no loss of the memory for words, and no difficulty in writing. It was, so far as the aphasia was concerned, entirely ataxic in character, and accompanied by right hemiplegia.

My opinion was, that the symptoms were to be attributed to thrombosis of the left middle cerebral artery.

Case VI.—R. M., aged twenty-five, noticed one day that his right foot was unusually cold. A few days afterward he had his first attack of hemiplegia of the right side. Suddenly, and without the least warning, except a severe vertigo, he fell, but immediately arose. There was no loss of consciousness, and with assistance he was able to walk to his residence, a short distance off. His face was drawn to the left side, and speech and memory were slightly impaired.

In February, 1869, having recovered motility, he was seized with another attack of right hemiplegia. This time he partially lost consciousness, and his speech again became affected. By April, 1869, he was able to resume his work as a weaver, but his arm was still weak.

In July he had another attack, which was slight.

In May, 1870, he again suddenly became hemiplegic on the right side. There was no loss of consciousness. The face and tongue were affected. With assistance he walked home, and in a week had quite recovered.

In July, 1870, he had his fifth and thus far last attack. While chopping wood he was suddenly seized with a violent pain in the head, followed by vertigo. He fell, but did not lose consciousness. There were right hemiplegia again, difficulty of speech, and dilatation of the left pupil. For five days afterward he was delirious, but finally recovered, with loss of power in the right arm and leg, and increased difficulty of speech. September 1st, he was admitted to the New York State Hospital for Diseases of the Nervous System. At this time the paralysis had entirely disappeared; the tongue could be moved freely in any direction, and his articulation was perfect. But his memory for words was greatly impaired, though facts and circumstances were remembered perfectly well. His speech was therefore hesitating, and if asked to repeat a sentence of three or four words he could not do it. Thus he could not repeat the words "sugar, coffee, crackers," although he began immediately after I had finished saying them.

Examination showed that the patient had hypertrophy of the heart, with aortic insufficiency.[1] My diagnosis was, repeated attacks of embolism of the left middle cerebral artery, or its branches.

This case was one of partial amnesic aphasia, with ataxic aphasia, which had disappeared with the hemiplegia.

Case VII.—Mrs. S. H. W., aged thirty-two, married. On the 26th of June, 1860, about three weeks after the birth of her child, she was suddenly seized with a severe pain in the right shoulder, which extended down the arm. Symp-

[1] I have condensed the histories of this and the preceding case from the reports of Dr. Cross, in my clinical lecture on Partial Cerebral Anæmia, published in the Journal of Psychological Medicine for January, 1871.

toms of albuminuria, accompanied by general dropsy, immediately ensued, and in a few weeks the dyspnœa from hydrothorax was alarming. Coma and a convulsion followed. Soon after the fit, which marked the height of her disease, as she was sitting by the bed, resting her head on her folded arms, her right side became completely paralyzed, and she lost the ability to speak. She was not entirely clear in her mind for a week after the attack, but gradually the dropsy disappeared, her intellect improved, and the paralysis became less.

At the time of the seizure, the face was drawn to the right side, the tongue deviated in the same direction, and there were strabismus and partial ptosis and paralysis of the orbicularis palpebrarum muscle on the right side. Motility and sensibility in the right arm and leg were, at first, completely abolished, but at the end of ten days she was able to move about, by holding on to a chair. During three years she continued to improve as regarded the paralysis, but for all that period did not speak a word. In the summer of 1863 she became able to say the word "no," and a few months later she could say "yes."

At my request, she allowed me to present her before the class of the Bellevue Hospital Medical College, in November, 1870, on the occasion of a clinical lecture on aphasia.

She was then, and is now, enjoying good health, with the exception of frequent headache. Her countenance is remarkably bright and cheerful, and her whole expression is exceedingly intelligent. She comprehends every word that is said to her, and attends to all her household duties. Yet she is unable to utter any words but "no," "yes," and "dado." The latter is seldom employed, but in her vocabulary signifies affirmation. She uses "yes" for affirmation, "no" for negation, and both for doubtful or indifferent conditions. Thus, if asked how she is, she answers "Yes, yes, no, no," which means that she is tolerably well. Sometimes she employs these words quite indiscriminately. If asked

what that is, pointing to a fan, she cannot tell, nor can she repeat the word fan. She shows, however, that she knows, by making the gesture of fanning herself. She can neither read nor write, although on one occasion she succeeded, after great difficulty, in writing "no." Not long since, she suddenly ejaculated, "I don't know!" and a few days ago exclaimed, "How do you do?" but she was not able to repeat either of these phrases, nor did she appear to be aware that she had said them. Her gestures are very intelligent and expressive. The right arm and leg are weaker than on the left side, and the sensibility is less.

There is a murmur at the apex of the heart with the first sound.

Ophthalmoscopic examination showed the vessels of the retina of the left eye to be much larger than those of the right.

In this case I diagnosticated embolism of the left middle cerebral artery.

The aphasia was of both the amnesic and ataxic forms, and was accompanied by right hemiplegia.

Case VIII.—Mr. B. consulted me in November, 1870, for loss of the memory of words, and fulness and pain in the head, with occasional vertigo. Over a year previously, while in the woods of Minnesota buying timber, he had suddenly lost consciousness for a few moments, and on recovering found that he had become hemiplegic on the right side, and had lost the power of speech. For a short time he could not utter a word, but gradually the memory of language, and the ability to coördinate the muscles of speech, returned to him, and he could articulate sufficiently well to be understood. For several months, however, his recollection of words was bad.

For some time he had been under the care of Dr. Hale, of Chicago, a homœopathic physician, who advised him to place himself under my charge. When I first saw him, he could talk quite well, but there was still a hesitancy in his

speech, and occasionally words were misplaced or miscalled. Articulation was distinct, and the hemiplegia had disappeared. There was pain, almost entirely confined to the left temporal region. There was the history of acute articular rheumatism, and there was aortic insufficiency.

In this case there had been at first amnesic and ataxic aphasia, with right hemiplegia. As the latter disappeared, the ability to coördinate the muscles of speech was increased, until at last articulation became perfect, and only amnesic aphasia remained.

Case IX.—H. I., a merchant, consulted me in August, 1869, for hemiplegia, with inability to speak. While sitting at his desk, six weeks previously, he suddenly became vertiginous, and lost consciousness for a few moments. On recovering his senses, he discovered that he was paralyzed on the right side, and that he could not speak a word. He was exceedingly anxious to make known some wish, and one of his clerks brought him paper and a pencil, but he could not write a letter. An alphabet was then written, but he was unable to select the letters to form the words he wanted to use.

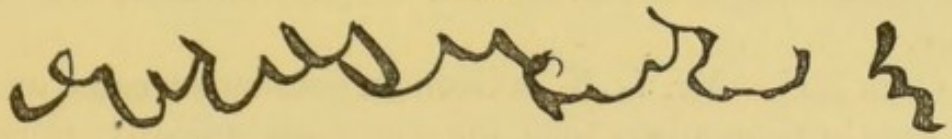
A physician was sent for, and Mr. I. was bled to the extent of sixteen ounces, without any favorable result. He remained hemiplegic and completely aphasic for about two weeks. He then began to walk, and acquired the ability to say "what," "certainly," and "saw my leg off," which he contracted into "sawmelegoff," accentuating strongly the ultimate syllable. These words he used without apparent intelligence, though he clearly understood all that was said to him, and laughed at any joke as heartily as ever. His condition was about the same when I saw him.

He could protrude his tongue, and move it actively in all directions, but could not articulate any words but those mentioned. Thus, when I asked him to say "table," he said, "Certainly;" and when I said, "Well, say it then," he exclaimed, "Sawmelegoff!" at the same time, to show that

he understood what I said, he went across the room, and put his hand on a table, uttering, at the same time, his full stock of words, "what," "certainly," "sawmelegoff."

I then asked him if he could write; he replied, "Certainly." I placed paper before him, and gave him a pen with ink, but he was unable to write his name as I requested, although he could use his fingers for other things tolerably well. I asked him to draw a series of parallel lines, and he did so without difficulty. On my insisting that he should now make an effort to write his name, he made the attempt with this result:

Fig. 7.



I told him that was not his name, at which he gesticulated violently, exclaimed, "Sawmelegoff!" and gave me one of his visiting-cards. This gentleman continued under my care for some time, but with no perceptible change. He had had two attacks of acute articular rheumatism, and had, when I saw him, both aortic and mitral insufficiency.

Here, then, was right hemiplegia, with fully-developed ataxic and amnesic aphasia. My diagnosis was, embolism of the left middle cerebral artery.

Case X.—Miss C. R., of strongly-marked hysterical diathesis, suddenly became aphasic while sitting at the breakfast-table. I saw her about two hours subsequently, when she drove to my office with her mother. There was no paralysis, the tongue could be moved freely in all directions, articulation was perfect, and she could pronounce any word mentioned before her. The memory of words was, however, entirely abolished.

Case XI.—Mr. S., a retired merchant, consulted me in September, 1870, for the effects of cerebral hæmorrhage. He was hemiplegic on the right side, and unable to talk.

His intelligence was good. He could read, but he was not able voluntarily to pronounce a word. The tongue was not in the least paralyzed, nor had it been. Occasionally ejaculations of various kinds would come forth. On one occasion, as he entered my office, he exclaimed—he was a German gentleman—"Guten Morgen, mein Herr," but by no effort could he repeat that or any other expression. His attempts to speak were continuous while he was with me; and his son who came with him said he was almost always trying to talk while he was not sleeping.

This case was, therefore, one of ataxic aphasia, and was marked by the existence of right hemiplegia. Cerebral hæmorrhage, involving the corpus striatum, was the cause.

Case XII.—Mr. L. N., a German gentleman, came under my care in September, 1869, for symptoms indicative of cerebral softening. He was slightly paralyzed on the right side. His speech was affected both amnesically and ataxically. Soon afterward, in consequence of maniacal symptoms making their appearance, I sent him, with the concurrence of my friend Prof. Flint, to the Lunatic Asylum, at Flushing. He remained there till September of the present year, gradually failing in mental and physical power, when, as he was no longer in a condition to injure himself or others, his friends, with my approval, removed him to their own home. At the present time he can scarcely remember a word, and his articulation is very defective. A remarkable feature of his conversation is that he calls every thing "kazze," "cat." He appears to have forgotten every other word.

The history of this case points to thrombosis as the probable lesion.

Case XIII.—This was a very remarkable and instructive case, one which I have already mentioned under the head of embolism.

The patient was a retired officer of the army, and consulted me in the autumn of 1869 for paralysis, vertigo, and

slight difficulty of speaking, from which he had suffered for some months. Several years previously he had been under the care of my friend Dr. Metcalfe for acute rheumatism, with cardiac complications. The history of the case pointed strongly to embolism, and, as the paralysis involved the right side, I diagnosticated a previous attack of embolism of the left middle cerebral artery.

The difficulty of speech was slight; there were both amnesic and ataxic aphasia.

Under the treatment employed he improved very much in the ability to walk, to use his arm, and to speak, so much so that he and his friends considered him better than he had been for several years. But about six weeks after he came under my charge he had another attack. This time the left side was paralyzed, and there was no difficulty of speech. Galvanism was employed, as before, and he recovered sufficiently to go to Washington City. While there he had a third attack, characterized by right hemiplegia and aphasia. He soon recovered his power of speech, and soon afterward had a further attack, involving the left side, and unattended by aphasia. He recovered under the care of Dr. Basil Norris, of the army, and soon afterward came again to New York. A short time after his arrival I requested my friend Prof. Flint to see him in consultation, with the special view of having him examine his heart. This was done with thoroughness, but no abnormal sounds were detected. While in New York he had two other attacks, during both of which he was delirious; both were characterized by hemiplegia. That of the left side was unaccompanied by aberrations of language; that of the right was attended with ataxic and amnesic aphasia. He forgot the names of the most ordinary things, and there were many words that he could not articulate at all. Thus, when he wanted a fan, he called it "a large, flat thing to make a wind with." He forgot my name, and could not pronounce the words beetle, general, physician, and many others. I sent him to Newport greatly improved, but he had other

attacks there, and finally died in the autumn of the present year, of, I presume, cerebral softening.

The interesting features of this case are the concurrence of hemiplegia and ataxic and amnesic aphasia, and the striking fact that there was no aphasia when the paralysis involved the left side. Thus, according to my views of the case, the patient had repeated attacks of cerebral embolism. When the embolus lodged in the left middle cerebral artery, there was aphasia accompanied by right hemiplegia; when the embolus obstructed the right middle cerebral artery, there was left hemiplegia, but no aphasia.

Case XIV.—In the early part of December, 1870, J. M., a patient of Bellevue Hospital, was, at his request, brought to my clinique at the college. His history, as given me by Dr. Judson, showed that he had repeated attacks of unconsciousness or semi-unconsciousness, which were accompanied with hemiplegia. Dr. Flint had also detected a bellows murmur, but it was at the apex of the heart. The patient had suffered from several seizures of acute articular rheumatism.

Upon inquiry, I ascertained that he had had altogether eleven attacks of vertigo, unconsciousness, and hemiplegia. His intelligence was good, and he spoke tolerably well, though with hesitation and occasional difficulty of articulation. His speech was much better than it had been, and there was no well-marked hemiplegia.

As in the case last mentioned, whenever the hemiplegia had been on the left side there was no aphasia, but when it was on the right side there was always well-marked difficulty of speech, both amnesic and ataxic.

The only other case, similar to these last two, that I have been able to find, is one reported by Dr. Stewart,[1] of a man who was admitted into the Middlesex Hospital, suffering from left hemiplegia, without aphasia. A week later he became affected with right hemiplegia and loss of speech. He

[1] Medical Times and Gazette, July 9, 1864.

died, and on post-mortem examination both middle cerebral arteries were found plugged with emboli.

The views which the cases I have observed have led me to form, have been confirmed by my recent study of the subject of aphasia. These have already been given in part, but the detail of the foregoing histories enables me to express the remainder with more confidence.

It cannot have failed to strike the reader that, in all the cases of which hemiplegia formed a feature, the aphasia was of the ataxic form, while when there was no hemiplegia the aphasia was amnesic. In the one the individual was deprived of speech because he could not coördinate the muscles used in articulation, in the other because he had lost the memory of words.

This is a point which has not hitherto been noted. The phenomena indicate, I think, very clearly the seat of the lesion, and the physiology of the parts involved.

The gray matter of the lobes presides over the *idea* of language, and hence over the memory of words. When it only is involved, there is no hemiplegia, and there is no difficulty of articulation. The trouble is altogether as regards the memory of words.

The corpus striatum contains the fibres which come from the anterior column of the spinal cord, and is besides connected with the hemisphere. A lesion, therefore, of this ganglion, or other part of the motor tract, causes paralysis of motion on the opposite side of the body. The cases I have detailed show, without exception, that the power of coördinating the muscles of speech is directly associated with this hemiplegia. A lesion, therefore, followed by hemiplegia and ataxic aphasia, indicates the motor tract as the seat. If amnesic aphasia is also present, the hemisphere is likewise involved. An analysis of the cases reported by Ogle, Jackson, and some other observers, shows that the association existed in their cases, although they have not noticed it as of any physiological or pathological bearing.

Another important feature of the foregoing cases is the constant association of the aphasia with right hemiplegia where there was any paralysis at all. This indicates, perhaps, only the more frequent occurrence of embolism on the left side, but the last two cases, as well as the one quoted from Dr. Stewart, show that the left hemisphere is more intimately connected with the faculty of speech than the right. In fact, it appears to me impossible to avoid this conclusion.

So much for some of the various theories which exist relative to the localization of the organ of language and for the clinical history of aphasia. I have not thought it necessary to discuss the view of Schroeder van der Kolk,[1] that the faculty of articulate speech resides in the corpora olivaria, because there is little if any physiological or pathological evidence to sustain it. Nor the hypothesis of Brown-Séquard,[2] that speech is a reflex phenomenon, because there is no evidence in support of that opinion. Neither have I, though much tempted, ventured into the philosophy of the subject to any considerable extent.

As to the causes, the prognosis, diagnosis, morbid anatomy, and pathology, they have been sufficiently considered in the remarks made, and the treatment is of course that of the pathological condition to which it is due, whether this be cerebral hæmorrhage, embolism, thrombosis, softening, hysteria, wounds, the bites of poisonous serpents, or other cause. One point, however, should be mentioned in this connection, and that is, that constant efforts should be made to exercise the vocal organs, by attempts to speak, and by the application of the galvanic or faradaic currents to the tongue and other muscles concerned in articulation.

[1] On the Minute Structure and Functions of the Spinal Cord and Medulla Oblongata. New Sydenham Society Publications, p. 140.

[2] Seguin's Memoir, already quoted

CHAPTER VIII.

ACUTE CEREBRAL MENINGITIS.

By acute cerebral meningitis is understood inflammation of two membranes of the brain—the pia mater and arachnoid. Some writers have made the attempt to discriminate between inflammation of the arachnoid and inflammation of the pia mater, but there are no diagnostic marks by which such a distinction can be made, and we find from post-mortem examination that neither membrane can be inflamed without the other participating in the morbid process. Inflammation of the dura mater is never included under the term meningitis.

The ancients made no distinction between the several inflammatory affections of the intra-cranial organs, but comprehended them all in one disease which they called frenzy —*φρήν*, the brain. Morgagni, however, showed that the membranes of the brain were the parts generally involved, and gave a very accurate account of the phenomena of an attack of acute meningitis. Since then, Rostan, Lallemand, Andral, Bouillaud, and others, have added to our knowledge.

Symptoms.—The symptoms of acute cerebral meningitis may be divided into three groups, arranged in chronological order: the stage of invasion, the stage of excitation, and the stage of collapse.

1. The Stage of Invasion.—The most prominent initiatory symptom is headache, which may be diffused or confined to a limited part of the head. When this latter is the

case, the frontal region is more generally its seat; next in order of frequency is the occipital, and next the temporal. At the same time, the face is flushed, the eyes are red and suffused, and there is a decided elevation in the temperature of the head, which is not only felt by the patient, but may be perceived by the hand of the physician. Vomiting is generally present.

As might be expected, these symptoms are accompanied by fever. This, however, rarely runs high, so far as the force or the frequency of the pulse is concerned, or as regards the heat of the skin. It is mainly characterized by restlessness and insomnia. Occasionally there is a tendency to somnolence.

This stage may last a few days or only a few hours, or may be so slight as not to attract attention. In general features it resembles the prodromatic stage of cerebral congestion.

2. Stage or Period of Excitement.—A chill ushers in this stage, and an increase in the intensity of several of the symptoms of the first stage, and the development of others, soon take place. Thus the fever becomes higher, the skin hotter, and the temperature of the body is elevated several degrees. The pulse is frequent, quick, and hard, and the face becomes redder than in the first stage. The pain in the head augments in violence, and is increased by pressure on the scalp, or even the slightest movement.

The eyes are bright, the pupils contracted and painfully sensitive to light. The hearing becomes morbidly acute, loud noises cause great agony, and even slight sounds are unbearable. The general sensibility of the body is increased, and hence the patient is rendered uncomfortable by the contact of the bedclothes with the skin. Delirium is generally present from the first, and is often of furious character. Hallucinations of sight and hearing are almost constant, and the irrationality of the ideas is marked by the incoherence of the speech. The patient when awake is continually talk-

ing, gesticulates violently, and weeps and laughs alternately over imaginary evils. It is sometimes necessary to use restraint to prevent him injuring himself or others, and the attendants should always be prepared for any emergency of the kind. As the disease advances, the delirium becomes more subdued, and the patient may exhibit some evidences of sanity.

Even when there is no delirium, as occasionally happens, the influence of the morbid action over the mind is shown in the irritability of the patient, and the change which he undergoes in character and disposition.

Convulsions rarely occur in adults, but motility generally is nevertheless disordered. The limbs are in almost continual action, as are likewise the jaw and the eyelids. Twitchings of the facial and other muscles, such as those of the forearm, are usually well marked, and occasionally there are irregular movements of the eyeballs. Convulsions, when they occur, may be either clonic, or tonic, or both. Thus there may be a gradually-increasing rigidity of some muscles, followed by relaxation and disordered movements. Sometimes there is opisthotonos as well marked as in some cases of tetanus. Hemiplegia or paraplegia may occur, but are infrequent complications. I have seen two cases in which one lateral half of the body was paralyzed during the whole course of the disease.

Contractions of the limbs sometimes take place, and may be confined to one side or a single limb. In this case the forearm is usually strongly flexed on the arm.

The muscles of organic life participate, and the bowels are obstinately constipated. There may be difficulty of swallowing, from spasm of the pharynx, and irregularity of breathing, from implication of the respiratory muscles.

The most characteristic symptom of this stage is, however, the obstinate and violent cephalalgia, of which mention has already been made, and yet there are cases in which it is entirely absent from first to last. Several such instances

have been under my own charge, and post-mortem examination has verified the existence of the evidences of meningitis. This stage lasts from a few days to two weeks.

3. Stage or Period of Collapse.—The beginning of this stage is marked by the occurrence of somnolence, which often shows a tendency to pass into coma, and by a subsidence of the delirium and muscular agitation. There are times, however, during which the stupor remits in profundity, and the patient appears to be somewhat conscious of his condition, but these periods only occur in the first part of the third stage. Ere long the coma becomes constant.

Paralysis then supervenes and is first manifested in the ocular or facial muscles. Thus from paralysis of one of the muscles of the eyeball strabismus ensues, or the upper eyelid may drop from paralysis of the levator palpebræ superioris. The pupils dilate and become insensible to light, and the mouth is drawn to one side from implication of the muscles of the face. Before long the contractions of the limbs relax, and paralysis takes place. The sphincters of the bladder and rectum also lose their power, and the urine and fæces escape involuntarily. The pulse becomes slow and irregular, but the temperature, as Jaccoud has shown, and as I have lately verified in several instances, does not fall. The insensibility becomes more and more profound, and the patient dies in a state of coma, sometimes from asphyxia produced by paralysis of the respiratory muscles, but generally from the gradual engorgement of the lungs.

Such is the ordinary course of an attack of simple acute meningitis occurring in a young and healthy person. But there are modifications often met with which require consideration. Of these, epidemic cerebro-spinal meningitis is scarcely to be considered a disease of the nervous system, and tubercular meningitis will be discussed under a separate head, but the differences due to acute rheumatism and old age may very properly be noticed in the present connection.

CEREBRAL RHEUMATISM.

By this term is signified the inflammatory condition induced in the membranes of the brain by an attack of acute articular rheumatism. The relation has been recognized from the very earliest period, but, though alluded to by Flint and other American writers, does not seem to have attracted marked attention in this country. My own experience, however, satisfies me that meningitis of a distinct form is often caused by rheumatism.

The membranes of the brain may become affected during the second week of an attack of acute rheumatism. The swollen and painful joints become less swollen and less painful, and the patient may be convulsed, or, what is more common, exhibits choreiform movements in the limbs.

The delirium, which is generally present, is similar to that met with in alcoholism. There are the same trembling of the limbs and tremulousness of the lips.

A marked point of difference between rheumatic and simple meningitis consists in the entire absence in the first-named of cephalalgia and vomiting, but it is frequently characterized by the presence of severe pain in the back, which may extend as high as the occiput. This was a prominent feature in more than half the cases that have been under my observation. The subsequent cause of rheumatic meningitis does not differ from that of the simple form of the disease. Coma and collapse ensue, with a general remission of the violence of the symptoms. Death, however, generally ensues, and in the way already described.

SENILE MENINGITIS.

In old persons the symptoms of acute meningitis are rarely so pronounced as in individuals of middle age. The affection comes on more gradually, and may have made considerable progress before its existence is suspected. There is little or no pain, no fever, and no gastric or intestinal derangement. The mental symptoms are very similar to those

due to softening. The patient has imperfect articulation, his memory is impaired, and he does things which show that he is not in his right mind. The delirium is of the low muttering kind, and there is a tendency to coma even in the first stage. There is a more or less general paresis in all the limbs, and subsultus is commonly present. Death is usually due to pulmonary engorgement.

Causes.—Among the predisposing causes of acute cerebral meningitis, age is first to be considered. Guérsant[1] asserts that the period of life between sixteen and forty-five is that during which acute meningitis is most liable to occur, not including children, who are far more prone to the disease than adults. Rilliet and Barthez[2] have, however, shown that very young infants are not so subject to simple acute meningitis as children of from five to eleven years of age. The very opposite opinion is expressed by Drs. Meigs and Pepper.[3]

Nine cases of acute simple meningitis have come under my observation. Of these, all were between the ages of thirty and forty.

Men are more subject to it than women. Of my cases, seven were males and two females. Parent-Duchatelet and Martinet,[4] however, think women are more predisposed to the affection than men.

Temperature, either very high or very low, predisposes to acute meningitis. Five of the cases under my care occurred in summer and four in winter.

Certain professions and habitudes appear to favor the occurrence of the disease. Among the former are all those which require the head to be exposed to strong and direct heat; among the latter are excessive intellectual exertion,

[1] Art. Méningite, in Dictionnaire de Médecine, Paris, 1839.

[2] Traité des Maladies des Enfants, Paris, 1853.

[3] A Practical Treatise on the Diseases of Children, Philadelphia, 1870, p. 464.

[4] Recherches sur l'Inflammation de l'Arachnoide, Paris, 1821.

and abuse of alcoholic liquors. Tertiary syphilis, gout, and rheumatism, are likewise predisponents.

Of exciting causes, injuries of the head from falls or blows of different kinds stand first. Next is exposure to the direct rays of the sun, or other source of great heat, and then recession of an exanthematous affection, such as scarlatina, measles, or erysipelas, and the irritation of dentition, or intestinal worms.

Diagnosis.—Acute meningitis may be confounded with partial or circumscribed encephalitis, but the distinction is made by considering that in the latter the headache is less severe, the delirium less marked, and the convulsions and contractions weaker. Moreover, the febrile excitement is much greater in acute meningitis than in partial encephalitis, and the whole disease more pronounced.

The meningitis of the aged bears a considerable degree of resemblance to cerebral softening; but the fact that the first-named affection is more rapid in its progress, and is not preceded by symptoms due to other morbid conditions, will generally enable the practitioner to make a correct diagnosis.

From delirium tremens it may be distinguished by the history of the case, by the greater tendency to insomnia exhibited in alcoholism, and by the general character of the delirium. The febrile excitement of acute meningitis, the pain in the head, the heat of the skin, the absence of clammy perspiration, and the increased temperature, as shown by the thermometer, are conclusive diagnostic marks.

From typhoid fever meningitis is diagnosticated by the existence in the former of meteorism, abdominal tenderness, and petechiæ, by the facts that the headache and febrile excitement are less, and that diarrhœa is present and vomiting is not.

Prognosis.—This is always grave. Occasionally death takes place in a few hours, and generally before the tenth day. When the disease is prolonged beyond this latter pe-

riod, the prognosis becomes more favorable. The occurrence of strabismus or other paralytic affection lessens the hope of a favorable termination. Prof. Flint, however, has cited two cases occurring in the hospital practice of himself and Dr. Thomas, in which there were strabismus, hemiplegia, and coma, both of which recovered. He also cites another case in which there was strabismus, and in which recovery took place. Hiccough is an unfavorable event.

Of the nine cases observed by myself seven died. In all of these fatal cases there was strabismus. In the two cases which recovered there was no squinting. The deaths in the fatal cases all occurred before the tenth day, and two took place before the end of the third day.

Morbid Anatomy.—If death occurs during the second stage of the disease, the most marked appearance found in the membranes is redness from increased hyperæmia. If, however, it is delayed till the third stage, effusion of serum is the prominent feature. In a case of which I made a post-mortem examination last summer, and which was caused by the great heat of the season, there was an extensive collection of bloody serum in the cavity of the arachnoid, and the pia mater was so adherent as to bring with it a layer of the gray matter of the brain as it was stripped off.

The fluid may consist solely of pus, or this may be mingled with serum in all proportions. The pus, with the fibrine of the exuded serum, forms thin plates of membraniform texture, which are scattered over the surface of the inflamed region or may entirely cover it.

If death has taken place late in the course of the disease, evidences of the implication of the cerebral substance will generally be discerned. These consist in the gray substance becoming of a pinkish color, and the white, when cut, showing numerous puncta vasculosa. The ventricles rarely contain any considerable amount of fluid, and are often entirely empty. The latter was the case in the instance above mentioned.

Pathology.—The symptoms of the first and second stages are due to congestion; those of the third mainly to effusion and consequent pressure.

An important question connected with the pathology relates to the determination, from the symptoms, what part of the brain is the seat of the lesion. The upper convex portion of the hemispheres is intimately related to the purely intellectual functions of the brain, while the under surface, or base, is connected with the motility of various parts of the body. Thus, if the inflammation be strictly limited to the upper surface of the brain, the predominant symptoms are those involving intellectuality, and consequently there is delirium, marked by incoherence of ideas and irrationality of language. If, on the contrary, the base of the brain alone is affected, the chief manifestations of disease are seen in the muscular system, and there are contractions, spasms, convulsions, and paralysis. When the morbid action extends to both regions, there is a combination of these phenomena.

But, as Jaccoud[1] states, there are some stubborn facts which stand in the way of the unreserved acceptance of the law laid down, for it occasionally happens that the symptoms are not in direct relation with the seat of the lesion. Thus in the case, the post-mortem examination of which I have referred to, there had been spasms and paralysis, yet the convex surface of the right hemisphere was alone involved, and that to an extent not exceeding a third the size of the hand. Jaccoud explains such cases by attributing to the cerebral symptoms a double origin; one set being due directly to the part affected, the other resulting from secondary reflex excitation. The explanation seems logical.

Another fact should also be taken into consideration. In acute cerebral meningitis there is very frequently a large effusion of serum or an extensive formation of pus. If either be collected on the upper convex surface of either

[1] Op. cit., p. 212.

hemisphere, the pressure exerted through the intervening brain-substance upon the motor tract at the base must produce more or less derangement of motility on the opposite side of the body.

Guyot,[1] who has given very careful study to the localization of the lesion from a consideration of the symptoms, declares that it is possible to define the seat very accurately, but his manner of looking at the subject places it in altogether a different position from that which Jaccoud gives it. Thus, tracing the fibres of the motor tract through the white substance to the convex surface of the hemispheres, he associates lesion of this region, not only with disturbances of ideation, but with derangement of motor functions. In this view he is supported by the experience of MM. Parent-Duchatelet and Martinet,[2] who state that in eight subjects who had exhibited hemiplegia in the beginning of paralysis on one side of the body, they had discovered, on post-mortem examination, effusion on the convexity of the opposite hemisphere.

When, however, the lesion is limited to the base, the functions of the hemispheres will not be affected, except upon the principle of reflex irritation or of the transmission of pressure. It is evident, however, that further researches, based upon post-mortem examinations, are necessary to the satisfactory solution of the interesting questions involved.

Treatment.—To afford any chance of a favorable result, the treatment should be energetic from the first.

General bloodletting may be practised with advantage in subjects of good constitution and of the middle period of life. As much as twelve or sixteen ounces may be taken from the arm if the pulse is hard, the cephalalgia intense, or the delirium furious. Leeches applied behind the ears or to the inside of the nostrils are more generally of advantage. The same may be said of cups to the nucha.

[1] Du Rapport des Symptoms avec les Lesions dans la Méningite. Thèse de Paris, 1859.

[2] Op. cit.

The hair should be cut off short, and ice kept constantly applied to the scalp during the first and second stages. It is better than the cold douche, for the reason that it is almost impossible to continue the latter without intermissions, during which the head again becomes hot. Compresses wrung out of cold water will not answer; they soon get heated, and act as poultices. Irrigation, by a small stream of ice-water falling from a vessel placed above the head of the patient, is a useful means of applying cold, but is often inconvenient.

Purgatives are generally advantageous and should be effective. Nothing is better than croton oil, although calomel and podophyllin, grs. x with grs. ij, make a good combination for the purpose.

My experience has satisfied me of the good effects of mercurialization. I have administered calomel in doses of a grain every two hours until the breath became fetid, and I am sure the effect has been beneficial.

The iodide of potassium is well spoken of by Dr. Flint,[1] who says he has witnessed the good effects of the drug in several cases. Dr. F. R. Lyman[2] has reported two cases in which it formed a prominent feature of the treatment, and in which recovery took place.

Within late years in the few cases of acute cerebral meningitis that have been under my charge, I have found the greatest benefit from the bromide of potassium, and the three cases that recovered were instances in which it was administered in large doses. The theory upon which its employment is based has already been fully considered in the chapter on cerebral congestion. It should be administered in doses of at least thirty grains three or four times a day, from the very beginning of the affection to the end of the second stage or the appearance of coma, should this symptom supervene.

The head should be kept well elevated, the chamber cool,

[1] Op. cit., p. 601. [2] American Medical Times, 1862, p. 334.

and well ventilated, the light in a great measure excluded, and the utmost quiet enjoined.

The food, without being stimulating, should be nutritious. Nothing is superior to strong beef-tea, made either from fresh beef or from some one of the extracts in the market.

In the third stage the treatment should be almost the reverse of that indicated as proper for the first and second stages. The mercury, iodide of potassium, bromide of potassium, ice to the head, and purgatives should be omitted, and attention should be given to the maintenance of the strength. To this end brandy, whiskey, or other alcoholic liquor, should be administered in such quantities as the occasion seems to require. It often happens in this stage that the delirium and excessive motility return. It must be remembered that this is not from any renewal of morbid processes within the cranium, but is entirely due to debility. At the moment of writing this, a young lady of this city is under my charge for acute cerebral meningitis, whom I did not see till the third stage was well advanced, and who for several days previously had exhibited a return of the delirium, for which depletive measures and hydrate of chloral had been employed. The free administration of brandy, champagne, and beef-tea soon dissipated the symptoms of relapse, and she bids fair to recover.

Blisters may be used in this stage with advantage. They are best applied between the shoulders, and should be six or eight inches square.

In the rheumatic form of the disease little special treatment is necessary. It is, perhaps, advisable to endeavor, by means of blisters or other revulsives, to bring back the disease to the joints.

In the acute meningitis of the aged, active depletive treatment is not so generally admissible, and if apparently indicated should be carried out cautiously. It may even be proper to treat some cases with stimulants from the very first.

CHAPTER IX.

CHRONIC CEREBRAL MENINGITIS.

Although sometimes a consequence of an acute attack, chronic cerebral meningitis is more generally an original affection.

Symptoms.—The symptoms of chronic cerebral meningitis are very similar to those indicative of softening. These are headache, somnolency, trembling, defective articulation, feebleness of the limbs, paralysis of the bladder, or of the sphincters of the bladder and rectum, producing involuntary discharges of urine and fæces, weakness of the memory, and general enfeeblement of all the mental faculties. The progress of the disease is slow, and therefore these symptoms may extend over several months or even years. They may, however, at any time be intensified by the development of a more acute form of inflammation.

Chronic cerebral meningitis, when due, as it may be, to syphilitic infection, runs its course more rapidly, and the symptoms are more decided. The third nerve is particularly liable to be involved, and hence there are ptosis, strabismus, double vision, and alterations in the normal size and motility of the pupils. The facial may likewise be involved, causing paralysis or spasms, and the fifth, giving rise to neuralgia, anæsthesia, or destruction of the eyeball from deranged nutrition.

Under the name of "general paralysis" Calmeil[1] described an affection which is not uncommon among the in-

[1] De la Paralysie Considerée chez les Aliénés, Paris, 1826.

sane, and which may be developed in persons not the subjects of mental alienation. A prominent morbid condition in such cases is frequently chronic meningitis, but the peculiarities of general paralysis are sufficiently well marked to require for it a separate consideration.

Causes.—The etiology of chronic cerebral meningitis frequently cannot be ascertained. It is sometimes the result of an acute attack, and, when it arises spontaneously, may be due to the excessive use of alcoholic liquors, or to syphilitic infection. Beyond this our knowledge does not extend.

Diagnosis.—This is often impossible to be made out, with even a moderate degree of exactness, and is always difficult. The affection may be confounded with cerebral softening, and the most careful study will in many cases fail in making the discrimination between them. The difficulty is frequently heightened by the fact that the two diseases coexist.

Prognosis.—This is almost invariably unfavorable, unless a syphilitic origin can be discovered, in which latter case the prospect of recovery is very materially enhanced.

Morbid Anatomy and Pathology.—The essential feature in the morbid anatomy of chronic cerebral meningitis is a new formation of connective tissue, by which the membranes adhere to each other and to the brain, and by which they are rendered opaque and thicker than normal.

In addition there may be deposits of exudation on the convexity or base of the brain, which, though intimately connected with the alterations of the membranes, are yet distinct from them.

The seat of the lesion may probably be determined with more exactness than in the acute form of the disease, for the reasons that the morbid process is extended over a longer period, and that the symptoms accordingly are more discrete, and that they are not marked by delirium and fever. The same principles are applicable to the inquiry, as have already been laid down.

Treatment.—The very active treatment adopted by some practitioners never leads to favorable results, and only seems to annoy or exhaust the patient. The syphilitic variety of the affection may generally be successfully combated by the iodide of potassium and mercury; but the simple, uncomplicated form is quite unamenable to therapeutic measures. For the former there is no better combination than that of bichloride of mercury, with iodide of potassium in solution, so that the one-sixteenth of a grain of the bichloride is taken, with from ten to thirty of the iodide, three times a day. Even if the disease have not a syphilitic origin, this is probably the best treatment which can at present be suggested. In either form antiphlogistic measures are contraindicated. On the contrary, wine and highly-nutritious food are frequently productive of amelioration.

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CHAPTER X.

TUBERCULAR CEREBRAL MENINGITIS.

Inflammation of the membranes of the brain, attended with or due to a deposit of miliary tubercles, was for many years considered as a disease peculiar to infancy, and was known as acute hydrocephalus before its morbid anatomy and pathology were clearly comprehended. It is now well understood to be an affection to which adults are liable.

By some authors, especially Robin and Bouchut, it is regarded as not being tubercular in character. It has hence occasionally been termed granular meningitis. Although mentioned by the ancient medical writers, no clear and systematic description of tubercular meningitis was given till Whyte[1] published his essay on the subject of dropsy of the brain. Since that time it has received the attention of many writers in this country, Great Britain, France, and Germany.

Symptoms.—Whyte defined three periods of the disease, which he marked by the state of the pulse. I think the symptoms may be properly arranged in four stages: 1. The prodromatic stage; 2. The stage of excitement; 3. The stage of depression; and 4. The stage of recurrence.

1. The Prodromatic Stage.—This period may be altogether wanting, or may be so slightly manifested as not to be noticed. Generally, however, it is well marked.

[1] Observations on the most Frequent Form of the Hydrocephalus Internus, viz., Dropsy of the Ventricles of the Brain. Works of Robert Whyte, edited by his son. Edinburgh, 1768, p. 725.

If the child be sufficiently advanced in years, a change of disposition is among the first symptoms perceived. Thus the temper becomes irritable, caresses are disregarded, and dislike is shown for those amusements which formerly gave pleasure. At the same time the appetite disappears, and the child loses flesh rapidly. This latter is not noticed about the face, but is mainly confined to the abdomen and limbs. The bowels are generally obstinately constipated, but occasionally there is diarrhœa. Headache is not often complained of; neither is vomiting a common symptom of this period. Fever is not continuous, although it is generally present at irregular times of the day.

The prodromatic stage may last only a few days, or may be prolonged for two or three months.

2. The Stage of Excitement.—This period is ushered in by obstinate vomiting, which is present in many cases, even though no food be taken. Intense pain in the head is a coincident symptom, and is so severe that the child puts his hands to his head and cries out or awakes screaming. Convulsions may also occur. They do not differ in general appearance from the ordinary epileptic paroxysms, and may be repeated several times.

Very early in this stage the fever becomes more persistent than in the first stage, although it may still be irregular. The pulse, however, is not hard and resisting, as in other inflammatory affections, but is soft and compressible.

Trousseau [1] has called attention to a condition of the skin present in tubercular meningitis, which he at first regarded as peculiar to this disease, but which subsequent investigation showed was likewise found in simple meningitis, in typhoid fever, and some other affections. If the fingernail be passed lightly over the surface of the abdomen or the thorax so as to trace a series of lines, in about thirty seconds the skin becomes red—the color being at first diffused, but very soon the lines made by the nail are indicated

[1] Op. cit., Leçon lv., Fièvre Cérébrale.

by a still redder color, which persists a long time. Trousseau calls this appearance the "cerebral stain" (*tache cérébrale*). The phenomenon he attributes to a profound modification in the vascularization of the skin; and although it is not to be regarded as absolutely pathognomonic, it is a sign of very great importance.

The intellectual faculties are not yet affected to any considerable extent, but the changes of character and disposition, and indifference to things which formerly excited interest, are still well marked.

The physical strength, though lessened, is still not yet so far reduced as to oblige the patient to remain in bed.

The tongue is usually coated and red at the edges, the appetite diminished, and the bowels are obstinately constipated.

The temperature of the body is elevated, but not to an extreme degree; the thermometer indicating from 101° to 103° F. Sometimes there are distinct remissions in the violence of all the symptoms, but the disease nevertheless goes on to its full development. The transmission from the second to the third stage is often marked by an amelioration which may last several days.

From what has been said, it will be seen that the characteristic phonomena of this stage are headache and vomiting. Its duration varies from seven to fourteen days.

3. Stage of Depression. — The pulse, which in the previous stage was sometimes as high as 140, and sometimes as low as 80, now becomes less rapid than is normal, and may even fall below 50. At the same time the beat is quick, but the interval between the pulsations is at times so great that the observer is, as Dance[1] says, fearful that the action of the heart has stopped. The interval between the pulsations is often irregular, and this may be regarded as a sign of unfavorable import.

In young infants there is a reduction in the temperature

[1] Mémoire sur l'hydrocéphale. Archives Gén. de Méd., 1830.

of the body below the normal standard, which lasts throughout the whole of this period. Roger regarded this reduction, preceded as it is by a higher temperature, and followed during the succeeding stage by another elevation, as pathognomonic of tubercular meningitis.

The continued excitement of the previous stage is replaced in this by a strong tendency to somnolence, which alternates with a rather quiet delirium. The patient lies on his back, with the eyes fixed, but yet not looking at any object with attention. Events transpiring around him no longer attract notice, and, though when addressed in a loud tone he may turn his gaze toward the speaker, it is very evident that the words convey no idea to his mind.

The fingers are kept in almost continual motion, picking up threads and other small objects from the bedclothes, and occasionally clutching at imaginary things. Again, the fingers are alternately opened and shut without any real or apparent motive, and again the head is turned restlessly from side to side of the pillow. Convulsions are very generally present from time to time during this stage, and may be so frequently repeated as to leave scarcely any interval between the seizures. Even if the attacks do not involve the body generally, the eyes scarcely ever escape; there being strabismus, convulsive movements of the pupils, and constant motions of the eyeballs. The facial muscles are likewise often affected.

In the intervals of wakefulness, the cephalalgia continues, and causes the peculiar scream which is so characteristic as to have received the name of the "hydrocephalic cry." It is a sound such as might be produced by mingled emotions of terror and grief. Although probably excited by the pain, it is more or less automatic, and is not exactly such a cry as would be produced by unmixed physical suffering. It is accompanied, however, by that contraction of the muscles of the face indicative of suffering.

The paleness of the countenance continues, but at times

there is a sudden redness, which disappears as rapidly as it comes.

The conjunctivæ are generally injected, and photophobia is present. M. Bouchut,[1] who has given great attention to the subject of ophthalmoscopy in diseases of the nervous system, finds peripapillary congestion, dilatation of the retinal vessels, and deformation of the papillæ.

There is often a general hyperæsthesia of the skin, for which, however, anæsthesia may be substituted. When this latter is the case the conjunctivæ participate, and inflammation results.

The limbs are weak, and, should the patient attempt to walk, the gait is staggering. The speech is hesitating, is rarely indulged in except in response to questions, and then with the least possible expenditure of words.

The vomiting, which formed so prominent a symptom of the previous stage, has ceased, but the constipation still persists.

The respiration is irregular, sometimes being rapid and sometimes slow. Occasionally there are deep sighs, followed by numerous quick inspirations, and again the respiratory movements may be so slight as scarcely to be perceived. This variation from the normal action, as well as the irregularity of the heart's movements, is due to the implication of the pneumogastric nerves at their origins.

This stage may last for from two or three days to two weeks.

4. Stage of Recurrence. — The characteristic phenomena of this stage are the return of the fever and the increase in the violence of the symptoms indicative of cerebral disturbance. Before its onset there may be a period of nearly complete intermission, so that the impression may be formed that recovery is taking place. This apparent cessation of the morbid action, however, only serves, with

[1] Du Diagnostic des Maladies du Système Nerveux par l'Ophthalmoscopie, Paris, 1866, p. 45, *et seq.* Plates iv., v., vi., vii., viii., ix., and xi., of the Atlas.

the experienced observer, to make the reappearance of the symptoms more striking.

Convulsions are more frequent and violent than in the previous stage, and tonic contractions of the limbs are not uncommon. These contractions are more generally met with in the muscles of the neck and upper extremities, and vary from time to time in their intensity. The head is thus thrown backward, and, as the morbid action frequently extends to the muscles of the back, an appearance in the patient not unlike that present in tetanus is produced.

Paralysis eventually supervenes. At first this is incomplete, affecting only a single limb or the muscles of the face, but it extends, and both limbs on one side or an arm and a leg of opposite sides become involved. Voluntary power is lost, but reflex movements can be excited by pinching or tickling.

The delirium acquires increased intensity, and alternates with the somnolence, which likewise becomes more profound, and which gradually masks all the other symptoms, till at last the coma is persistent and general, and spinal sensibility is lost.

Before death the pulse rises in frequency, a cold sweat makes its appearance, and the patient dies either by a slow process of asphyxia, or in convulsions.

The fact that tubercular meningitis is not confined to infants is now generally admitted. Dance[1] was the first to recognize its occurrence in adults, and Gerhard,[2] of Philadelphia, a few years subsequently reported several cases. Ledibuder[3] also pointed out the analogy between the tubercular meningitis of infants and that of adults, and still later Valleix[4] gave the weight of his authority to the same effect.

[1] Op. cit. [2] American Journal of the Medical Sciences, 1834.

[3] Essai sur l'Affection Tuberculeuse Aiguë de la pie-mère, Paris, 1837.

[4] De la Méningite Tuberculeuse chez l'adult. Archives Générale de Médecine, 1838.

So far as the symptoms are concerned, I have never been able to perceive any essential points of difference between the tubercular meningitis of children and that of adults.

The affection is, of course, modified, as are all other diseases, by the age of the patient, but, when allowance is made for this factor, the morbid process is one and the same in character. In adults, however, it generally supervenes in the course of tuberculosis of the lungs, whereas in infants it is ordinarily a primary manifestation of the tubercular diathesis.

Causes.—Tubercular meningitis is an expression of a general state of the system. To enter at length into the question of its etiology would necessarily involve a discussion of the cause of the diathesis to which it is essentially due. Nevertheless, there are a number of determining causes that may be appropriately considered. Age is an important factor in determining the accession of tubercular meningitis. It is rare during the first year of infancy, but is more common during the period extending from the second to the seventh year than any other time of life. From eight to ten it is much less frequent, and from ten to fifteen is rarely seen.

In adults it is most common between the ages of seventeen and thirty. From thirty to forty it is rare, and after forty is scarcely ever met with.

Males are more frequently the subjects of tubercular meningitis than females, and this holds good for all ages of life.

The season of the year appears to exercise no influence.

As to many other exciting causes alleged by authors, such as blows, emotional excitement, and previous diseases, nothing very definite is known. The same cannot, however, be said of the morbific influence of bad air, insufficient food, improper clothing, neglect of cleanliness, and a disregard for other sanitary requirements.

Diagnosis. — Tubercular meningitis is liable to be confounded with several other affections, and can sometimes only be distinguished with difficulty.

From simple meningitis it may be diagnosticated by the facts that the onset of the former is sudden, while the latter is insidious in its approach, and slow in the development of its symptoms; the one goes on steadily through its course, the other halts and remits; in the one the temperature of the body rises several degrees, in the other the elevation is scarcely ever more than two degrees; in the one there is no hereditary tendency, while in the other inquiry will usually reveal the existence of hereditary tubercular predisposition.

The mental symptoms show a marked difference. In simple meningitis the delirium is often furious, and is always very active; in the tubercular form of the disease the delirium is quiet, and alternates with stupor.

In typhoid fever there may be vomiting and headache, but the bowels are not constipated, and there is tenderness over the right hypogastric region. Moreover, the epistaxis, the eruption, and the swelling of the spleen, which occur in typhoid fever, will aid in making the diagnosis more certain.

Worms in the alimentary canal may give rise to a set of symptoms very similar to those which form the prodromata of tubercular meningitis. As Jaccoud observes, therefore, it is well, whenever a child exhibits these symptoms, to administer one or two doses of a strong vermifuge.

A peculiar affection, to which young infants are liable, may be mistaken for tubercular meningitis. It was first described by Dr. Gooch,[1] but derived its name—"hydrocephaloid disease"—from Dr. Marshall Hall. I have already alluded to this disorder under the head of cerebral anæmia. In it the child is irritable, restless, starting at every noise, moving in sleep, and often waking screaming. Vomiting is

[1] On Some Symptoms in Children erroneously attributed to Congestion of the Brain. Gooch's Essays, New Sydenham Society, 1859, p. 179.

frequently present, but the bowels are loose. The whole appearance of the child betokens exhaustion, and, if due care be not taken, death may ensue. The absence of constipation, the history of the case, and the depressed state of the fontanelle, if this be yet open, will suffice to render the diagnosis clear.

Trousseau considers the irregularity of the respiration the most important sign indicating the presence of tubercular meningitis. "In no other disease," he says, "will you meet with this singular anomaly. You will not observe this unequal and irregular respiration either in the essential convulsions of infancy or in typhoid fever. I have reason, then, for insisting on the importance of the symptom."

Prognosis.—There is not much to say under this head. The ordinary termination of the disease is death. I have never seen a case recover; and, though instances with a favorable result have been reported, I am disposed to think the diagnosis of such has been erroneous. Drs. Meigs and Pepper,[1] of thirty-one cases, had no recovery, though they report a case of tuberculosis of the meninges—not tubercular meningitis—in which recovery appears to have taken place, though the child died a year or two afterward with dysentery.

It seems contrary to reason to expect a radical cure in a disease in which the cause cannot be removed. Do what we will, the tubercular deposit remains; and, as Jaccoud remarks, the reported cases of recovery were rather instances of a long remission in the intensity of the symptoms.

Morbid Anatomy and Pathology.—A question arises at the outset of an inquiry relative to the morbid anatomy of tubercular meningitis, which refers to the essential character of the disease; and that is, whether the gray semi-trans-

[1] A Practical Treatise on the Diseases of Children. Philadelphia, 1870, p. 452.

parent granulations met with on post-mortem examination are tubercles, or whether they are an entirely distinct morbid product? Valleix, Rilliet and Barthez, Barrier, Grissole, Meigs and Pepper, and others, regard them as tubercles. Grissoll expresses himself clearly on this point. "We have no doubt," he says, "that these granulations are tubercles in a rudimentary state; for we have many times, in the same subject, followed the morbid product in its different phases of evolution from the amorphous condition to the fully-developed tubercle."

On the other hand, Bouchut, basing his conclusions mainly on the microscopical observations of Robin, is of the opinion that the granulations are formed: 1. Of fibroplastic elements, consisting of free nuclei and fusiform cells, and ovoid cells. The nuclei are ovoid or spherical, and generally very small, not exceeding 0.008 to 0.009 in. in diameter. 2. Of a great quantity of granular amorphous homogeneous matter, which keeps the other elements strongly united. 3. Of a few vessels and fibres of connective tissue. Among all these elements the tubercular corpuscles of micrographers are not to be found; and, therefore, the disease cannot be regarded as tubercular in character. M. Empis[1] also contends that the microscopical analysis shows that the gray granulations are entirely distinct from tubercle. On the other hand, it is alleged—and I am disposed to think with force—that the most which the investigations of M. Robin and others in accord with him show, is, that there is no special characteristic of tubercle which will enable us to declare with certainty that it is present, and that it does not possess a structure which is the same in all stages of its development. The collateral evidence goes very far to support the view that the granulations are tubercular in character.

The question which also arises, as to whether the inflammation precedes the tubercular deposit, or vice versa, is gen-

[1] Traité de la Granulie. Paris, 1865.

erally decided in favor of the prior appearance of the tubercles. The granulations are met with in the course of the vessels of the pia mater. This membrane is always more or less inflamed, and is thickened by the infiltration of sanguinous, serous, plastic, or purulent exudations. The granular or tubercular matter is likewise deposited at the base of the brain, and in this position is doubtless the cause of the derangements of motility which constitute so prominent a feature of the disease.

The tissue of the brain is not generally much involved, although on section the red points, indicative of the situation of blood-vessels, are very much increased in number. Occasionally there are small extravasations of blood found in the gray substance.

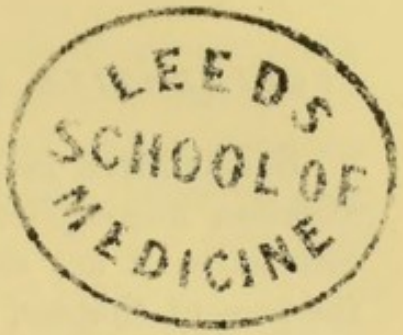
The ventricles are distended by serum, and this is sometimes so great in quantity as to cause the rupture of the septum lucidum. The liquid is either clear and limpid, milky from the presence of pus-globules, or bloody from containing red corpuscles.

The morbid anatomy of the lungs and other organs, although interesting in the present connection, need not be dwelt upon; suffice it to say that tubercular deposits are always met with in some one or more of the viscera and especially in the lungs.

Treatment.—In regard to a disease so uniformly fatal as tubercular meningitis, there is not much to say. The principal advice I have to give is, to refrain from blisters, antimonial ointment, leeches, and drastic purgatives, which have no other effect than to shorten the life of the patient, and to make his existence still more intolerable than it is made by disease. Iodide of potassium does less harm, but I have never known it do any good. Niemeyer, however, recommends it, and many will doubtless continue to employ it on his authority.

When we have any reason to suspect an hereditary tendency to tubercular meningitis, prophylactic measures may

be used with hope of success. These consist in providing for pure air, ample clothing, nutritious food, and in the administration of cod-liver oil, iron, iodine, and quinine. A climate not subject to sudden vicissitudes, and of such a character as regards warmth and dryness that the patient can spend a great portion of the day in the open air, is also a matter of prime importance.


LEEDS SCHOOL OF MEDICINE

CHAPTER XI.

SUPPURATIVE ENCEPHALITIS OR CEREBRITIS.

Suppurative inflammation of the brain is a very rare affection uncomplicated with meningitis. In this latter connection it has already been sufficiently considered. In the present chapter, therefore, I shall discuss it solely as an independent lesion, and mainly in reference to the subsequent formation of abscess.

Symptoms.—The symptoms of suppurative inflammation of the brain vary according to the seat of the lesion, and are rarely of such a character as to enable us to say, with any great degree of certainty, that we have a case of uncomplicated encephalitis before us. Nevertheless, certain phenomena have been recognized, and, after death, the evidences of inflammation of the brain have been discovered. But these symptoms are, many of them, met with in other cerebral disorders, and therefore cannot be regarded as pathognomonic. It is difficult, if not impossible, to arrange them in stages; and therefore, after the prodromata, I shall consider the phenomena of acute encephalitis in accordance with their relation to the several functions of the organism liable to be affected.

The premonitory symptoms are similar to those of cerebral congestion, and doubtless depend upon a like pathological condition. Thus there are vertigo, pain in the head, noises in the ears, troubles of vision, numbness, and difficulties of speech. They never, however, last as long as they do in simple congestion.

Sometimes the first-observed symptom of approaching encephalitis is an epileptiform convulsion.

In the fully-established disease the phenomena are very decided, but at the same time have no necessary or constant relation with the pathology, as similar symptoms are met with in other very different affections.

Disorders of Sensibility.—At first, there is generally hyperæsthesia; subsequently, anæsthesia. Headache is a common symptom, as it is in so many other cerebral diseases. There is no particular location of the pain—sometimes the frontal region, at others the occipital, and again the vertical or parietal regions, being its seat. It varies, likewise, as regards intensity and form, and may consist of a feeling of fulness or constriction only. It is present from the very beginning of the disease, and usually continues through its whole course.

Pains are felt in various parts of the body, are sharp and lancinating, and often attended with cramps. Cutaneous hyperæsthesia is also occasionally met with.

In the next place, there is anæsthesia, with all its accompaniments of formication, numbness, and other abnormal sensations of the kind, mainly affecting the face and upper extremities. As to the special senses, the sight is almost always deranged. There are bright flashes of light, iridescent appearances, and photophobia, all showing increased irritability of the retina. The pupils are contracted, the conjunctivæ suffused, and the eyeballs are the seat of a dull, aching pain. Subsequently, the pupils become dilated, and vision is lost. Ophthalmoscopic examination shows, in the early stages, papillary infiltration, with retinal congestion, and later, papillary atrophy and granular degeneration, the results of optic neuritis. There is also, generally, double vision, to which allusion will be more fully made directly.

The hearing is at first very acute, and even slight noises are more or less painful. Noises in the ears, of various kinds, are present. As the disease advances, the hearing

becomes much impaired, and is gradually lost, in one or both ears.

The taste and smell are rarely affected.

Disorders of Motility.—As with the sensibility, the motor organs of the body at first exhibit evidences of increased excitability. Thus, there are twitchings of the muscles, mainly of those of the face, and clonic or tonic spasms. Sometimes these last for several days. Subsultus is especially noticed in the flexor tendons of the wrist.

General convulsions may take place, with or without loss of consciousness. Frequently the action is limited to one side of the body, or implicates one side of the face, or a single limb. Strabismus occurs, and double vision is produced, at this stage, from spasms of one of the ocular muscles.

This period of muscular excitation corresponds very accurately with the stage of augmented sensibility.

It is succeeded by a period of diminished motor power. Paralysis generally begins in a distant part of the body, and slowly involves one side. Thus, there may at first be a difficulty in raising the toes, or in grasping things with the fingers; then the knee becomes weak, the flexors of the thigh follow, and the whole limb drags. If the arm be the first member affected, the difficulty advances from the fingers to the elbow, and thence to the shoulder. Sometimes the morbid action extends equally on both sides of the body, and then the gait becomes weak and shuffling. The legs are spread wide apart, so as to increase the base, and keep the centre of gravity more easily within it. The knees are bent, the pelvis is flexed on the thighs, and the whole body is inclined forward. The face rarely escapes. It may be affected on one side only, in which case there is distortion, or there may be a gradual failure of muscular power on both sides. The muscles connected with the eyes almost always suffer. Ptosis is common, and external strabismus, causing double vision, accompanies it, both being produced by the implication of the third or motor oculi nerve.

One side of the face sometimes becomes permanently contracted, and thus an appearance is produced somewhat resembling that which is caused by paralysis of the opposite side. It may be distinguished from this latter condition, however, by the fact that in it the eyelids are spasmodically closed, and the side of the face much more distorted than when there is paralysis of the opposite side. The tongue is always, in my experience, prominently affected. The first sign of diminished motility is the frequency with which it is bitten, in conversation or mastication, and sometimes it is made quite sore, on one or both sides, or at the tip, from this cause. Then the patient discovers that long-continued speaking causes a sensation of fatigue, at the root of the tongue, and that a feeling as if this organ were too large for the mouth is experienced. Then articulation becomes indistinct, the words are clipped or slurred over, so that at times it is difficult for others to understand what he says.

Disorders of Intelligence.—The first indication of mental weakness is the susceptibility experienced to the influence of emotions. The patient will thus get uncontrollable fits of laughing or crying from very slight causes, and sometimes from no apparent cause. These paroxysms are frequently of mixed character, the patient passing from laughing to crying, and *vice versa*.

The memory begins to fail at a very early period, especially as regards the names of things. The enfeeblement is by no means, however, confined to words, but facts and circumstances likewise fail to be remembered. Gradually a condition of complete dementia ensues, and finally coma, with or without previous or alternating delirium.

Disorders of the Functions of Organic Life.—There is always febrile excitement in encephalitis. At first the pulse is frequent, rising to 120, but as the disease advances it falls till toward the close it goes below the normal standard. It is characterized, according to Barras,[1] by a characteristic

[1] Bulletin de la Société Médicale d'Émulation, Juin et October, 1823.

tremulousness (*tremblottement*), which he compares to the unequal vibrations of a cord moderately stretched. This peculiarity he attributes to irregular arterial dilatation. According to my experience, the symptom is by no means constantly met with, and it certainly is not pathognomonic, for the same peculiarity of pulse is found in several other disorders. In a case, however, now under my care, in which there is reason to suspect encephalitis and abscess, the phenomenon is present in a marked degree, not only in the radial artery but in the temporal and the angular, as it passes between the nose and the inner angle of the orbit.

The respiration in the first stages is not materially deranged, but later it becomes irregular and stertorous, and finally asphyxia may take place.

The temperature of the body is elevated till the fever abates, and paralysis makes its appearance. The thermometer rarely, however, goes above 103° Fahr., and is generally a degree below this point.

The digestive organs usually show more or less evidence of derangement. Constipation is always a prominent feature, and the appetite is capricious. At times the patient refuses to eat, at others he will cram his stomach with all kinds of edibles. Digestion is often troublesome, and occasionally dangerous from paralysis of the pharyngeal muscles. Cases are on record in which death has occurred by the food becoming impacted in the throat, and several cases have come under my own notice, in which, from a like cause, a fatal result was barely prevented by the use of very energetic measures.

Moreover, the secretions of the mouth are almost always altered either in quantity or quality, or both, and the sensibility of the tongue and faucial mucous membrane is often impaired. Hence, the patient is not aware that he has filled his mouth, and goes on cramming it with food, which makes an alimentary mass larger than can pass through the œsophagus. This course, even without the pharyngeal paralysis, in-

terferes with the act of swallowing. The fæces are sometimes passed involuntarily, but this is almost entirely a feature of the last stage. Nausea and vomiting are present more or less from the very first.

There may be either retention of urine from paralysis of the bladder, or incontinence from paralysis of the sphincter. Or both conditions may coexist, giving rise to a constant dribbling.

These symptoms may be arranged in five classes, designated by the most prominent feature of each : the *paralytic*, the *comatose*, the *epileptiform*, the *apoplectiform*, and the *maniacal*.

Complications may and often do arise. Thus there may be meningitis, temporary congestions, extravasation of blood, effusion of serum, or some intercurrent visceral affection.

The tendency of acute encephalitis is to suppuration and the consequent formation of abscess, and many of the symptoms enumerated are due to the supervention of this condition. Death ensues gradually from exhaustion or asphyxia, or may take place suddenly from the bursting of the abscess into the ventricles, or upon the surface of the brain.

Causes.—No age is exempt from the disease, although it is more common in old persons than in adults of middle age or young persons.

It is probably more common in males than females solely from the fact that they are more subject to the exciting causes of the disease. Among these are the inordinate use of alcoholic liquors, venereal excesses, extreme intellectual exertion, great emotional disturbance, and exposure to extreme heat.

It may also be induced by disease of the internal ear, by erysipelas affecting the head, or by severe attacks of scarlet-fever, small-pox, or other eruptive disease.

The most common cause, however, is injury of the brain.

Diagnosis.—The diagnosis of suppurative encephalitis is, in the first stages, difficult if not impossible; the symptoms

being common, as I have already said, to several other disorders. From cerebral hæmorrhage the distinction can be made without difficulty, for, although encephalitis may be developed with rapidity and by an apoplectic seizure, the tendency is for the subsequent phenomena to become progressively more marked, while in hæmorrhage there is a gradual amelioration. The pulse in hæmorrhage is from the first slow and regular, unless the medulla oblongata be the seat, while in encephalitis it is rapid and irregular.

Meningitis is always associated with superficial encephalitis, and hence the symptoms bear a certain amount of resemblance to those of the affection under consideration. But the latter is, in general, characterized by the facts that the paralysis is more defined, both in intensity and location; that the delirium is less acute; that the cephalalgia is not so intense, nor the delirium so prominent or constant a phenomenon.

In epilepsy the paroxysm is the main phenomenon of the disease. When this ceases, the patient in general recovers his ordinary mental faculties, but the epileptiform seizures of suppurative encephalitis are never followed by complete intellectual restoration.

The disease with which it is most likely to be confounded is that which, from its obvious characteristics, is denominated general paralysis. I know of no diagnostic marks between the two conditions, except that general paralysis is usually of longer duration, and is especially apt to affect the insane.

The symptoms due to tumors are often almost identical in character with those attendant on abscess. The history of the case is our only safe guide. The fact that the brain has received an injury of some kind will indicate suppurative encephalitis as the probable difficulty. A lady is, at the moment of writing this, under my charge, who has been successively treated by several of the most skilful diagnosticians of this city, at times for abscess, and again for tumor,

and I venture to say that no one, without the aid of a post-mortem examination, can say which lesion exists.

Prognosis.—Suppurative encephalitis is invariably fatal, if the disease does not terminate in resolution. As Jaccoud, however, remarks, cases of alleged cure before the stage of suppuration is reached must always have an element of uncertainty about them, and do not therefore permit us to mitigate the unfavorable character of the prognosis. Drs. Gull and Sutton,[1] while stating that there is nothing in the morbid anatomy of cerebral abscess which makes it necessarily an incurable affection, admit that practically it is irremediable. In this opinion I unhesitatingly concur.

Morbid Anatomy and Pathology.—Suppurative encephalitis is a local disease restricted in its action, and hence affecting a limited and well-defined region of the cerebral tissue. This may vary from the size of a walnut to that of the closed fist, and is ordinarily irregularly spherical in shape. Although never of a diffused character, there may be, at the same time, several centres of inflammation. The part most frequently affected is the gray matter of the cerebrum—the morbid process involving the white substance in its progress. Next, the cerebellum appears to be a favorite seat. The corpora striata, and the optic thalami, are also frequently involved.

It sometimes happens that the pus which results from the inflammatory action is not collected in a cavity, but is infiltrated into the subjacent tissue. In such cases there is no well-defined abscess, but a pulpy mass is found on examination after death, consisting of the elements of the brain-substance in a more or less disorganized condition, with those of the blood intermingled with pus—the whole of a greenish-yellow color.

Again, there may be a collection of pus, but at the same time the walls are imperfectly formed, and there is infiltration to some extent. Lastly, the puriform deposit is entirely

[1] Abscess of the Brain. Reynolds's System of Medicine, vol. ii., p. 544.

limited by a membrane consisting of connective tissue, and forming a cyst. The cerebral substance in contact with the walls of an abscess gradually breaks down, and hence the cavity undergoes constant enlargement in all directions, but especially in the lines of least resistance. If the abscess is near the surface of the hemisphere, the tendency is to enlarge toward the external periphery; if it is situated in the central part, in the corpora striata or optic thalami, the absorption of the peripheral tissue takes place in the direction of the ventricles. In the first instance, when the rupture ensues, the pus will be extravasated into the cavity of the arachnoid; in the second, it will be poured out into the ventricular cavities. In either case, coma and death will result if the amount of pus be sufficiently large. It has happened that the pus has escaped from the cranium by the nose or ear. A lady now under my charge experienced this result several weeks since; a large quantity of purulent matter making its exit through the posterior nares. She is still alive, in full possession of her reasoning faculties, and her articulation perfect, but with the loss of sight in both eyes, paralysis of the right side of the face, the left arm, and leg, and suffering the most intense and constant pain in her head. The seat of the lesion is probably partly in the right half of the pons Varolii. The suppurative action is doubtless still going on, and I regard her death as inevitable.

The substance of the brain in contiguity with the abscess, as already stated, undergoes disintegration. This is in the nature of softening.

CHRONIC CEREBRAL ABSCESS.

Suppurative inflammation of the brain, terminating in the formation of abscess, may be of a chronic character, the course of the disease extending over several months. This is especially apt to result from disease of the internal ear.

Cases have been reported by Abercrombie,[1] Lallemand,[2] Toynbee,[3] Ribière,[4] and others, and three have come under my own observation.

Chronic abscess may also result from injuries of the brain or skull, and from suppuration set up around a clot due to extravasation of blood.

As in the acute form of the disease, there are no very characteristic symptoms indicating the formation of abscess. Indeed, in some cases there are no symptoms at all referable to the brain for the whole period of the course of the disease, till a short time before death. A great part of a lobe may be destroyed, and even both anterior lobes almost entirely obliterated, and the patient continue to manifest his ordinary degree of intelligence.

Ribière[5] has collected a number of interesting cases, several of which almost overturn some of our most definite ideas of cerebral physiology and pathology. Thus, he cites (Observation II.) the case of a man who entered the Hôpital de la Pitié, January 27, 1866. The patient was depressed, answered questions with difficulty, and complained of a violent pain in the head. The symptoms were supposed to indicate the existence of typhoid fever. Two days subsequently a purulent discharge was noticed from the right ear, and, the pain in the head persisting, the diagnosis was changed to suppurative otitis, with probable caries of the petrous portion of the temporal bone. Leeches were applied behind the ears and purgatives administered, after which the patient felt so far well that he determined to leave the hospital. He went to work again, and, on the 12th of February, attended a ball. The following morning, pus, mixed

[1] On Chronic Inflammation of the Brain and its Membranes. Edinburgh Medical and Surgical Journal, vol. xvi., 1818, p. 265, *et seq.*

[2] Op. cit., p. 80, *et seq.*

[3] The Diseases of the Ear, etc., Philadelphia, 1860.

[4] Des Abcès de l'Encéphale Consécutifs à la Carie du Rocher. Thèse de Paris, 1866.

[5] Op. cit.

with blood, was discharged from the right ear, and, the tendency to stupor reappearing, he again presented himself at the hospital. It was then ascertained that the flow from the ear had begun several years previously, but had ceased for the two years immediately preceding his first entrance into the hospital.

On the 14th he was in a state of not very intense stupor, since he was able to complain of the pain in the head; his pulse was 60, full and hard, and pus was passing from the right auditory canal. By the 16th of February the stupor had increased. There was no paralysis, deviation of the face, nor alterations of sensibility. The patient understood questions put to him, but answered slowly and imperfectly. The eyelids were closed, light appeared to be unpleasant, and the purulent flow still continued. He died at nine o'clock that night, without convulsions.

The post-mortem examination of the head revealed the following condition:

The external auditory canal was filled with desiccated purulent matter; there was neither abscess nor abnormal redness about the ear.

The superior longitudinal sinus was gorged with blood, the veins were black and dilated; the brain appeared congested, but a yellow tint of the right cerebral lobe was noticed. At the inferior face of this lobe, where a rupture had occurred in handling the brain, a quantity of pus estimated at one hundred grammes (about three ounces) flowed out. This was of a greenish color, and of offensive odor. The cavity left was about the size of a hen's egg, and was bounded by red, indurated, and thick walls. The pus, which during life had flowed from the auditory canal, had not come from the abscess, but from the carious petrous portion of the temporal bone.

Around the abscess the substance of the brain was yellow and softened. Three-fourths of the middle and posterior lobes were infiltrated with pus and softened in texture.

The capillaries were not visible to the naked eye; the convolutions of the island of Reil were not recognizable, and the neighboring convolutions were not now distinct. The corpus striatum of the right side was healthy in its anterior fourth. In the rest of its extent it was softened. The optic thalamus was also softened, as were likewise the roots of the optic nerve. We see that, in this case, as Ribière remarks, a considerable abscess had destroyed, in great part, the corpus striatum and optic thalamus, and that, nevertheless, the patient had been able to work till within a few days of his death, and was so slightly paralyzed as to be able to attend a public ball. Aside from a certain hebetude, the intellectual faculties were not deranged.

Another patient observed by Ribière presented an entire absence of cerebral troubles, no paralysis, no contractions, no convulsions; the sensibility was intact, and the intelligence was active. Nevertheless, there was a degree of stupidity expressed in the countenance, and the expression was dull.

Still there is almost always some pain in the head, which may be irregular as regards its location and character, or may be confined to one particular spot.

In one of the cases under my observation, there was very acute pain, almost constant nausea or vomiting, a strong tendency to coma, and hemiplegia of the left side, coexisting with purulent discharge from the right ear. The patient, who had a short time previous suffered an attack of scarlet fever, to which the ear-trouble was due, died suddenly comatose, but without convulsion. Examination after death showed the existence of caries of the petrous portion of the temporal bone, and an abscess containing about two ounces of pus in the middle lobe of the right hemisphere. The right corpus striatum was softened in about half of its extent.

In the other case there had been profuse discharge from the right ear for several years, unattended by any cerebral

symptoms except occasional pain and headache, which were supposed by the family to be due to gastric derangement, and for which no medical advice was ever asked. One morning the patient, a young lady, twenty years of age, was suddenly roused from bed by an alarm of fire. In her hurry to dress herself, and in the confusion of the moment, she struck her head against the edge of an open door. She immediately felt a severe pain in the head and cried out, but almost instantly sank down to the floor in a stupor, from which she never emerged, death ensuing within five hours. On removing the calvarium a large extravasation of pus was discovered under the arachnoid, covering the right hemisphere, and it was discovered that an abscess, the cavity of which was as large as a small orange, had occupied the middle lobe, and had burst through the convex superior surface by rupturing the cerebral substance. The petrous portion of the temporal bone of that side was carious, and communicated by several very small openings with the abscess.

When speaking of cerebral hæmorrhage, I have referred to another case in which there was abscess of the cerebellum, produced by injury of the skull. In this instance there were notable symptoms, vertigo, convulsions, nausea, vomiting, and violent pain in the back of the head. At first there was no paralysis, but the patient subsequently became paraplegic, and died in convulsions. Examination after death disclosed an abscess, the cavity of which comprehended nearly the whole of the left lobe of the cerebellum.

Although recovery from chronic abscess of the brain never takes place, yet life is often prolonged for several years, even when there may be marked symptoms of cerebral disorder. And then when death occurs it is generally suddenly, with or without obvious exciting cause.

Treatment.—The treatment of acute suppurative encephalitis is altogether palliative. Symptoms, such as pain, vertigo, and vomiting, may be controlled to a certain extent. I have derived considerable benefit from the extract of In-

dian hemp, given in conjunction with the bromide of potassium. The doses of Squires's extract may range from half a grain to two grains three times a day, with from thirty to forty grains of the bromide, either of potassium or sodium. The pain and irritability of the nervous system are greatly lessened by these remedies, and thus the patient's condition rendered more tolerable.

When there is reason to suspect a syphilitic origin, mercury and iodide of potassium may be administered theoretically with some prospect of success, but practically with very little benefit. The medicines should be given in frequently-repeated doses—calomel being the preferable mercurial—so as to bring the system, as soon as possible, under their influence.

Bloodletting, local and general, blisters, tartar-emetic, and other measures calculated to depress the powers of the system, are worse than useless.

In suppurative disease of the internal ear, probably due to caries of the petrous portion of the temporal bone, preventive measures against chronic abscess may do something. Leeches applied to the mastoid process, and blisters behind the ear, are indicated, and mercury with iodide of potassium will afford a chance of a beneficial result. The solution of the bichloride of mercury with iodide of potassium in water constitutes an eligible preparation. The flow of pus should be facilitated, and the propriety of trephining the mastoid cells may be a question for consideration. The management of injuries, with a view to preventing abscess, is to be conducted upon very obvious surgical principles.

CHAPTER XII.

DIFFUSED CEREBRAL SCLEROSIS.

By diffused cerebral sclerosis is to be understood a morbid condition of some part of the brain characterized by induration and atrophy of the tissue, and not distinctly circumscribed except by the anatomical limits of the region affected.

It is not a disease which can be recognized with any great degree of certainty or even of probability during life. Is is, however, a well-marked pathological condition, giving rise to very prominent symptoms. Of late years the affection has not been much noticed, except incidentally, by a few writers of special treatises—though, under the name of "induration of the brain," it received considerable attention many years ago.

The symptoms by which it is characterized are by no means peculiar to it, though, when taken collectively, they give us some reason to diagnosticate sclerosis as their cause. A number of cases have come under my observation in which the lesion was probably diffused cerebral sclerosis; but I have never had the opportunity of verifying my diagnosis by post-mortem examination. The remarks, therefore, which I shall make on the morbid anatomy will mainly be based upon the studies and observations of other writers.

Symptoms.—The symptoms of diffused cerebral sclerosis, like so many other brain-affections, are connected with the mind, with sensibility, and with the power of motion. It generally makes its appearance during infancy, and produces

an arrest of development in the part of the brain affected, and consequently in certain parts of the body. The initial phenomena are those of congestion and inflammation, during the course of which epileptic convulsions frequently ensue. These may be few in number, and may cease in a few days, or they may be very frequently repeated and last for several years, or during the whole life of the patient. The mind remains undeveloped, speech, if already acquired, often becomes imperfect, and, if not yet present, may never be commenced. The limbs, usually only on one side of the body, become paralyzed, and do not grow with the same rapidity as those on the sound side. Contractions are very apt to take place, from the fact, probably, that the normal degree of antagonism between the muscles is destroyed, and that those not so much paralyzed as others draw the limbs in the direction of their action. It is quite common, therefore, in the affection under consideration, to find the fingers drawn into the palm of the hand, the wrist flexed on the forearm, the forearm on the arm, and the arm drawn backward by the action mainly of the latissimus dorsi. In the lower limbs, club-feet are produced in a similar manner.

It is not uncommon, too, to find one or more senses weak or altogether lost, and the general sensibility of the body diminished on one side.

The urine and fæces are often passed involuntarily, or else the patient, from never having acquired a sense of propriety or cleanliness, passes them whenever he chooses, at any time or place.

With this general idea of the symptoms, I proceed to refer somewhat at length to its history, in the course of which I shall quote several cases in illustration of its progress.

The first to direct specific attention to the disease under consideration was M. Pinel,[1] the younger, who, in a memoir read before the French Academy of Sciences, May 27, 1822,

[1] Recherches d'Anatomie Pathologique sur l'Endurcissement du Système Nerveux. Journal de Physiologie de Magendie, t. ii., 1822, p. 191, *et seq.*

brought forward several cases in illustration of what he denominated "induration of the brain." I quote the first case in full as a typical example of the affection:

Beler, aged eighteen years, an idiot from birth, was admitted into the Salpêtrière Hospital, June 1, 1821. The patient was paralyzed in the left arm and leg. She could not use this arm, for the hand was strongly flexed on the forearm, and could not be extended. She walked with great difficulty, dragging the left leg. Her intellectual faculties were very much restricted; she comprehended only the questions which were addressed to her relative to her health, her intelligence not extending beyond that point. She had also great difficulty in articulating the words yes and no, which were the only words she could speak. She had no particular habit, was always calm and tranquil, and had to be anticipated in all her wants. She was subject to occasional attacks of epilepsy; but, when the paroxysms came on, she had fits almost without intermission for thirty or forty hours. They returned about every twenty-five days. On the 4th of December, 1821, the patient was taken with a series of epileptic fits, almost continual in character, which lasted during four days, the paroxysms succeeding each other with inconceivable rapidity. During these continuous convulsions the right limbs were affected with violent movements. The left limbs, which had been paralyzed for a long time, were also strongly agitated, and the general sensibility was abolished. The face was red, the eyes were twisted, the dejections were passed involuntarily, the pulse was frequent and irregular, and the respiration unequal and jerking. The patient died on the fourth day, without there having been any remission in the symptoms.

Post-mortem Examination.—"General marasmus; remarkable emaciation of the paralyzed limbs. The cranium was thick, eburnated, and very hard to break. The meninges were pale and healthy. The right lobe [hemisphere] of the brain was very much smaller than the left, it was

atrophied; the convolutions were almost obliterated and very small, especially in the frontal and occipital regions. They were large and deep in the inferior part. The cortical substance was thicker than it generally is; the lateral ventricle was very small and dry. The substance of the brain, throughout the whole extent of this right lobe [hemisphere], and notably above the ventricle, was of remarkable hardness, and it was torn with difficulty by the fingers, the tissue separating in longitudinal bands which converged toward the corpus striatum.

"The left lobe [hemisphere] of the brain, much more developed than the right, was of the .softness and consistence of the healthy brain-tissue, and this condition made the alteration in the right lobe [hemisphere] more obvious."

The rest of the description refers to other organs.

In regard to this case, M. Pinel remarks that to the pathological condition, the loss of the power of motion in the whole of one side, the almost complete annihilation of the intellectual faculties, and probably the epileptic fits, are to be ascribed. The condition—which is frequent with idiots, but of which it is often difficult to estimate all the various symptoms—is ordinarily revealed less by the paralysis of the limbs than by the distortions which it determines in the feet and the hands. Three other cases are adduced, in one of which the cerebellum was also in part indurated. M. Pinel, as the result of his observations of the morbid anatomy, states that the nervous tissue resembles a compact inorganic mass; its consistence and density are those of hard-boiled white-of-egg; the cerebral substance is atrophied; it appears entirely deprived of blood-vessels—the eye perceiving no trace of capillaries. The induration appears to affect more particularly the medullary substance than the gray substance; it was never observed in this last-named tissue.

Griesinger,[1] under the name of "diffused hypertrophy

[1] Die Pathologie und Therapie der psychischen Krankheiten. Zweite Auflage, 1861, p. 301. Also New Sydenham Society translation, p. 359.

of the connective tissue of the brain," describes the affection now under consideration, and refers to an interesting case reported by Isambert,[1] in which a microscopical examination of the altered tissue was made. It occurred in an idiotic child, two years of age. The ventricular walls, the great ganglia, the pons and peduncles, were solid and hard; their tissue was elastic, like caoutchouc; the nerve-tubes in the white substance were almost completely destroyed and an amorphous granular substance occupied their place; there also existed newly-formed fibrous connective tissue. In regard to such cases, Griesinger remarks that, when we are told that a hitherto healthy and well-developed child, about the period of dentition, or during the second or third year, suddenly became feverish, was attacked with convulsions and delirium, fell into a slightly soporific state, and soon afterward apparently recovered, but with the intellectual and physical development checked, the condition may be due to one of two morbid processes: either there are slight congestion and inflammation of the membranes, or there is encephalitis, which, after passing out of the acute stage, suspends further development in the affected parts. The mind, therefore, ceases to expand; walking, if begun, is arrested; speech remains as it is, or is altogether lost; one side of the body does not grow so fast as the other; and convulsions, paralysis, and contractions, are present.

A case in point, referred to by Griesinger, I quote from Calmeil:[2]

"M. Alfred, born at Havre, single, aged twenty-two years, came to the Bicêtre, where he resided twenty-two months: he had been an invalid since infancy.

"Until about three years of age, he had exhibited no peculiarity as regarded intelligence—resembling other children of his years.

"At this period, however, he was attacked with measles,

[1] Compt. rend. et Mém. de la Société de Biologie, t. ii., 1856, p. 9.

[2] Traité des Maladies Inflammatoires du Cerveau, Paris, 1859, t. ii., p. 411.

which was considered mild in form, and from which he had nearly recovered, when he was seized with a succession of severe eclamptic paroxysms. During twelve hours, it was impossible to rouse him from the coma, and general convulsions were present almost without interruption.

"The day after, it was perceived that he was deaf, blind, and incapable of articulating the least sound; the convulsions had ceased.

"At the end of fifteen days he recovered his hearing; after a year he could say a few words; but the retinæ continued insensible to impressions of light.

"It was now perceived that he walked with a certain degree of difficulty, and that he could hardly use the right hand. At times, also, he lost consciousness, but without falling, and it was subsequently recognized that these attacks were epileptic.

"Until the age of thirteen, the intelligence of M. Alfred underwent scarcely any development, and he remained imbecile notwithstanding all the efforts made for his improvement. He nevertheless acquired a knowledge of a certain number of words, and he could make himself understood whenever he had a want to gratify.

"At the age of nineteen he presented the symptoms of an almost complete state of idiocy. He comprehended some things, and could imperfectly articulate a few words. He was not evilly disposed, but he was incapable of attending to his person, and even of eating without assistance.

"He could take a few steps by supporting himself against the wall, on articles of furniture, or a cane, but he dragged his feet on the ground, and his right leg appeared to be weaker than the left. The right arm was contracted and almost immovable. Tactile sensibility was not affected anywhere. He did not appear to perceive objects placed immediately before his eyes, and the pupils were dilated and insensible to the sudden accession of light. As regarded the bladder and rectum, he evacuated them without

seeming to exercise the least restraint of cleanliness or propriety.

"The epileptic paroxysms occurred with long intervals between them, and presented no characteristics worthy of special mention. The complexion was pale, and the body emaciated and notably weak.

"During the month of January, 1827, there was frequent cough, combined with abundant expectoration, diarrhœa, and other symptoms of phthisis." He died in February of the same year.

Autopsy.—The whole of the right side of the body was much less developed than the left side. The right arm and leg were especially emaciated and thin. "The face was free from distortion, and the cranium, without being deformed, was small and very narrow. The greater part of the cranium was abnormally thick, and contained an excessive amount of calcareous matter.

"The dura mater was without change, and did not adhere to the osseous surfaces.

"A very considerable quantity of serum was infiltrated into the meshes of the pia mater—principally toward the middle and convex surface of the two cerebral hemispheres. The pia mater was thickened, but was not adherent to the convolutions.

"The left cerebral hemisphere was notably smaller than the right; the posterior lobe being particularly remarkable for its diminution. The convolutions were flattened, and were as thin as the blade of a knife, were resistant to the touch, and were of a clear yellow color. The middle and anterior lobes were neither of them of ordinary size.

"The posterior lobe of the right hemisphere was less developed than in a healthy brain, but the number of atrophied convolutions was small.

"On cutting into the left posterior lobe with a bistoury, its tissue was found to be white, compact, homogeneous, and very resistant. It might be said that the cerebral sub-

stance had become doughy, and that an element, foreign to its nature, gave it an excessive degree of hardness.

"On the right, the atrophied convolutions of the posterior lobes were difficult to cut; their structure was compact, but the induration of the nervous tissue did not extend deeply into the thickness of the lobe.

"In all other parts of the brain the white and the gray substance, as well on the left as on the right side, were apparently, in all respects, in a healthy condition.

"The corpora striata and the optic thalami were free from change, either as regarded their volume or their structure.

"The pons Varolii, the tubercula quadragemina, and the peduncles of the cerebrum, and cerebellum, were in a normal state.

"The spinal cord relatively, and perhaps even absolutely, appeared to be larger than was natural.

"The optic nerves were atrophied, of a glossy white color, and very hard."

Other cases, similar in general features, are adduced by Calmeil.

In the very interesting monograph of Cotard,[1] to which reference has already been made, the relation of sclerosis to atrophy of the brain is clearly pointed out. As indicating a certain set of symptoms, in existence with a definite pathological state, I quote the following case, No. XXIX of his series:

"C., aged fifty-eight years, an inmate of the Salpêtrière since 1828, entered the infirmary April 25, 1865, under the charge of M. Charcot.

"She gave the following information, which she said she had from her mother, and from other persons who had brought her up: At the age of eighteen months she had three attacks of convulsions, which left her paralyzed on her right side. She had never had convulsions since. She

[1] Étude sur l'Atrophie partielle du Cerveau, Paris, 1868, p. 49.

had already begun to walk when the seizures took place, but she did not walk again till she was three years old.

"According to the information given by the superintendent of her ward, who had known her since her entrance into the hospital, her intelligence had always been weak; she was incapable of attending to herself; she could read tolerably well, and could sign her name; she had always spoken without difficulty.

"She had been employed with coarse sewing, and had invariably been docile and attached to those who took care of her.

"Her health had always been good, though she had, when about the age of twenty-five or thirty, several attacks of hysteria. Menstruation had been regular, and had ceased when she was forty-five.

"For about a year the patient had been the subject of frequent attacks of vomiting, or of epigastric pain. At the time of her admission to the infirmary, she was very much emaciated and very cachectic.

"Her intelligence did not appear to have been recently enfeebled; she could read, sign her name, and speak without difficulty.

"Her senses seemed to be intact; sight was good in both eyes, and the pupils were equal. There was no facial paralysis, and the tongue was protruded straight.

"The right arm was emaciated, atrophied, and contracted; the forearm was pronated and semi-flexed on the arm; the hand was flexed on the forearm, and inclined toward the ulnar side; the fingers were flexed in the palm of the hand, particularly the ring and little fingers; the index-finger was semi-flexed, and the thumb was extended.

"It was possible, without very great force, to bring the several parts of the limb almost into a state of extension, but, as soon as it was left to itself, it resumed its habitual position. The patient could execute a few movements with the shoulder and the elbow, but the wrist was absolutely

paralyzed, and the fingers could only be moved to a very limited extent.

"The right leg was less atrophied, and there was no other deformity than a talipes equinus. The patient walked with a cane.

"The sensibility of the right side was intact, and no very notable difference of temperature was observed between the healthy and the paralyzed sides.

"The patient died May 17th, after symptoms of acute peritonitis.

"*Autopsy.*—Cancer of the stomach, circumjacent abscess, purulent peritonitis.

"No exterior deformation of the cranium; on the left side its walls were thick, doubly and triply so at some points; the frontal sinus extended to the left of the mesial line, and communicated with a large cavity situated in the orbital arch, which was composed of two thin osseous lamellæ.

"The left middle fossa was smaller than the right, and the right cerebellar fossa was smaller than the left.

"The dura mater being incised, a large quantity of serum escaped from the left side. The left hemisphere was very small, shrivelled, and in length and breadth scarcely two-thirds the corresponding dimensions of the right hemisphere. The convolutions were pressed together, were hard, and of a whitish color.

"On the external face of the middle lobe, behind the posterior marginal convolution, and on the prolongation of the fissure of Sylvius, there was a deep depression running upward and backward, and three or four centimetres in length. At the bottom of this depression the convolutions were reduced to little ridges, which were hard, and of a yellow color. The ventricle was considerably dilated; the corpus striatum did not appear to be perceptibly diminished in volume, but the optic thalamus was hardly one-fourth as large as that of the opposite side. There was considerable

atrophy of the left crura of the fornix, and of the mammary tubercle.

"The olfactory and optic nerves of the left side were apparently healthy; the tubercula quadrigemina were not atrophied.

"The right hemisphere was healthy.

"The right hemisphere of the cerebellum and the middle cerebellar peduncle of the same side were atrophied."

Examined with the microscope, the indurated convolutions of the left hemisphere presented an enormous quantity of amyloid corpuscles and of nuclei of connective tissue.

The following cases I select from others of similar character which have occurred in my own practice:

Case I.—J. S., a boy, aged five years, was brought to me in the autumn of 1869, to be treated for epilepsy. The paroxysms occurred several times a day, and had originated when the child was two years of age, in consequence, as the mother thought, of a fall.

At that time he could say a number of words, and was rapidly learning to talk; his intelligence was good, and he had been walking for several months.

But after the first convulsion he ceased to speak and to walk, though he continued up to the time I first saw him to give his attention to very striking objects, such as noisy tops, bright-colored articles, and, above all, music and soldiers. During this period he had at least six exacerbations, characterized by pain in the head, repeated convulsions, and coma.

When he was about two years and a half old it was observed that he did not move the left arm and leg so freely as the right, and soon afterward he ceased to move them at all. The toes then began to be drawn under the sole of the foot, and the heel was raised. Then the leg became flexed on the thigh, and soon afterward the fingers of the left hand and thumb were gradually bent so as to press strongly against the palm. The wrist followed, and then the forearm. Both limbs were greatly atrophied.

When he came under my examination he was having epileptic convulsions, both of the *grand* and *petit mal*, every day. There was no deformity of the skull, though it was certainly small for his age. His mind was feeble, and he did not give attention to any remarks made to him, but bright objects at once attracted his gaze, and he made efforts to get hold of them.

I examined the fundus of the eyes with the ophthalmoscope, and discovered an anæmic condition of the retinæ and atrophy of both optic disks.

I gave it as my opinion that the child was suffering from diffused cerebral sclerosis, involving the left hemisphere; and that there was scarcely any prospect of material amelioration in his mental or physical condition.

Case II.—A female, aged eight years, entered the New York State Hospital for Diseases of the Nervous System, June, 1870, having previously been a patient at my clinic at the Bellevue Hospital Medical College. When quite an infant she had suffered from epileptiform convulsions, which had been almost immediately followed by paralysis of the right upper and lower extremities. The convulsions recurred at short intervals, and atrophy of the paralyzed limbs, with contractions of the fingers, hand, and forearm, supervened. She learned to walk, however, quite well, and also to talk without any very notable defects.

Her mind was weak, and she was extremely silly in her behavior; she had never learned to read.

Under the use of the bromide of potassium her epileptic paroxysms ceased, but the contractions and atrophy of the right arm resisted treatment by galvanism and mechanical appliances. The leg acquired much more power under the treatment than it had previously possessed.

Case III.—W. W., a gentleman, aged forty-three, came to me, December 11, 1869, to be treated for what his physician and friends regarded as softening of the brain.

About six months previously he had experienced, on

awaking in the morning, great difficulty in extending the left hand and fingers, and through the whole day there was a decided tendency manifested for the latter to close and the hand to be flexed upon the forearm; and this gradually, day after day, became stronger, till at last neither the hand nor fingers could be extended.

Then the corresponding lower extremity became involved in a similar manner, and about a month after noticing the first symptom he had an epileptiform convulsion, and this was repeated twice the following day. Since then the fits have occurred at intervals of four or five days. With the contractions in the limbs of the left side there was gradually-advancing paresis until, when he came under my observation, both arm and leg were almost completely paralyzed. Atrophy of both extremities was present to an extreme degree, and sensibility and electro-muscular contractility were almost entirely abolished.

His mind was also notably impaired. He laughed immoderately at every question I put to him, and had a decided expression of imbecility. His speech was not affected to any remarkable degree, except as regarded extreme slowness of utterance. He had previously to his illness been a ready and quick speaker. My diagnosis was diffused cerebral sclerosis, and I gave an unfavorable prognosis. The treatment, which will be considered under its proper head, was, however, successful to a very considerable extent.

It will be seen, from the foregoing account of the symptoms, that diffused cerebral sclerosis is characterized mainly by weakness of intellect, paralysis, and muscular contractions.

Causes.—The predisposing causes of the affection under consideration are not thoroughly understood. The disease appears to be much more frequent in infancy, although it lasts to the period of old age, and sometimes originates at an advanced time of life.

The exciting causes are likewise imperfectly known. In-

juries of the skull from falls or blows and hæmorrhagic cysts appear to have some influence in originating the disease, but more generally it is developed, so far as we can perceive, spontaneously.

Diagnosis.—The diagnosis of diffused cerebral sclerosis must always be more or less uncertain, for the reason that the symptoms are met with in other very different affections. In children a similar set of phenomena may be the consequence of arrest of development in the brain without any alteration of its structure recognizable by our means of observation. In the case of an idiotic child affected with convulsions, hemiplegia, and muscular contractions, I found, on post-mortem examination, the left hemisphere markedly smaller than the right, but I could detect no change of any part of its structure.

Symptoms like those met with in diffused cerebral sclerosis may result from brain-tumors of various kinds.

In adults the disease is readily discriminated from cerebral hæmorrhage and embolism by the gradual character of its advance, and by the mental symptoms being more strongly pronounced. But from softening the diagnosis cannot always be made out, and an opinion must be formed from the history and phenomena in each individual case.

From thrombosis the diagnosis is equally difficult. Perhaps the distinction may be made both as regards softening and thrombosis by the facts that, though contractions are met with in both these diseases, they are not such invariable accompaniments as they are in diffused cerebral sclerosis, and that they are never, as occasionally in the latter affection, a primary symptom.

Prognosis.—The prospect of complete recovery is very gloomy, and even amelioration has hitherto been regarded as out of the question. I am inclined, however, to think, as the result of my own experience, that the condition of patients, apparently suffering from the affection in question, may be decidedly improved by suitable medical treatment.

I have several times succeeded in arresting the convulsions, strengthening the mind, increasing the strength and sensibility of the paralyzed members, and relaxing the contractions. My success has been much more decided in cases which had originated late in life—probably, for the reason mainly that the disease was seen earlier in its course.

Morbid Anatomy.—This division of the subject has already been considered incidentally, to some extent, in the remarks made under the head of symptoms, and in the detail of cases quoted.

The most obvious feature detected by ordinary observation is the increased hardness and density which the cerebral tissue has acquired. This generally occupies a considerable portion of one lobe, or may extend through the whole of it, or may even affect a whole hemisphere. It is not distinctly circumscribed, but diminishes in intensity from the centre to the periphery, and, according to Pinel, never invades the gray substance.

The increased density is attended with atrophy when the disease affects the adult, and with atrophy and arrest of development when children are its subjects.

In order to understand the essential nature of the morbid process which causes the brain to become indurated, a few words in regard to cerebral histology are necessary.

Besides the nervous tissue of the brain, there is another anatomical element present which fulfils the function of binding the cells and fibres together, and giving the whole substance its normal degree of consistence. According to Virchow,[1] this, although analogous to, is different in some respects from ordinary connective tissue. He gave to it the name of neuroglia or nerve-cement.

Diffused cerebral sclerosis consists in the hypertrophy or increased formation of this tissue, and the atrophy or disappearance of the proper nervous substance. Atrophy of the brain may, however, be due to other causes than sclerosis,

[1] Cellular Pathology, Chance's translation, London, 1860, p. 277.

as in the case reported with great minuteness by Schroeder van der Kolk,[1] and several of those cited by Lallemand,[2] Turner,[3] and other writers.

Pathology.—The symptoms which result from diffused cerebral sclerosis are those which we might expect to be the consequence of a condition which essentially consists of a disappearance of that part of the brain-tissue capable of producing or transmitting nervous force, and the substitution of another histological element which is of secondary importance. They all indicate deficient cerebral power. It is with the brain as with a muscle undergoing atrophy: less force results from its action in correspondence with the advance of the process by which the characteristic anatomical elements disappear.

Doubtless, if we had the opportunity of more thorough study of the symptoms of diffused cerebral sclerosis, and comparing them with the condition of the brain as found by post-mortem examination, we should find that they varied considerably in character, according to the part affected, and we should probably have reason to believe that the nervous cells which had disappeared—motor, sensitive, or trophic—were in exact pathological relation with the symptoms observed. This special point has been well studied by MM. Duchenne de Boulogne and Jouffroy,[4] in a recent paper, devoted to a somewhat different disease, and to which I have recently been enabled to add a few important data.

Treatment.—This division of the subject has scarcely received any attention from authors. My experience, however, has sufficed to convince me that we can occasionally improve the condition of the patient.

[1] A Case of Atrophy of the Left Hemisphere of the Brain, etc. New Syd. Soc. trans. London, 1861.

[2] Op. cit.

[3] De l'Atrophie partielle ou unilatérale du Cervelet, etc. Paris, 1856.

[4] De l'Atrophie Aiguë et Chronique des Cellules Nerveuses de la Moelle et du Bulbe Rachidien, etc.: *Archives de Physiologie*, No. 4, Juillet et Août, 1870, p. 499.

If there are epileptic convulsions, they may be prevented by the administration of the bromide of potassium in doses of at least twenty grains, three times a day, to an adult. Larger doses may be necessary. On the cessation of the convulsions, it will sometimes be found that the intelligence at once begins to be developed.

The paralysis and contractions may sometimes be lessened by the persistent use of both the induced and primary galvanic currents. The first named will often in the beginning fail to act upon the muscles, in which case the latter should be employed. This is always better for the contracted muscles than the induced current. For the relief of the paralysis it should be interrupted, for the relaxation of contractions it should be constant.

As regards the central lesion, I think it may occasionally be reached, when it has not had time to become very extensive or profound. And the best and really only means I know of are the primary galvanic current passed through the brain, and the administration of the chloride of barium.

In using the galvanic current, the electrodes — wet sponges—should be applied over the mastoid processes, and kept there for a period not exceeding three minutes. Fifteen of Smee's cells will afford a current of sufficient intensity. The application should be made about every alternate day.

The chloride of barium may be given in doses of about a grain three times a day. I usually administer it in solution, according to the following formula: ℞. Barii chloridi ʒj, aquæ dest. ℥j, M. ft. sol.; dose, gtts. xij three times a day.

I am unable to say that these measures have actually removed the supposed sclerosis of the brain, and caused the reformation of the atrophied cells, but I am very sure that symptoms such as are attendant upon diffused cerebral sclerosis have several times been measurably dissipated by its influence. Thus, in the third case mentioned as occurring in my practice, the mind improved, the epileptic paroxysms

ceased, the contractions were relaxed, the paralysis lessened, the affected limbs increased in size, and the further progress of the disease was arrested. At the present date (December 30, 1870) the gentleman is able to take care of himself, to walk tolerably well, and to use the formerly-paralyzed arm for many purposes. In three other cases a like treatment has been productive of almost as marked a degree of benefit.

LEEDS SCHOOL OF MEDICINE

CHAPTER XIII.

MULTIPLE CEREBRAL SCLEROSIS.

In multiple cerebral sclerosis the lesion involves several parts of the same ganglion, and consists of plates or nodules of sclerosed tissue scattered throughout its substance.

It is only of late years that the affection in question has been partially recognized as a distinct pathological condition associated with certain symptoms. These symptoms were formerly, and still are to a great extent, confounded with other groups similar in several prominent features, but different altogether in anatomical relations, normal and abnormal.

Thus, under the designation of paralysis agitans, were comprehended the phenomena due to multiple cerebral sclerosis, multiple cerebro-spinal sclerosis, and muscular agitation general or local—the result of very dissimilar lesions, or without discoverable morbid changes of any kind—the one symptom of tremor sufficing to bind them together. Even by late writers the distinction is not clearly made out.

It is, in the present state of our knowledge, impossible to say in all cases what part of the intra-cranial mass is affected. Still, we are not altogether without data on this point, and an attentive consideration of the symptoms will often, at least, enable us to say what ganglion of the encephalon is the main seat of the lesion. But, mindful of the fact that this work is intended to be practical, I shall not venture to deal with pathological refinements, but will point

out, with as much succinctness as possible, one form of the morbid process under notice—a form which I think I am enabled to describe, from my own observations, with considerable accuracy. That form I shall designate—

MULTIPLE SCLEROSIS MAINLY AFFECTING THE HEMISPHERES.

Symptoms.—Among the first symptoms noticed in this affection is pain, which occurs in sharp paroxysms of short duration. Sometimes the sensation is as instantaneous as an electric shock. It is rarely the case that there is any extreme constant pain experienced, though a feeling of fulness or constriction is occasionally more or less permanent.

In a few cases the first observed symptom has been an epileptic paroxysm.

It is not uncommon to meet with disorders of sensibility in other parts of the body; and these may either be anæsthetic or hyperæsthetic in character. Probably the most common is a numbness of the ends of the fingers or toes, which gives the sensation of cushions when objects are touched, and which is generally confined at first to a single upper or lower extremity. Shooting pains, something like electric shocks, are also sometimes experienced. The progress of the disease is almost invariably slow, and hence several months may elapse before any disorders of motility are experienced. These, however, are the next symptoms to make their appearance, and are generally first manifested by the occurrence of tremor or trembling.

Tremor usually, but not always, is gradual in its development, and may be restricted to narrow limits. It may at first only be felt when the patient is unusually quiet, and has not his attention engaged. Thus a gentleman told me he had, for several months, only been sensible of a vibration in his arm when he lay down at night. It was then—from the description he gave me—limited entirely to the extensor indicis of the left hand, and was, in the beginning, not strong enough to move the finger. When I first saw him,

several years afterward, both arms and one leg were strongly agitated.

In another case, which I saw almost from the very beginning, the tremor was restricted to the same muscle for several months, and then gradually involved the extensors and flexors of the hand. And, in several other instances which have come under my notice, the onset was equally gentle. But, as I have said, this is not always the case. A gentleman consulted me in the summer of 1870, who, after having experienced severe darting pains in the head and through the limbs on the right side, was suddenly, while in his field overlooking some work, seized with a violent trembling of the right hand, which continued for several minutes, notwithstanding his efforts to prevent it. A few days subsequently, he had another accession of a similar kind in the same limb, and by degrees the intervals became shorter, until, in the space of a month, the tremor was constantly present except when he slept, and, when I saw him, had extended to the whole arm and to the lower extremity of the same side.

In another case, a gentleman, much addicted to excessive mental exertion, was awakened one morning by a violent agitation in his right foot. He had been under my care several months previously for severe headache and inability to sleep, for which, believing them to result from inordinate intellectual labor, I had recommended mental rest and horseback exercise. Under the use of these measures he had apparently quite recovered, but against my advice had resumed his literary labors.

He was not very confident how long the shaking of the foot had lasted, but thought it was not more than a few seconds.

Several days afterward, while writing, his right hand began to tremble slightly. He ceased his occupation, and rubbed his hand with the other. The tremor stopped for a moment only, again began, and has scarcely ever since

been absent. The whole side eventually became involved.

The tendency of the tremor is always to extend. Beginning in an extremity or a group of muscles, or only in a single muscle, it goes on attacking others, until at last all the limbs and even the head may become affected. By preference, the advance of the tremor is lateral, that is, if an arm be first invaded, the leg of the same side next suffers, then the other arm, and then the corresponding leg. Usually the head is the last part attacked; but this is not always so, as I have seen several cases in which the trembling began in it.

For a long time the tremor is to some extent under volitional control. A patient, for instance, will slap his tremulous hand on his knee and for a few seconds can manage to keep it quiet, but it soon begins to shake again, and, though perhaps a second time he may arrest its movements by a like process, the period of rest is shorter. Any change of position is calculated to quiet the tremor for a time, and thus the patient is every few minutes moving his arms or legs in the attempt to get a little respite.

It is always increased by emotional disturbance of any kind. A limb which may ordinarily be but slightly tremulous, will shake violently from the excitement or anxiety produced by making a visit to a physician. The effort to keep it quiet will also often increase the tremor.

For a very considerable period after the beginning of the disease, the shaking ceases during sleep, but eventually this state affords no respite, and the patient is thus deprived still further of his physical strength.

It is not often the case that the muscles of the face are affected very early in the disease, but they frequently become involved at a later period. In several cases I have seen a constant tremor in the upper eyelid of one or both sides, and in one instance this was the first manifestation of the disease.

In another very remarkable case the first indication of tremor was perceived in the left eyeball, which was by clonic spasms of the internal rectus muscle kept in a state of motion producing a kind of nystagmus. The upper lid of the same eye next became affected, and then the tremor appeared in the corresponding arm. The upper lip I have several times seen tremulous, causing thereby an indistinctness in the articulation.

I have never observed other muscles supplied by the facial nerve to be involved in the tremor.

Occasionally the lower jaw is rendered tremulous from the seat of the disease being at the origin or in the course of the fifth nerve.

The tongue is sometimes affected with tremor, generally at first on only one side, and I am inclined to think that the muscles of the pharynx and larynx do not invariably escape.

The tremor is not, as some authors have asserted, only manifested when voluntary movements are performed. This is probably the case at least in the first instance with multiple cerebro-spinal sclerosis, but it certainly is not in the purely cerebral form now under consideration. Jaccoud[1] calls attention to the error which has been committed relative to this point, and my own experience is uniformly in support of the opinion he expresses.

The next symptom of importance to make its appearance is paralysis; and, when the sclerosis is limited to the hemispheres or begins in them, it always follows the tremor. On this point I have insisted in my lectures to the class of the Bellevue Hospital Medical College, as an important indication of the fact that paralysis agitans is often a cerebral disease, and I am glad to find so exact an observer as Jaccoud[2] asserting that the paralysis is often preceded by muscular agitation or trembling.

At first the loss of power is slight, and, like the trembling, is limited to a single muscle or group of muscles, but

[1] Traité de Pathologie interne, p. 194. [2] Op. et loc. cit.

it gradually extends until it involves the limbs of one side, or even of both sides. According to my observations, it follows the course of the trembling, no limb being ever paralyzed till it has for some time been affected with tremor. In the face, however, the paralysis appears to be independent of the tremor.

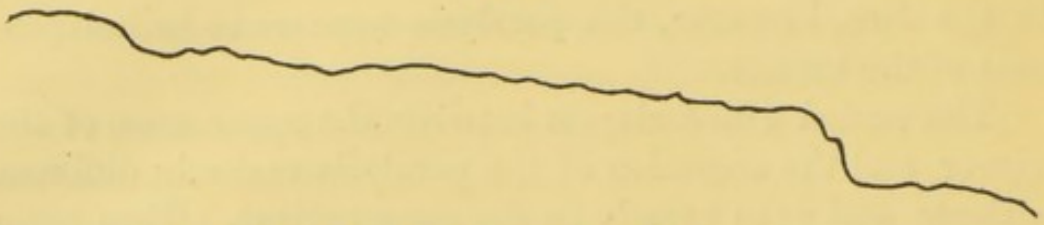
The period which elapses between the appearance of the tremor and the accession of the paralysis varies in different patients, and even greatly in the same patient. Thus some muscles may exhibit notable loss of power in a few weeks after they have begun to be agitated, while others remain free from paresis for many months.

When the loss of power affects the extensors or flexors—especially in the former event—contractions may take place, as in diffused cerebral sclerosis, and the limbs are thus more or less distorted. The most common seat of this phenomenon is in the upper extremity, and it generally begins in the fingers, extending gradually to the wrist and elbow. But in some cases, even though the antagonism between certain groups of muscles be destroyed, there are no contractions. The muscles of the head, face, and trunk, do not escape. Strabismus, ptosis, and facial paralysis, are thus produced, and the muscles concerned in speech, in deglutition, and in respiration, likewise become involved. The sphincters, according to my experience, are rarely paralyzed in the early stages of the disease, but I have several times witnessed paresis of the bladder among the primary symptoms.

A marked symptom which I have observed, and which can only be distinctly shown by means of the dynamograph, is the inability of the patient to maintain a continuous muscular contraction, for even a short period. I have noticed this as among the very first indications of paresis, and I am disposed to think it exists even before the tremor is noticed. Thus, a gentleman occupying a prominent public position, and in whom I had diagnosticated multiple cerebral sclerosis mainly affecting the hemispheres, instead of making a

straight line with the pencil of the instrument, traced one of which the following cut is a *fac-simile* ·

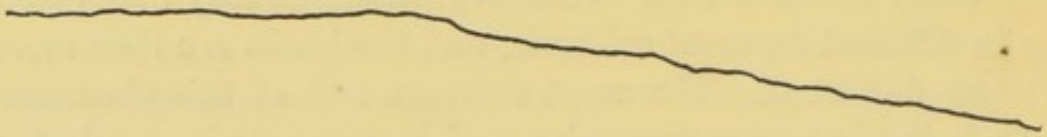
FIG. 8.



Repeated efforts only gave worse results.

In another case, that of a gentleman referred to me by my friend Dr. Van Buren, the line made was as follows:

FIG. 9.



Here the patient was able to maintain the contraction at its original force for only about the sixth of a minute—the time required for the paper to traverse the pencil being exactly half a minute, and a third part of the line being horizontal.

The ability to coördinate the affected muscles is always impaired, and thus in voluntary movements there is agitation independently of the esoteric tremor. This is seen not only in active movements but in passive muscular contractions, such as those by which an article is held in the hand. In such a case the fingers cannot be kept in apposition with the object, but are moved about in a disorderly manner. The incoördination is manifestly connected with the inability to maintain a lengthened muscular contraction to which reference has just been made.

Sometimes, by the strong effort of the will, assisted by the sense of sight, these last two difficulties may for a little while be overcome. A gentleman now under my charge, suffering from the affection in question, cannot, for instance,

carry a glass of water to his lips except by looking at it fixedly and concentrating all his volitional power upon the act. His lower limbs are not yet affected, and he consequently can coördinate them, in walking and other movements, perfectly well.

In another case, a lady, affected with multiple cerebral sclerosis, undertook to help her invalid husband to rise from his chair; a band of music happening to pass the window, she turned to look at it, and, at once relaxing her hold, let him fall to the floor and injured him severely.

Zenker[1] reports a case in which there was a similar loss of the appreciation of the state of the muscles; and another is mentioned by Reynolds,[2] under the head of "muscular anæsthesia." I am very sure that many cases of this last-named affection are instances of multiple cerebral sclerosis of other ganglia, and I shall presently more specifically refer, under a different head, to two remarkable cases which have occurred in my own experience.

Another phenomenon closely related with this incoördination is generally present in multiple cerebral sclerosis, and that is, that the patient loses that innate or early-acquired knowledge of the exact situation of the several parts of his body. We can all of us, not thus affected, close our eyes, and touch, with the end of the finger, any particular point on the face or rest of the body, with the utmost exactness. But a person with multiple cerebral sclerosis involving the hemispheres cannot do this. Thus, in attempting, with the eyes shut, to place the end of the index-finger on the middle of the eyebrow, he misses that point, sometimes by as much as two inches; and, no matter how frequently he tries, he succeeds no better. It would appear that, in such cases, the normal instinct of topographical relation between the fingers and the cutaneous surface gen-

[1] Ein Beiträg zur Sklerose des Hirns und Rückenmarks. Henle und Pfeufer's Zeitschrift für Rationelle Medizin, Bd. xxiv., 1865.

[2] System of Medicine, vol. ii., p. 330.

erally, which all persons and many animals seem to possess, is impaired.

The electro-muscular contractility is never, according to my experience, diminished in multiple cerebral sclerosis, uncomplicated with similar lesions in the spinal cord.

The attitude and gait of a person affected with multiple cerebral sclerosis are peculiar. In standing the body is generally inclined forward, the head falling toward the chest, the trunk flexed at the pelvis, and the knees slightly bent. In walking the action is similar to a jog-trot, the body being still inclined forward, and the patient often moving with considerable rapidity. I have had several persons with the disease under my charge who could not walk at all, but who could run with surprising agility. One of these, a gentleman advanced in life, sent to me by my friend Prof. Sayre, was unable to take a step in my consulting-room. He was carried down-stairs by his attendants with some difficulty, and when he reached the front-door he was put on his feet. He then told his servant to give him a push, which the man did with all his might, and the old gentleman, being started, went at a full run and jumped into his carriage without the least difficulty.

There is often a strong tendency to plunge forward, and at times there is an impossibility of controlling it except by catching hold of some fixed object. Not long since I was walking down Broadway, when I saw in front of me a gentleman who was then under my charge, and in whom I had diagnosticated multiple cerebral sclerosis. Although aware of his peculiar impulsive gait, I had never seen it so strikingly manifested as it was then. He went at a full trot, threading his way among the numerous people in the street, until, apparently exhausted, he would lay hold of a lamp-post or awning-post and cling to it till he had recovered his breath, to start off again in a similar manner.

This impulsion of the body forward makes it easy for the patient to ascend a staircase, but, on the contrary, very diffi-

cult to go down one. The first case of the disease in question which I saw in this city, over six years ago, was characterized by an extreme degree of festination. It was that of a maiden lady, over fifty years of age, who had been affected for several years. When she was going up-stairs no one could perceive the least irregularity in her gait, but to go down was impossible.

Sometimes, however, the tendency is to go backward. This was the case, to a remarkable extent, in a gentleman, a resident of this city, who was sent to me by my friend Prof. Van Buren. Every time he rose from his chair he was forced to take several steps backward, and it was only by constant mental effort that he was able to go forward at all.

The tactile sensibility is generally impaired from a very early period in the course of the affection, and thus, the two points of the æsthesiometer must be more widely separated than in the normal condition of the system, in order to get two separate impressions. This anæsthesia bears no necessary relation to the region of skin covering the affected muscles. According to my experience, it is most marked at the terminal extremities of nerves.

Numbness of different degrees, pains of various kinds, increased or diminished temperature, and excessive hyperæsthesia of the skin, may also exist.

The special senses may be affected to a variable extent. Thus there may be amblyopia, or even complete blindness; the taste is very often impaired or abolished, and the hearing rendered less acute.

The ophthalmoscope should always be employed to examine the fundus of the eye. The condition generally found to exist is white atrophy of the optic disk, which is identical in general features with sclerosis. The vessels of the retina will usually be found small, the branches of the veins few in number, and the choroid of a paler hue than is natural.

The course of multiple cerebral sclerosis is progressive. The patient is finally unable to walk, the friction of his

shaking body against the bed abrades the skin, the dejections are passed involuntarily, and he dies either in coma, in convulsions, or by a gradual process of asthenia, his mind participating in the general decay. The duration of the disease varies from a few months to eight or ten years. Generally it runs its course in about five years.

Causes.—Age is certainly one of the most powerful predisposing causes of multiple cerebral sclerosis mainly affecting the hemispheres, and causing the symptoms heretofore classed as paralysis agitans. Thus, of nine cases in which I diagnosticated the disease in question, all were over fifty years of age, and three were over sixty. I have seen numerous cases of paralytic tremor in younger persons, but the morbid condition had scarcely any points in common with that now under notice. Cases, however, are on record in which young persons were the subjects. There is some evidence to support the theory that it is sometimes hereditary, but the whole subject is so confused in the minds of most authors that it is difficult to make out clearly what they refer to under the designation of paralysis agitans. Of the nine cases occurring in my own practice, private and hospital, four had immediate ancestors who had suffered from some form of tremor and paralysis. Whether the lesion was purely cerebral, cerebro-spinal, or whether the disease was entirely functional, I was not able to decide from the information given.

The influence of sex is more readily ascertained and is very evident. Seven of my cases were males and only two females.

Of exciting causes there are many. In two of my cases it followed immediately on attacks of scarlet fever, in one it was a sequence of typhoid fever, in one it ensued after rheumatism, in one it was probably syphilitic, in two it was apparently excited by great emotional disturbance, in one by inordinate muscular exertion, and in two no cause could be assigned, or at least there was not, in my opinion, any sufficient exciting cause to be discovered.

Diagnosis.—Multiple cerebral sclerosis has heretofore been confounded with other diseases, and its very existence as an independent affection is doubted by my friend Dr. M. Clymer,[1] and other writers. To this point I will return when the morbid anatomy and pathology are discussed, and, as in the foregoing account of the symptoms and causes, will base my remarks under the present head mainly on the results of my own experience.

The occurrence of "head-symptoms" is sufficient to diagnosticate multiple cerebral sclerosis from the functional paralysis agitans, which is never a very serious affection, and the seat of which is not centric. Besides, in the latter there are no festination, alterations of sensibility, incoördination, muscular anæsthesia, or inability to maintain a continuous muscular contraction, while the paper of the dynaomgraph traverses the pencil of the instrument. The functional disorder is more liable to occur in persons under fifty than in those over that age. From the cerebro-spinal form of multiple sclerosis, which will be fully considered in another section of this work, it is distinguished mainly by the facts that the tremor makes its appearance before the paralysis, and that the agitation is present whether voluntary movements are being made or not.

With the purely spinal form it is not likely to be confounded by any one paying the slightest attention to the phenomena of the two diseases.

From chorea it might in some cases not be readily discriminated without a thorough study of the clinical history and existing symptoms. But, though chorea sometimes occurs in adults, and is generally accompanied by "head-symptoms," the two affections possess few other phenomena in common.

In the first place, the mental symptoms in chorea are indicative of feebleness from the very first, while in multiple

[1] Notes on the Physiology and Pathology of the Nervous System, with reference to Clinical Medicine, New York, 1870, p. 11.

cerebral sclerosis imbecility supervenes late in the course of the disorder. In chorea there are no vertigo, pain in the head, or other evidences of congestion, while in the disease under notice these are among the very earliest symptoms. In chorea there is no actual tremor, but the disorderly movements are more extensive and irregular than in multiple cerebral sclerosis; neither is there festination or bending of the body forward.

Tremor is sometimes met with after cerebral hæmorrhage or other cause producing hemiplegia, but in such cases the clinical history, and the fact that the trembling comes on after the paralysis, will suffice to render the diagnosis sure.

Prognosis.—The prospect of recovery is always unfavorable, but not, I am induced to think, absolutely hopeless if the patient be seen sufficiently early in the course of the disease and submitted to proper medical treatment. The probability of an arrest of the onward tendency is by no means small under like circumstances. Still, in the great majority of cases, all means fail, and the affection gradually and persistently goes on to its termination, death.

Morbid Anatomy.—The membranes of the brain are sometimes opaque in patches and occasionally contain an abnormal amount of serous fluid. The cerebral convolutions are occasionally flattened, and the gray substance is thinner than in the normal condition. It may also be changed in color, being pale, and scarcely, according to Jaccoud, to be distinguished from the white substance.

On cutting into the tissue of the hemispheres, plates or nodules of hardened matter are found scattered throughout its extent. These are well defined and vary in size from that of a cherry-stone to that of a small walnut. In the only case in which I have had the opportunity of making a post-mortem examination, they were confined entirely to the white substance of the hemispheres. Their color is white or grayish-white, and they are of varying degrees of consist-

ency from that of hard-boiled white of egg to that of cartilage.

Examined with the microscope, they are seen to consist of the neuroglia, which, to a great extent, has taken the place of the nervous tissue, and of the *débris* of this latter in the forms of fibres, nucleated cells, and free nuclei. They are formed, therefore, by the hypertrophy of the connective tissue of the brain at the expense of the nervous tissue proper.

Sometimes there are very few of these deposits—indeed, there may only be one—and at others they are present in large numbers. In the case examined by myself there were seven in the left hemisphere and eleven in the right, of sizes varying as previously stated.

They may be found in other parts of the cerebral mass besides the hemispheres, though in the form under consideration these are their most prominent and constant seats. Thus, they may exist in the hemispheres and in the medulla oblongata, the pons Varolii, and the cerebellum, at the same time. When they occupy, likewise, the spinal cord, another disease is produced which differs anatomically and pathologically from multiple cerebral sclerosis.

Sometimes large numbers of amyloid corpuscles are met with, but their presence is not constant.

Pathology.—The first question to be considered under this head relates to the existence of multiple cerebral sclerosis as an independent affection—that is, without lesions of like character being at the same time produced in the spinal cord.

The weight of authority is probably against the view expressed in this chapter, and, as I have, so far as I know, made the first attempt to identify a certain group of symptoms with multiple sclerosis limited to the cerebral ganglia, I am the more desirous to place the reasons by which I have been actuated before the reader.

Andral,[1] under the designation of partial induration of the brain, describes the morbid anatomy of an affection which is probably the same as that under present consideration, although his account of it is by no means full or precise.

Valentiner,[2] citing a number of cases observed by himself and Frerichs, details one in which the lesions were limited to the brain, and in which the symptoms were similar to those I have specified in this chapter.

Jaccoud declares that certain cases establish the possibility of sclerosis limited to the encephalon. In a note he refers to several writers who have stated the parts affected, in some of which, however, the spinal cord was also involved. In the following it appears to have been restricted to the brain:

Stœhr, hemispheres corpora mamillaria; Dumville, protuberance medulla oblongata and corpora olivaria; Pool, hemispheres centrum ovale; Cruveilhier, anterior face of the medulla oblongata, protuberance, cerebral peduncles, corpus callosum, walls of the lateral ventricles, and the origins of the pneumogastric glosso-pharyngeal and hypoglossal nerves; Duplay, hemispheres, particularly in the vicinity of the ventricles, optic thalami, and corpora striata; Van Camp, protuberance; Obertimpfler, hemispheres; Barthez and Rilliet, hemispheres, particularly one convolution; Cohn, hemispheres in two cases; Gunsburg, hemisphere, gray substance of the convolutions; Valentiner-Frerichs, cerebellar peduncles, corpora olivaria, protuberance, and medulla oblongata; Meynert, cerebellum and protuberance.[3]

Bourneville and Guérard,[4] while asserting that the exist-

[1] Préces d'Anatomie Pathologique, tome ii., 2e Partie, Paris, 1829, p. 810.

[2] Über die Sklerose des Gehirns und Rückenmarks. Deutsche Klinic, B. xiv., 1856.

[3] I quote this note from Jaccoud, without vouching for its correctness, as, from the fact that he does not cite the works in which the details are to be found, I have not been able to verify his statements.

[4] De la Sclerose en Plaques Disséminées, Paris, 1869. Analyzed by Dr. E.

ence of multiple cerebral sclerosis as a separate and distinct affection rests on only one case—that of Valentiner—which they further declare was probably imperfectly reported, admit that the cerebral form may be regarded as established. But none of the authors who have referred to it identify a form of paralysis agitans with a lesion characterized by the presence of bodies of sclerosed tissue in the brain, and especially in the hemispheres. Thus, Dr. Clymer expresses the opinion that, excluding the tremor, which may accompany hemiplegia and certain other disorders of which it is an altogether secondary phenomenon, there are but two varieties of paralysis agitans: 1. That which results from multiple (disseminated) sclerosis, affecting the encephalon and spinal cord; and, 2. A purely functional disorder, first fully described by Parkinson.[1] Now, in my opinion, Parkinson has described two very distinct affections under the name of paralysis agitans. One of these is certainly functional so far as this; that the tremor shows no disposition to extend to distant parts of the body, that it is the only symptom present, that no lesion has been discovered, and that it is readily cured; the other is characterized by the phenomena which I have detailed in this chapter, and which, though imperfectly described by other authors, have either been confounded with multiple cerebro-spinal sclerosis, or regarded as constituting an aggravated form of the functional disorder. My views of its true pathology have been formed from careful observation of the course of the disease in nine cases, in one of which I was enabled to make a post-mortem examination.

P. B., male, aged sixty-five, formerly a drummer in the army, and latterly an instructor of buglers, came under my observation at Ceboleta, New Mexico, in the winter of 1849–

C. Seguin in Archives de Physiologie, etc. No. 4, Juillet et Août, 1870, p. 524, *et seq.*

[1] Essay on the Shaking-Palsy, London, 1817. I have not been able to find a copy of this work in New York, and have for several years been unsuccessfully trying to obtain it. My citations from it are therefore second-hand.

'50. While milking a cow, one evening, he suddenly experienced a severe pain in his head, which lasted only a few seconds. He soon afterward had an epileptic paroxysm, during which he bit his tongue severely. He had no other fit, so far as was known, but the pain in the head recurred at different times, never, however, lasting longer than a minute or two.

No other symptom appeared for several weeks, and then he experienced severe darting pains in the arms, and soon afterward the left hand began to shake. On examination I found the tremor limited entirely to the extensor communis digitorum, and that the motion was entirely in the line of extension and flexion. Little by little the other muscles of the forearm became involved, and then the disorder extended upward, affecting the biceps coraco-brachialis triceps, deltoid, and the muscles of the shoulder generally. The arm was much weaker than the other, although he was left-handed.

In about three months after first noticing the tremor in the left hand, the left foot began to shake, and, as in the first instance, the agitation gradually extended upward, until, so far as I could see, all the muscles of the extremity were involved.

He now complained of numbness in the ends of the fingers of the affected extremity, and this slowly extended to the whole arm. The sensibility of the leg remained intact.

Next the right arm went through a similar sequence of phenomena, then the right leg, and finally the head.

There was no decided tendency to forward impulsion till both legs were involved, though there was difficulty in maintaining the erect posture, and the body was inclined forward before either inferior extremity became affected. But, with the accession of tremor in both lower limbs, a marked disposition to trot and a corresponding difficulty of walking slowly made their appearance.

For over a year the tremor ceased as soon as the patient went to sleep, and it generally became less troublesome as soon as he lay down and tried to sleep. But at last it continued night and day, and thus apparently hastened the termination of the disease, for he lost strength rapidly from deprivation of sleep. This debility was still further increased by innutrition from improper food, it being impossible, in the then state of the country, to get any fresh vegetables.

During the whole period from the occurrence of the first paroxysm of pain, there was a gradual but marked failure of the mental powers, until a condition of very decided imbecility was reached. Death finally took place about two years and one month after the epileptic fit, which occurred on the same day with the first pain felt in the head.

I made the post-mortem examination with great care, but without any clearly-preconceived idea of what I should find, except that I expected to discover lesions of some kind in the brain and spinal cord. On removing the calvarium, the membranes covering the surface of the hemispheres were found to be healthy. I removed the entire brain from the skull, and carefully examined the base. There was no appreciable lesion of any kind. No tumor, no induration, no softening of any of the ganglia. The membranes were dissected off, and the convolutions on the superior surface were, I thought, less distinctly marked than was normal. I then cut through the right hemisphere horizontally an inch from the surface, and was surprised to find the course of the scalpel resisted by a hard body. This I discovered to be a mass of dense tissue one inch and a quarter long, half an inch wide, and about half an inch thick. I then very thoroughly examined the hemisphere, not allowing any part of it to escape observation, and discovered eleven of these nodules of variable size—the smallest as large as a cherry-stone, the largest about the size of a walnut—in the white

substance. In the left hemisphere I found seven similar masses.

There were none in the peduncles, in the optic thalami, in the corpora striata, in the medulla oblongata, pons Varolii, cerebellum, or any other part of the encephalic mass.

I then examined the spinal cord in like manner, making several hundred sections of it, but found no alteration anywhere. It was perfectly healthy in every respect, neither congested, softened, nor indurated in any part of its extent.

The sclerosed bodies were, many of them, dense and as hard as cartilage, others were like hard-boiled white of egg, and others like cheese. No microscopical examination was made.

In this case the lesions were entirely limited to the hemispheres, a circumstance which I can well believe is not common—other ganglia of the brain generally participating and giving rise to corresponding modifications of, or additions to, the symptoms.

Thus, when the medulla oblongata is involved, there is difficulty of swallowing and implication of the muscles of respiration; when the pons Varolii is affected, we have among other symptoms facial paralysis; when the corpora striata, more intense paralysis; when the optic thalami, derangement of vision and perhaps of hearing; when the crura cerebri, various unilateral convulsive movements and participation of the muscles supplied by the third pair of nerves; and when the cerebellum, especially the crura, the tendency to go backward instead of forward; and so on with the other important parts of the encephalic mass.

Other relations connected with the pathology will be considered when the subjects of multiple cerebro-spinal sclerosis, and what, for want of a better name, may be called paralysis agitans, are reached.

Treatment.—To detail all the various methods which have been employed in the treatment of the group of symp-

toms which I have classed together as multiple cerebral sclerosis mainly affecting the hemispheres, would be a fruitless piece of labor. Many of the cases of cure which have been reported were not instances of the disease now under notice, but of the milder, and, so far as we know, functional disorder; and, therefore, it would be useless to adduce them as guides in the present connection. I shall therefore confine my remarks to the results of my own experience.

I am very sure that the condition of the patient is generally improved by the simultaneous administration of the chloride of barium and hyoscyamus. The former may be employed according to the formula given in the immediately preceding chapter; that is, in doses of a grain three times a day; the other in the form of the tincture, in doses of from one to two drachms morning, noon, and night. Care should be taken that the latter preparation be fresh and properly made. As sold in the apothecaries' shops, it is often inert.

By these two remedies alone, the tremor is often markedly diminished, and the paralysis and other disorders of motility and sensibility greatly lessened.

Thus, in the case of a distinguished gentleman, a Senator of the United States, who consulted me in the spring of 1870 for what was designated shaking-palsy, but in whom I diagnosticated the disease under consideration, amendment was perceived from the very first day of the treatment. The tremor and paralysis diminished, the mind became stronger and more able to endure exertion, and the physical strength much increased. He was soon able to write and to attend to his official duties, and he has continued in his advanced stage of improvement to the present date. He still, however, takes his medicines, and will probably be obliged to do so for a long time yet.

In another case—that of a gentleman living in the interior of this State—no means have been so successful in improving the general health, and arresting the progress of

the disease, as the chloride of barium and tincture of hyoscyamus. I have given these remedies alone or in conjunction with others in six cases, and never without a decidedly favorable effect.

Electricity is, however, a powerful adjunct, and I always employ it when the opportunity exists for so doing. The primary current, from fifteen of Smee's cells, should be passed through the brain antero-posteriorly and laterally, as previously described, and the sympathetic nerve should likewise be acted upon by a current of similar intensity.

The tremulous muscles should also be subjected to the influence of a primary current of low tension. I am not sure that it makes any difference in which direction the current be passed, but it is important that it should not be so intense as to cause any considerable pain.

For the paralysis the induced current—not too strong—is to be recommended, and for any contractions that may be present it is the preferable form to use.

A gentleman, over sixty years of age, from Tennessee, consulted me in September, 1870, for tremor associated with paralysis. His physician, Dr. W. W. Yandell, came with him, and gave me much valuable information in regard to the progress of the disease. In the first place, there had been, several years previously, symptoms of a disordered cerebral circulation, indicated by pain and vertigo. Soon afterward tremor supervened in the left hand, and gradually extended to both limbs of that side. There were also paralysis and loss of sensibility. When he came under my notice, the upper extremity was more affected than the lower; contractions had taken place, and the fingers were strongly pressed against the palm of the hand, the hand was bent on the forarm, and the elbow was flexed to its utmost extent. The limb was somewhat atrophied, but electro-muscular contractility was not sensibly impaired.

The voice was exceedingly weak, but there was no paralysis of the tongue or facial muscles, and, though the patient

could not speak above a whisper, every word was articulated distinctly, and was appropriately used. The body was greatly bent forward, the attitude being that of a person ascending a steep hill, and there was decided festination. The tremor and paralysis were much more marked on the left side than the right, and the agitation was altogether independent of voluntary movements.

The mind, except as regarded the memory, was not essentially impaired, and the sight and hearing were unaffected by the disease. There had never been any convulsive attack or loss of consciousness, and the course of the disease had been extremely gradual. Ophthalmoscopic examination revealed nothing beyond an anæmic condition of the retinæ and choroids.

I diagnosticated multiple cerebral sclerosis mainly affecting the hemispheres, but probably involving also the right corpus striatum, and I prescribed the chloride of barium, tincture of hyoscyamus, and electricity. He remained in New York a few days, and then returned to his home with the tremor abated, the contractions partially overcome, the muscles improved in strength, and the tendency to festination lessened.

A month afterward Dr. Yandell, who had continued the treatment, wrote me, of the patient, that the improvement was more decided than his most sanguine friends had anticipated, and still continued; that the agitation was scarcely perceptible; that he could more than half extend the fingers of the left hand, could straighten the wrist and elbow, and could lift a chair, or put on his hat, with the right hand. From what I have since ascertained, he bids fair to recover entirely.

If the general health be materially impaired, cod-liver oil, iron, and strychnia, may be administered with advantage.

The food should always be highly nutritious, and a glass or two of wine, if not particularly contraindicated, may be

taken daily with advantage. Passive exercise in the open air is always beneficial, but excessive walking or strong muscular exertion of any kind should be carefully avoided. Emotional excitement or mental labor must be rigidly avoided.

Under the treatment thus indicated, the patient may at least be relieved of a great deal of his suffering.

CHAPTER XIV.

TUMORS OF THE BRAIN.

Though tumors of the brain differ greatly in character, they all, when they are accompanied by any notable symptoms, present many features in common. It will, therefore, be convenient to consider them under one head, and point out their differences when the morbid anatomy and pathology are discussed.

Symptoms.—It is possible for a person to have a tumor of the brain as large as an orange, and present no symptoms of it during life. One such case came under my observation several years ago, and many others are on record. In the instance referred to, the patient, a teamster, was twice shot in a quarrel, one ball grazed the skull, ploughing up the right parietal bone for the extent of an inch; the other entered the left breast, wounding the heart. Death ensued almost instantly. The brain was examined, and a tumor of an elliptical form, two inches in its long diameter, and one and three-quarters in its short diameter, was found, involving the white substance of the left posterior lobe. The character was that which Virchow has since called gliomatous, and contained no nervous tissue.

Again, it sometimes happens that tumors of large size exist in the brain, and produce no symptoms till a few days before death. Then very violent manifestations ensue, and the patient dies convulsed or comatose. And it is always the case that the symptoms are entirely different, as one or other part of the brain is involved, or the tumor is large or

small. Thus, we know very well that a morbid growth, seated in the pons Varolii, will cause very diverse symptoms from those produced by a similar formation in one of the anterior lobes of the hemispheres. We may say, in general terms, that tumors situated in the medulla oblongata, the pons, the optic thalami, the corpora striata, the crura cerebri, the cerebellum, and the convex surface of the hemispheres, give rise to more decided manifestations than when the white substance of the hemispheres is the seat.

Pain is probably the first symptom which attracts attention. It is generally confined to a definite region of the head corresponding to the location of the disease, but this is not always the case. It may be either a dull ache, lasting the greater portion of the day, or a sharp, lancinating paroxysm, which ensues but for a few moments and recurs frequently. As the morbid process goes on, the cephalalgia becomes more severe, and finally reaches a stage of great intensity. So great is the suffering that the patient cries out with the agony, and in a case under my observation suicide was attempted. Mental excitement, physical exertion, noises, and bright lights, aggravate the pain.

The special senses rarely escape. The sight is among the first to suffer derangement, and vision may be irretrievably lost from pressure exerted upon the optic nerve, or through congestion of the retina and choroid and consequent disorganization of these structures. The eyeball of the affected side is often rendered more prominent than the other, even when the tumor does not involve the orbit.

The hearing is also often affected, and the taste not unfrequently perverted or lessened in acuteness.

Disorders of sensibility in various parts of the body are common. These are either of the nature of anæsthesia or hyperæsthesia, and are usually experienced in the face or extremities.

Vertigo is a very general symptom, and may be of all degrees of intensity, sometimes preventing the patient stand-

ing, walking, or even sitting. It is often observed very early in the course of the disease, and is frequently accompanied by nausea or vomiting.

The disorders of motility are shown either as paralysis or convulsions. In several cases under my observation the loss of muscular power was first exhibited in the muscles of the eyeball and its appendages, causing external strabismus, ptosis, and permanent dilatation of the pupil, from paralysis of the third nerve, or internal strabismus from the lesion involving the sixth nerve.

In a case now under my charge, the muscles supplied by the right facial nerve are alone affected, and in another the left side of the face and right side of the body are paralyzed. When there is paralysis, it is generally of the hemiplegic form, though occasionally it is paraplegic. Whatever its form, it is almost always of slow progress. Paralysis may be entirely absent. It is only a *necessary* attendant when the tumor involves some part of the motor tract.

When the muscles concerned in articulation are implicated, the speech is rendered indistinct, and some sounds may be impossible of utterance, not from any defect in the idea of language or of its expression, but simply from paresis of the vocal organs.

Convulsions are other prominent symptoms, and they may be among the initial phenomena. It is not at all unusual for the first evidence of intra-cranial disturbance to be an epileptiform convulsion, and similar paroxysms may occur at intervals for many years. They may be general, or, what is more common, limited to one side of the body.

Sometimes consciousness is not lost, but there are various convulsive movements of the limbs, tonic or clonic in character. Occasionally these are confined to the muscles of the face or eyeball.

Disturbances of equilibrium, manifested by tendency to advance, to go backward, or to turn round to the right or left, are sometimes present.

With these symptoms there are generally others not so palpably connected with the morbid intra-cranial process. Thus there may be disorders of the stomach, bowels, and kidneys, and of the respiration and circulation, which add much to the discomfort of the patient.

As to the intellectual faculties, it is not uncommon to find that they do not become involved to any considerable extent till a late period of the disease. Then the change is usually a gradually-advancing imbecility.

Death takes place either by convulsions or coma, or a combination of both. The following cases, which I select from my note-book, are interesting in several relations:

J. H., male, aged thirty-seven, came under my observation January 15, 1856, at Fort Riley, in Kansas. A few months before he had received an injury of the left hip by being thrown from his horse, and was stunned for a few minutes. A few days afterward, as he was lying in bed, he suddenly became vertiginous, and at the same time had noises in his ears and some pain not very definitely located. He never had vertigo again, but the pain never left him night or day for several weeks. It then suddenly ceased, and did not recur till the morning of December 31st, when a sharp twinge was experienced in the front of the head, and he immediately saw every thing double. Ptosis and dilated pupil of the left eye soon supervened, and the arm of the right side became weaker. When I saw him the grasp of his hand was very feeble, and the ocular troubles very noticeable. The pain was almost constantly present, and was of the most intense character. He said it seemed as if a red-hot iron were being thrust through his brain.

He had come several miles to see me, and went home after I had given him a palliative medicine. A few days afterward a messenger came for me in great haste, with the information that the patient was dying, and requesting my attendance. On my arrival, I found that he had been dead several hours, having had repeated severe convulsions. On

post-mortem examination, a tumor, spheroidal in shape, with an average diameter of an inch and a quarter, was found occupying the middle third of the inner surface of the left middle lobe, so as to press on the left crus and third nerve.

The points of interest in this case are the sudden cessation of the pain and its recurrence simultaneously with the paralysis of the third nerve, the slight paralysis of the body, and the absence of convulsions till just before the fatal termination. The ptosis, diplopia, and dilatation of the pupil, doubtless occurred at the very instant that the tumor encroached on the crus.

The history of the following case, which I saw in September, 1864, at the request of my friend Prof. Van Buren, I take from the report of Dr. F. N. Otis,[1] under whose immediate care the patient was:

Miss E., aged twenty-six, was of healthy parentage, and, though of delicate organization, had enjoyed good health up to February, 1861, when she received a fall on the ice, striking violently upon her elbow. She was not conscious of receiving any other injury at the time. At three A. M. of the following day she awoke with an intense pain in the top of her head, of a throbbing, lancinating character, which continued throughout the day. By night she obtained relief. No further effect from the fall was experienced until about two weeks subsequently, when she discovered a small firm circumscribed swelling on the crown of the head at the point where the pain had previously been felt. This swelling, which was painless, increased gradually, until, after a year, it had attained the size of half a lemon. Soon after the appearance of the tumor, Miss E. began to suffer with severe pain, confined chiefly to the vertex, of the same character as that experienced immediately after the fall. This pain would continue almost without cessation for two or three weeks, after which for a like period she would be quite free from it.

[1] New York Medical Journal, vol. i., 1865, p. 26.

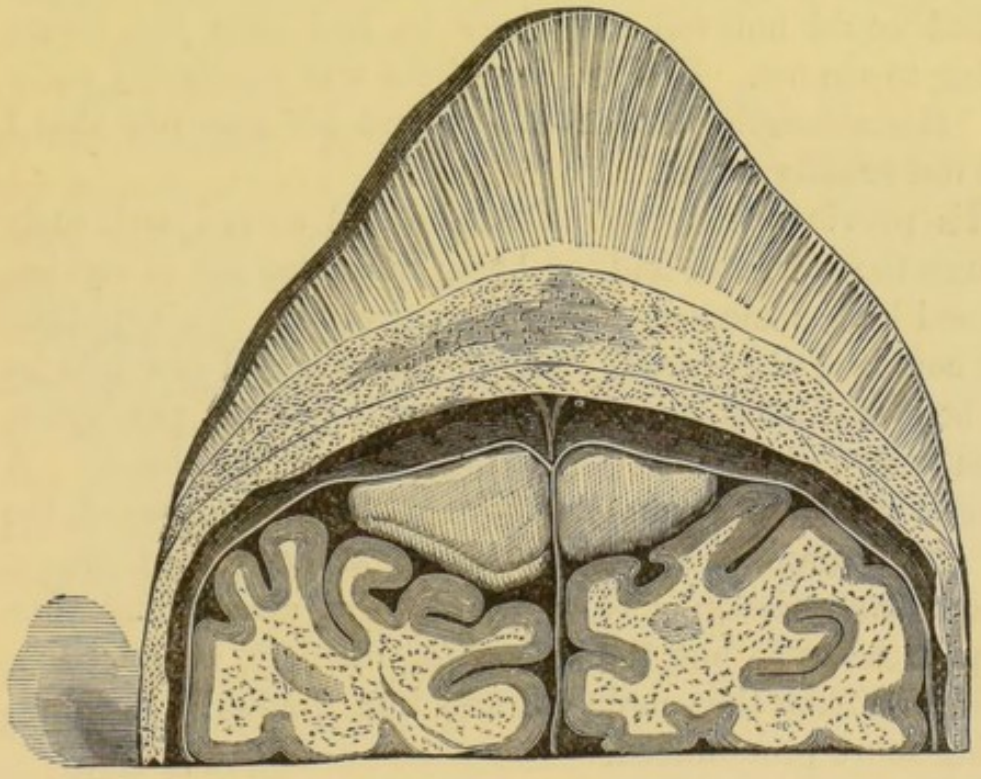
She had also occasional attacks of numbness, preceded by great drowsiness, and a cold, creeping sensation, succeeded by total loss of the power of motion, sometimes confined to a single extremity, and at others involving the entire body. These attacks usually came on at night, or after rest in a recumbent position, and generally, though not invariably, were precursors of severe headache. They were always followed by great nervous prostration. At first rare, they increased in frequency as the tumor enlarged, so that by February, 1863, she was seldom free from them for more than ten or twelve days, and the tumor had doubled in size within the year. She now began to be much annoyed by tingling, crawling sensations in her face and through the head after any unusual exertion in writing, reading, or singing, but rode daily on horseback with apparent benefit. As time passed, she had frequent dizzy turns, with nausea, and sudden flashes like electric shocks passing over the entire body, lasting only for an instant, but leaving her much prostrated. The headache, which was always of the most agonizing description, came to be referred chiefly to the tumor, though often associated with pain through the temples and other parts of the head. The muscles of the neck sometimes became rigid, and the vision, as well as the sense of taste and smell, often became very imperfect and continued so for weeks. Sometimes the power of speech would be lost, but she always retained perfect consciousness. These attacks rarely lasted more than an hour or two.

On the 23d of October, 1864, she was attacked with a peritoneal inflammation, from the effects of which she died on the ninth day thereafter. Leaving out the details of the post-mortem examination of other parts of the body, we find that an incision was made across the vertex from ear to ear, and the skin dissected from the tumor, at the apex of which it was found to be firmly adherent. The calvarium was then sawn in a line one inch above the orbital margin around to the occipital protuberance; the hemispheres of

the cerebrum were then sliced, and the whole raised at the same time.

On removing the two hemispheres, which were adherent above, a tumor one and a quarter inch in thickness and three inches in diameter, of a dull lemon-yellow color, a little softer than the cerebral substance, and separated into two lateral halves, was seen springing from the central surface of the dura mater. This intra-cranial tumor had insinuated itself into the sulci between the convolutions, and the dura mater could be traced between it and the bones. The situation of the tumor, and the relation to the exterior growth, are shown in the accompanying cut:

FIG. 10.



The microscopical examination by Dr. Gouley gave indications that both formations were encephaloid in character.

Similar cases to the foregoing have been reported by Mr. Paget,[1] of London, and by the late Dr. Isaacs,[2] of this

[1] Surgical Pathology, London, 1853, vol. ii., p. 221.

[2] Transactions of the Medical Society of the State of New York, 1859.

city. It will be noticed that, in the case just cited, there were neither convulsions, paralysis, anæsthesia, mental derangement, nor difficulties of speech. When I saw the young lady, not long before her death, there were no symptoms present from which it could have been inferred that a tumor occupied any part of the intra-cranial cavity.

I. R., a general officer of volunteers during the late war, consulted me in the spring of 1870, through his brother, for what was thought to be softening of the brain. The patient was stout and well made, had no difficulty of speech, no derangement of sensibility, and no paralysis of any part of the body. His senses were remarkably acute. His memory, however, was almost entirely gone, he had forgotten the names of his children, did not even know what city he was in, and could not tell me where he had been just before coming to see me. Besides this there was absolutely nothing. His strength was enormous, and his grip one that I shall not readily forget.

His previous history was that he had served arduously through the war, and had, on being mustered out of service, resumed his business as a lumber-merchant. No syphilitic taint could be discovered. Six months before I saw him he had been suddenly seized with an epileptiform paroxysm, which was followed by agonizing pain in the head. A second convulsion ensued in about a month afterward, the pain continuing to be of the utmost severity, and almost without intermission. There was a third attack, and then the pain ceased; but the failure of memory began to be manifested from that moment, and had gradually been becoming more pronounced.

I diagnosticated a tumor involving mainly the white substance of one of the hemispheres, situated probably in the posterior lobe, and not affecting the motor tract, or the course of any of the cranial nerves. My principal reasons for not regarding the lesion as softening were the absence of paralysis or even paresis, the integrity of all the special

senses, and the absolute perfection of articulation. At the same time I regarded the matter as extremely doubtful, and I cite the case here merely as one of interest in which the difficulty was probably a tumor. The patient died during the first week in January of the present year (1871), but I have received no details of any post-mortem examination.

In May, 1870, I was requested by Dr. Hermann Knapp to meet him in consultation in the case of a gentleman suffering from a cerebral tumor. The morbid growth apparently occupied the right anterior lobe of the brain, and involved also the temporal region of the skull on the same side. The sight of the right eye was destroyed, and that of the left so much impaired that only strong lights or shadows could be distinguished. The lymphatic glands of the neck were very much enlarged.

The pain was most acute night and day, with scarcely an intermission. The right arm was numb and paralytic, but there was no absolute paralysis anywhere except in the ocular muscles. The mind was intact, and there had never been a convulsion.

Under the use of the iodide of potassium and the protiodide of mercury the swelling of the cranium diminished, the swollen lymphatic glands were reduced, and the pain almost entirely abolished. I saw him several times afterward, and, when I discontinued my visits, he was doing wonderfully well. Since then I have lost sight of him.

There was no history of syphilis in this case.

The following account of a case, in which there was a tumor of the cerebellum, I have from my friend Prof. Austin Flint, M. D.:

"In June, 1842, I was present, by invitation of Dr. James P. White, of Buffalo, at the autopsy in the case of W. R., aged about forty years. I noted at that time the following brief account of the history as stated by Dr. White the attending physician:

"The illness was dated from the preceding February

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(five months), but he had previously complained of pain in the head, and lassitude. In February he had had chills, which were at first attributed to malaria. Subsequently vomiting was a prominent symptom; it occurred in the morning immediately after rising from bed. Cephalalgia was a frequent, not a constant, symptom. He referred the pain especially to the occiput. In April he left Buffalo to visit friends in Rochester. He was prostrated by the journey, and, his condition now being alarming, he returned home. Notwithstanding the treatment adopted, he gradually failed, and died June 7th.

"There had never been convulsions nor paralysis.

"*Post-mortem Examination.*—The body was considerably emaciated. There was slight opacity of the arachnoid, and in some situations a small quantity of serum was effused beneath this membrane. The effusion within the ventricles was somewhat greater than usual. With these exceptions there were no morbid appearances, except in the cerebellum. Here was a tumor of the size of an English walnut. It was of fine consistence, and supposed to be tuberculous. There was no appearance of inflammation or softening of the cerebral substance around the tumor, which was situated in the right lobe of the cerebellum.

"It was ascertained in this case that the venereal appetite had been wanting for many months before death. I recollect that Dr. White informed me at the time that vertigo was a feature in this case, and that it induced unsteadiness in the voluntary movements. Dr. White has since informed me that his recollection is now distinct as regards this point."

A very important paper on intra-cranial tumors is that of my friend Prof. Roberts Bartholow, M. D.,[1] of Cincinnati. Dr. Bartholow has discussed the relations of symptoms to lesions with great perspicuity and fulness.

[1] Report on Intra-cranial Tumors; their Symptomatology and Diagnosis, with Illustrated Cases, Columbus, 1869.

This able observer divides the symptoms produced by cerebral tumors into two orders:

1. Those common to morbid growths or adventitious products in general.

2. Those peculiar to tumors in special situations.

In the first order are to be placed headache, vertigo, amaurosis, convulsions, and derangement of the intellectual and reflective faculties; in the second alterations of sensibility, disturbances of the special senses, disorders of motility, vomiting, and urinary disorders.

Causes.—The causes of cerebral tumors are so intimately connected with their character that a classification becomes at once necessary. Following Jaccoud[1] in this respect, I shall divide them into four groups: the vascular, the parasitic, the diathetic or constitutional, and the accidental. Even with this division we shall find that our knowledge of their etiology is not extensive.

Vascular tumors are aneurisms of the cerebral arteries. The term does not include the capillary aneurisms of Bourchard and Charcot, referred to under the head of cerebral hæmorrhage, but applies only to dilatations of the larger arteries. According to Gouguenheim,[2] they are more common between the ages of fifty and sixty than at other periods of life, though cases were met with under the age of puberty. Tables given by Durand[3] are to the same effect, as is likewise the experience of Lebert,[4] Gull,[5] and others. This is what might be expected from the known proclivity of the arteries to disease after the age of fifty.

Sex appears to exert but little influence, though aneurisms of the cerebral arteries seem to be somewhat more frequent with men than women.

[1] Op. cit., page 247.

[2] Des Tumeurs anévrysmales des artères du Cerveau. Thèse de Paris, 1866, p. 12.

[3] Des anévrysmes du Cerveau. Thèse de Paris, 1868, p. 87.

[4] Klinische Wochenschrift, Berlin, Nos. 20 to 42, 1866.

[5] Guy's Hospital Reports, third series, vol. v., 1859, p. 281, *et seq.*

As exciting causes, blows on the head, falls, sudden and great physical exertion, intense emotion, or mental labor, embolism, and concentric hypertrophy of the heart, are to be mentioned.

Parasitic tumors are caused by the migration of the embryos of the cysticercus and echinococcus from other parts of the body.

Diathetic tumors are either cancerous, tuberculous, or syphilitic in character. The first named are more common during the adult period of life than any other, though they are met with at all ages. Although women are more subject to some forms of cancerous tumors than men, yet in the brain they are far more common in the male sex. Of forty-eight cases studied by Lebert, cancer of the brain was primary in forty-five, that is, made its first appearance in this organ.

Ogle,[1] of twenty-five cases of cerebral cancer, found that in thirteen the disease was confined to the brain, while, on the other hand, contrary to the generally received opinion, Dr. Mackenzie Bacon[2] found but ten primary cases out of seventy-three.

There is no doubt that cancer of the brain is sometimes the result of traumatic cause.

Tuberculous tumors of the brain are generally met with in young children, though they do occur, as in the case related by Dr. Flint just cited, in adults. They are almost always secondary to similar products in the lungs.

Syphilitic tumors are, of course, the result of the syphilitic infection of the system.

Accidental tumors may be caused by injuries, as was probably the case in one of the instances cited. Jaccoud, however, expresses the opinion that such an apparent relation is purely accidental, and that all we know of their etiology is that they are more common after the age of forty than before that period.

[1] British and Foreign Medico-Chirurgical Review, July, 1865, p. 223.

[2] On Primary Cancer of the Brain, London, 1865.

Diagnosis.—The diagnosis of cerebral tumors is sometimes almost self-evident, in others it is equally impossible. This difference is due, not only to the various situations they may occupy, but also to their diverse nature.

The presence of severe pain in the head for a long time is of itself some indication of the existence of a tumor if it is unaccompanied by febrile excitement. Epileptiform convulsions, occurring after the age of forty, should excite suspicion that their cause is to be found in a morbid growth of some kind. The character of the convulsive seizures will aid us in forming an opinion of their etiology. When produced by a tumor they are generally unilateral, the loss of consciousness is not so complete, and there is rarely subsequent stupor. The diagnosis from epilepsy is rendered more evident by the fact that, in tumor, the convulsions are seldom accompanied by mental weakness, and never by periods of active unconsciousness. From softening the distinction can be made without much difficulty in the majority of cases. The acute pain, the integrity of the mind, and the absence of general paresis, will usually suffice. But sometimes the discrimination cannot be made, for there are cases of tumors in which there is very little pain, in which the mind is involved, and in which the paralysis is not very strongly marked.

The occurrence of very limited paralysis points to the existence of a tumor, rather than any other affection. A gentleman is now under my care, who, several years ago, had a cerebral hæmorrhage, from which he was rendered hemiplegic. He regained to a great extent his mental and physical powers, but a few days ago suddenly had diplopia from paralysis of the external rectus muscle of the left eye, by which internal strabismus was produced. As yet there have been no other head-symptoms except vertigo, with which he has suffered a great deal in the last two years, and which was excessive when the diplopia appeared. In other respects the health is good, and the mind gives no evidence

of being affected. The paralysis of the external rectus is on the same side with the general hemiplegia.

In my opinion, though I express it, of course, without positiveness, there is an aneurismal tumor pressing upon the sixth nerve after its emergence from the medulla oblongata, and probably affecting the left internal carotid artery. If this view be correct, other symptoms will certainly arise ere long. These will probably consist in the more extensive implication of cranial nerves, and in the supervention of hemiplegia.

The diagnosis of the character of the tumor is of interest, and sometimes of importance with a view to the prognosis.

Aneurismal tumors are more common in persons of advanced age than in the young, they are more frequently accompanied by vertigo, and they are more generally indicated by paralysis of one or more of the cranial nerves. The mental symptoms are not often marked.

Parasitical tumors usually first manifest themselves by the occurrence of epileptiform convulsions, and the mental faculties do not long remain unaffected, for the reason that such products are more commonly seated in the gray substance of the brain than in the white tissue or the ganglia at the base. As these latter generally escape, troubles of motility are rare. Diathetic tumors are more easily recognized than any others, for the reason that we have other evidence of the existence of constitutional infection in the great majority of cases. As regards cancer, however, this aid is not generally afforded, the affection being usually primary, and not producing the ordinary indications of the cancerous cachexia. But, as in the case cited in full, and the others referred to, the existence of an external tumor is some indication, in connection with head-symptoms, that there is a corresponding growth within the cranium.

Tubercle may be suspected in cases presenting the symptoms of cerebral tumor, when there are indications of similar deposits in the lungs or other parts of the body, when

the subject exhibits evidence of possessing the tuberculous diathesis, or when the history shows hereditary tendency.

In a patient presenting the symptoms of a tumor of the brain, its nature may safely be considered syphilitic, if, in addition, his clinical history shows that he is tainted with syphilis, or has, at some former period, suffered from it.

In regard to accidental tumors or those of various anatomical characteristics, there is not much to be said of their diagnosis. There are no means by which one species can be distinguished from another, and no positive indications which can enable us to discriminate them from other tumors, except by the way of exclusion.

Prognosis.—Cerebral tumors uniformly lead to a fatal result, except they be syphilitic in character. In these latter there is a very considerable prospect of recovery if the proper medical treatment be adopted.

Morbid Anatomy and Pathology.—*Vascular Tumors.*—The most common seat of cerebral aneurisms is the basilar artery, and they are larger here than when any other vessel is affected. Gouguenheim [1] gives the following table, based upon sixty-eight cases :

Basilar	17 cases.
Middle cerebral	14 "
Internal carotids	12 "
Anterior cerebral	8 "
Posterior communicating	5 "
Cerebellar	4 "
Anterior communicating	2 "
Posterior cerebral	3 "
Middle meningeal	2 "
Arterio-venous	2 "

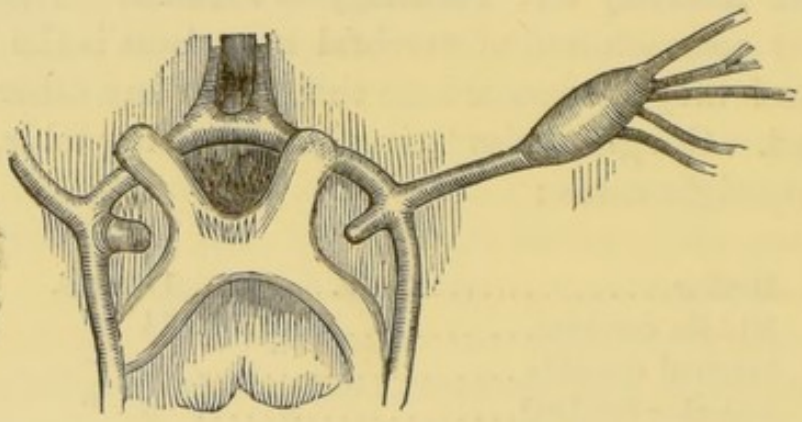
Cerebral aneurisms do not differ in any essential particular from similar formations in other parts of the body. They are, however, smaller, rarely being as large as a walnut, and

[1] Op. cit., page 21.

generally ranging in size from that of a cherry-stone to that of an almond.

Lebert ascertained that they were more frequently met with in the arteries of the left side of the brain than in those of the right. Gouguenheim confirms this observation. Thus of forty-one cases in which the side was determined, twenty-seven were on the left, and fourteen on the right. This difference is doubtless in part at least due to the fact that one of the causes of cerebral aneurisms, embolus, is more common on the left side than on the right. In a very interesting paper, Prof. W. R. Smith[1] calls attention to the fact that aneurisms of the encephalic arteries may be thus produced. The following figure, which I take from his memoir, gives an excellent illustration of such an aneurism in the left middle cerebral artery:

Fig. 11.[2]



LEEDS SCHOOL OF MEDICINE

In regard to the post-mortem examination of the patient, from whom the preparation was taken, Prof. Smith says:

"Upon tracing the left middle cerebral artery into the fissure of Sylvius, it was found to be obstructed (just where it branches into twigs surrounding the island of Reil) by a plug of fibrine of a yellowish color and oblong form, fully a quarter of an inch in length and about the eighth of an inch

[1] Cerebral Aneurism. Reports of the Dublin Pathological Society. Dublin Quarterly Journal of Medical Science, November, 1870, p. 443.

[2] The drawing shows the position of the aneurism reversed to the right side.

in breadth. At the seat of obstruction the vessel was dilated into an oblong tumor half an inch in length and a quarter of an inch broad, the space intervening between the original plug and the arterial tunics being occupied by coagulated blood."

The theory sustained by Prof. Smith was, as he freely states, first proposed by Dr. Senhouse Kikes [1] in the paper to which I have already referred under the head of embolism.

The idea was formerly very generally entertained, that cerebral aneurisms were always true, that is, caused by the uniform dilatation of all the coats of the artery. Hodgson [2] sustained this view on the ground that the tunics of the encephalic arteries were of such extreme tenuity that they readily dilated, and Albers,[3] Crisp,[4] Gull,[5] and others, held similar opinions, but the recognition of the fact that the arteries of the brain are peculiarly subject to disease in persons advanced in age, and the researches of Lebert, Virchow, and Kölliker, go to show that such a view is erroneous. Three other kinds are known to exist, the mixed external in which the interior and middle coats are ruptured and the sac is formed by the external coat; the dissecting, in which the internal tunic is ruptured and the blood is to a certain extent forced between the layers of the middle tunic; and the arterio-venous. This latter is seated in the cavernus sinus, and is produced by the rupture of a small carotid aneurism, or it is the result of wound or injury.

Aneurismal tumors may cause death either by the pressure which they exert on important parts of the brain or by the giving way of the sac and the consequent extravasation of blood, producing pressure and disorganization.

Parasitic tumors are of two kinds, those produced by the

[1] Medico-Chirurgical Transactions, vol. xxxv., p. 852.

[2] A Treatise on the Diseases of Arteries and Veins, London, 1815

[3] Mémoire sur les Anévrysmes du Cerveau et ses Meninges, Bonn, 1836.

[4] A Treatise on the Structure, Diseases, and Injuries of the Blood-vessels, London, 1847.

[5] Guy's Hospital Reports, 1857.

cysticercus and those caused by the echinococcus or hydatids. The former are small, scarcely ever being larger than a small bean. They are rarely encysted, as in other parts of the body, but are in close apposition with the brain-substance. They are generally met with in numbers ranging from ten to twenty. Cruveilhier [1] reports a case in which there were over one hundred.

They are found in all parts of the cerebrum and cerebellum; fifty of those discovered by Cruveilhier, in the case just cited, were in the cerebellum. Generally they are near the surface of the brain—often in the pia mater, in which situation they press upon the gray matter, and often in this latter substance. When situated in the ventricles, there is less impediment to the growth of the parasite, and hence it may become developed into a more or less perfect tape-worm.

Cobbold [2] states that there are about one hundred cases on record of cysticerci being found in the brain after death. Of these, Griesinger [3] reports between fifty and sixty.

Echinococci, or hydatids, though much larger than the foregoing-described parasites, are less numerous. Generally there is only one, and rarely are there two cysts. Each cyst may contain a single hydatid, as is usually the case, or there may be more in different stages of growth. In size, the cysts vary from that of a marble to that of an orange, and consist of a vascular membrane enclosing the parasite.

Of one hundred and thirty-three cases occurring in the human subject and analyzed by Cobbold, sixteen were situated in the brain. All were of course fatal.

Both of these species of parasitical tumors may be primary, or they may be accompanied by similar growths in other parts of the body.

[1] Anat. Pathol. Gén., t. ii., p. 83, Paris, 1852.

[2] Entozoa: An Introduction to the Study of Helminthology, with Reference more particularly to the Internal Parasites of Man, London, 1864.

[3] Cysticerken und ihre Diagnose, Archiv der Heilkunde, 1862.

Diathetic tumors are either cancerous, tuberculous, or syphilitic.[1]

Cancer may affect any part of the brain, though it more generally attacks the hemispheres, the cerebellum, the optic thalami, the corpus striatum, or the pons Varolii. It may begin in the bones of the cranium, in the membranes, or in the brain itself. A common seat is the orbit. According to Dr. Mackenzie Bacon, of seventy-three cases of brain-tumors occurring in the London hospitals during the period from 1854 to 1863, ten were cancerous. Ladame,[2] of three hundred and thirty-nine cases of cerebral tumors, collected from various sources, found that sixty-seven were cancerous.

The dimensions of cancerous tumors are very variable. Generally they do not much exceed the size of an English walnut, though they may be twice as large.

Either variety of cancer, encephaloid, scirrhous, or colloid, may have its seat in the brain. Primitive cancer is usually single; secondary, multiple. In a case reported by Dr. Webber,[3] of Boston, in which there was a preëxisting cancerous tumor of the vagina, the brain was found to contain several deposits of cancerous growths — one quite large, situated in the left hemisphere, and two in the cerebellum.

Ogle[4] has shown that the brain-substance surrounding the cancerous growth undergoes softening. Frequently it is not changed at all.

The tumor itself does not often undergo softening, but a kind of fatty degeneration and atrophy occur, and the tissue becomes hard and compact, with no traces of blood-vessels remaining.

Tubercular tumors may be either single or multiple. In the former case, they are often as large as a cherry; in the

[1] Op. cit.

[2] Symptomatologie und Diagnostik der Hirngeschwülste, Würzburg, 1865.

[3] Journal of Psychological Medicine, vol. iv., 1870, p. 569.

[4] Journal of Mental Science, 1864, p. 229.

latter, they may be as small as a grain of wheat. Very large tubercular tumors result from the fusion of two or more smaller ones. They are generally seated in the hemispheres or cerebellum, though the other parts of the encephalon are not exempt. They are the most frequently met with of all the forms of cerebral tumors.

Syphilitic tumors are in general seated in the membranes, or in these and the gray matter. They are very rarely entirely confined to the substance of the brain, and are never encysted. They are, therefore, not distinctly circumscribed, but the elements of which they are composed are infiltrated into the surrounding brain-tissue. In size they vary, rarely being as large as a walnut. Histologically they consist of nuclei and cells. The former contain nucleoli and occupy the periphery of the tumor, while the cells are found mainly in the centre. Syphilitic tumors are ordinarily accompanied by like growths in other parts of the body, especially the lungs and liver.

Accidental Tumors.—Under this head are included all formations not diathetic or vascular. Among them are the *fibro-plastic* tumors, which may attain to the size of an orange and which are generally growths from the dura mater at the external part of the base of the cranium. They are composed of fusiform cells, nuclei, and blood-vessels. They are of variable consistence, sometimes being almost fluid, and at others gelatiniform in character.

Under the name of *gliomata*, Virchow described a cerebral growth due to an abnormal development of the neuroglia or connective tissue of the brain. They are more generally found in the posterior cerebral lobes, and may attain to the size of an orange. Usually there is but one. There are two kinds of these tumors, one soft, being about the consistence of the brain-substance, the other much harder. They consist of cells and nuclei, but never contain any of the nervous elements. *Cholesteatomata*, sometimes called pearly tumors, may arise from the cranial bones, from the mem-

branes, or from the brain itself. They rarely attain to the size of a walnut, and are generally very much smaller. Histologically they consist of a limiting membrane of extreme tenuity, the contents of which are disposed in concentric layers. These strata are epidermic cells which have undergone degeneration. There are no vessels either in the envelope or the contents, which, in addition to the elements just mentioned, consist of cholestrine and stearine.

In addition to these there are *osseous tumors* (exostoses), growing from the cranial bones, and which may or may not be syphilitic, *lipomatous*, *enchondromatous*, *mucous*, and several other species of tumors, which are treated of fully in the special monographs on the subject, but which need not detain us in the present connection.[1]

Two bodies cannot occupy the same space at the same time. In a state of health, the brain so nearly fills the cranial cavity that there is barely room for those variations in the amount of blood and ventricular fluid which occur within the normal limits. The growth of a tumor, therefore, is at the expense of the brain. As the former increases in size, the latter diminishes, and hence some of the symptoms resulting from tumors are similar to those which follow atrophy or sclerosis. Besides, we have other consequent effects, such as œdema, congestion, anæmia, hæmorrhage, inflammation, or softening.

When cerebral tumors press upon the cranial nerves they produce fatty degeneration and atrophy. This effect is manifested by alterations of sensibility or of motility in the parts supplied by these nerves. In the eyes, however, in addition, the changes can be seen with the ophthalmoscope. They consist in the main of atrophy of the optic disk, disap-

[1] For a very full and complete essay on the subject of Cerebral Tumors, the reader is referred to Dr. J. W. Ogle's cases illustrating the Formation of Morbid Growths, Deposits, Tumors, Cysts, etc., in connection with the Brain and Spinal Cord and their investing Membranes, British and Foreign Medico-Chirurgical Review, 1864–'65.

pearance of the vessels, congestion of the retina, or hæmorrhage or serous infiltration with detachment. As Jaccoud remarks, easily appreciated by the ophthalmoscope, these lesions have a real importance in clinical diagnosis.

As to the relation between the symptoms and the seat of the lesion, the principles enunciated under the head of cerebral hæmorrhage are applicable to cerebral tumors.

Treatment.—There is no treatment calculated to cure the patient, unless a syphilitic taint can be ascertained. It is well, however, even when there are no positive indications of the existence of such a diathesis, to act upon the presumption that it does exist, and to administer mercury in some form with the iodide of potassium. By adopting this principle, I have several times succeeded in curing patients who exhibited the most positive indications of suffering from tumor of the brain. One very remarkable case was that of a gentleman who consulted me several months since for ptosis, double vision, dilatation of the pupil, vertigo, and cephalalgia. The opinion was expressed by other physicians that there was a cerebral tumor, and I entirely accorded with the view. The gentleman had no recollection of ever having had a chancre of any kind, but I nevertheless administered the bichloride of mercury and iodide of potassium, according to the following formula: ℞. hyd. bichlor. (corros), grs. ij, potass. iodidi ʒ v, aquæ ʒ iv, M. ft. sol. Dose, teaspoonful three times a day. At the next visit of the patient he remembered that when in China, several years previously, he had contracted a chancre for which he was treated. I continued the treatment, conjoining it with the use of electricity to the eye so as to act upon the paralyzed muscles, and had the satisfaction to see a gradual but steady improvement take place, till eventually in the course of a few weeks the cure was complete.

Another case was that of a lady who consulted me in July, 1870, for agonizing pain in the head, vertigo, and paralysis of the third nerve of the left side, the latter producing

ptosis, external strabismus, and consequent diplopia. I could discover no evidence of syphilis, but I nevertheless administered the bichloride of mercury and the iodide of potassium, as in the foregoing case. The induced or faradaic current was applied to the eye, and the patient soon began to mend. The headache disappeared first, then the vertigo, and eventually the paralysis. Subsequently I ascertained from the lady's husband that it was barely possible he might have infected his wife. I have no doubt whatever that he did.

The medication recommended can do no harm. There is, therefore, no reason why the patient should not have the chance of being benefited by it.

The prescription mentioned is a very eligible form for administering both the mercury and iodide of potassium. Salivation is never caused by it, and the stomach generally tolerates it well. Of course the proportions of the ingredients can be altered, as may seem best in individual cases.

The induced galvanic current is beneficial in restoring contractility to the paralyzed muscles. When applied to the eye the lids should be closed, one electrode, a wet sponge, is placed on them, the other is held in the hand or placed on the nape of the neck, and a current not so strong as to cause any considerable pain is then allowed to pass through the intervening tissues. For the relief of the pain attendant on cerebral tumors, morphia may be administered hypodermically, or, what I have found advantageous in several cases, the extract of Indian hemp, as recommended by Reynolds, may be used.

Counter-irritation, as produced by the actual cautery or other less powerful means, can do no possible good, and only adds to the discomfort of the patient.

CHAPTER XV.

INSANITY.

GENERAL PRINCIPLES.

The brain is the chief organ from which the force called the mind is evolved, and, so far as the present inquiry goes, may be regarded as the only one. For, though, wherever there is gray nerve-tissue, nervous force is generated, and though all nervous force partakes more or less of the character of that which we call mind, its qualities are not of such a nature as to bring their aberrations within the scope of this chapter.

By mind, therefore, we understand a force developed by nervous action, and especially by the action of the brain. The modifications which this force, in its cerebral relations, undergoes outside of the limits of health, either as regards excess, deficiency, or variation of quality, are embraced under the term insanity. Some authors have doubted the connection between the brain and the mind. Though we all feel that the relation does exist, it is perhaps as well to state briefly the facts which tend to establish the dependence of the one upon the functionation of the other. They have been well set forth by Mr. Bain :[1]

1. The action of an organ, even within the limits of health, frequently gives rise to sensations of various kinds,

[1] The Senses and the Intellect, second edition, London, 1864, p. 11. Also Mental and Moral Science; a Compendium of Psychology and Ethics, London, 1868, p. 5.

SCHOOL OF MEDICINE

and slight functional derangements are very distinctly felt. Thus, the pain of indigestion is referred to the stomach or bowels, as the case may be; difficulties with the urinary excretion cause uneasiness in the kidneys; derangements in the secretion of the bile cause pain in the liver; loud noises produce unpleasant feelings in the ears, and excessive or improper use of the eyes causes pain in these organs. So it is with the brain. Though ordinarily we are not conscious by any particular sensation that we are using it when we think (and the same is true *mutatis mutandis* of the other organs mentioned), yet inordinate mental exertion gives rise to headache, vertigo, and other derangements of sensibility, referable to the brain. I have had many patients under my charge in whom very slight mental action invariably produced pain in the head. It is well known that the brain becomes diseased when it is unduly taxed, just as does the spinal cord, the eye, or a muscle.

2. Injury or disease of the brain impairs in some way or other the powers of the mind. A blow on the head causes confusion of thought, and, if hard enough, may abolish consciousness or the power of thought altogether. A piece of fractured bone, or a bullet, pressing on the brain, likewise destroys the ability to think; and though, as in cases cited in another part of this treatise, there are examples of terrible wounds of the brain without, for a time, notable impairment of the mind, there is some loss from the first, and eventually the patients die with head-symptoms. The various affections of the brain which have been considered in this treatise, without exception produce, at some time or other of their course, derangement in the evolution of mind. Insanity, too, very often is shown, after death, to have been accompanied by structural changes in the brain.

3. The action of the brain, like that of other organs, results in the disintegration of its substance, and this destruction of tissue is in direct proportion to the amount of mental work done. We find, therefore, that the alkaline

phosphates, which are mainly derived from the destructive metamorphosis of the nervous tissue, and which are excreted by the kidneys, are increased in quantity after severe intellectual labor, and are diminished by mental quietude. In a memoir published several years ago, I gave the results of a series of experiments performed upon myself, which show very conclusively that increased use of the brain causes increased decay.[1]

4. The size of the brain is well known to bear a direct relation to the intelligence of the individual; and, when all other conditions are alike, it may be said that the largest brain will produce the greatest amount of mental energy. Quality is, however, also an important factor, and when with great size we also have a large amount of gray matter, the intellectual capacity is at its maximum.

Thus, Dr. Thurnam[2] has shown that the average weight of the brain in Europeans is 49 ounces, while in ten men remarkable for their intellectual development it was 54.7 ounces. Of these, the brain of Cuvier, the celebrated naturalist, weighed 64.5 ounces, Spurzheim's 55.06, and Daniel Webster's 53.5. On the other hand, the brain is small in idiots. In three idiots whose ages were sixteen, forty, and fifty years, Tiedemann found the weight of their respective brains to be $19\frac{3}{4}$, $25\frac{3}{4}$, and $22\frac{1}{2}$ ounces. Mr. Gore[3] has reported the case of a woman, forty-two years of age, whose intellect was infantine, who could scarcely say a few words, whose gait was unsteady, and whose chief occupation was carrying and nursing a doll. After death, her brain, carefully weighed, was found to weigh but 10 ounces and 5 grains.

Mr. Marshall[4] has also reported a case of microcephaly

[1] Urological Contributions, American Journal of the Medical Sciences, April, 1856, p. 330. Also Physiological Memoirs, Philadelphia, 1863, p. 17.

[2] Journal of Mental Science, April, 1866.

[3] Notes of a Case of Microcephaly, Anthropological Review, No. 1, May, 1863, p. 168.

[4] Brain and Calvarium of a Microcephale. Transactions of the Anthropological Society of London, in Anthropological Review, No. 2, August, 1863, p. 8.

existing in the person of a boy twelve years old, whose brain weighed but 8½ ounces. The convolutions were strongly marked, though few in number and narrow.

5. Experiments performed upon the nerves and nerve-centres show that from the brain proceeds the force by which muscles are moved, and that it is the organ by which sensations are perceived. Thus, division of a nerve supplying a certain muscle cuts off the connection between the brain and that muscle, and hence the will can no longer act upon it. Division of the optic nerve, for instance, prevents the perception of visual images.

From all of which considerations the connection between the brain and the mind is as clearly made out as any other fact in physiology.

The mind differs from forces in general, in being compound; that is, in being made up of several other forces. These are perception, the intellect, the emotions, and the will. All the mental manifestations of which the brain is capable are embraced in one or more of these parts. Either one of them may be exercised independently of the other, though they are very intimately connected, and in all continuous mental processes are brought more or less into relative and consecutive action. As constituting the basis of my classification of the several forms of insanity, it is expedient to describe these four sub-forces of the mind.

1. Perception.—By perception is to be understood that part of the mind whose office it is to place the individual in relation with external objects. For the evolution of this force the brain is in intimate relation with certain special organs which serve the purpose of receiving impressions of objects. Thus an image is formed upon the retina, and the optic nerve transmits the excitation to its ganglion or part of the brain. This at once functionates, the force called perception is evolved, and the image is perceived. If the retina be sufficiently diseased, the image is not formed; if the optic nerve is in an abnormal condition, the excitation is

not transmitted; if the ganglion be disordered, the perceptive force is not evolved.

Like reasoning is applicable to the other senses—hearing, taste, smell, and touch.

Perception may be exercised without any superior intellectual act—without any ideation whatever. Thus, if the cerebrum of a pigeon be removed, the animal is still capable of seeing and of hearing, but it obtains no idea from these senses. The mind, with the exception of perception, is lost.

2. The Intellect. — In the normal condition of the brain the excitation of a sense and the consequent perception do not stop at the special ganglion of that sense, but are transmitted to a more complex part of the brain where the perception is resolved into an idea. Thus the image impressed upon the retina, the perception of which has been formed by a sensory ganglion, ultimately causes the evolution of another force by which all its attributes capable of being represented upon the retina are more or less perfectly appreciated according to the structural qualities of the ideational centre. To the formation of the idea several important faculties and modes of expression of the intellect contribute.

Thus, if we suppose the retina to have received the image of a ball, a higher ganglion converts this into a perception, and a still higher one into an idea; and this idea relates to the size, the form, the color, the material, etc., primarily, and the origin, uses, ownership, etc., secondarily. In gaining this conception of the thing impressed upon the retina, the memory, judgment, and other faculties of the intellect are brought into action, and the process of reasoning is carried on.

3. The Emotions.—An idea in its turn excites another part of the brain to action, and an emotion is produced, or this last-named force may be evolved under certain circumstances without the intermediation of the idea, but solely

from the transmission of a perception to the emotional ganglion.

An emotion is that pleasurable or painful feeling which arises in us in consequence of sensorial impressions or intellectual action. According to Bain, the word emotion is used to comprehend all that is understood by feelings, states of feeling, pleasure, pain, passion, sentiments, affection, etc.

Within the limits of health the emotions act powerfully on certain organs of the body, and thus express their own activity. Thus grief is exhibited by the flow of tears from over-excitation of the lachrymal gland; extreme joy may also cause weeping; the jaw falls, and the angles of the mouth curve downward in mortification or sorrow, while in pleasure the face expands laterally. The eyes, the nose, and the mouth, are the three facial centres from which emotional expression is mainly produced. Other organs of the body, as the salivary glands, the heart, the mammary glands, the liver, the kidneys, and, in fact, nearly every viscus of the body, may exhibit the effects of emotion by the transmission of excitations through the sympathetic nerve. Most of the resulting effects are due to the fact that the sympathetic nerve especially presides over the vaso-motor system, and thus regulates the calibre of the blood-vessels.

The Will.—By volition acts are performed. Some acts are automatic, but all done in consequence of intellection are the result of willing, and are for some specific purpose connected with an idea. Volition in the series of mental manifestations may precede emotion, but it always follows ideation.

To sum up these outlines: A person walking in the street sees a man on the opposite side of the way—*Perception;* he recognizes him as a friend whom he has not met for many years—*Intellect;* he determines to go across and speak to him—*Will;* he does so, and exhibits joy at the reunion—*Emotion.*

Or, to alter the sequence somewhat:

A person at a theatre sees and hears an actor on the stage—*Perception;* the attitudes, gestures, and words of the player call up certain ideas—*Intellect;* he is moved to great joy or grief—*Emotion;* and, determining to recognize the ability of the actor—*Will*, claps his hands, or throws him a bouquet.

The mind, therefore, as before stated, is a compound force evolved by the brain, and its elements are perception, intellect, emotion, and will. The sun likewise evolves a compound force, and its elements are light, heat, and actinism. One of these forces, light, is again divisible into several primary colors, and the intellect of man, one of the mental forces, is made up of faculties. It would be easy to pursue the analogy still further, but enough has been said to indicate how clearly the relationship between brain and mind is that of matter and force.

In individuals whose brains are well formed, and free from structural changes, and are nourished with a due supply—neither excessive nor deficient—of healthy blood, the perception, the intellect, the emotions, and the will, act in a manner common to mankind in general. Slight changes in the formation or nutrition of the brain induce corresponding changes in the several parts of the mind, or in it as a whole. As no two brains are precisely alike, so no two persons are precisely alike in their mental processes. So long, however, as the deviations are not directly at variance with the average human mind the individual is sane. If they are at variance, he is insane. But within the limits of mental health marked irregularities are met with in different parts of the mind. Thus some persons are noted for never perceiving things as the majority of people perceive them. Others have the emotional system inordinately or deficiently developed. Others are weak in judgment, defective in memory, feeble in powers of application, or vacillating in their opinions. Others, again, are lacking in volitional power—in the ability to perform certain acts, to refrain from others,

or to follow a definite course of action which the intellect tells them is expedient and wise.

Eccentricity.—Persons whose minds deviate in some one or more notable respects from the ordinary standard, but yet whose mental processes are not directly at variance with that standard, are said to be eccentric. It is not always easy to draw the line between strong eccentricity and mild insanity. About the former, however, there is this marked characteristic: that its manifestations are according to a fixed system, are not founded on delusions, and are generally excited by those emotions or desires which are reflected back to the individual, such as vanity, pride, the love of approbation, or of notoriety, etc. Eccentric persons stand upon the verge of insanity with a decided predisposition to mental disease, and ordinarily do not pass the limit merely for want of a sufficient exciting cause. Several instances of eccentricity passing into undoubted insanity have come under my observation. In one of these, a lady had since her childhood shown a singularity of conduct as regarded her table-furniture, which she would have of no other material than copper. She carried this fancy to such an extent that even the knives were made of copper. People laughed at her, and tried to reason her out of her whim, but in vain. In no other respect was there any evidence of mental aberration. She was intelligent, by no means excitable, and in the enjoyment of excellent health. An uncle had, however, died insane. A trifling circumstance started in her a new train of thought, and excited emotions which she could not control. She read in the morning paper that a Mr. Kopperman had arrived at one of the hotels, and she announced her determination to call on him. Her friends endeavored to dissuade her, but without avail. She went to the hotel, and was told he had just left for Chicago. Without returning to her home, she bought a ticket for Chicago, and actually started on the next train for that city. The telegraph, however, overtook her, and she was brought back

from Rochester, raving of her love for a man she had never seen, and whose name alone had been associated in her mind with her fancy for copper table-furniture. She died of acute mania within a month.

In another case a young man, a clerk in a city bank, had for several years exhibited peculiarities in the keeping of his books. He was exceedingly exact in his accounts, but after the bank was closed for the day he always remained several hours, during which he ornamented each page of his day's work with arabesques in different-colored inks. His fellow-clerks amused themselves at his expense, but his superior officers, knowing his value, never interfered with him in his amusement. Gradually, however, he conceived the idea that they were displeased with him, and at last this became so firmly rooted in his mind that he resigned his position, notwithstanding the protestations of the directors that his idea was erroneous. Delusions of various kinds soon supervened, and he is now hopelessly insane.

Inquiry will frequently disclose the fact that the insane have been eccentric for several years before becoming affected with cerebral disease to such an extent as to produce decided mental aberration.

Definition of Insanity.—Every medical witness, who appears in a case involving the mental capacity or responsibility of an individual, is expected to give a definition of insanity. It is extremely difficult to do this satisfactorily, as it is also with a great many other terms which are applied to complex forces. It is difficult to give such a meaning to the word as will cover all possible cases of deficiency or aberration of the mental faculties, and yet not include those instances of cerebral disease which cannot properly be classed under this head. For the purpose of showing how authors have varied in their ideas of the signification of the word, as well as for the instruction of the reader seeking for information on the point, I quote a number of definitions from some of the most eminent authorities:

Dr. John Haslam,[1] who has written one of the most lucid treatises on insanity in the English language, and who was for many years one of the physicians to Bethlehem Hospital, confesses his inability to give a thoroughly comprehensive and yet a sufficiently exclusive definition of madness; and Dr. Prichard[2] frankly admits that it is better to give up the attempt to define insanity in general terms. Notwithstanding the reluctance of these and other medical authorities to formularize the phenomena of insanity, the attempt has frequently been made with more or less approach to completeness. If the word can be even imperfectly defined in simple language without conveying erroneous ideas, it is certainly advisable to make an effort in this direction.

According to Hoffbauer,[3] an individual is insane when the understanding is diverted or changed in its operations; when he is powerless to avail himself of his intellectual faculties, or to make known his wishes in a suitable manner.

This definition, though embracing all cases of insanity, is not satisfactory, for the reason that it is applicable to certain cerebral disorders which are not properly classed under this head. Among these may be mentioned apoplexy, and concussion and compression of the brain.

Dr. Bucknill, in his "Essay on Criminal Lunacy," defines insanity as "a condition of the mind in which a false action of conception or judgment, a defective power of the will, or an uncontrollable violence of the emotions and instincts has separately or conjointly been produced by disease." This definition is a very excellent one, but still includes those diseases of the brain attended with unconsciousness which are not insanity.

Dr. Guislain,[4] an eminent Belgian authority, says that

[1] Observations on Madness and Melancholy, etc., second edition, London, 1809, p. 37.

[2] Article Insanity, in Cyclopædia of Practical Medicine.

[3] Untersuchungen über die Krankheiten der Seele. Halle, 1803, p. 11.

[4] Leçons sur les Phrénopathies, tome i., p. 45.

"insanity is a morbid derangement of the mental faculties unattended by fever, and chronic in its character, which deprives man of the power of thinking and acting freely as regards his happiness, preservation, and responsibility."

The objections to this definition, in addition to those applicable to the others given, are that insanity is not necessarily unaccompanied by fever, and that it is not always a chronic affection.

Drs. Bucknill and Tuke,[1] quoting from Maimon, say that "mental health consists in that state in which the will is free, and in which it can exercise its empire without obstacle. Any condition different to this is a disease of the mind, and if it is asked, What is the will? it may be replied, according to the definition of Marc, that it is a moral faculty which originates, directs, prevents, or modifies the physical and moral acts which are submitted to it."

The late Prof. Gilman, of this city, who had given a great deal of study to the subject, declared that the best definition he had been able to make was, that "insanity is a disease of the brain by which the freedom of the will is impaired." This has the advantage of being short and of being to the point. Other diseases, however, are included in its terms.

It would be easy to go on and quote numerous other authorities on this point, but enough have been cited to show the general import which physicians give to the word insanity. I will therefore dismiss the further consideration of this division of the subject, by stating that my own idea of insanity is based entirely on the fact, that as the healthy mind results from a healthy brain, so a disordered mind comes from a diseased brain. Insanity, therefore, strictly speaking, is only a symptom, and I would define it as—

A manifestation of disease of the brain, characterized by a general or partial derangement of one or more faculties of

[1] A Manual of Psychological Medicine, etc., London, 1858, p. 79.

the mind, and in which, while consciousness is not abolished, mental freedom is perverted, weakened, or destroyed.

An essential feature of the definition of insanity here given is, that it depends directly upon a diseased condition of the brain. This is the immediate cause, and may consist of structural changes due to injury, disease, or malformation, or of malnutrition, the result of excessive intellectual exertion, the action of powerful emotions, irritations in distant parts of the body, the sudden stoppage of the digestive process, the introduction into the system of certain drugs, such as opium, alcohol, belladonna, etc., the retention in the organism of substances poisonous in character, but which in health are excreted, and of other factors capable of altering the quantity or quality of the blood circulating through the cerebral vessels, or of accelerating or retarding the metamorphosis of tissue which the brain undergoes in common with all the other organs of the body. These causes, with others, will be more fully considered hereafter.

Classification.—Many classifications have been made of the several manifestations of insanity. As is well known, mental disease appears under different characters, just as does the healthy mind. Some authors have been exceedingly minute in their arrangement, making a type of the disease from each particular symptom or delusion the patient may show. Others, again, are metaphysical and unpractical.

The classification of Esquirol[1] has been very generally adopted, with more or less modification according to the peculiar ideas of the authors. Although arbitrary, and based upon the principle of regarding the symptom as the disease, it is certainly the best of its kind; and, though made more than thirty years ago, is still followed by many writers. The terms employed by Esquirol are so frequently met with in works on insanity, that I give his classification as a part of the history of the subject:

[1] Des Maladies Mentales, tome i., p. 11, Paris, 1838.

1. *Melancholia.*—Perversion of the understanding in regard to an object or a small number of objects, with the predominance of sadness and depression of mind.

2. *Monomania.*—Perversion of understanding limited to a single object or a small class of objects, with predominance of mental excitement.

3. *Mania.*—A condition in which the perversion of understanding embraces all kinds of objects, and is attended with mental excitement.

4. *Dementia.*—A condition in which those affected are incapable of reasoning, from the fact that the organs of thought have lost their energy and the force necessary for performing their functions.

5. *Imbecility or Idiocy.*—A condition in which the organs have never been sufficiently well-conformed to permit those affected to reason correctly.

One of the latest and most able writers on the subject of mental derangement is Dr. Maudsley,[1] and he classifies the several forms of insanity according to the mental symptoms, as follows:

I.—Affective or Pathetic Insanity.

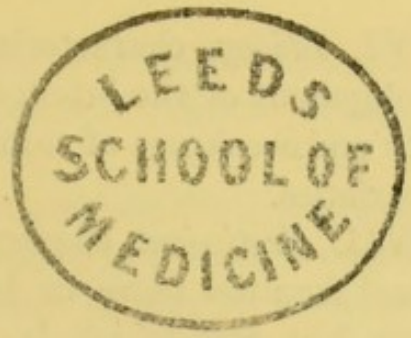
1. Maniacal perversion of the affective life. Mania sine delirio.
2. Melancholic depression without delusion. Simple melancholia.
3. Moral alienation proper. Approaching this, but not reaching the degree of positive insanity, is the insane temperament.

II.—Ideational Insanity.

1. General.
   a, Mania.
   b, Melancholia, { acute, chronic.

[1] The Physiology and Pathology of the Mind. London and New York, 1867, p. 323.

2. Partial.
   a, Monomania.
   b, Melancholia.
3. Dementia, { primary, secondary.
4. General Paralysis.
5. Idiocy or Imbecility.



In 1867, an International Congress of Alienists was held in Paris, and a committee, appointed by that body to make a classification, reported the following:

I. *Simple insanity*, embracing the different varieties of mania, melancholia, and monomania, circular insanity and mixed insanity, delusion of persecution, moral insanity, and the dementia following these different forms of insanity.

II. *Epileptic insanity*, or insanity with epilepsy, whether the convulsive affection has preceded the insanity and has seemed to have been the cause; or whether, on the contrary, it has appeared during the course of the mental disease only as a symptom or a complication.

III. *Paralytic Insanity.*—This commission regards the disease called general paralysis of the insane as a distinct morbid entity, and not at all as a complication, a termination of insanity. It proposes, then, to comprehend under the name of paralytic insane all the insane who show in any degree whatever the characteristic symptoms of this disease.

IV. *Senile dementia*, which we would define as the slow and progressive enfeeblement of the intellectual and moral faculties consequent upon old age.

V. *Organic dementia;* a term by which the commission means to designate a disease which is neither the dementia consequent upon insanity or epilepsy, nor paralytic dementia, nor senile dementia, but that which is consequent upon organic lesion of the brain, nearly always local, and which presents, as an almost constant symptom, hemiplegic occurrences more or less prolonged.

VI. *Idiocy*, characterized by the absence or arrest of development of the intellectual and moral faculties. Imbecility and weakness of mind constitute hereof two degrees or varieties.

VII. *Cretinism*, characterized by a lesion of the intellectual faculties more or less analogous to that observed in idiocy, but with which is uniformly associated a characteristic vicious conformation of the body, an arrest of the development of the entirety of the organism. Outside of these typical forms there are others, such as—

1. Delirium tremens.
2. Delirium of acute diseases; traumatic delirium.
3. Simple epilepsy.

My own classification is much simpler than most that have been proposed, and is based entirely on the division of mind given. In part it has been brought forward by other authors, though with different explanations of the terms employed.

I. *Perceptional insanity*, characterized by the tendency to the formation of erroneous perception either from false impressions of real objects (illusions), or from no external excitation whatever (hallucinations).

II. *Intellectual insanity*, characterized by the existence of delusions.

III. *Emotional insanity*, characterized by the uncontrolled or imperfectly-controlled predominance of one or more of the emotions.

IV. *Volitional insanity*, in which there is an inability to exert the full will-power either affirmatively or negatively.

V. *Mania*, characterized by the union of two or all four of these forms in the same individual.

VI. *General paralysis*, a peculiar form of insanity, attended with progressively-advancing loss of mental and motor power.

VII. *Idiocy and dementia:* the first due to the fact that

there are original structural defects in the brain; the second resulting from the supervention of organic changes in a brain originally of normal power.

In a work like the present, embracing the diseases of the whole nervous system, it is, of course, impossible to consider at full length the very interesting subject of insanity. I shall endeavor, however, to give certain prominent features, referring the reader, for more complete information, to the monographs treating specifically of diseases of the mind.[1]

But, before proceeding to describe the several types mentioned, there are some important symptoms of mental disorder, the character and import of which must be clearly understood. These are illusion, hallucination, delusion, incoherence, and delirium.

Illusion.—An illusion is a false perception of a real sensorial impression. Thus a person seeing a ball roll over the floor, and imagining it to be a mouse, has an illusion of the sense of sight; another, hearing the pattering of the rain on the roof, and perceiving in this sound the voice of some one calling him, has an illusion of the sense of hearing; another, having some bitter substance placed upon his tongue, and forming the perception of a sweet flavor, has an illusion of the sense of taste; and so on as regards the other senses. In all such cases there is a material basis for the perception, but this latter is not in exact relation with the former.

Illusions are not always indicative of cerebral disorder; indeed, they are very common with all of us under certain circumstances. It is, perhaps, never the case that the perception is precisely in accordance with the real properties of the substance making the sensorial impression. We never see, hear, taste, smell, or feel things exactly as they are. This imperfection may be due to surrounding circumstances not being favorable. Insufficient light may thus make our

[1] No work is better calculated to give philosophical views of the subject of insanity than the treatise of Dr. Maudsley, on the "Physiology and Pathology of the Mind." His little work on the "Body and Mind" is also admirable.

vision imperfect; loud noises may render us incapable of appreciating gentle sounds. A strongly-sapid substance previously rubbed over the tongue and fauces prevents our distinguishing delicate flavors; a powerful odor may make such an impression on the schneiderian membrane that other odors for a long time smell like it, and exposure to very cold weather interferes markedly with the discriminating power of the sense of touch.

Imperfect perceptions are often formed in consequence of the perceptive ganglia being otherwise occupied. Thus, if we are looking intently at some object of interest, we are apt not to attend to the sounds which reach our ears, and consequently no clear perception of them is formed.

Illusions of all the senses, but especially of sight and hearing, are met with in insanity, and particularly in those acute forms characterized by the presence of delirium.

Hallucination.—An hallucination is a false perception without any material basis, and is centric in its origin. It is more, therefore, than an erroneous interpretation of a real object, for it is entirely formed by the mind. An individual, who, on looking at a blank wall, perceives it to be covered with pictures, has an hallucination; another, who, when no sounds reach his ear, hears voices whispering to him, also suffers from an hallucination, and such false perceptions may be created as regards all kinds of sensorial excitations. The organs of the senses, in fact, are not necessary to the existence of hallucinations. Thus, if the eyes be closed, images may still be seen; if the hearing be lost, voices may still be heard, and the reason for this is found in the fact that the erroneous perception constituting the hallucination is formed in that part of the brain which ordinarily requires the excitation of a sensorial impression for its functionation. Hallucinations are always evidence of cerebral derangement, and are common phenomena of insanity. They may be excited by emotions of various kinds, by which the character or quantity of the blood circulating in the brain is changed,

by intellectual exertion, by certain drugs, and many other factors to be presently more fully considered.

Delusion.—Illusions and hallucinations may exist, and the individual be perfectly sensible that they are not realities. In such cases the intellect is not involved. But, if he accepts his false perceptions as facts, his intellect participates, and he has delusions. A delusion is, therefore, a false belief. It may be based upon an illusion or an hallucination, may result from false reasoning in regard to real occurrences, or be evolved out of the intellect spontaneously by the result of imperfect information, or of an inability to weigh evidence or to discriminate between the true and the false. Delusions are not a test of insanity, as most lawyers and many physicians believe. If they were, one-half the world would be trying to put the other half in lunatic asylums! They may be present without coexistent insanity, and many cases run their course without them.

To be indicative of insanity, a delusion must be contrary to the customary mode of thought of the individual. Thus a believer in spiritualism is not necessarily insane because he sees and converses with the spirit of Benjamin Franklin, for it is a part of his mentality to believe in the existence of spirits and in the possibility of evoking them so as to see them and talk with them. But, if a non-believer in spiritualism should imagine that he was in the habit of seeing Franklin's spirit and of conversing with it, it would be good evidence of his insanity. And further, though the spiritualist might think he had interviews with Franklin, and still be sane, yet if he believe, without foundation and contrary to evidence, that his brother had tried to poison him, he would have a delusion sufficient to indicate his insanity.

At a former period of the world's history, a belief in the possibility of seeing devils and demons of various kinds, and of suffering from their torments, was commonly entertained. Indeed, it is religiously held now by a great many otherwise sensible people. Such a belief is, according to my mode of

thought, a delusion, and probably nine-tenths of those who read this treatise will agree with me in so regarding it. But it certainly would not be safe to consider every one holding such a creed as insane. A like reasoning applies to the holders of every other form of belief not in accordance with our own. A delusion, to be indicative of insanity, must be such a belief as would not be entertained in the ordinary normal condition of the individual, must have been formed without such evidence as would have been necessary to convince in health, and must be held against such positive testimony as would have in health sufficed to eradicate it.

Insanity may exist without delusions at any time being present. Thus there may be emotional insanity, the main feature of which consists of mental depression with an unreasoning tendency to suicide; or there may be volitional insanity, characterized by an inability to refrain from setting fire to neighbors' houses, or from committing homicide.

Incoherence.—A person is said to be incoherent when the words he utters are without proper relation to each other, or when his language is not in accordance with his ideas. As an example of incoherence, I cite the following letter which I received a few days since from a patient:

"In the Neck, *January* 7, 1871.

"Dear Sir: I said he was in my own conscience that the book was confined I quote the long time with eccentricity in the common way. This is in memory to my upshot which was incorrect at the final oblivion. Dogs and money consistency with foundlings without ante bellum which was in statu quo.

"This is passive in contiguity with the works met in the creation of existence.

"Very commingle

"in good faith

"J. S. W———."

This exhibits an extreme case, as there is not an idea to

be obtained from the language used. Such instances are, however, common enough.

Incoherence is a prominent feature of delirium, and is sometimes met with in the chronic insane. It is directly due either to the impossibility of keeping the attention sufficiently long on one idea for its full consideration, or to a like difficulty in coördinating those parts of the brain which are concerned in the formation and expression of thoughts.

Delirium.—Delirium is that condition in which there are illusions, hallucinations, delusions, and incoherence, together with a general excess of motility, an inability to sleep, and acceleration of pulse. In acute delirium these phenomena are well marked; in the low and chronic forms they are less strongly indicated. Sometimes one or the other of these elements notably predominates. Delirium is present in the early stage of acute mania, and may exist as an accompaniment of certain diseases of the brain which do not ordinarily cause insanity, such as cerebral congestion or anæmia. It is also common in fevers and in several other disorders of the system.

I.—Perceptional Insanity.

In uncomplicated perceptional insanity, those parts of the brain only are disordered which are concerned in the formation of perceptions. It constitutes the primary form of mental aberration, and of itself is not of such a character as to lessen the responsibility of the individual, or to warrant any interference with his rights. It consists entirely in false perceptions; and if the intellect is for a moment deceived, the error is immediately corrected. As already stated, these are either illusions or hallucinations. In some cases these erroneous perceptions may coexist in the same individual. They may be related to all the senses, but are especially common as regards sight and hearing.

Illusions, as already mentioned, are not necessarily due to any centric difficulty, though such an origin is common.

Thus, it is an illusion if a person on looking at an object sees two images. This result is due to some cause destroying the parallelism of the visual axes, and may be produced by a tumor of the orbit or by paralysis of one or the other of the ocular muscles. Even in such a case, if the paralysis were due to central lesion, the higher ganglia of the brain might escape implication. Illusions are often excited by emotional disturbance, and are then probably directly due to some disturbance of the cerebral circulation. The false perceptions called hallucinations are of more importance than illusions, in the symptomatology of insanity in general. In the purely perceptional form of mental aberration they are also exceedingly interesting, and are very often troublesome symptoms. Thus, a gentleman, who had overworked himself in financial business, was subject to hallucinations of hearing, which, however, did not in the least impose on his intellect. As he walked through the streets to his place of business, he heard a voice continually whispering to him, "Take care—take care!" So strong was the impression made, that he often involuntarily turned round to see who was speaking to him. In another case, a gentleman saw images of various kinds as soon as his head touched the pillow, though they were never present when he was standing or sitting.

The case of Nicolai, the German bookseller of the last century, is well known as a remarkable example, and others are afforded in the cases of Jerome Cardan, Pascal, and many other noted personages.

Like illusions,[1] the immediate cause of hallucinations is generally derangement of the cerebral circulation, either as regards quantity or quality.

As is well known, they are frequently produced by alco-

[1] For a fuller account of the subjects of illusions and hallucinations, the reader is referred to the author's works on Sleep and its Derangements: J. B. Lippincott & Co., Philadelphia; and the Physics and Physiology of Spiritualism: D. Appleton & Co., New York.

holic liquors, opium, belladonna, Indian hemp, and other drugs. They may also result from mental exertion and emotional disturbance, from an overloaded stomach, or may occur in the course of various diseases, especially those of a febrile or exhausting character.

Perceptional insanity may make its appearance suddenly, the first evidence of its presence being the illusion or hallucination. Usually, however, there are prodromata indicating cerebral derangement. These are pain in the head, irritability of temper, suffusion of the eyes, noises in the ears, a general restlessness, and some febrile excitement. The skin is generally dry, the mouth parched, the bowels costive, and the urine high colored and scanty. If not arrested, it may pass into one or the other of the following types of mental aberration.

II.—Intellectual Insanity.

The essential feature of intellectual insanity is delusion. It may be developed suddenly, or, as is generally the case, preceded by evidences of cerebral disorder, which, though at the time of their occurrence not attracting particular attention, are called to mind by the observers after the disease has become fully developed.

In the first stages of intellectual insanity it is not often that the delusions are fixed, and they may succeed each other with such rapidity that the patient resembles one affected with mania. They may be based on illusions or hallucinations, or may arise from the reasoning of the patient from purely imaginary premises not connected with the senses. Sometimes they are spontaneous, and at others they appear to come from dreams.

Thus, a gentleman, who had for several days been singular in his behaviora, woke in the night and imagined that he saw his wife standing by his bedside with a phial of prussic acid which she was about to empty into his mouth. The hallucination took such strong hold of him that he went

into the adjoining room, where his wife slept, to see if she were there or not, and, though he found her sleeping quietly, he awoke her, and accused her of having attempted to poison him. No amount of argument or persuasion could eradicate the false belief from his mind.

Another for several days had been spending money very freely in articles of little or no use to him, when one morning he announced to his family that for several days he had been thinking a great mistake had been committed in his conception, and that his soul had got into the wrong body. He was therefore convinced that he was not the man he should have been, and hence he had done a great many things which were altogether repugnant to his physical senses. So long as the antagonism continued between his mind and his body, there was no hope of any happiness for him in this world. In this case there had never been any hallucination or illusions of any of the senses. The delusion was therefore entirely the result of the patient's own perverted thoughts.

When rapidly following each other, delusions are clearly spontaneous—are not the result of any series of thoughts, but come on the spur of the moment and upon very slight suggestions. As they are readily formed, they are not fixed in character. A lady, for instance, after receiving some very sorrowful intelligence relative to her husband, imagined that she had lost her eyesight. For a few hours she remained with her eyes shut, alleging that there were two deep cavities behind the lids. Suddenly she opened them, said she saw perfectly well, but that the top of her head had been cut off, and this was almost immediately changed to the belief that she was perishing with cold, and so on, no one delusion lasting longer than a few minutes. In many cases like this the erroneous beliefs are excited by sensations in various parts of the body, but this was not so in the present instance.

The connection between dreams and insanity is very

close. Most of us have at times had such vivid dreams that they have been removed from our minds with difficulty. There appears to be no doubt that many of the delusions of the insane have dreams for their cause.

The delusions of the insane are, in the great majority of cases, connected more or less directly with themselves. Thus a person believes that his leg is made of glass, that his head is reversed on his shoulders, that he is some great personage, that a large fortune has been left to him, or that some misfortune has deprived him of his property or his friends. He will often reason logically and forcibly from the premises he has assumed, and will give no evidence of insanity outside of his delusion. Such cases are embraced under the term of reasoning mania, and the skill and acumen exhibited by persons thus affected are often surprising. When it is important, in their estimation, for them to conceal their delusions, they will often do so for a long time, and stratagems of various kinds are necessary to their speedy detection. Sooner or later, however, the delusion comes out.

The designation monomania can properly be applied to many of the cases of intellectual insanity. In the uncomplicated form of the disease it is rare, after it is fully established, that more than a single object, or a small class of objects, are the subjects of the delusions.

The delusions of the insane may be comprehended under two categories—those which are of a pleasant or exalted character, and those which are unpleasant or morbid. These usually leave their impress on the countenance of the patient, and his actions and manner are in accordance with them.

It would be strange if this were not the case. The only guide which man has for his actions is his reason. He weighs arguments and motives, and determines according to the bearing which they may have on his mental processes. A delusion is, in many cases, simply a false premiss; the

conclusions which the individual draws from it are entirely logical. Taking, for instance, the case of the gentleman who had imbibed the idea that his wife had attempted to poison him; and, admitting that he was correct in this notion, his subsequent conduct—his denunciations, his refusal to live with her, his efforts to have her imprisoned, etc.—is perfectly reasonable. The line of conduct was such as most men would have pursued under like circumstances. In such cases, therefore, there is no fault in the intellectual processes after the first step is taken. It is this first step which constitutes the disease—it is the delusion which enslaves the mind.

Intellectual insanity is often uncomplicated by any other form of mental derangement. There are no illusions, no hallucinations, no overpowering influence of the emotions, and no loss of control over the will. Even when the delusion is of such a character as apparently to be connected with some one of the senses, and thus to be based upon a false perception, full inquiry will often show that there is no error of the sensorial processes, centric or eccentric. Thus, a lady under my care had the delusion that she had lost her palate, as she called it. I held a mirror to her face, and, while she opened her mouth, I pointed out to her that all the parts were present. "Yes," she replied, "I see all that; the form is there, I know very well, but the substance is gone;" and no arguments could convince her to the contrary. A gentleman conceived that his right hand was made of glass, and therefore, to prevent its being broken, he kept it carefully enclosed in a stout case made to fit it accurately. On my calling his attention to the physical qualities of his hand, and pointing out how they differed from those of glass, he said: "I once thought just as you do. My brain was then incapable of appreciating minute differences as well as it can now; and, though I confess that my senses still convey to me the idea that my hand is like other people's, yet I know the conception is erroneous, and I cor-

rect it at once by my reason. My hand looks like flesh and blood, but it is glass for all that. Nothing is more calculated to deceive than the senses."

Persons affected with uncomplicated intellectual insanity may go through the world without giving any considerable evidence of mental derangement, unless the subject of their delusions be touched upon. Still, there is no telling to what extremes a delusion may carry its subject. Like a sane idea, it may extend further with each day of life. A person, for instance, imagines that he is the Emperor of Russia. At first he does not comprehend the full importance of his supposed position, and, if of moderate reasoning power, possessing deficient information, and naturally of a quiet disposition, he may never go further than dressing himself in some tawdry finery, and strutting pompously through the wards of the hospital. But, under other circumstances, he reflects upon the greatness of his station, and thus, from time to time, he conceives new ideas of his powers and importance, and may thus become a very troublesome patient. He comes to believe, perhaps, that he has the power of life and death, and may attempt to exercise his imaginary prerogative.

Delusions in regard to relatives and friends are very common, and hence the conduct of the person entertaining them is changed as it relates to the objects of his erroneous ideas. It is a usual thing, therefore, for such an insane person to disinherit those who would naturally be heirs of his property. This point is of importance in its medico-legal relations.

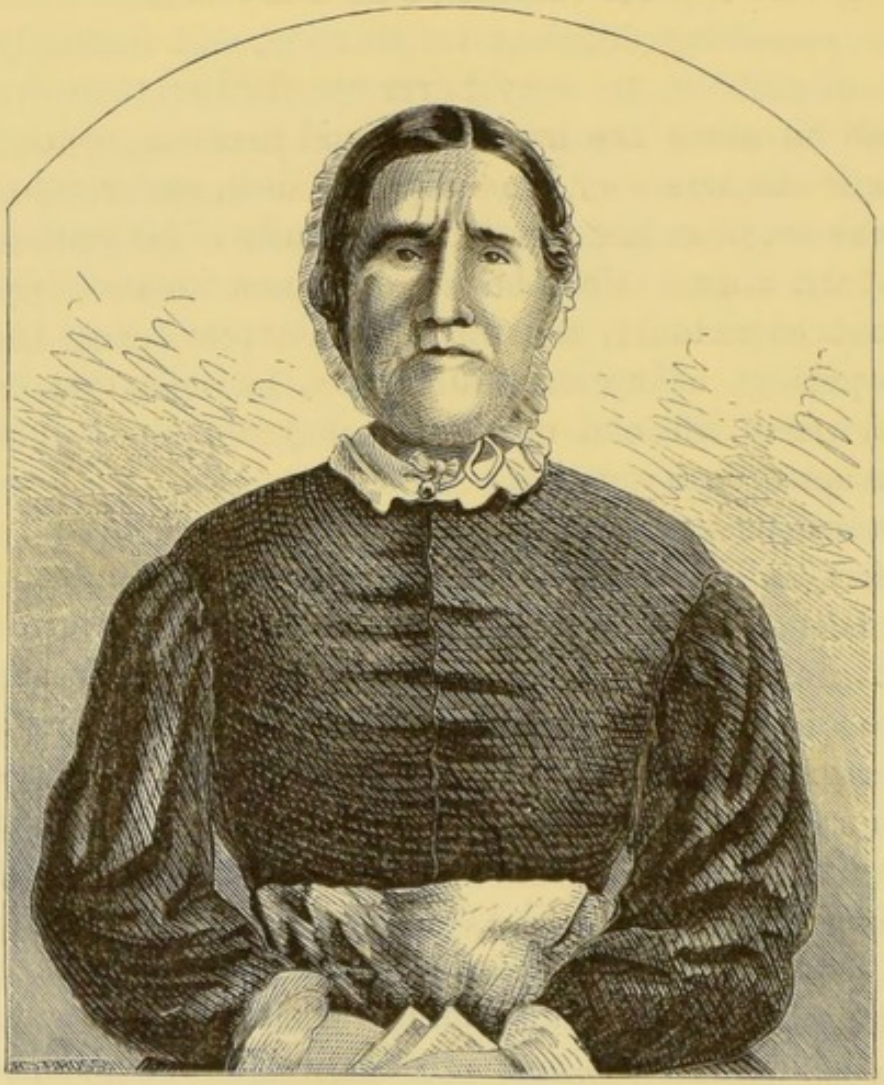
Delusions may be of such a character as to affect the emotions secondarily. A very common delusion is that of having committed the unpardonable sin, and accordingly the patient suffers great emotional disturbance. This influence upon the emotions is perfectly natural and logical, for, if the person really had committed a sin for which there is no hope of pardon, and had thus incurred the punishment of

eternal damnation, it would be strange if the emotions of sorrow and despair were not excited into activity. Such cases, however, are not to be embraced under the head of emotional insanity; and, though at first sight they may appear to be of that type, inquiry will reveal the fact of the preëxistence of the delusion.

Intellectual insanity is often the sequence of an attack of acute mania, which form of mental aberration will be presently considered.

I subjoin the accompanying portrait, engraved from a

FIG. 12.



photograph, of a typical case of intellectual insanity. The patient was, for many years, an inmate of the New York

City Lunatic Asylum on Blackwell's Island. Her delusion was, that she was the wife of the late President Buchanan. She assumed his name, and was exceedingly tenacious of her rights and dignities. All visitors were received by her with as much formality as though she were the real mistress of the White House. It will be seen, upon examination, that there is no trace of emotional disturbance to be perceived in her countenance. The expression of her face is intelligent and shrewd, and she might have walked Broadway every day of her life without exhibiting as much evidence of insanity as many of those who perambulate that thoroughfare and are considered perfectly sane.

III.—Emotional Insanity.

The emotions are at all times difficult to control, but they may acquire such undue prominence as to dominate over the intellect and the will, and assume the entire mastery of the actions in one or more respects. This effect may be produced suddenly, from the action of some cause capable of disturbing the normal balance which exists among the several parts of the mind, or it may result from influences which act slowly but with gradually-increasing effect. In either case there is not necessarily either delusion or error of judgment, but it very generally happens that the intellect sooner or later becomes involved.

Emotional insanity may be produced without there being any discoverable cause, and without the patient being able to allege a motive.

Some emotions are more frequently disordered than others. Those of a sorrowful character are preëminent in this respect, and, when they are affected, the type of insanity called melancholia is the result. This may be either acute or chronic in its course. The first is rarely uncomplicated, and hence will be more properly considered under the head of mania.

Homicide, suicide, and other crimes, may be the result

of emotional disturbance as well as of intellectual insanity. The most common of these is undoubtedly suicide, the individual committing self-destruction in order to escape from the depressing influences which act upon him. It more frequently happens, however, that the emotions are disordered through the morbid operations of the intellect. A person, for instance, to cite the example previously given, imbibes the delusion that he has committed the unpardonable sin, or that God has deserted him, and, in consequence, passes into a condition of settled melancholy, during which he may attempt self-destruction to escape from his harrowing thoughts, or commit a homicide, in order that the same end may be accomplished by his being hanged for murder. Other emotions may of course be excited into morbid activity by derangement of the intellect. Delusional jealousy, anger, hatred, or love, may thus urge their unfortunate victim to the perpetration of crime, plunge him into a depth of unhappiness from which there is no escape, or lift him into an ecstasy of bliss far exceeding that derivable from the realization of all his wishes.

Under the head of moral insanity, Dr. Prichard, several years ago, described a form of mental derangement which embraced several species which are now more properly placed under other heads. Several of these are clearly emotional in character, and most of them relate to altered modes of feeling or of the affective faculties, and therefore, in the largest sense of the word, may also be called emotional. Careful and thorough inquiry will, however, often show that the primary difficulty is one of defect, and not of aberration or exaggeration, and that, therefore, these instances of deficient moral sense, leading the subjects to the perpetration of crimes of various kinds, should be classed under the head of imbecility.

Many cases of what are called temporary insanity, mania ephemera, transitory mania, and morbid impulse, are really instances of emotional insanity. That such a condition ex-

ists there can be no doubt, and it is important, both as regards the subject and society, to be able to recognize or to disprove its presence.[1] A few words, therefore, on this point, will not be out of place.

The state with which transitory emotional insanity is most apt to be confounded is that which has been designated heat of passion. Passion is emotional activity. It refers to that mode of the mind in which certain impressions or emotions are felt, and which is accompanied by a tendency or impulse, often irresistible, to act in accordance with these impressions or emotions irrespective of the intellect. An act performed in the heat of passion is one prompted by an emotion which for the moment controls the will, the intellect not being called into action. It is an act, therefore, performed without reflection. The passions are, to a certain extent, under the control of the will, and this power of checking their manifestations is capable of being greatly increased by self-discipline. Some persons hold their passions in entire subjugation, others are led away by very slight emotional disturbances. The law recognizes the natural weakness of man in this respect, and wisely discriminates between acts done after due reflection and those committed in the midst of passional excitement.

The acts performed during temporary emotional insanity, in their more obvious aspects, and when viewed isolatedly, resemble those done in the heat of passion. But they are so only as regards the acts themselves. Thus a person, entering a room at the very moment when one man was in the act of shooting another, would be unable to tell whether the homicide was done in the heat of passion, or under the influence of an attack of temporary insanity; he would be equally unable to say whether it was committed with malice aforethought or in self-defence. The act, there-

[1] The best monograph on temporary insanity with which I am acquainted is that of Krafft-Ebing, Die Lehre von der Mania Transitoria, für Aerzte und Juristen dargestelt. Erlangen, 1865.

fore, by itself, can teach us nothing. We must look to the attending circumstances, and to the antecedents of the perpetrator, for the facts which are to enlighten us as to the state of mind of the actor. Now, the conditions of temporary emotional insanity are so well marked that the act which indicates the height of the paroxysm may almost be disregarded, for it is always preceded by symptoms of mental aberration, while acts done in the heat of passion are not thus foreshadowed.

And, as regards the subsequent state of the individual, the distinction is equally apparent. The one who has committed a criminal act in the heat of passion soon subsides to his ordinary condition of equanimity, and generally begins to think of his safety. The one who has perpetrated a similar act during an attack of temporary emotional insanity never thinks of escape, nor even avoids publicity. He may even boast of his conduct, or deliver himself into the hands of the law. What is, however, of greater importance is the fact that, though he may subside into a condition of comparative sanity, the evidences of disease are still present, and remain in him for days, weeks, or even months and years. These symptoms are generally those of cerebral congestion, to which attention has already been directed.

In heat of passion, the act follows immediately on the excitation of which it is the logical sequence. In temporary insanity, the act is the culmination of a series of disordered physical and mental manifestations, and may or may not be in relation with the emotional cause. The distinction is, therefore, clear and precise. The case of Henriette Cornier, so fully detailed by Georget,[1] is a striking instance of the action of emotional disturbance and morbid impulse. This woman was twenty-seven years of age, was of a joyous disposition and gentle in her ways, and particularly fond of young children.

In June, 1825, a notable change ensued in her; she be-

[1] Discussion Médico-Légale sur le Folie, ou Aliénation Mentale, Paris, 1826.

came sedate, seldom laughed, sighed often, was taciturn, and neglectful of her work. She was accordingly discharged from her service as domestic, and returned to her friends. She soon afterward made an attempt at suicide by throwing herself from the parapet of a bridge, but was prevented.

She then entered the service of a Madame Fournier, still being disposed to melancholy, notwithstanding all efforts made to restore her.

On the 4th of November, her mistress went out, leaving Henriette at her work, and directing her to go to a shop kept by a woman named Belon, and get some cheese. This woman had a very beautiful little daughter not two years old, for whom Henriette had always manifested a great liking. On this occasion she fondled the child as usual, and persuaded her mother to let her take it out to walk. Henriette took the child to Madame Fournier's house, and, going first to the kitchen, obtained a large knife, with which, and the child, she went to her own room. On the staircase she met the porteress, and, before her, embraced with every evidence of love the little child she held in her arms. Arrived at her own chamber, she laid the infant on its back on the bed, and, seizing its head with one hand, she with the other drew the knife rapidly across the neck and severed the head from the body before her victim could utter a cry. Before, during, and after this crime, she had, as she declared, no emotion or feeling of horror. On the contrary, she was calm, collected, felt neither pleasure nor sorrow, but apparently acted mechanically.

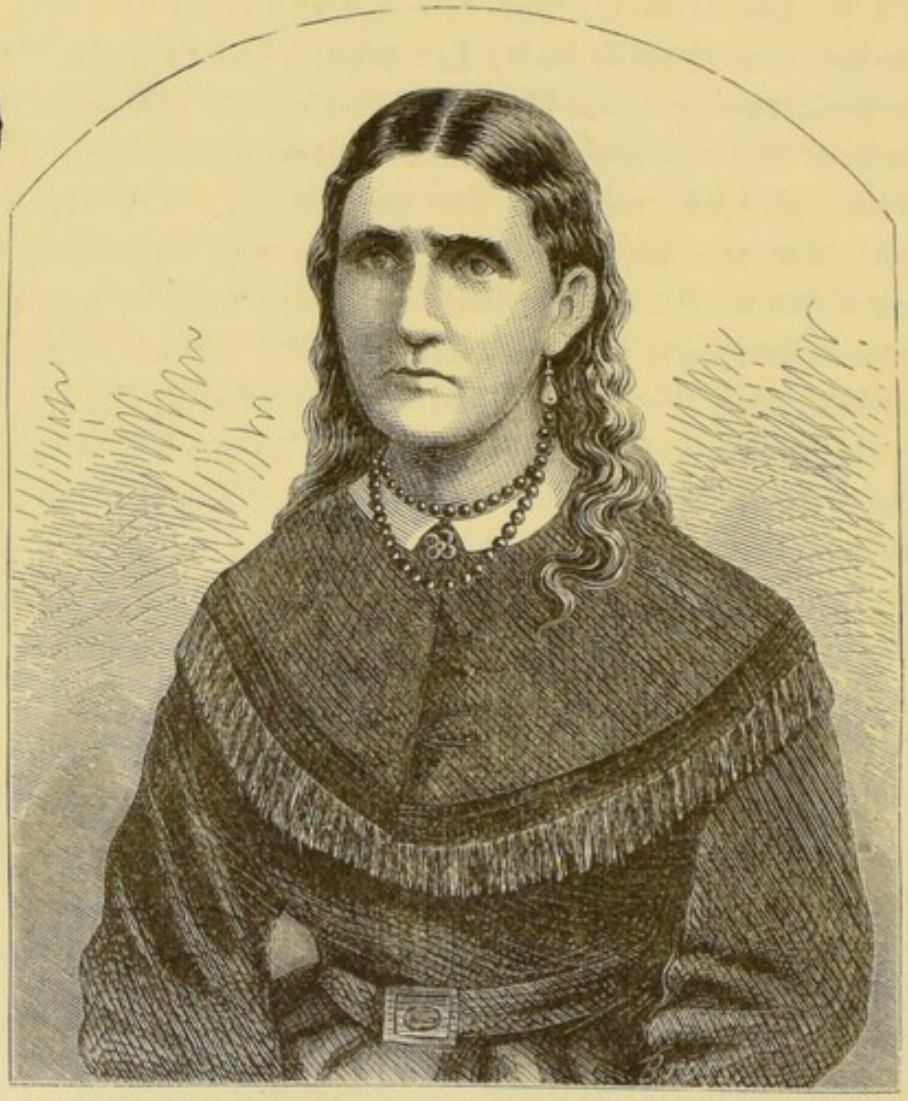
Two hours afterward the mother came for her child; Henriette stood at the door. "Your child is dead," she exclaimed, and then, entering the room, seized the head of the murdered infant and threw it into the street.

On the arrival of the officers, she was found sitting in the room with the dead body, gazing at it, her hands covered with blood and the knife near her. She did not deny her crime, and exhibited neither penitence nor remorse. "I in-

tended to kill it," she said, and, on being further interrogated, declared that she had no particular motive; that she had experienced the inclination, and she was destined to perpetrate the act.

She was suspected of insanity, and was examined by a commission consisting of Adelon, Esquirol, and Léveillé, who reported that they were unable to determine whether she was sane or not. This report not being satisfactory, a second examination was ordered, but still no definite opinion could be obtained from the commission. She was tried and found guilty, very illogically, of voluntary but unpremeditated homicide, and was sentenced to hard labor for life.

FIG. 13.



The above likeness (Fig. 13) is that of a woman affected

with pure emotional insanity, of a depressing character, but without delusions of any kind. She could assign no cause for the intense melancholy with which she was affected, and which caused her to pass the greater part of the day crying and wringing her hands. She had twice attempted suicide before she came under my care, not from any delusion, but solely that she might escape from her overpowering emotions and the mental anguish they caused her. She was fully sensible of her situation, knew how groundless was her grief, and constantly lamented her inability to control her feelings.

IV.—Volitional Insanity.

In uncomplicated volitional insanity, there are no delusions and no emotional disturbance, but solely an inability to exert the will in accordance with the intellect. Many cases of morbid impulse are instances of volitional insanity, in which an idea suddenly flashing across the mind is immediately carried out by the individual, although his intellect and his emotions are strongly exerted against it. Thus, a person who previously has not exhibited any very obvious symptoms of mental derangement—though careful inquiry will invariably show that slight evidences of cerebral disease have been present for some days—instantaneously feels a morbid impulse to commit a murder or perpetrate some other criminal act, and is forced to yield, notwithstanding all the efforts he may make. Numerous cases of the kind are on record.

Thus Esquirol[1] relates the case of a man thirty-two years old, of a nervous temperament and quiet disposition, who had been well educated, and who was fond of the fine arts. He had suffered from a brain-disorder, but had been several months cured. After being in Paris for about two months, during which time he led a perfectly regular life, he one day entered the Palais de Justice and attacked an advocate

[1] Des Maladies Ment. Paris, 1838, t. i., p. 380.

with great fury. The next morning, when seen by Esquirol, he was perfectly tranquil and composed, showed no anger whatever, and had slept well all night. The same day he designed a landscape. He recollected what he had done the previous day, and spoke of it with coolness. He declared that he had entertained no ill-will against the advocate, had never even seen him before, and had no business with him or any other lawyer. He could not understand, he said, what had actuated him to make the assault. Subsequently he exhibited no indications whatever of being insane.

Many instances of the so-called moral insanity may properly be placed under the head of volitional insanity, for they are characterized by an inability to so exert the will as to refrain from the perpetration of acts known to be crimes. Of such are cases of kleptomania, dipsomania, pyromania, etc.

The will in insanity is often secondarily affected through disturbance originating in the intellect or the emotions, and acts are hence performed which give evidence of the existence of mental aberration. In mania of all kinds, and especially in dementia and general paralysis, there is either a loss of volitional control, or an inability to exert the normal will-power.

V.—Mania.

In mania the mind is affected in several, generally all of its parts. There are illusions, hallucinations, delusions, emotional disturbance, and loss of volitional power or control. The patient is either morbidly excited or depressed, and is often violent in his language and actions. Acute mania is the more common species of mental aberration, and in its two types of exaltation and depression constitutes the form which it is most important for the physician to understand. I shall therefore consider them at some length, so far as their symptoms and cause are concerned.

Acute mania with exaltation has its prodromatic stage, the symptoms of which are very similar to those which pre-

cede an attack of fully-developed cerebral congestion. These in the main are pain or fulness in the head, confusion of ideas, increased irritability of the mind, and, above all, wakefulness. In addition, there are restlessness of body and a singularity of behavior, which strike those thrown into intimate relations with the subject, and cause them to suspect that something is wrong with him.

The character and disposition undergo a change, and it is very common for unfounded prejudices to be formed against persons formerly highly esteemed.

Before very long there are illusions and hallucinations. At first the patient struggles against them, but eventually he accepts them as true, and hence becomes subject to delusions. These are rarely fixed in the earlier stages, and may not be so through the whole course of the disorder.

With these symptoms there are evidences of derangement in other organs besides the brain. Thus, the appetite is lessened, the bowels are torpid, the kidneys fail to eliminate the normal quantity of urine, the heart becomes irregular in its action and beats with increased frequency, a certain sign of a weak and excited nervous system, and the skin is either bathed in perspiration or is dry and hard.

With the full development of the disorder the patient becomes incoherent and rambling, showing a great disposition to talk, to laugh, and to sing, and indulging in antics of various kinds. His delusions mainly have reference to himself: he imagines that he is some great personage, that he has suddenly become very rich, or that he has been specially singled out for some other piece of good fortune.

Not unfrequently he is exceedingly troublesome, destroying the furniture of his room, tearing his clothes, attacking those around him, and making all kinds of attempts to escape from restraint, but at the same time there is rarely any serious effort to do great bodily harm either to himself or others. Sometimes, however—and this fact should always be born in mind by the attendants—there is a disposition to

perpetrate acts of extreme violence, and such a tendency, even when not previously manifested, may very suddenly be developed.

Thus, a lady under my care, who had a few days before become insane, behaved with propriety, merely making continual efforts to get into the street to attend court, where, as she believed, she had an important lawsuit. Without any warning, however, she went into an adjoining room where her infant child was sleeping, and threw it out of the window before she could be stopped in her act, exclaiming: "Well, if I can't go out, my baby shall." Fortunately, the child fell on a thick grape-vine, and was not injured.

In another case, a gentleman, whom I saw in consultation with my friend the late Prof. George T. Elliot, became affected with acute mania of the most hilarious and exalted character. While playing on the piano and singing with the utmost glee, he expressed a wish for a cracker, and went to the dining-room to get one. While apparently looking for something to eat, he suddenly seized a knife and attempted to cut his throat. The close proximity of his attendant alone prevented his inflicting serious injury on himself.

As a rule, patients with acute mania lose all sense of decency, and become exceedingly filthy in their habits and obscene in their language and conduct.

At times such lunatics exhibit a surprising degree of cunning, and are able to exercise great control over their conduct when they have an end to accomplish. They may thus readily deceive the young and inexperienced physician, and induce him to forego the idea of putting them under permanent restraint, or they may so impose on him as to induce him to relax his vigilance, and thus allow of their committing some outrageous act.

It must be remembered that acute mania is not suddenly cured, but runs a definite and allotted course.

It is rare that the memory of the patient suffers to any considerable extent in acute mania. The patients are per-

fectly conscious of their surroundings, and are seldom deceived by the subterfuge so frequently and so unjustifiably employed that they are to be taken to a hotel or a country-seat when about to depart for an asylum. If the stratagem does for the moment impose upon them, they recollect the fraud, and will not again repose confidence in those who have perpetrated it.

Their appetites are generally unchanged. If in the habit of smoking or drinking, they still want their tobacco and their wine, and are usually able to eat a full allowance of food.

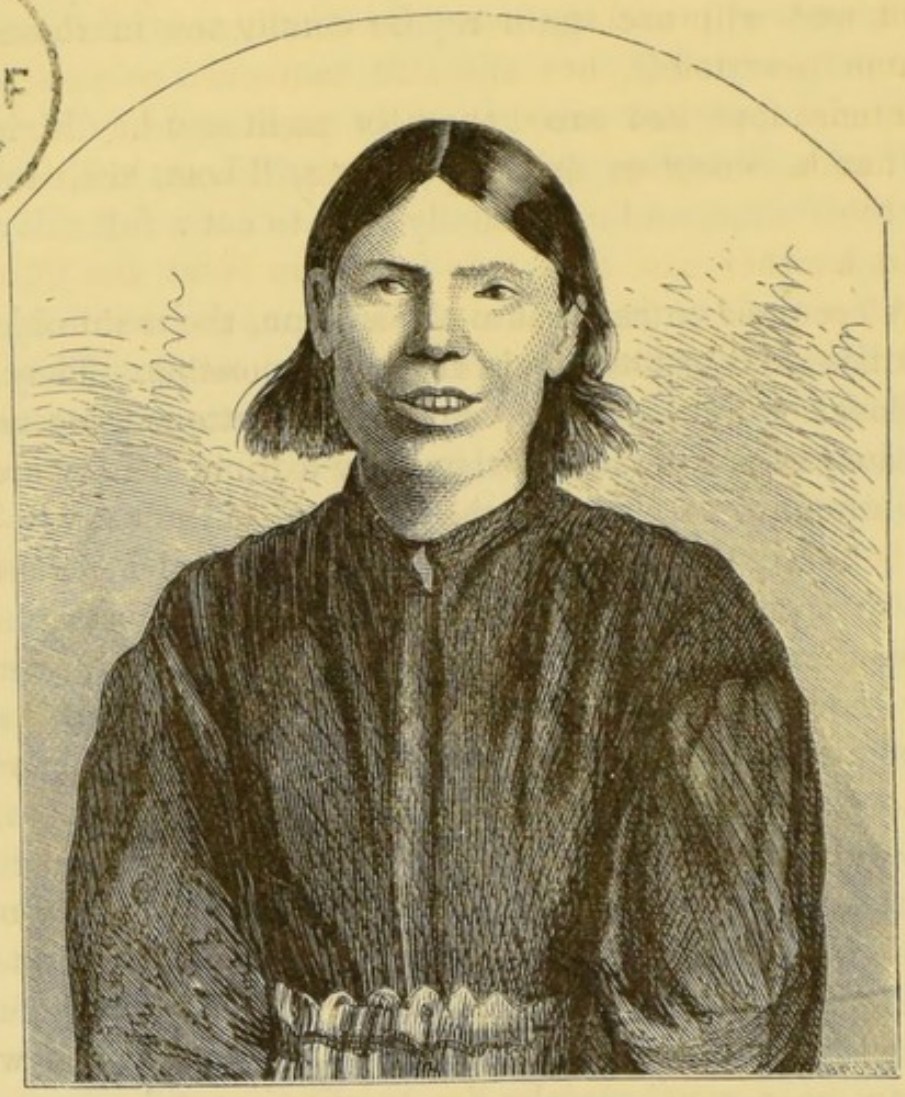
After their entrance into the asylum, the main object of their lives is to get out again as soon as possible. They often recognize their condition, and will call attention to any indications of improvement they may exhibit. They are not for a moment deceived by the delusions of their fellow-lunatics. "Doctor," said a patient to me whom I had sent to a lunatic asylum, and was visiting, "this is the best place in which to study the infirmities and humbugs of human nature of which I have any knowledge. Everybody here is insane except myself. There is a fellow I used to know before he lost his mind, a good, clever fellow he was too, and as sharp as a steel trap. Now he is a d—d fool, and thinks he takes his breakfast off the top of the capitol every morning. And there is a lady holding that bunch of rags to her breast and thinking it's a baby. These lunatics are funny, very funny, but I've had about enough of them, and would like to go somewhere else." At the time this gentleman thought he was General Grant, and was going to be inaugurated President in a few days.

It is rarely the case that the sleep is regular and sound. Often they will lie awake all night, talking of their plans, or else will annoy their attendants in every conceivable way. Although having usually uncomfortable feelings in the head, they rarely suffer from acute pain in that part of the body.

The accompanying woodcut represents a case of acute mania, with general mental exaltation. No one can fail to perceive the expression of happiness on the face.

EEDS
HOOL OF
DICINE

FIG. 14.



Acute Mania, with Depression.—The acute melancholia of many authors is a very terrible form of mental aberration. Like that just described, it is generally preceded by prodromata, which indicate, by their character, the type of insanity which is about to be developed, but it often appears with great suddenness. In the case of a lady now under my charge, the first evidence of mental disorder was a violent scream, due to the fact that an idea had instantaneously

flashed through her mind that she had committed the unpardonable sin, and had consequently lost all hope of saving her soul. For several days she continued, with scarcely an intermission, to scream, to cry, and to sob, at the same time showing the greatest terror from the apprehension that the devils were approaching her. Gradually this extreme state became less violent, but she still continued to be actuated by intense fear, and paced the floor night and day, wringing her hands, weeping, and exclaiming, "Lost, lost, lost forever!"

In another case of a lady from the West, the idea suddenly occurred to her that she was about to be killed. She screamed, and begged, and prayed, to those around her not to allow her to be injured. In the furniture and attendants she saw her murderers, and to escape from them made several attempts to throw herself out of the window. Then she believed that she was to be poisoned, and refused all food with the utmost pertinacity—closing her teeth so firmly together that it was only by the use of all my strength that I could succeed in prying them open.

Of all the forms of insanity, this is the one in which illusions and hallucinations of the senses are most common. These are particularly so as regards sight and hearing, and do not, as a general thing, refer to the body of the patient—although generally in direct relation with his delusion.

A gentleman, who, within a short period after becoming affected with the present variety of insanity, came under my care, was controlled by the delusion that he had committed so many sins that atonement must be made. He had, therefore, several times attempted suicide, and, when I entered the room where he was, he was in the act of struggling with his friends, who were using all their strength to prevent him throwing himself out of the window. As soon as he saw me, he fell on his knees, held up his hands in the attitude of prayer, and mumbled out a few words which showed that he took me for a priest, and was asking for in

tercession with the offended Deity. On arriving at the asylum, to which I recommended him to be immediately sent, he went at once to an open coal-fire, and, before any one knew what he was about, thrust his hand into the mass of burning coals, and succeeded in injuring it terribly.

Fig. 15.

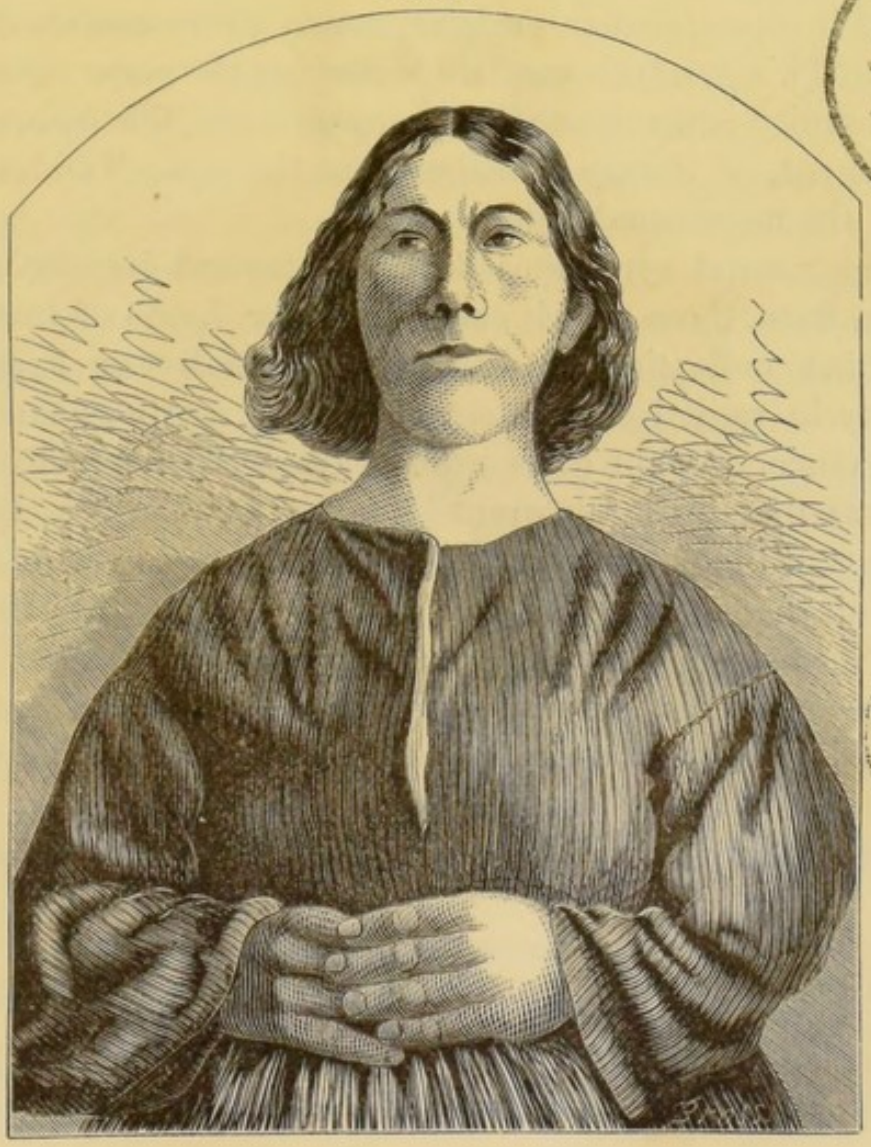
In all cases of acute mania with depression, too great care cannot be taken to prevent self-injury or suicide. It must be constantly kept in mind that the idea is a very common one with this class of patients, and that frequently they manifest great astuteness in concealing it till they are ready to make the attempt.

The physician, in general practice, should always urge that patients affected with the form of insanity under consideration should, as soon as possible, be placed in an asylum, for it is almost impossible to manage them in ordinary houses, or with their friends about them.

The preceding woodcut (Fig. 15) is an admirable likeness, taken from a photograph of a young woman in the New York City Lunatic Asylum, suffering from acute mania, with depression. Apprehension and terror are plainly depicted on her countenance.

Fig. 16 represents a female inmate of the same asylum,

Fig. 16.



LEE
SCHOOL
MEDICI

whose history I have not been able to obtain, but whose expression of countenance, though less pronounced than that of the preceding, is nevertheless sufficiently indicative of the existence of mania with depression.

VI.—General Paralysis.

The affection now known as general paralysis was first described by Delaye,[1] in 1822; then by Bayle,[2] in the same year; and then, with much more thoroughness and exactness, by Calmeil,[3] in 1826. It is a very common form of mental derangement, and, aside from the implication of the mind, presents the very striking feature of a gradually-advancing paralysis, which derives its name of general from the fact that it involves, sooner or later, nearly every muscle of the body. This paralysis may show itself at the same time that the insanity is manifested; it may precede the mental derangement, or it may be subsequent thereto. The latter is much the more usual order.

The mental symptoms differ, in several important respects, from those which occur in other forms of insanity. The first indication of disease is generally an excessive anxiety in regard to matters which are really of no great importance. Of the cases which have come under my care, one was first made apparent by a morbid apprehension on the part of the patient that he was not managing some trust-funds in the best possible way; another by the idea that he was constantly wounding the feelings of his friends; and another was continually changing his mind about the most trivial things, and apparently thinking that the world watched, with great anxiety, all his movements.

At first the general mental type is that of depression.

[1] De la Paralysie Générale Incomplète. Thèse de Paris, 1822.

[2] Recherches sur les Maladies Mentales, Paris, 1822; and Traité des Maladies du Cerveau et des Membranes, Paris, 1826.

[3] De la Paralysie Considerée chez les Aliénés. Recherches faites dans le service de feu M. Roger-Collard et de M. Esquirol, Paris, 1826.

The emotions are easily excited, and the delusions which soon make their appearance are of the melancholic form. The idea of propriety in the every-day acts of life seems to be lost, and the patient will commit all kinds of indecent acts without appearing to be aware that he is doing any thing unusual. His memory fails rapidly, and his intellectual vigor declines from the very first. Hence he is not able to argue in defence of his delusions, but attacks with physical force those who venture to differ with him. His acts are in other respects eccentric and absurd. He spends money in things which are of no manner of use to him, and at the same time refuses to pay his small debts; he harasses in every possible way those who are about him, gives them impossible orders, and then abuses them if they are not at once obeyed; he is whimsical at the table, his likes and dislikes are changed, and he either eats and drinks voraciously, or declares that nothing is cooked to suit him, and leaves the table in a rage. Gradually the form of his mental aberration changes; he becomes more cheerful, forms all kinds of impossible schemes for suddenly acquiring great wealth, and these are quickly abandoned for others equally impracticable. Thus delusion after delusion rapidly succeeds each other, and these, in the great majority of cases, relate to the grandeur, the wealth, the physical strength, or some other great quality of the patient constituting the *délire des grandeurs* of the French. One will tell of his immense palaces built of gold and inlaid with precious stones, and in the next breath will descant of his great weight or his extreme lightness, or of the number of children he has, or of the millions of operas he has composed. Another urges his great importance in the political world, tells us that he has elected all the members of Congress himself, that he has paid off the national debt, and that in consequence he is to be made Emperor of the United States, with a salary of a thousand millions a year; that he is going to have a thousand physicians, who are to be clothed

in blue-velvet uniforms embroidered in gold and diamonds; that he has chartered the Great Eastern for a pleasure-trip, and engaged ten thousand musicians and a similar number of ballet-dancers to go with him. The next day he has forgotten all these fancies, and is off on another series of absurd ideas. In no respect is he restrained in the extent of his delusions. Impossibilities are not regarded. While scarcely able to drag one leg after the other, he will brag of his great fleetness of foot, and in the very death-grasp will mutter about his extreme strength and endurance.

The symptoms connected with sensation are equally well marked. In the early stage headache is often very severe, so much so that, as Westphal[1] has remarked in his excellent monograph on the subject of general paralysis, the patient often dashes his head against the wall. At other times the feeling in the head is that of fulness or tightness, and these sensations are often accompanied with vertigo. Neuralgia in various parts of the body is common, and some of my patients have complained of the different degrees of numbness, especially in the hands and feet.

But still more strongly manifested are the disorders of motility, due to the progressive paralysis. According to my experience, the first sign of loss of power—one which is very often observed before any evidence of mental derangement is perceived—is a slight defect of articulation due to paralysis of the lips. At first this is scarcely perceptible; there is merely a little trembling, an action such as that seen in persons who are endeavoring to restrain their emotions, but it is sufficient to give indistinctness to the utterance of those words which contain labial letters.

The tongue is the next to be affected. Examination shows that there are fibrillary contractions of its muscles, and it is moved with less facility. The articulation is slovenly, words are slurred over, and there are both stammering

[1] Ueber den gegenwärtigen Standpunct der Kenntnisse von der allgemeinen progressiven Paralyse der Irren. Griesinger's Archiv, Heft. i., p. 44.

and stuttering. The patient notices these difficulties, and in endeavoring to obviate them makes matters worse, by his inability to be exact, contrasting strongly with his efforts. The paralysis of the tongue gradually becomes more complete, and at last this organ can only be moved with great difficulty. The other facial muscles participate, and a blank, somewhat sorrowful, expression is constantly present. The voice loses its fulness, and there is difficulty of swallowing.

The muscles of the eye are also generally involved, producing ptosis from paralysis of the levator palpebræ superioris diplopia from implication of the internal rectus, and contraction of the pupil—all of these effects, except the last, being due to difficulty existing at the point of origin, or in the course of the third nerve. The pupils are often unequal, and Austin[1] declares with all seriousness that contraction of the right pupil is associated with melancholic delusions, and contraction of the left pupil with elation. Further investigation has not confirmed this theory. The gait of patients affected with general paralysis is very peculiar, and is of two distinct kinds. In the one it is similar to that of a person suffering from sclerosis of the posterior columns of the spinal cord (locomotor ataxia). The feet are lifted high, and are thrown down with a good deal of force, the heel striking the ground first. As Westphal remarks, patients with this gait cannot stand with the eyes shut and the feet close together.

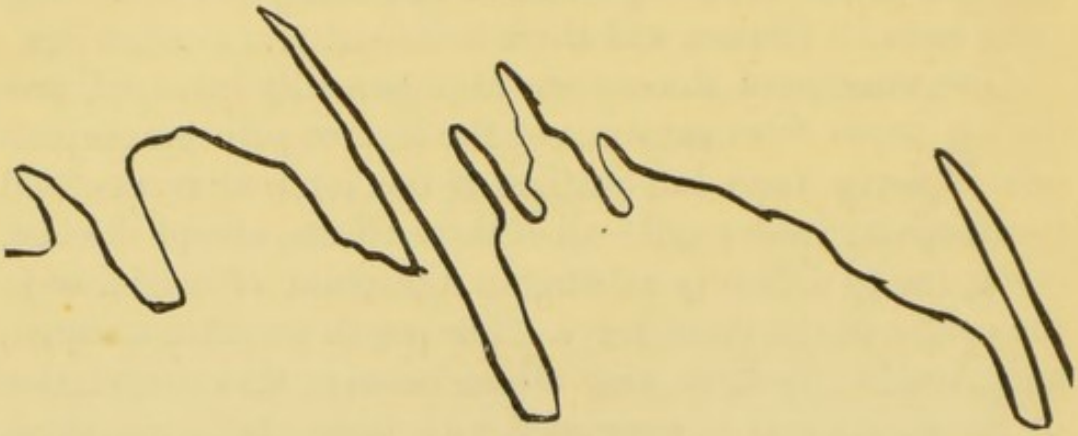
In the other kind the feet are scarcely lifted from the ground, but are shuffled over it, and the action is somewhat like that of a person attempting to balance himself on a tight-rope. Patients with this gait can without difficulty stand with the eyes shut.

As regards the upper extremities, the fingers lose their strength and delicate coördinating power. The handwriting is shaky, and there is awkwardness in buttoning the

[1] A Practical Account of General Paralysis, its Mental and Physical Symptoms, Statistics, Causes, Seat, and Treatment, London, 1859, p. 31, *et seq.*

clothing. The grip of the hand is still strong, but there is an impossibility, as shown by the dynamograph, of maintaining a continuous muscular contraction for even a few seconds. The following is one of the tracings made by a patient affected with the disease under consideration:

Fig. 17.



In analyzing this tracing, we see that it is not from feebleness of the muscles that the line is descending, for there are spasmodic elevations which show considerable force. It proves, however, that, no matter at what point the pencil is placed, the patient cannot keep it there.

The senses, with the exception of sight, do not often become materially affected. Atrophy of the optic nerve causes amaurosis or amblyopia. Ophthalmoscopic examination will very generally detect this condition of the papilla at a very early stage of the disease, together with retinal and choroidal anæmia.

Convulsive seizures occur, and these are generally epileptiform in character, though occasionally they are of the nature of apoplexy. They vary greatly in character, sometimes resembling the *petit mal* of epilepsy; at others, characterized by strong convulsive movements or coma. Besides these, there are attacks of complete paralysis of certain muscles, or groups of muscles, which, however, rarely leave any permanent effects, the usual degree of power being regained in a few days.

The course of general paralysis is often marked by periods of great improvement, and the patient's friends imagine that he is certainly recovering. The symptoms, mental and physical, all abate in violence, and may even disappear to such an extent as not to be evident to general observers. But the physician must not be deceived, for the amelioration is merely temporary, and sooner or later the disease regains its former ascendency. At no time, even during the height of the remission, is the mind of the patient in such a condition as to admit of any considerable intellectual exertion. There may be an absence of delusions, but the mental weakness still exists.

Progressively this decline in the force of the mind becomes more strongly marked, until at last a condition of extreme dementia is reached. Simultaneously the physical power diminishes, until finally the patient, unable to walk, to stand, or even to sit, is confined to the bed for the rest of his existence. Bed-sores form, and deglutition becomes more and more difficult. From this cause, the food may become impacted in the fauces, and thus death produced by interruption to the respiratory process; or the food may enter the larynx. The sensibility of the lining membrane of the cheeks and fauces is notably diminished, and hence the patient, in eating, goes on filling his mouth, not knowing that he is doing so. When he at last attempts to swallow, the mass of food is greater than can pass down the œsophagus, and, unless some one is near to assist him, he chokes to death.

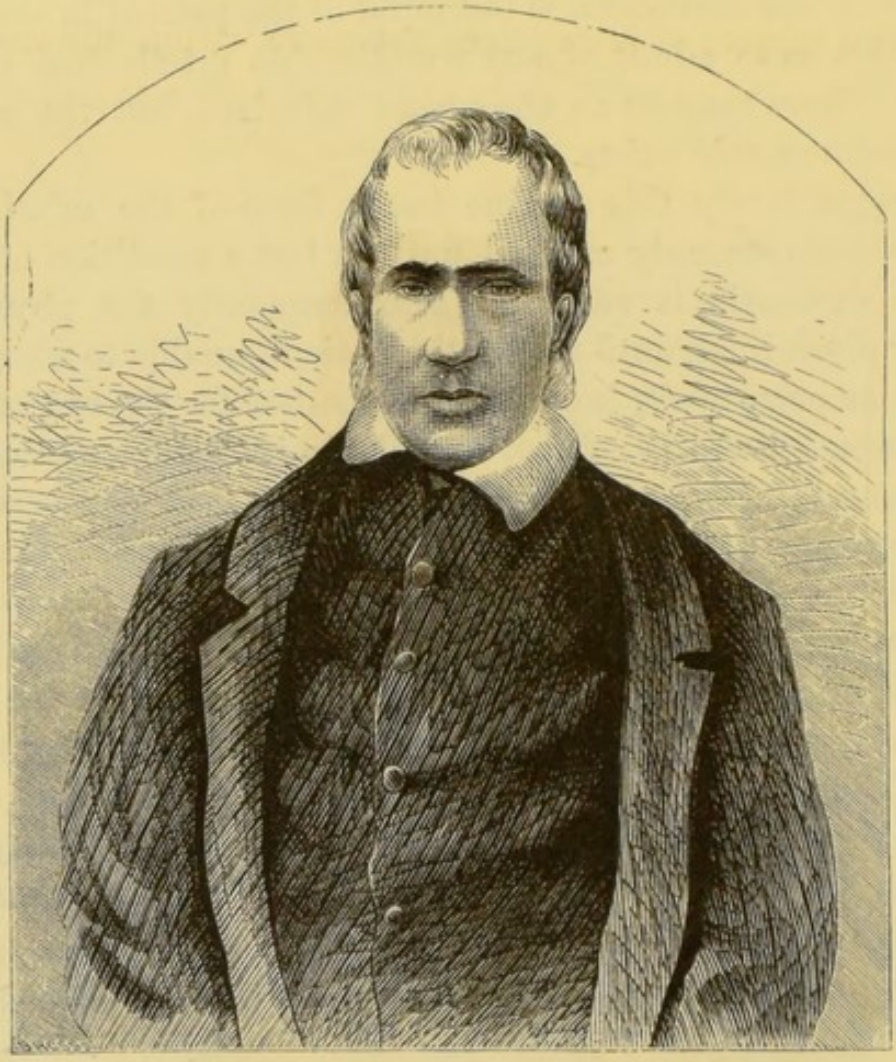
Death may otherwise take place from a gradual cessation of the respiratory process, or from sheer exhaustion.

The duration of general paralysis is variable. Sometimes death results in a few months, and at others it may be deferred for five or six years. The average period is about three years. General paralysis is not likely to be confounded with any other affection than chronic alcoholic intoxication, from which the history of the case and its general

progress will suffice to distinguish it. With lead paralysis it has scarcely any features in common.

General paralysis is almost invariably fatal. A few cases of recovery have been reported, but there is room for doubting that most of them were actual cases of the disease, and the others were probably, as Griesinger suggests, instances in which the remission was long. A complete recovery in

FIG. 18.



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which the patient has been able to resume his ordinary occupation, and to exert his natural mental and physical powers, has certainly not come under my notice. At the same time, recognizing the skill brought to bear upon modern medical science, I do not despair of eventually being able to give a more hopeful statement. That the life of the patient

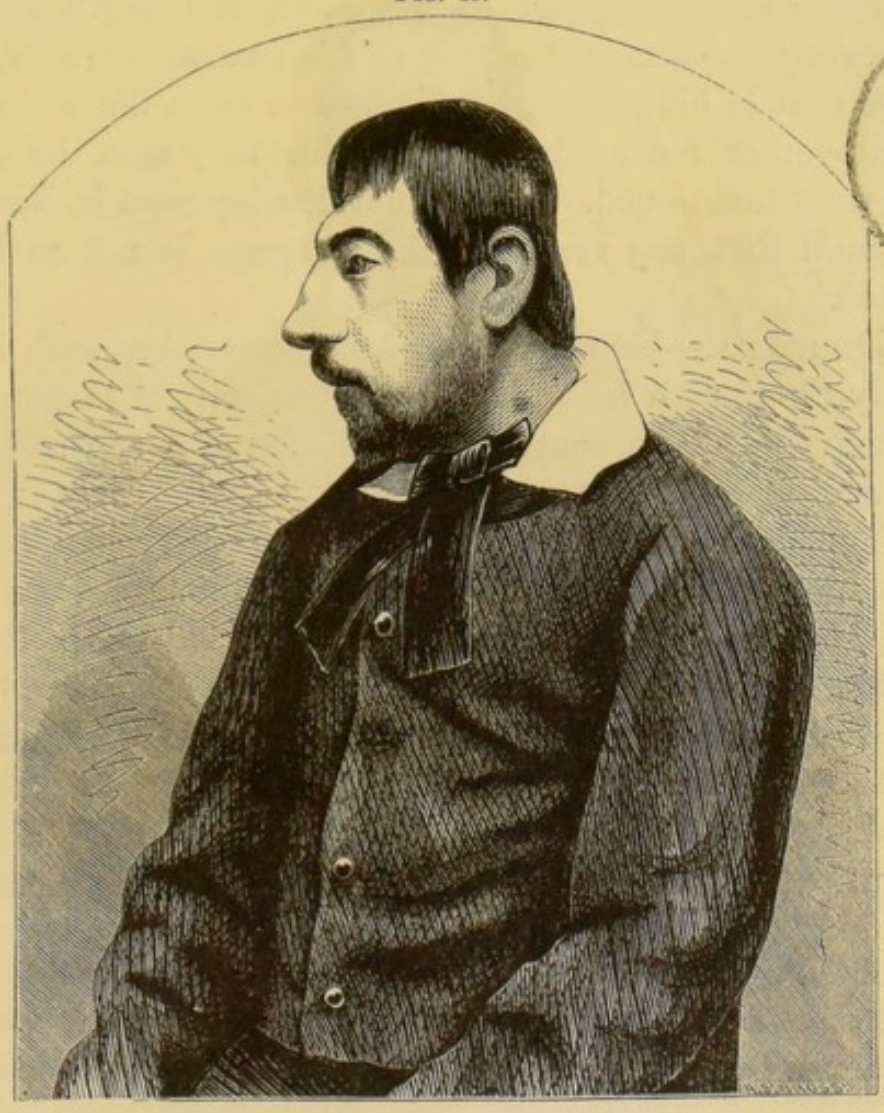
can be prolonged and his condition improved by judicious management I am very sure.

The preceding cut (Fig. 18), which I take from Tuke and Bucknill's "Manual of Psychological Medicine," gives a more typical representation of the face of a patient suffering from general paralysis than any I have been able to find elsewhere.

VI.—Idiocy and Dementia.

The subject of idiocy does not come within the scope of

Fig. 19.



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the present work, and I merely mention it as an element in the classification I have proposed. The treatment of those

so unfortunate as to be originally weak-minded, is a particular branch of medical science which requires special training, and which could not be sufficiently considered in any of its relations in a work intended for the general practitioner. There is not so much to cure in an idiot, as there is to develop. As an illustration of the facial expression and general appearance of an extremely idiotic person, I give the preceding likeness of a young man (Fig. 19) in the New York City institution on Randall's Island. When I first saw this individual several years ago—when the photograph was taken—he was filthy in his habits, and had not as high a degree of intelligence as a well-trained dog. Through persevering efforts on the part of his instructors, he has become neat in his person, has learned to say a few words, and is altogether more advanced intellectually than he was at the time mentioned. His cranial development is, however, so small that any very material progress is not to be expected.[1]

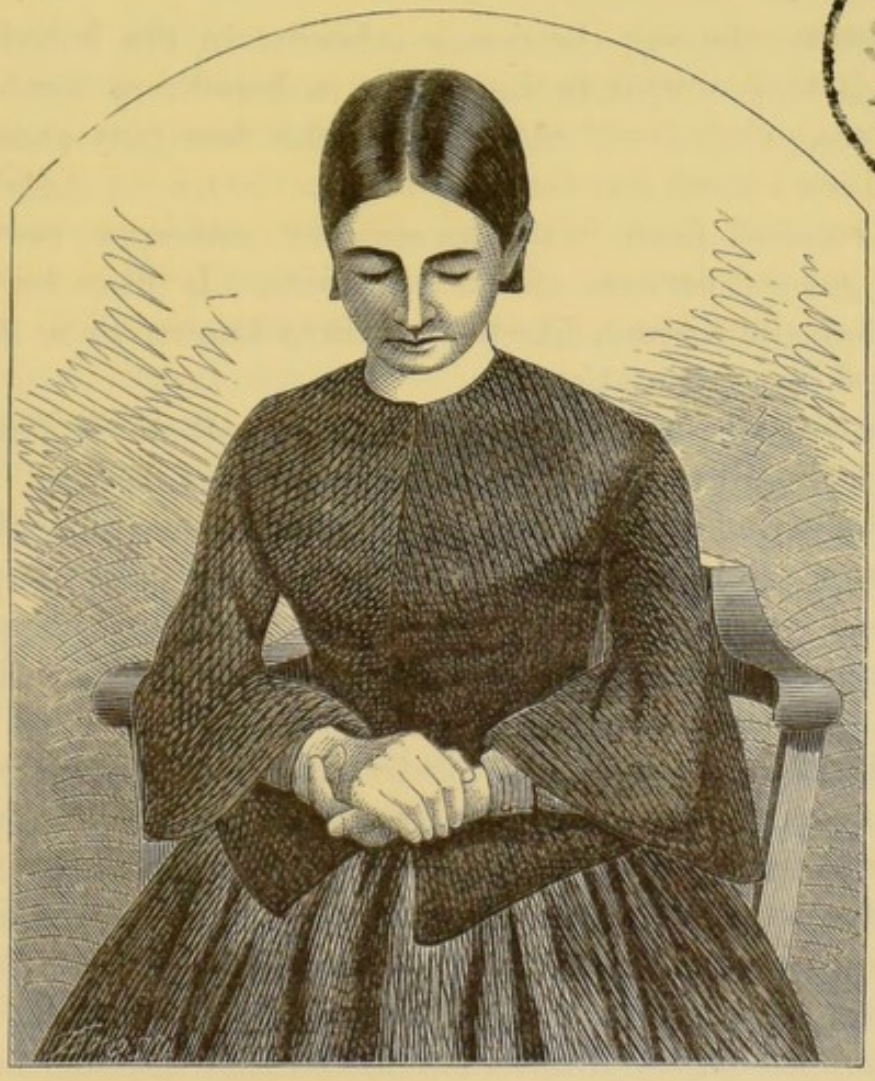
Dementia.—Dementia may be primary, but such is very rarely the case, it being in the vast majority of instances the consequence of an acute attack of insanity, or incident to old age.

The characteristic feature of dementia is mental weakness, and this is shown as regards the emotions, the intellect, and the will. The former are not held under control, slight matters bring them into inordinate action, and tears are shed, and laughter excited, when there is neither adequate cause for one or the other. The intellect is affected in all its parts. The power of application, or of fixing the attention, is materially lessened, and this is doubtless one reason why imperfect ideas are formed of very simple mat-

[1] For full information on the subject of idiocy, the reader is referred to Idiocy and its Treatment by the Physiological Method, by Edward Seguin, M. D., New York, 1866, and a Manual for the Classification, Training, and Education of the Feeble-Minded, Imbecile, and Idiotic, by P. Martin Duncan, M. D., and William Willard, London, 1866.

ters, and why it is so difficult to conceive a series of connected thoughts. The memory, especially for recent events, is weakened to an extreme degree, and the delusions of the patient, if still present, are constantly undergoing change from the impossibility of recollecting them. Volition is almost entirely abolished. The patient is altogether controlled by others, the idea of offering opposition to their wishes never entering his mind.

FIG. 20.



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The facial expression of a patient affected with dementia is not always characteristic, and this mainly for the reason that the physical health is generally good. The deficiency of mental power is, however, readily perceived in the ma-

jority of cases, when the attempt is made to excite the brain to action. The failure of the face to respond to the ideas sought to be conveyed becomes very evident.

An excellent representation of a patient affected with dementia is given in the preceding woodcut (Fig. 20).

Having thus very cursorily considered the symptoms and course of insanity in its several forms, I come in the next place to the discussion of certain points common to all the varieties. In so doing I shall still refer only to general principles.

Causes.—Among the causes inherent in the individual, none is so powerful in its action as hereditary tendency. This may show itself not only by the fact that ancestors have been insane, but that insanity in the descendants may have resulted from hysteria, epilepsy, catalepsy, or some other general nervous affection in them. It often happens, too, that the disease, like many others known to be hereditary, skips a generation.

Insanity is more common in males than in females, though the difference is not so great as many suppose.

The period of life between twenty-five and forty-five is that at which insanity is most liable to make its appearance. Cases are on record of infants having manifested unequivocal symptoms of mental aberration, but the affection is not often met with under the age of puberty.

The civil condition of the individual, as regards marriage or celibacy, exercises an effect over the causation of insanity. Statistics show that celibates of both sexes are more liable than the married. So far as males are concerned, this result is probably due to the fact that in celibacy, as a rule, the mode of life is more irregular. Insanity is assuredly more common among civilized than uncivilized nations, but, as regards the different classes of individuals who go to make up a civilized community, it is very certain that the refined, educated, and wealthy classes, are not so liable to insanity as the lower orders. The exciting causes are both moral and

physical. Of the former, emotional disturbance, grief, terror, disappointed affection, anxiety, great joy, etc., stand first in influence. It is doubtful if moderate intellectual exertion ever, of itself, causes insanity. It is only when the brain is worked night and day, to the deprivation of sleep, and without sufficient change, that insanity results from mental labor. Continual thinking on one subject is the most effectual way of producing insanity by the action of the brain.

Among the physical causes, drunkenness, the use of opium and other narcotics, excessive venereal indulgence, masturbation, blows on the head, exposure to severe heat or cold, the puerperal state, and certain diseases, may be referred to.

Diagnosis.—The principal point to be considered under this head is the discrimination of the real from the feigned disease. The necessity for making the distinction, generally arises from the simulation of insanity by criminals. Such pretenders are, in general, discovered without difficulty, from the facts that they overact their parts, are not possessed of sufficient knowledge to carry out the fraud consistently, and are deficient in the perseverance and force of character requisite to success.

The antecedents of the individual must be carefully inquired into, as well as his actual condition. Watching him when he thinks he is not observed, and the administration of ether by inhalation, will generally suffice to expose the pretender.[1]

Prognosis.—This depends very greatly upon the form of insanity present, and upon the possibility of procuring suitable treatment. Acute mania, with depression, general paralysis, and dementia, are the most unfavorable types with which we have to deal. Either of the uncomplicated forms,

[1] For an exellent monograph on this subject, the reader is referred to Étude Médico-Legale sur le simulation de la Folie, par le Docteur Armand Laurent, Paris, 1866.

or acute mania with exaltation, is much more readily cured. The older the patient when the disease appears, the more unfavorable the prognosis for all ages over puberty, except for very early life. The existence of hereditary tendency, and the habit of using alcohol and other narcotic substances to excess, likewise lessen the prospect of recovery.

Morbid Anatomy and Pathology.—The fact that after death, in cases of insanity, no appearances are to be found within the cranium to which the disease existing during life could fairly be attributed, is no proof of the absence of changes from the normal condition. The difficulty may be with the blood circulating through the brain, and in the inadequacy of our means of examination, and it is well known that we are as yet scarcely on the threshold of inquiry into alterations of cellular structure, only discoverable by careful preparation of the nervous tissue, and through microscopical examination. Still, even with all these unfavorable circumstances, in the great majority of fatal cases of insanity palpable deviations from the normal structure are observed. One fact may be considered as established, and that is, that the morbid alterations are by preference found in the membranes and cortical substance of the brain, and that those changes are the result of congestion and inflammation. Thus there are thickening and opacity of the membranes, adhesions, effusion of serum, and softening of the cortical substance. Histologically the changes are those of defective nutrition and degeneration—leading to the formation of connective tissue in superabundance at the expense of the nervous substance, and the degradation of this latter into fat, amyloid bodies, pigment, etc.

The pathology of insanity is readily deducible from the morbid anatomy, and from an attentive consideration of the symptoms observed during life. Many cases, especially those of temporary insanity, are clearly the result of congestion, and others, such as those of acute mania, owe their first and most prominent symptoms to a like cause. In

other cases, exhaustion of the brain-tissue is the immediate cause. From excessive use, and insufficient repair, there is actually what may be called a chemical atrophy of the cerebral substance, and healthy brain-action is, moreover, in such cases generally rendered much more difficult from the fact that the products of nerve disintegration are not removed.

Insanity is often the result of alterations in the normal qualities of the blood. The influence of many substances in producing mental aberration is well known, and there is no doubt that their power is, to a great extent, due to their entrance into the blood, and their consequent circulation through the cerebral vessels. Among these, alcohol, opium, Indian hemp, belladonna, and bromide of potassium, may be mentioned. I have, in a memoir[1] upon the subject, cited several cases of positive mania caused by the excessive use of bromide of potassium. The accumulation of either urea or bile in the blood is well known to produce mental derangement, and there is no doubt that other morbid agents, such, for instance, as malaria and the poison of typhus, will give rise to a like result. Irritations in different parts of the body may, by reflex action, give rise to insanity. The influence is probably exerted through the sympathetic nerve on the vaso-motor nerves. I have seen two cases of acute mania caused by worms in the alimentary canal, and one by indigestible substances. Temporary insanity is frequently the result of such causes.

The insanity of women generally has a reflex origin. The so-called puerperal mania is a variety which differs in no essential respect from ordinary cases of acute mania. Irritations of the genital organs, especially of the ovaries, may give rise to all the forms of insanity mentioned, with the exception, perhaps, of general paralysis. A few weeks ago I was consulted in the case of a lady who had become

[1] On some of the Effects of the Bromide of Potassium, when administered in Large Doses. JOURNAL OF PSYCHOLOGICAL MEDICINE, January, 1869, vol. iii., p. 46.

insane clearly from the irritation caused by chronic metritis. A more marked case of acute mania, with depression, and a more determined disposition to suicide, I have rarely witnessed.[1] In these cases of blood-poisoning and reflex irritation it is not to be expected that morbid changes will generally be discovered in either the brain or its membranes. Occasionally, however, such alterations are eventually produced.

Treatment.—The fact is daily becoming more evident and more generally admitted that insanity is to be treated as a material disease, and not as a metaphysical nonentity. It is therefore incumbent on the general practitioner to make himself acquainted with the principles involved in the pathology and treatment of the very important class of diseases now under consideration. Besides, although he may not have the facilities for continuing the treatment of insane patients, he is called to them, in the first instance, at a time when they are most susceptible of cure, and it is on his opinion that measures as to personal restraint, or custody of property, are taken. The first thing to do in the case of an insane person is to provide competent attendance; for, as I have already said, there is no knowing to what extremes his delusions or impulses may carry him. Even in the simple, uncomplicated cases of illusions and hallucinations, this must be secured, for in an instant the false perceptions may be accepted as realities, and acts performed in accordance with them.

Then inquiry should be made as to the cause, and efforts directed to its removal. If this can be ascertained to be eccentric, a good prospect of success may be entertained.

Examination should always be made with reference to the state of the cerebral circulation, and here the ophthal-

[1] For a very full account of this important division of the subject, the reader is referred to Die Beziehungen der krankhaften Zustände und Vorgänge in den Sexual-Organen des Weibes zu Geistesstörungen, by Dr. C. E. Louis Mayer, Berlin, 1870.

moscope is capable of giving very definite indications. It will very often be found that there is hyperæmia of the brain, and then the treatment recommended under the heads of cerebral congestion and cerebral hæmorrhage must be carried out. Latterly I have used the bromide of lithium in cases of acute mania, and have more reason to be satisfied with it than with any other medicine calculated to diminish the amount of blood in the cerebral vessels, and to calm any nervous excitement that may be present. The rapidity with which its effects are produced renders it specially applicable in such cases. The doses should be large —as high as sixty grains or even more—and should be repeated every two or three hours till sleep be produced, or at least till half a dozen doses be taken. After the patient has once come under its influence, the remedy should be continued in smaller doses, taken three or four times in the day.

In cases of cerebral congestion attended with illusions and hallucinations, but without mania, the other bromides will answer the purpose—preferably the bromide of sodium. They may also be given in the more violent forms if the bromide of lithium cannot be obtained.

When the mental excitement is in a measure subdued, and the patient sleeps reasonably well, the dilute phosphoric acid of the pharmacopœia is an admirable remedy for restoring strength to the exhausted system. It may be given in doses of half a drachm, properly diluted with water, after each meal.

Schroeder van der Kolk[1] has pointed out the efficacy of aloetic purges in certain cases of emotional insanity of a depressing character due to accumulations in the colon. I have frequently had occasion to be gratified at the success obtained. The aloes should be administered in doses of

[1] Die Pathologie und Therapie der Geisteskranken auf anatomisch physiologischer Grundlage, Braunschweig, 1863, p. 185, *et seq.*

Also translation by Dr. James T. Rudall, London, 1870, p. 134.

about five grains three times a day. I am in the habit of combining it with three grains of inspissated ox-gall, and with excellent results. Under this very simple system of medication I have frequently seen the most intense melancholy disappear in a few days.

In calming maniacal excitement I have occasionally used opium. When it is necessary to obtain an immediate effect, nothing can equal morphia hypodermically administered. But its continuance, I am sure, is almost always productive of bad results by increasing cerebral congestion. Indian hemp, digitalis, and hyoscyamus, are occasionally useful. The application of cold water, in the form of the douche, is a rational method of relieving intra-cranial hyperæmia and moderating nervous excitement. It should be used with caution. The warm bath will also prove beneficial in some cases.

In general paralysis, whatever means of treatment are employed should be used in the earliest stages to obtain the greatest measure of success. I have several times derived benefit from the use of the iodide of potassium in large doses—not that it has cured, but that it has mitigated the violence of the symptoms, and put some check to the advance of the disease. Liedesdorf[1] declares that, though he has only used it himself in syphilitic cases, there is evidence in favor of its utility as opposing some obstacle to the onward course of the affection. He also recommends the cold douche in all stages. In my own practice I have employed phosphorus and strychnia with beneficial results.

In dementia, tonics and stimulants are generally useful. Of the former, iron and quinine may be employed with advantage, and especially phosphorus, phosphoric acid, and strychnia, the latter being combined with either of the others.

A great many more remedies might be mentioned, and in a work specially devoted to insanity I should call attention to them, but I have said enough to indicate the course

[1] Lehrbuch der psychischen Krankheiten, Erlangen, 1865, p. 229.

of the treatment so far as medication goes.[1] There are a few remarks yet to make under this head in regard to means which, in my opinion, should not be used.

Counter-irritants are rarely if ever useful. I have never seen any benefit derived from them.

General bloodletting is never necessary. It will calm a highly-maniacal patient, but so will a sufficiently severe blow on the head.

The hydrate of chloral is a dangerous remedy. I have seen it produce great increase in the maniacal excitement. Its first effect is always to augment cerebral congestion, and, though it eventually lessens the amount of blood in the brain and calms nervous irritability, the dose must be large for these results to be obtained, and the same ends can be accomplished by safer means.[2]

The moral treatment can only be effectually carried out by secluding the patient from society at large, and especially from his immediate family. It is not always necessary to confine him in an asylum, but it is necessary in the great majority of cases to place him in such a situation as will secure for him safety, the companionship of sensible people, and the influence and control of some one skilled in the philosophy of the human mind, in the anatomy and physiology of the brain and nervous system, and in medical science generally. The great difficulty with asylums is, that they contain only insane people, and the prevalent idea among the public—and it is often carried out by the officers of asylums—is, that institutions for the insane are simply places in which dangerous or troublesome maniacs are to be kept in safety. My own idea is, that the best of all places

[1] Of all writers on psychological medicine, no one seems to have more unbounded faith in drugging than Kieser—Elemente der Psychiatrik, Breslau und Bonn, 1855.

[2] For my views on this subject, based on experiments, the reader is referred to my memoir entitled: On the Physiological Effects and Therapeutical Uses of the Hydrate of Chloral, New York Medical Journal, February, 1870, p. 469.

for a lunatic of any kind is the family of a physician, of such a one as I have just mentioned. The association of an insane person day after day, year after year, with others similarly affected, with scarcely the least contact with people of sound minds, is certainly in opposition to the first principles of true psychological medicine.

Asylums are, however, for the present at least, necessary, and it is with great pleasure that I am enabled to state, after having visited a great many institutions for the insane in Great Britain and on the Continent, that American asylums are very far superior in every respect.

It has been said that it is useless to attempt to reason a lunatic out of his delusion. Perhaps there is truth in this when serious structural lesions exist in the brain. The false intellectual conception is then a fixed result of the altered brain-tissue, and is just as direct a consequence of cerebral action as is a thought from a healthy brain. Still, we know that in health it is sometimes possible by argument to counteract the most firmly-rooted ideas, and there is no reason why the same result may not occasionally be produced by arguments addressed to a person with an insane mind. We know, in fact, that this end is at times accomplished, and that, by never for an instant admitting the truth of an insane delusion, and at suitable times—not obtrusively, but when occasion offers—urging such arguments against it as would be convincing to persons of sound minds, the lunatic comes at last to see the falsity of his ideas, and to laugh at them himself. Little by little he loses faith in his perverted reason, and, though he may take up another delusion, the last is held with much less tenacity than the first.

Amusements are generally of service, and a proper system of rewards and punishments is understood by all but the most furious maniacs. Kindness and forbearance, supported by firmness, will not altogether fail in their influence with even the most confirmed and degraded lunatics.

SECTION II.

DISEASES OF THE SPINAL CORD.

CHAPTER I.

SPINAL CONGESTION.

Though congestion of the spinal cord, like that of the brain, is of two kinds, active and passive, yet the symptoms and general course of the two varieties are so generally alike, that nothing would be gained by considering them separately.

Symptoms.—The symptoms of spinal congestion are referable to the cord and to those parts of the body below the seat of the lesion. The most prominent local phenomenon is pain, which is rarely acute, but is described as a dull, aching sensation similar to that experienced in the back after severe and long-continued muscular exertion in a stooping attitude. This pain is increased by the recumbent posture and by standing, if the lower part of the cord be its seat; but pressure, if steadily applied, does not augment it. A sudden blow or a shock, such as that produced by making a false step, aggravates it to a considerable extent.

A sensation of heat is occasionally experienced in the cord, which is not unpleasant, and which is not affected by pressure.

With the local symptoms there are others still more notable perceived in the parts of the body below the seat of the disease. Thus, if, as is very generally the case, the lesion be situated in the dorsal or lumbar region, there are disturbances of sensibility and motility in the lower extremities. The various sensations indicating anæsthesia are present, and are usually first experienced in the skin covering the under surface of the toes. Formication, "pins and needles," tingling, and a feeling as if the toes are swollen, are noticed. It is rarely the case that the anæsthesia is complete. Its extent and exact situation may be accurately determined by the æsthesiometer.

Sometimes there is hyperæsthesia, and occasionally both conditions coexist. The extent of either may be accurately measured with the æsthesiometer. Shooting pains in the limbs and along the course of the nerves coming from the diseased part of the cord are now and then present, but they are not a prominent feature in simple congestion.

A sensation of constriction is at times complained of, and is referred to the body or one or both of the limbs. It is compared to the feeling which would be produced by a tight cord or encasement in an unyielding garment. It is rare in uncomplicated spinal congestion. According to the situation of the lesion, there are pains either in the abdomen, chest, or both, and there may be dyspnœa and palpitation of the heart. In three cases under my care, the difficulty of breathing and irregular cardiac action were prominent features. Similar cases are cited by Ollivier[1] (d'Angers). The temperature of the parts of the body below the lesion is always reduced, from the fact that the vaso-motor nerves are involved.

Erections of the penis are common, especially after the patient has been in the recumbent position for some time.

The most striking phenomena of spinal congestion are

[1] Traité des Maladies de la Moelle Épinière. Troisième édition. Paris, 1837, tome iii., pp. 1–137.

those connected with the alterations of motility. Paraplegia is always present to some extent, though it is rarely complete. Thus the patient, though unable to walk, can generally move the legs when sitting down or lying in bed. Twitchings of the muscles are occasionally present, but not often to a severe degree.

The loss in the power of motion, like the alterations in sensibility, is only present in those parts of the body situated below the diseased parts of the cord. The bladder is very generally affected, either in its own muscular tissue or in its sphincter. In the first case, there is a difficulty of expelling the urine, owing to loss of expulsive power, and this is aggravated by paralysis of the abdominal muscles, or there is incontinence of urine from paralysis of the sphincter. Both conditions may coexist, and then, when a sufficient quantity of urine has accumulated in the bladder, it dribbles away. In such a condition, the bladder is never entirely empty, and the urine is passed alkaline and fetid.

The sphincter of the rectum is sometimes involved, producing involuntary evacuation of the fæces, but obstinate constipation from paralysis of the abdominal muscles, and consequent loss of expulsive power, are much more common. Reflex excitability is, according to my experience, invariably lessened, and is sometimes entirely abolished.

The electro-muscular contractility of the paralyzed muscles is always more or less diminished, though not to the same extent as in some other affections of the cord. As a general rule, the farther the muscle is from the centre the less is its electro-muscular contractility.

The tendency of spinal congestion is to extend itself and eventually to involve the whole cord. In the active form of the disease, this process often takes place with great rapidity, and the symptoms generally are more pronounced and succeed each other with more promptness. The phenomena of spinal congestion are always rendered more decided by the patient's assuming the recumbent posture. He

is hence more paralyzed in the morning before rising from bed than in the evening before he retires. This is due to the fact that the position in question, especially if he lies on his back, allows the spinal blood-vessels to become more readily distended. It is the same thing, as regards the cord, that keeping the head in a dependent position would be as regards the brain.

Bed-sores are not common. Radcliffe [1] seems to assert that they are never met with. Brown-Séquard [2] says an ulceration upon the sacrum or nates is not rare in this affection. Ollivier [3] does not mention them in his account of the disease. Of sixty-two cases of spinal congestion which have been under my care during the last six years, or in which I have been consulted, bed-sores occurred in but two, and in these there was reason to believe they were not the special result of the lesion of the cord.

According as the antero-lateral or posterior columns are mainly affected, the symptoms of spinal congestion differ. Thus, in the former case, the phenomena are chiefly manifested as regards motility, in the latter as regards sensibility. Generally both sets of columns are involved. In spinal anæmia, as we shall presently see, this is not the case.

Causes.—The most common cause of spinal congestion, according to my experience, is exposure to intense cold. This was the alleged cause in eighteen of the sixty-two cases previously referred to as having come under my observation. Fevers appear to be next in frequency, especially those of malarious origin, nine cases being referred to this influence.

Venereal excesses were the apparent cause in five cases, and maintaining the erect posture for a long time was the obvious cause in three cases. This last influence was very well marked in the case of an eminent lawyer of this city, who became suddenly affected with spinal congestion after

[1] Reynolds's System of Medicine, vol. ii., p. 622.

[2] Lectures on the Diagnosis and Treatment of the Principal Forms of Paralysis of the Lower Extremities, Philadelphia, 1861, p. 69. [3] Op. cit.

making a speech of several hours' duration. The suppression of a customary discharge, such as the menstrual flow or a hæmorrhoidal bleeding, is likewise liable to induce congestion of the cord. Three cases under my charge were thus caused. Two were the result of blows, one of severe muscular exertion, and in the remainder no cause could be with any degree of certainty ascertained.

Among the effects of working under compressed air, spinal congestion must be included. Drs. Babington and Cuthbert,[1] of Dublin, have called attention to this fact, and Dr. Clark,[2] of St. Louis, has recently brought forward several additional cases occurring in the workmen in the caisson used in building the bridge over the Mississippi River.

Passive spinal congestion may be caused by any obstruction to the return of blood by the veins, such as cirrhosis of the liver, pregnancy, abdominal tumors of various kinds, diseases of the lungs or right side of the heart, and the long-continued maintenance of the dorsal decubitus.

Diagnosis.—Spinal congestion is liable to be confounded with several other affections, and with some to the great injury of the patient. Thus it may not be distinguished from spinal anæmia, a condition likewise giving rise to paraplegia, but of which the treatment is very different.

It may be diagnosticated from this affection by the facts that in spinal anæmia there is pain in the cord, increased by pressure on the spinous processes of the vertebræ, or, if there is no spontaneous pain, such pressure causes it; by the disturbance induced in the cranial, thoracic, or abdominal viscera, according to the part of the cord affected, being much more prominent; by the circumstance that women are more generally its subjects; that there is often a previous affection generally of the urinary organs which has caused the anæmia; but, above all, by the fact that in spinal anæmia

[1] Paralysis caused by working under Compressed Air, Dublin Quarterly Journal of Medical Science.

[2] St. Louis Medical and Surgical Journal.

the symptoms are less strongly marked after the patient has been lying down some time, whereas the reverse is the case in congestion.

Spinal anæmia never produces any urinary difficulty, although such trouble may cause spinal anæmia. In a case, therefore, in which there was doubt as to the spinal cord being in a state of congestion or anæmia, the order of sequence, as regards the paraplegia and bladder-difficulty, would seem to render the diagnosis exact. In spinal anæmia the bladder is affected before the paraplegia appears; in spinal congestion the paraplegia comes on before the bladder is involved.

In spinal anæmia there is no formication, pricking, tingling, or other sensation indicative of anæsthesia. Hyperæsthesia is, on the contrary, exceedingly common.

The further diagnostic marks will be considered when we come to the subject of spinal anæmia.

Congestion is distinguished from inflammation of the cord by the facts that in it the jerkings of the limbs are slight, that the paralysis is not so extreme, that the urine is never alkaline, that the pain in the cord is less, and by the infrequency of the feeling of constriction at the upper limit of the lesion.

From meningitis it is diagnosticated by the absence of spasms in the muscles of the back, and by the fact that movements of the paralyzed limbs do not cause pain.

Prognosis.—In simple uncomplicated spinal congestion the prognosis is not unfavorable, if, in addition, the case be put under suitable treatment at an early period. It must be remembered, however, that there is a tendency to interstitial changes, and that, if the vessels of the cord be left for a long time in a state of turgidity, it may be impossible to prevent structural alterations of greater severity. Very few of the cases that have come under my own immediate notice were in the first stage of the affection, but yet twenty-three were entirely cured, nineteen were greatly relieved, seven

are still under treatment, six are known to have died, and the rest were lost sight of, most of them not having been under my immediate charge.

Morbid Anatomy.—The post-mortem appearances in cases of congestion of the spinal cord are either in the cord proper or its membranes. As regards the first, section shows increased vascularity both of the gray and the white substance, especially if microscopical examination be made. The capillaries will be found increased in size and more numerous than in the normal condition.

The membranes of the cord contain very large and very tortuous vessels, and in congestion they are rendered still larger and more complex in their anastomoses. The pressure which they are capable of exerting upon the cord is not inconsiderable.

It is almost invariably found that the cerebro-spinal fluid is increased in quantity.

These evidences of congestion are sometimes extremely limited in their extent, at others the whole length of the cord is involved.

Pathology.—The symptoms which result from congestion of the cord are of two distinct classes: increased excitability from hyperæmia, and interruption of the proper functions of the cord from pressure. The former, in the main, results from the increased amount of blood in the gray matter and white substance; the latter from the enlarged meningeal vessels and the increased amount of cerebro-spinal fluid, which, in the form of serous effusion, is the result of their turgidity. As one or the other of these conditions predominates, we have some symptoms more prominent than others. Thus hyperæsthesia indicates rather hyperæmia of the gray substance, anæsthesia pressure upon the white substance. Twitchings, when present, are likewise the result of over-excitation of the gray tissue; while motor paralysis is induced by pressure upon the antero-lateral columns.

The modifications which may be produced in the intensity of the symptoms by the position of the body show the effect of pressure very clearly. In the recumbent posture on the back, the blood gravitates in large amount to the spinal vessels, pressure on the cord is increased, and the phenomena of anæsthesia and paralysis are more strongly marked. Again, causes which increase the activity of the circulation, such as alcoholic stimulants, and others which directly augment the amount of blood in the cord, such as strychnia and phosphorus, invariably increase the hyperæsthesia and induce muscular twitchings, even if they have not previously been observed.

Treatment.—In cases of spinal congestion which come on suddenly, and which are therefore acute in their character, such as result from the sudden arrest of an habitual discharge, blood may be drawn locally from the spinal region by cups or leeches. The best place for the application of the latter is the verge of the anus, and I have several times witnessed very decidedly satisfactory results from their use in this situation.

Purgatives are likewise beneficial, and preference should be given to those which produce watery evacuations, as thereby the overloaded vessels are relieved, and the absorption of the superabundant cerebro-spinal fluid facilitated. Nothing can be better for this purpose than the sulphate of magnesia given in doses of a drachm two or three times a day.

In this form the ergot of rye may be given with advantage from the very inception of the disorder. In the more chronic form it is indispensable. It should be administered in very much larger doses than are laid down in the text-books on materia medica. I am in the habit of using it, in this and analogous spinal diseases, in doses of a drachm of the fluid extract three times a day. The action of the ergot is to lessen the diameter of the blood-vessels of the cord by its constringing power over the organic muscular fibre en-

tering into the composition of their walls. Ten years ago[1] I spoke as follows: "But I have recently ascertained by actual experiment that ergot does exert the influence in question. I prepared a weak aqueous infusion of this substance and placed it on the web of a frog's foot under the microscope. In a few moments contraction of the capillaries ensued, and they became so small as not to allow of the passage of the blood-corpuscles. This experiment I have repeated several times, and am perfectly satisfied that the result is as I have stated. More, I have frequently injected small quantities of the infusion into the stomach of frogs, and contraction of the capillaries of the web always followed."

This was certainly the first demonstration of this very important action of ergot.

Since that time I have given it in a large number of cases of diseases of the spinal cord, congestion among them, in which it was necessary to diminish the amount of blood in the spinal vessels, and I am entirely satisfied that such is its effect; but I never obtained its full influence till, in accordance with the suggestion of my friend Dr. A. Jacobi, of this city, I adopted the practice of giving it in what may be called very large doses. Among the cases which first came under my care, since my residence in New York, was that of Mr. W., of Tennessee, who had become affected with congestion of the cord, from exposure to cold and dampness. When I first saw him he was unable to walk without the assistance of crutches, and a man on each side of him holding his shoulder. He had paralysis of the bladder, which had come on after the paraplegia, and a constant, dull, aching pain in the loins. There were also occasional startings of the legs, especially after he had gone to bed. All his symptoms were worse in the morning. I at first gave him ten drops of the fluid extract of ergot three times a day,

[1] A Clinical Lecture on Chronic Myelitis, delivered in the Baltimore Infirmary, March 16, 1861. American Medical Times, June 15, 1861, p. 379.

but, continuing this for two weeks without effect, I at once increased the doses to a teaspoonful. In less than a week the effects were manifest. Sensibility began to return in the extremities, the strength increased, the bladder began to contract on its contents, the lumbar pains ceased, and by the end of a month he had entirely recovered. A few weeks afterward he had a relapse, but the ergot, taken as before for ten days, again restored him, and he has since remained perfectly well.

I cite this case because it is one in which nothing else but the ergot was given, and as one in which its influence was twice distinctly manifested.

Belladonna is also a valuable remedy in spinal congestion, especially when there is paralysis of the sphincter, or when the pain in the back is severe. The tincture, in doses of fifteen drops three times a day, may be employed, and a belladonna plaster may be applied to the painful region of the spine.

The hot douche—the water being of the temperature of 98° Fahr.—to the spinal column is an excellent means of determining the blood from the deep to the superficial vessels. The water should be allowed to fall from the height of about two feet upon the naked back over the diseased part of the cord every day for about five minutes. Dry cups are also valuable adjuncts.

Electricity is always useful. The constant current should be applied to the spine over the affected part of the cord, the positive pole being held at the upper limit of the lesion, and the negative rubbed up and down all the part below. The duration of the application should not exceed ten minutes. By this means the calibre of the spinal vessels is probably lessened. At any rate the downward current certainly is beneficial, while the upward increases the intensity of the symptoms.

The induced current should be used to the paralyzed muscles, so as to excite them to contract. In this way their

nutrition is promoted, and any tendency to atrophy from disuse obviated.

The primary current should not be employed more frequently than every alternate day. The induced may be used every day for half an hour or longer, short of causing fatigue.

I will only add that strychnia and phosphorus should never be administered in congestion of the cord, as their action is the very reverse of that desired, and irreparable damage may be done by their use.

CHAPTER II.

SPINAL ANÆMIA.—ANÆMIA OF THE POSTERIOR COLUMNS.—ANÆMIA OF THE ANTERO-LATERAL COLUMNS.

A DEFICIENT quantity of blood in the spinal cord, or a depravation in the quality of the blood circulating through its tissue, gives rise to two cognate, but, so far as their phenomena go, different affections. In one of these, which has hitherto been known as spinal irritation, the morbid action is in a great measure confined to the posterior columns of the cord; in the other, which embraces several differently-named disorders, characterized by paralysis, such as reflex paralysis, inhibitory paralysis, spinal paresis, paralysis from peripheral irritation, etc., the antero-lateral columns are mainly affected.

In thus specifically locating the lesions in these affections, I am aware of the fact that post-mortem examinations are wanting to support them. Nevertheless, the symptoms characteristic of each are so distinctly marked, and are in such intimate physiological relation with the regions of the cord specified, that I do not think I am at all exceeding the limits of probability.

Retaining the name of spinal irritation, as one well known to the profession, it will nevertheless be understood that, in my opinion, the proper designation of the disease would be anæmia of the posterior columns of the spinal cord. I have arrived at this view after a very careful consideration and analysis of the symptoms observed in a large number of cases.

The same remarks are applicable, *mutatis mutandis*, to reflex paraplegia, a symptom which I am very sure results from anæmia of the antero-lateral columns of the cord.

ANÆMIA OF THE POSTERIOR COLUMNS OF THE SPINAL CORD.—SPINAL IRRITATION.

History.—It has been questioned by several distinguished authors whether such an affection as spinal irritation really exists as a distinct disease. Thus Valleix[1] ascribes the most important of its manifestations to hysteria, and regards the spinal tenderness present as being due to simple intercostal neuralgia; Inman[2] considers the pain produced by pressure over the spinous processes of the vertebræ as existing in the muscular attachments, and as indicative of what he calls myalgia. Mr. Skey[3] evidently looks upon all cases of spinal irritation as hysterical in their character, and Niemeyer[4] speaks incredulously on the subject, without giving any very decided opinion. It would be easy to bring forward other authorities who have expressed similar views, and I may have to allude to some of them more fully hereafter. In the recently-published nomenclature of the Royal College of Physicians,[5] the affection has no place unless it be included under the head of hysteria.

The first author who distinctly grouped together the symptoms of spinal irritation was J. Frank,[6] who, under the name of rachialgia, described the disorder with considerable accuracy, and laid the principal stress upon the local pain.

[1] Traité des névralgies, ou affections douloureuses des nerfs. Paris, 1841, p. 345.

[2] On Myalgia: its Nature, Causes, and Treatment, etc. Second edition, London, 1860, p. 225, *et seq.*

[3] Hysteria, etc., New York, 1867, p. 72, *et seq.*

[4] A Text-Book of Practical Medicine. American edition, New York, 1869, vol. ii., p. 258.

[5] The Nomenclature of Diseases drawn up by a Joint Committee appointed by the Royal College of Physicians of London. London, 1869.

[6] De Rachialgitide, in Prax. med. univ., P. II., t. i., p. 37.

He was followed by Stiebel,[1] who, however, contributed little to our knowledge of the subject.

Mr. J. R. Player[2] was among the first English physicians, if not the very first, to call attention to the fact that eccentric derangement of function may be the result of irritation of the spinal cord. Thus he says: "Most medical practitioners who have attended to the subject of spinal disease must have observed that its symptoms frequently resemble various and dissimilar maladies, and that commonly the function of every organ is impaired whose nerves originate near the seat of disorder. The occurrence of pain in *distant parts* forcibly attracted my attention, and induced frequent examination of the spinal column; and, after some years' attention, I considered myself enabled to state that, in a great number of diseases, morbid symptoms may be discovered about the origins of the nerves which proceed to the affected parts, or of those spinal branches which unite them; and that, if the spine be examined, more or less pain will commonly be felt by the patient on the application of pressure about or between those vertebræ from which such nerves emerge."

The term "spinal irritation" appears to have been first used by Dr. C. Brown,[3] of Glasgow, who, in a very excellent paper, gives a picture of the disorder which cannot fail to be recognized as truthful and exact by those who have witnessed several cases of the affection. He insists upon not confounding the complaint with those organic diseases of the vertebræ and spinal cord which some of its symptoms cause it to resemble, points out the variation of the phenomena according to the seat of the spinal tenderness, and inculcates the employment of rest and counter-irritation as the most effectual remedies. His ideas of the pathology of

[1] Über Neuralgica Rachitica, Rust's Magazine, t. i., c. xvi., p. 549.

[2] Quarterly Journal of Science, vol. xii., p. 428. Quoted by Teale.

[3] On Irritation of the Spinal Nerves. Glasgow Medical Journal, No. II., May, 1828.

the disease are: "That the immediate cause of the pain of the back and breast is spasm of one or other of the muscles arranged along the spine altering the position of the vertebræ, or otherwise compressing them as they issue from the spinal marrow.

"That this spasm in many instances is strictly a *local* disease, produced by fatigue, wrong posture, or other causes, and quite unconnected with the state of the brain, spinal marrow, or nervous system in general.

"But that, in other formidable instances, this partial, spasmodic, or wrong action of the muscles, is owing to a faulty state, perhaps an enlargement, of the vessels of the brain or spinal marrow. This state of the brain, as in many other diseases, gives rise to spasm or even to convulsion of certain muscles; which partial symptom, from its severity, attracts the chief attention. This local affection is confined to those portions of the spine where there is the greatest motion, and where, of course, the muscles having the greatest activity are most liable to deranged action or spasm. I imagine that this view of the subject is illustrated and perhaps confirmed by various symptoms which were observed in the different cases, and which without it were very incomprehensible. The partial palsy, the affection of the sight, the giddiness of the head (for I find that this was a prominent symptom in several cases, especially in that of A. S.), all give some confirmation to the notion that the brain is affected in these severe cases."

Dr. Darwall,[1] of Birmingham, describes several features of the affection with accuracy, such as those simulating cardiac and gastric diseases. He is inclined to believe that the morbid condition of the spinal cord depends mainly upon irregularity of the circulation, generally congestion.

But no essay upon the subject of spinal irritation, which had yet appeared, was equal in thoroughness to that of Mr.

[1] On some Forms of Cerebral and Spinal Irritation. Midland Medical Reporter, May, 1829.

Teale,[1] and it is to him that the views now generally held relative to the connection between various eccentric phenomena, such as pain, spasm, and visceral disturbance, and a peculiar condition of the spinal cord, are to be attributed. He, however, committed the great error of regarding the affection as being due to inflammation, and, in what for those days was logical accordance with this theory, he combated it with strong antiphlogistic measures. His book may be studied with advantage, as presenting an admirable account of the many diverse phases which spinal irritation may assume.

Mr. Tate,[2] in his work on hysteria, attributes many of the protean manifestations of this disorder to spinal irritation, limited, however, to the dorsal region. He fails to recognize it as an independent disease. His treatment consists in the application of tartar-emetic ointment along the whole length of the dorsal vertebræ, and strong purgation. He discountenances the use of leeches and blisters.

Mr. W. R. Whatton[3] insists chiefly upon the liability to mistake spinal irritation for disease of the vertebræ. He gives a very excellent account of the symptoms. The treatment he recommends consists in the abstraction of blood, by leeches or cups, from the parts where the tenderness is felt, repeated every three or four days, and the application of small blisters on each side of the painful spots. Any debility ensuing in consequence of this treatment is to be remedied by the preparations of iron and quinine.

In a clinical lecture delivered in Dublin, Dr. Corrigan[4] relates the particulars of several cases of spinal irritation, successfully treated by local antiphlogistic measures, and

[1] A Treatise on Neuralgic Diseases dependent upon Irritation of the Spinal Marrow and Ganglia of the Sympathetic Nerve. London, 1829.

[2] Treatise on Hysteria. London, 1830.

[3] On Spinal and Spino-Ganglial Irritation. North of England Medical and Surgical Journal, No. III., 1831.

[4] Medico-Chirurgical Review, July, 1831, p. 182.

the internal use of iron. He does not, however, add any thing of importance to our previous knowledge of the subject.

Dr. Isaac Parish,[1] of Philadelphia, appears to have been the first American author who called attention to the affection in question. He relates the details of several cases, recommends the use of counter-irritants, especially tartar-emetic ointment, and concludes:

"First, that tenderness on pressure in some portion of the spinal cord is an attendant on many chronic neuralgic affections, and that, by relieving it in the manner proposed, these complaints are either entirely eradicated or temporarily suspended.

"And, secondly, that the precise indications which this circumstance affords are not sufficiently understood at the present time to justify the establishment of any definite pathological principles applicable to the whole class of neuroses."

Dr. W. Griffin and his brother, Mr. D. Griffin,[2] of Limerick, were the next to write upon the subject. The joint work of these gentlemen is based upon one hundred and forty-eight cases, all of which are thoroughly analyzed, and from which very definite deductions of pathology and treatment are drawn. The essay is not excelled in importance by any previous contribution, and constitutes a really valuable study. The conclusions which they draw are so instructive that I do not hesitate (though by no means indorsing them all) to transfer them without abbreviation:

"1. That tenderness at one or more points of the spine is an attendant on almost all hysterical complaints, on numerous cases of functional disorder when the hysteric dispo-

[1] Remarks on Spinal Irritation as connected with Nervous Diseases: with Cases. American Journal of the Medical Sciences, vol. x., 1832, p. 223.

[2] Observations on the Functional Affections of the Spinal Cord and Ganglionic Nerves, in which their Identity with Sympathetic, Nervous, and Simulated Diseases is illustrated. London, 1834.

sition is not so obvious, and in many nervous or neuralgic affections.

"2. That many of the symptoms of these affections evidently depend upon a peculiar state of certain nerves, probably at their origin, may be reproduced at any moment by pressure, and are often relieved by remedies applied there.

"3. That, in all cases of tenderness of the cervical and upper dorsal spine, there was nausea, or vomiting, or pain of stomach, or affections of the upper extremities; but no pain of the abdomen, dysury, ischury, hysteralgia, or affections of the lower extremities.

"4. That, in all cases of dorsal tenderness, pains affecting the abdomen, bladder, uterus, testes, or lower extremities, were usual symptoms; while nausea, vomiting, or affections of the upper extremities, were never complained of.

"5. That nausea and vomiting appeared to have more relation to tenderness of the cervical spine, pain of stomach to tenderness of dorsal; but that, when there was soreness of both, nausea or vomiting was still more frequent, and pain of the stomach scarcely ever absent.

"6. That, when several points or a great extent of the spinal column is painful and tender on pressure, local remedies are generally less effectual, and there is a strong disposition to transference of the disordered action from one organ to another; the pain or tenderness, in all such cases of transference, shifting its place to a corresponding part of the spinal column, leaving the original point free, or with a very diminished degree of tenderness.

"7. That spinal tenderness is seldom or never met with in cases of pure inflammation, except when these accidentally occur in persons previously suffering from irritation of the cord; and that, when appearances of inflammation present themselves in any organ accompanied by a corresponding spinal tenderness, they cannot commonly be removed by the remedies applicable to inflammatory cases, and are often rendered worse by them.

"8. That there does not appear to be a complaint to which the human frame is liable, whether inflammatory or otherwise, which may not be occasionally irritated in disturbed states of the cord; and hence that this disturbed state is one vast source of those complaints called hysterical or nervous.

"9. That those functional disorders connected with spinal tenderness are very often attended by some disturbance of the functions of the uterus, but that they are by no means always so, since they occur in those who are regular in this respect: in girls long before the menstrual period of life, in women after it has passed, and, lastly, in men of nervous susceptible habits, and in boys.

"10. That in fact they are not necessarily dependent upon any one organ; since they are found indifferently coexisting with disturbance of the digestive organs solely, or the uterus solely, or of the circulatory or respiratory system.

"11. That from the cases detailed we have reason to suppose spinal tenderness may arise from uterine disorder, from dyspepsia, from worms in the alimentary passages, from affections of the liver, from mental emotions, from the poison of typhus, from marsh miasmata, from erysipelatous, rheumatic, and eruptive fevers, and from the irritation arising from local injury.

"12. That it is almost invariably found, in connection with gastric or abdominal tenderness, in fever; and this tenderness is, probably, like the soreness of scalp, pains in the limbs, etc., dependent on the morbid state of the cord.

"13. That, whether in fever or in other complaints, it is met with in the situation of the eighth or ninth dorsal vertebra much more frequently than at any other part of the spine.

"14. That affections attended by spinal tenderness are seldom fatal; that, even in those cases of intense irritation of the cord under which patients suffer extremity of pain for years, the event is generally favorable.

"15. That they frequently, as well as hysteria, occur with all the appearances of a primary affection of the nervous system.

"16. That affections are occasionally met with presenting all the marks of the hysteric character, and perfectly resembling cases described as those of spinal irritation, but unattended by spinal tenderness or any other direct indication of a morbid state of the cord."

The treatment recommended consists in the removal of the cause if this still continues in action, purgatives, the application of blisters and leeches to the skin, the internal administration of hyoscyamus and belladonna, to lessen the nervous irritability, alum in cases of gastric derangement, and change of air and scene.

In a subsequent work, the Messrs. Griffin[1] again discuss the subject, but bring forward no additional facts.

Dr. John Marshall[2] is confident that many visceral affections, such as heart-diseases, asthma, phthisis, dyspepsia, diabetes, chorea, and even phlegmasia dolens, are frequently really produced or simulated by spinal irritation. Some of his cases of supposed functional disorder of the spinal cord are, however, obviously organic, consisting of congestion, inflammation, or softening of the organ.

In his classical work, Ollivier[3] devotes considerable space to what he calls "*an Affection described under the name of Spinal Irritation.*" He considers the pathological condition to be one of congestion of the meninges of the cord, and bases this opinion in great part on the success which, according to him, ensues on the use of leeches, blisters, and

[1] Medical and Physiological Problems: being chiefly Researches for correct Principles of Treatment in Disputed Points of Medical Practice. London, 1845.

[2] Practical Observations on Diseases of the Heart, Lungs, Stomach, Liver, etc., occasioned by Spinal Irritation, and on the Nervous System in General as a Source of Organic Disease. London, 1835.

[3] Traité des Maladies de la Moelle Épinière. Troisième édition. Paris, 1837, t. seconde, p. 209.

counter-irritant ointments. In addition, he favors the administration of opium, digitalis, hyoscyamus, belladonna, and subcarbonate of iron.

Türck[1] regards the phenomena of spinal irritation as being due, first, to disorder of other organs, whereby a morbid impression is propagated along the incident excitor nerves to the spinal cord, or, second, to derangement of the capillary circulation of the cord. That is, the disease may be either of eccentric or centric origin. He does not advance our knowledge beyond the point reached by previous authors.

Coming again to our own country, we find that in 1844 a very valuable paper was published by Prof. Austin Flint,[2] based upon fifty-eight cases of functional disorder connected with an abnormal condition of the spinal cord. In this memoir, without going into any discussion relative to the pathology of the affection, Dr. Flint considers the disorder as giving rise to tenderness over the vertebral column, causing alterations of sensibility, as affecting the muscular system, as producing abnormal mental manifestations, as affecting the digestive organs, the genito-urinary organs, the heart and circulation, and as causing paroxysms of sinking. He then considers the physical habits of the patients, the results of medical treatment, the probable remote causes, and then, at some length, the remedial measures which he has found most successful. Under this head, Dr. Flint advises the use of counter-irritants to the spine, especially cupping, and generally without scarification. Issues he found inapplicable, death ensuing in the one case in which he used them. There is no doubt, however, that in this instance he had an organic disease to deal with, and that the issues had nothing to do with the fatal result. Tonics, especially iron, he found to be of great advantage.

[1] Abhandlung über spinal Irritation. u. s. w. Wien., 1843.

[2] Observations on the Pathological Relations of the Medulla Spinalis. American Journal of the Medical Sciences, April, 1844, p. 269.

In a very full analysis of the medical reports of the Stockholm Hospital, by Dr. Magnus Huss,[1] the subject of spinal irritation receives due consideration. Dr. Huss classes the symptoms of the disorder as follows: 1. Pain of various parts of the vertebral column, existing either idiopathically or developed by pressure. 2. Cramps, either of a clonic or tonic nature, in those parts subjected to the influence of the spinal cord. 3. Loss of power in the same portions of the body, ranging from simple stiffness and weakness to complete paralysis. 4. Altered sensibility, either by excess or by great diminution of sensation.

It will be observed that in this enumeration the author confines his specification of morbid phenomena to those which relate to sensation and the power of motion.

The treatment is fully and philosophically considered. Of external remedies he prefers counter-irritants, using the milder forms first, and then the severer, such as the moxa and the actual cautery, should the first fail. Venesection, either general or local, should be cautiously employed, and is not generally indicated. He is the first, so far as my researches extend, to mention electricity, a means which he thinks may be employed with advantage in chronic and debilitated cases. Potash-baths are also recommended.

Of internal remedies he specifies iron, opium, strychnia, phosphorus, and valerian, as being preëminently useful.

Axenfeld[2] devotes a considerable portion of his treatise to spinal irritation. He regards it as being produced either by a trouble of innervation or congestion. In the treatment, leeches occupy the first place, and in light cases blisters, sinapisms, dry cups, and stimulating frictions, are useful. Internally he recommends nothing but quinine and iron.

Dr. Radcliffe[3] writes very sensibly on the subject of spinal irritation, and gives a typical case which is quite in-

[1] British and Foreign Medical Review, October, 1846, p. 463.

[2] Des Névroses, Paris, 1863, p. 284.

[3] Reynolds's System of Medicine, London, 1868, vol. ii., p. 640.

structive. He incidentally gives it as his opinion, that the pathological condition is one of anæmia, and he consequently discourages the use of leeches, relying mainly on blisters and tonics.

I have thus cited the principal authorities upon spinal irritation, without, however, by any means, exhausting the bibliography of the subject. Notwithstanding the eminence of many of those who have contended for the existence of a definite affection of the spinal cord, characterized by tenderness on pressure over one or more of the vertebræ, and certain eccentric disorders involving sensibility, the power of motion, and functional derangement of many of the viscera, it must be confessed that the great mass of the medical profession has regarded the whole theory with suspicion, if not with absolute distrust. The principal reason for this is undoubtedly to be found in the fact that, like many other new theories, that of spinal irritation has been applied to explain conditions which it could not logically be made to cover. Thus many cases of disease or disorder of the heart, due to organic difficulties of that organ, or excited by disease of other viscera through the sympathetic system, have been attributed to spinal irritation. The same is true also of the uterus, stomach, liver, and other organs, and even of the spinal cord itself, which often, when the seat of organic diseases, such as congestion, meningitis, inflammation, tumors, etc., has been regarded as simply in a state of irritation. It is very certain, also, that numberless cases of hysteria have been attributed to irritation of the spinal cord. In the following remarks I will endeavor to be as explicit as possible, and not to claim too much for a pathological condition which I am very sure exists, and which I therefore think is entitled to recognition. If I contribute any additional information, it will be mainly due to the fact that our means of examination are much more perfect and extensive, and our knowledge of physiology, pathology, and therapeutics,

more thorough than when most of the authors I have quoted wrote upon the subject. My observations are based upon a careful study of one hundred and twenty-seven cases which have occurred in my private practice during the last six years, and of which I have full notes, and twenty-nine cases of which I have less complete data—in all, one hundred and fifty-six cases.

Symptoms.—Centric Symptoms.—1. *Tenderness at some one or more Points over the Spinal Column, increased by Pressure.*—This is the essential symptom of spinal irritation, though varying in intensity from the slight degree of pain experienced upon strong pressure to the acute hyperæsthesia, which does not allow of even the contact of the clothing without the production of great suffering. It is generally complained of by the patient, though occasionally it has to be sought for by the physician. The brothers Griffin found this symptom present in all but five out of one hundred and forty-eight cases, and it is very probable that these five were not cases of spinal irritation, a supposition which the authors themselves evidently entertain. Certainly the details of the cases do not support the view which would ascribe their phenomena to any affection of the spinal cord. Most of the other authors I have cited refer to this tenderness as a prominent feature. Parish thinks it alone is to be relied upon as indicating irritation; Mr. Whatton declares that it is never wanting; Axenfeld regards it as the dominant and characteristic symptom; and Radcliffe, while admitting that it is not equally well marked in every case, states the rule to be that spinal tenderness and spinal irritation go together.

On the other hand, Flint does not regard tenderness as an invariable and essential element of the affection under consideration. He found it absent or indistinct in five of his fifty-eight cases, while the other attendant circumstances furnished unequivocal evidence that the diagnosis was correct.

My own opinion would lead me to consider no case as one of spinal irritation in which tenderness on pressure over the vertebræ was absent. In the one hundred and twelve cases noted by me, this symptom was present in all. There are diseases of the spinal cord, which produce derangements of other organs of the body, and which are not characterized by vertebral tenderness, but these are far more serious affections than spinal irritation, and of altogether different pathology.

The seat of the tenderness is generally in the dorsal region of the spine. The Griffins found cervical tenderness in twenty-three cases, cervical and dorsal tenderness in forty-six, dorsal alone in twenty-three, dorsal and lumbar in fifteen, lumbar in thirteen, the whole spine tender in twenty-three, and no tenderness in five. Of one hundred and forty-eight cases, therefore, one hundred and seven exhibited tenderness in the dorsal region.

Dr. Flint found cervical and dorsal tenderness in three cases, lumbar and dorsal in ten, and dorsal alone in twenty-one cases.

Of my own cases, twenty-five had cervical tenderness only, thirty-seven cervical and dorsal, forty-five dorsal only, nineteen dorsal and lumbar, fifteen lumbar only, and in fifteen the whole spine was tender. One hundred and sixteen cases, therefore, of one hundred and fifty-six were characterized by dorsal tenderness, and in forty-five it was limited to this region.

The degree and character of the tenderness are subject to great variation. In some cases strong pressure is required to develop it, while in others the least touch is insupportable. Sometimes there are shooting pains, which radiate from the tender spot, while at others the hyperæsthesia is quite circumscribed. In a gentleman now under my care with well-marked spinal irritation, and who has a tender spot over the third lumbar vertebra, pressure not only causes intense suffering at that point, but develops pain along the

whole course of the crural nerves and their branches as far as their terminations on the inner sides of the feet. Another, a lady, who has spinal tenderness over the eighth cervical and first dorsal vertebræ, experiences, from pressure, intense pain along the course of the first intercostal, the internal anterior thoracic, and all the nerves of the left upper extremity. Why in these and other cases particular nerves should be affected, is a question which will be more fully considered hereafter.

The pain developed by pressure is not always of the same character. Sometimes it is dull and aching, and at others sharp and lancinating. I have not noticed that any very definite relation exists between the character of the pain and the severity of the other symptoms, though, as regards the degree of pain of each kind, there is a marked connection. By this I mean that a dull, aching sensation may indicate as profound a pathological condition, and be accompanied by as intense eccentric phenomena, as a sharp and lancinating pain, though a severe aching pain and a severe lancinating pain always indicate more serious disorder than when these sensations are not so emphatic.

The character of the pain varies in accordance with the tissue in which it is felt. The dull aching sensation is only developed by strong pressure, and is seated in the muscular, tendinous, or cartilaginous structures about the vertebræ. The sharp, piercing twinges excited by slight pressure arise from the skin, and subcutaneous cellular tissue. With these species of sensations, the æsthesiometer always shows increased sensibility of the skin over and in the vicinity of the painful centres.

To ascertain whether or not the tissues outside of the spinal canal are in a state of hyperæsthesia, the pressure should be applied with gradually-increasing force, by means of the thumbs applied to the spinous processes and the intervertebral spaces, as recommended by Flint. The examination should be thorough, and extend throughout the

whole extent of the vertebral column. The fact that the patient denies the existence of tenderness should have no weight with the physician. Only a few days ago a young lady consulted me for severe infra-mammary pain, headache, and nausea. I at once suspected spinal irritation, but she declared, in answer to my inquiries, that there was no sign of tenderness anywhere over the spinal column. I insisted, however, on a manual examination, and to her great surprise found three spots that were exceedingly painful to slight pressure. This young lady had been treated for dyspepsia for several years, without deriving any benefit from the measures used, but was cured by the treatment which I shall presently fully consider. Occasionally it happens that the tenderness is not perceived for some time after the pressure is made. In a recent case I found the interval to be over a minute, and then acute pain, following ing the course of the nerves, was experienced. I am not prepared to offer an explanation of this phenomenon.

2. *Pain in the Spinal Cord.*—The tenderness just noticed is seated primarily externally to the vertebral canal, and is developed by pressure. That which is now to be considered is located in the spinal cord, and is, therefore, capable of being produced by pressure upon non-tender spots. It is a very common symptom, having been present in one hundred and one of my cases. Generally it is confounded with spinal tenderness, from which, however, it is quite distinct. It is aggravated by motion of the spinal column, by action of the muscles which have their attachments to the spinous and transverse processes, by percussion, and sometimes by the erect posture. In the case of a gentleman of this city, it was so great when he stood up that he was forced to keep the recumbent position nearly the whole time. When I first saw him he was wearing an apparatus designed to keep the weight of the head from the vertebral column, and to prevent the vertebræ pressing upon each other, under the idea that he had disease of the inter-

vertebral substance. I removed the instrument, and, treating him for spinal irritation, he recovered his health in a few weeks.

Pain in the spinal cord, in the disorder under consideration, is usually seated near the point of external tenderness, though it is often at a distance, and sometimes is felt throughout the whole extent of the cord. The eccentric phenomena bear a distinct anatomical and physiological relation to it, as do those which are connected with spinal tenderness. There is likewise a similar connection existing between the pain in the cord and the vertebral tenderness.

To ascertain the existence of spinal pain, when it is not spontaneously felt or superinduced by muscular exertion, percussion should be practised. The ends of the fingers will answer for this purpose, though I prefer a little vulcanized india-rubber hammer, and a plessimeter, such as are sometimes used for percussing the chest. Even over spots which exhibit much tenderness, the deep-seated pain in the cord itself can clearly be distinguished.

Eccentric Symptoms.—By far the most important and noticeable symptoms of spinal irritation are to be found in distant parts of the body. These vary in their character and seat, according to the part of the spinal cord affected. Following the example of the Griffins, I shall consider these symptoms as they depend upon irritation of the several regions of the cord with which they are connected.

a. The Cervical Region.—Of the cases upon which this paper is based, in twenty-five the irritation existed in the cervical region only, of the spinal cord; in thirty-seven, the cervical tenderness was conjoined with dorsal tenderness, and in fifteen with tenderness of the whole spine. Taking the uncomplicated cases as presenting the clearest features, the following would appear to be the more prominent symptoms of cervical spinal irritation.

Vertigo was an accompaniment in eleven cases, and *headache* in fifteen; *noises in the ears* in eight, and *disturb-*

SCHOOL OF MEDICINE

ances of vision in four. *Fulness* and a *sense of constriction* across the forehead were complained of in several cases, as was also tenderness of the scalp. In addition, the *mind* was more or less affected in every case, and in seven the aberration was of such a character as almost to amount to insanity. In one of these, a married lady, aged thirty, there were several paroxysms of maniacal excitement every day; and in another, that of a young lady aged twenty-three, so furious were the exacerbations that, for fear she would injure herself or others, she had to be restrained by two strong nurses, who held her while the fits lasted. The predominant type, however, was melancholia.

Sleep was deranged in every case, generally in the form of insomnia, though in three cases the tendency to somnolence was excessive. In every case the dreams were of an unpleasant character; in two there was nightmare, and in one somnambulism.

Neuralgic pains were present in seventeen of the twenty-five cases. If the upper part of the cervical region was the seat of the irritation, these pains were experienced in the scalp and face; if the lower, they were seated in the neck, the shoulders, upper part of the chest, and the upper extremities. Sometimes the pain was of a dull, burning character, and was then generally seated in the muscles of the nucha. Muscular effort always increased the suffering. In accordance with Teale's experience, it several times occurred that the neuralgia was intermittent, the paroxysms coming on about sundown and lasting through the night. In none of these cases was there anæsthesia.

Motility was interfered with in eighteen cases. Sometimes there were *fibrillary twitchings;* in five cases there were *clonic spasms* of the muscles of the face and neck; in three, *general chorea;* in two, *contractions* of the flexors of the arm on one side, so that the elbow was rigidly bent; in two, the contractions were in the flexors of the hands, and in four, of the fingers. In one case there was *complete loss*

of power over the hand; in four, *aphonia;* and in one, almost constant *hiccough* while the patient was awake.

Nausea was present more or less in fifteen cases, and, in one, part of every thing taken into the stomach was almost immediately rejected. *Pain* in the stomach was not met with in any case.

b. The Dorsal Region.—I found the dorsal region of the spine tender in one hundred and sixteen cases. In thirty-seven of these it was conjoined with cervical, in nineteen with lumbar tenderness, and in fifteen it was affected with the whole spine, leaving forty-five uncomplicated cases.

The most prominent symptoms in these cases were connected with the viscera, the stomach being the organ commonly involved. Thus, *gastralgia* was present in every case, *nausea* and *vomiting* in nine cases, *pyrosis* in three, *gastric flatulence* in forty, and *acidity*, as evidenced by heartburn, in twenty-six.

Next in order came the heart. There were *palpitations* in twenty-six cases, *fits of oppression*, during which the heart beat with irregularity as regarded force and rhythm, in ten cases, and *attacks of syncope* in five.

There was *difficulty of breathing* in fifteen cases, and *cough* in fifteen.

Intercostal neuralgia existed in ten, and *infra-mammary pain* in thirty-one cases.

There were no muscular spasms, contractions, or paralysis.

In the thirty-seven cases in which the dorsal tenderness was conjoined with cervical tenderness, the symptoms characteristic of each region were more or less intermingled. In two cases there was *epilepsy*, and in three *chorea paralytica.*

c. The Lumbar Region.—This portion of the spine exhibited tenderness in forty-nine cases. In nineteen of these it was accompanied by dorsal tenderness, in fifteen the whole spine was affected, and in fifteen the tenderness was

confined to the lumbar region alone. Of these latter all were characterized by *neuralgic pains* in the lower extremities, and in three of them there were similar pains in the muscles of the back and abdomen. In six there was *spasm of the neck of the bladder*, accompanied with severe pain, and causing great difficulty of urinating, in one there was *incontinence of urine*, in five *pain in the uterus and ovaries*, and in one *neuralgia of the rectum.*

Motility was affected in eight cases. In four of these there were strong *tonic contractions* of the muscles of the lower extremities, and in four *paralysis.* In all of these there were occasional *clonic spasms* simulating chorea. Of the nineteen cases in which there was also dorsal tenderness, the symptoms were in general those characteristic of spinal irritation of both regions.

d. The whole spine was tender in fifteen cases, and so extensive was the hyperæsthesia that it was scarcely possible to press upon the most limited spot without producing pain. Of these cases the most prominent symptom in three was *epilepsy*, in one *paralysis*, sometimes of the upper and sometimes of the lower extremities, and in three *contractions* of the limbs. *Neuralgic pains*, either in the scalp, face, neck, chest, upper extremities, abdomen, and lower extremities, were present in every case, according to the part most severely affected for the time being. The heart was disordered in five cases, the stomach in ten, in three there was *difficulty of swallowing*, from alternating paralysis, and spasm of the muscles of the larynx, and in two *aphonia.*

Causes.—The most powerful predisposing cause is *sex.* Of the one hundred and fifty-six cases, one hundred and forty were females. Age is likewise influential in determining to the disorder. Of one hundred and thirty-seven cases in which I have recorded the age, seventy-two were between fifteen and twenty-five, thirty-two between twenty-five and thirty-five, fifteen under fifteen, and eighteen over

thirty-five. The period of life between fifteen and twenty-five is therefore that at which spinal irritation is most apt to occur.

Hereditary influence was ascertained to exist in thirty cases.

The exciting cause of spinal irritation is not always easy to ascertain. In thirty out of one hundred and thirty-seven cases I could not, by the most careful inquiry, find any circumstance likely to have given it origin. In twenty-one it was manifestly produced by blows, falls, or strains, in twelve it was obviously caused by sexual excesses, and four by onanism. In ten there was reason to ascribe it to anxiety and grief, in two to excessive mental exertion, in twenty-one to insufficient physical exercise, in fourteen to innutritious and insufficient food, in three to over-indulgence in alcoholic liquors, and in one to the use of opium. In the remaining nineteen cases it followed exhausting diseases, such as typhoid, scarlet, and intermittent fever, dysentery, and diphtheria, and was probably directly the result of their influence.

It may also be caused by obliteration of the aorta or spinal vessels, by tumors, thrombosis, or embolism, by hæmorrhage from vessels in relation with those of the cord, or by exposure to severe cold.

In general terms, it may be said that any cause capable of reducing the powers of the system may produce spinal irritation.

Morbid Anatomy and Pathology.—I have already stated it as my opinion that the essential condition of spinal irritation is anæmia of the posterior columns of the cord. Other writers have ascribed it to inflammation, congestion, hysteria, and numerous other factors. The reasons which have induced me to arrive at this conclusion are briefly as follows: Owing to the fact that spinal irritation is not *per se* a fatal disease, we rarely have the opportunity to verify any views we may hold in regard to its pathology. In the few cases in which

post-mortem examinations were made, nothing abnormal was found, a circumstance, however, far more compatible with the idea I have expressed than with any other:

1. It is a well-recognized fact that irritation is often a result of a deficient supply or a poor quality of blood. Thus headaches are frequently caused by cerebral anæmia, and are promptly relieved by increasing the amount of blood in the cerebral blood-vessels. Irritability of the mind is also a constant accompaniment. A feebly-nourished stomach rejects food, and is the seat of pain. An anæmic heart beats with great rapidity, weak muscles are affected with tremor, and an exhausted generative system is brought into a state of unnatural erethism by the slightest kind of excitation. Analogy, therefore, supports the theory I have suggested.

2. The diagnosis of diseases of the spinal cord has become so perfect that we are able to distinguish congestion, meningitis, myelitis, softening, tumors, etc., by their symptoms and by the means of research at our command. We see, therefore, that the morbid phenomena which result from such conditions are not such as we now class under the head of spinal irritation. This division of the subject will be more fully considered under the head of diagnosis.

3. I have repeatedly ascertained, by actual experience, that those agents which are known to diminish the amount of blood in the spinal vessels invariably increase the severity of the symptoms due to spinal irritation, while they are as effectually lessened in intensity by remedies which tend to produce spinal hyperæmia.

4. The general condition of patients the subjects of spinal irritation is always below par, and the exciting causes are all such as tend to the production of asthenia.

5. The character of the symptoms points decidedly to the greater, and at times sole implication of the posterior columns. There are cases of the disorder in which there is no derangement of motility in any part of the body, and in all cases aberrations of sensibility are the prominent fea-

tures. Moreover, the viscera are generally affected in their functions, a circumstance of itself strongly indicative of the situation of the lesion in the posterior columns.

These circumstances, I think, go very far toward confirming the view I have expressed, that in spinal irritation the vessels of the cord, especially those of the posterior columns, contain less blood, and that this fluid is inferior in quality to that when the organ is in a healthy condition. Now that the function of the sympathetic nerve, as regards its action in regulating the calibre of the blood-vessels, is so satisfactorily proven, we can partially understand how local congestions and anæmias may be superinduced. It is probable, therefore, that the original difficulty in many cases of spinal irritation resides in the sympathetic system, and the intimate anatomical relations existing between the two nervous centres are strongly in favor of this suggestion.

On the other hand, many of the phenomena of spinal irritation point strongly to the secondary involvement of the sympathetic system. It is thus that the visceral disturbances which form such prominent features are mainly to be explained.

The pathology of several others of the more striking symptoms of spinal irritation has been a subject of frequent discussion, but at the present day presents no difficulties. Thus the excitation of pain in the tissues to which the cutaneous nerves are distributed results from the law that irritation at a nervous centre induces pain at the points in which the nerves arising from that centre end. Each compound spinal nerve sends a twig to the skin contiguous to it, and these twigs terminate immediately over the spinous processes. Now, whenever an irritation is thus transmitted to the periphery, it may be reflected back to the centre whence it came, by local irritations. Thus a patient is suffering from chronic inflammation of the spinal cord, and in consequence has pain and muscular spasms in his lower extremities. An irritation applied directly to the cord in-

creases the pain and spasms; an irritation applied to the lower extremities augments the pain in the cord, and may induce pain and spasms in distant parts of the body. Hence it is that pressure on the skin over the spinous processes not only causes cutaneous pain, but also gives rise to spinal pain, and neuralgic sensations in those nerves which come from the irritated part of the cord.

The pain existing in the cord is aggravated by percussion or muscular action. The spinal cord, it is true, is enclosed in a strong and thick, bony canal, which, however, is entirely filled by its contents. A blow, therefore, on the exterior of the column causes a vibration, which is propagated through the bony structure to the cord and its membranes. If this blow be very violent, the concussion may be such as to inflict irreparable damage on the cord. When any portion of the cord is in a state of irritation, a very light blow upon the spinous processes, over the disordered part, will cause severe pain, or notably add to that already present. The vertebral column is flexible, and therefore muscular action may, by producing deviations from the ordinary line followed, occasion pressure, and, in the abnormal condition of the cord, excite pain.

Diagnosis.—Recollecting that no case is to be regarded as one of spinal irritation which is not characterized by spinal tenderness, we have our diagnostic inquiries limited to the distinguishing of spinal irritation from other spinal affections. It is certainly true that the distinction has often been overlooked, and that at times there is a real difficulty in forming a correct judgment. Nevertheless, by carefully estimating all the circumstances, permanent errors of diagnosis are not likely to occur.

There are three diseases of the spinal cord which may in their earlier stages be confounded with simple spinal irritation. These are chronic myelitis, meningitis, and congestion. As the treatment of these affections is in many respects the exact reverse of that proper for spinal irritation,

and as they are of far more serious character, it is important to make as early and as correct a discrimination as possible.

In both spinal irritation and myelitis there is tenderness over some part of the vertebral column, which tenderness is increased by pressure, but this tenderness is never due to hyperæsthesia of the skin, whereas in spinal irritation it often is.

In spinal irritation there is never, so far as my experience goes, anæsthesia, whereas this is a constant accompaniment of myelitis.

The contractions which take place in some cases of spinal irritation are painless, while those due to myelitis are attended with great suffering.

In myelitis there is a sensation as if a tight cord were tied around the body at the upper limit of the paralysis, a sensation which is absent in spinal irritation. It is true that Mr. Teale has described several cases which he classed as spinal irritation and in which the sensation of constriction was present, but careful examination of the histories leaves scarcely a doubt that these were really cases of myelitis.

The bladder is never paralyzed in spinal irritation, whereas in myelitis it generally is, if the inflammation be located in the lower dorsal region of the cord. The same is true of the sphincter ani. Myelitis is always productive of paralysis, and there is always more or less atrophy of the paralyzed muscles. Spinal irritation seldom gives rise to paralysis, which, when it does result, is always incomplete, and is never productive of atrophy.

The progress of myelitis is generally, unless arrested by appropriate treatment, toward a worse condition, whereas no such tendency is manifested by spinal irritation.

From spinal meningitis, spinal irritation is distinguished by the circumstances that in the former disease there are constant painful spasms of the muscles of the back, pain

in the cord, and no spinal tenderness increased by pressure.

From congestion of the spinal cord and its membranes, spinal irritation is sufficiently distinguished by the facts that there is generally little or no pain in the cord in the first-named affection, and no spinal tenderness. In congestion, likewise, the paralysis and other symptoms are always worse after the patient has been lying down, while in spinal irritation the recumbent position always alleviates the condition.

Another means, which in doubtful cases will invariably lead to a correct diagnosis, is afforded by the known effects of certain medicines. Thus spinal irritation is, as I have several times ascertained, made worse by the administration of ergot, while each one of the other diseases I have named is alleviated. The reverse is true of strychnia, which in all cases aggravates the symptoms of myelitis, meningitis, or congestion, while it is an efficient means of cure in spinal irritation. A hypodermic injection of the thirtieth of a grain is sufficient to settle the matter in cases where the diagnosis is of difficult formation.

The flatulence, eructations, and vomiting, are very symptomatic of spinal irritation, while they are rarely phenomena of either of the other affections.

One other disease is liable to be confounded with spinal irritation, and that is angular curvature, in which there is spinal tenderness increased by pressure. The facts, however, that strumous disease of the vertebræ generally occurs in children, that the scrofulous diathesis is always present, that an angular prominence can be detected by careful examination, that the paralysis progressively becomes more profound, that the constitutional effects are more severe, are sufficient, even in doubtful cases, to guide to a correct diagnosis.

Prognosis.—The prognosis in cases of spinal irritation is generally favorable. In fact, so far as my experience extends, I have never seen a case which entirely resisted treat-

ment, and very few in which a cure was not ultimately effected. When remedies suitable for the difficulty do not prove successful, it is because the patient does not steadfastly persevere in their use.

Of the one hundred and fifty-six cases forming the basis of this chapter, one hundred and thirty-three were thoroughly cured, ten were lost sight of soon after treatment was commenced, but were materially improved, and thirteen were relieved for the time being, but continued to have relapses.

Treatment.—The principles of treatment applicable to spinal irritation are four:

1. To remove the cause.
2. To improve the general tone of the system.
3. To increase the amount of blood in the spinal cord, and improve the nutrition of this organ.
4. To set up a counter-irritant action in the vicinity of the disordered region of the cord.

In regard to the first indication, I have nothing special to say. The cause once ascertained, common-sense would dictate its removal as speedily and as effectually as possible, by the proper means according to its character.

The second indication is to be met by tonics, such as quinine and iron, and especially stimulants judiciously administered. I am as well convinced of the general applicability of alcohol in some form, in the treatment of spinal irritation, as I am of any thing. Whiskey, brandy, and rum, are to be preferred on account of their less liability to disagree with the stomach, and as containing a greater percentage of alcohol than vinous or malt liquors. Among the tonics the preparations of zinc are valuable, and I think the oxide is to be preferred. Cod-liver oil is also of great service.

The third indication is easily fulfilled by strychnia, phosphorus, phosphoric acid, and opium. The two first-named remedies may be very satisfactorily combined in a pill con-

taining half a grain of extract of nux-vomica and the tenth of a grain of the phosphide of zinc, which may be given three times a day. Strychnia may also be given by solution of the sulphate in dilute phosphoric acid, and in doses of about the thirty-second of a grain to half a drachm of the acid. The beneficial effects of these remedies are perceived in a few days. Opium is especially useful in those cases in which there are contractions of the limbs, and here its action is, of course, not solely that of an agent increasing the amount of blood in the cord. I prefer to give it either in the form of suppositories, composed each of half a grain of the aqueous extract and a sufficient quantity of the butter of cacao, or by hypodermic injection of morphia. I have frequently seen contractions, which had persisted with obstinacy for several weeks, relax in a few minutes under the influence of opium thus administered.

The application of hot water to the spine is also an admirable adjuvant. It should be used as hot as can be borne. Nothing is better for the purpose than Dr. Chapman's india-rubber bags.

But there is a remedy which apparently either contracts or enlarges the diameter of the blood-vessels, and which is more efficacious in removing spinal irritation than any other with which I am acquainted, and that is the direct galvanic current. The method I follow in cases of spinal irritation is the application of the negative pole at some point above the seat of the pain, and the positive at another, an equal distance below. An ascending current is thus brought to bear upon the cord, and this seemingly conduces to the dilatation of the blood-vessels and the improvement of the nutrition of the cord. The current should not be passed at any one *séance* for more than fifteen minutes, and no one application should last longer than three or four minutes. For the relief of the spinal tenderness the negative pole should be applied directly to the painful part, and the positive to a point distant laterally from it a few inches.

In inflammation or congestion of the cord or its meninges, the method of application is directly the reverse of this.

The fourth indication is one of great importance, and, when properly carried into effect, a cure will often result in slight cases without any other means of treatment being employed. The *rationale* of the action of counter-irritants in this and similar derangements is by no means clearly understood. It is a question which I do not, however, propose to discuss. Of counter-irritants my experience leads me decidedly to the employment of blisters in preference to any others. They should be applied to the skin, immediately over the painful part of the spine, and should be renewed as often as may be necessary. Tartar-emetic ointment, though useful, is more painful and I think not so efficacious as blisters. Dry cups are more admissible, and almost always do good. They should be applied on each side of the spinous processes for an extent of four or five inches above and below the painful spot. Leeches, or any other means for the abstraction of blood are, according to my experience, always prejudicial.

Besides these therapeutical means, there are others of a more strictly hygienic character, which cannot be overlooked. Thus the food should be of a highly-nutritious character, moderate physical exercise should be taken, and as much time as possible should be spent in the open air.

Patients almost always feel more comfortable in the recumbent position than any other, because thereby the blood is allowed to settle in the spinal vessels. They should not therefore be prevented lying down during the greater part of the day, but at the same time they should be encouraged to take exercise, and especially so when there is any loss of power in the lower extremities. The induced or faradaic current is almost always of service, when applied to the affected muscles, and the direct is of great efficacy when passed through neuralgic nervous trunks.

In illustration of the views inculcated in this memoir, I append the following details of cases:

Case I. *Irritation of the Cervical Region of the Spinal Cord.*—Mrs. J. S. consulted me, May 7, 1868, for what she had been informed was a cerebral disorder. The patient was thirty-eight years of age, had had five children, and had always enjoyed good health till two years previously, when she had been thrown from her carriage. She was not stunned or otherwise seriously injured. Soon after the accident she noticed a rumbling noise in one ear, and in a few days subsequently the other ear became similarly affected. About the same time there were flashes of light before the eyes, and a dull, heavy pain in this point of the head. Vertigo was also frequently present. There was insomnia, and when she did sleep she was very apt to be attacked with nightmare.

These symptoms continued to annoy her for several months, without, however, compelling her to seek for medical advice, until at last she had a seizure which was certainly epileptic in its character. This was followed with disturbance of vision, and intense neuralgia of the fifth pair of nerves. She now placed herself under the charge of a physician in a neighboring city, where she was then residing, who diagnosed a tumor of the brain, and gave an unfavorable opinion as to the ultimate result. He, however, advised the use of iodide of potassium. She took this in large doses faithfully for three months—during which period she had two more epileptic attacks—without perceiving any benefit, and then she went to Europe. While there she consulted a number of physicians and surgeons of eminence, all of whom gave a very guarded prognosis. By the advice of several of these she took the bromide of potassium, with, at first, some advantage, but this was eventually lost, and her symptoms became as severe as before. She had several epileptic paroxysms during the four months she was taking the bromide. Finally, she travelled through Germany and

Italy, and, still obtaining no relief, returned home. I saw her a few days after her arrival. She was then suffering from facial neuralgia, excessive tenderness of the scalp, so that she could not have her hair brushed without enduring great pain, obscureness of vision, pain in the eyeballs, redness of the conjunctivæ, vertigo almost constantly, great mental irritability, amounting at times to positive insanity; wakefulness, nightmare, and contraction of the fingers, the nails being strongly pressed against the palm of the hand.

Ophthalmoscopic examination showed dilatation of the retinal vessels, arterial and venous pulsation, and congestion of the optic disks of both eyes. The pupils of both eyes were contracted.

Perhaps I should not have suspected any spinal difficulty, if she had not herself called my attention to a pain which she said she constantly felt between the shoulders. I therefore examined the upper part of the spine very carefully, and found deep-seated pain developed by percussion over the seventh cervical vertebra, and great hyperæsthesia of the skin over the eighth. Her symptoms were not those in the least indicative of congestion of the cord or its membranes, of meningitis, or myelitis, and the apparent severity of the cerebral symptoms, and the general good condition of her mind and sensorial and motor functions, were so incompatible, that I could not, upon reflection, bring myself to the belief that she was affected with any organic disease of the brain. My inquiries and examinations all led me to the conclusion that she was laboring under spinal irritation of the lower cervical region.

I therefore prescribed for her five drops of the phosphorated oil three times a day, applied a blister to the painful spot, and daily passed the direct galvanic current through the cord, by applying the negative pole to the fifth cervical, and the positive to the sixth dorsal. My object was, not only to improve the nutrition of the cord, but also, by irritation of the sympathetic, to contract the vessels of the

brain. Budge and Waller had shown, several years previously, that, when that portion of the spinal cord situated between the seventh cervical and sixth dorsal vertebræ is acted upon by the galvanic current, the pupils are dilated. Now, dilatation of the pupils is produced by excitation of the sympathetic, and excitation of the sympathetic within the limits mentioned likewise causes contraction of the vessels of the brain, as can readily be seen by ophthalmoscopic examination while the current is passing.

Under the influence of this treatment the amendment was rapid, and at the end of three months she was entirely cured. It was necessary, however, to apply eleven blisters.

Case II. *Irritation of the Cervical Region of the Cord.*—M. S., a gentleman of sedentary habits, consulted me, August, 1867, for intense headache and facial neuralgia, with which he had suffered for several months. The disease had come on gradually, and, although now never entirely absent, was paroxysmal in its character, being more severe at night than through the day. The external pain followed the course of the fifth pair of nerves through all its branches; the internal was fixed in the posterior part of the head, and was evidently due to cerebral anæmia, as it was relieved by stimulants and by holding the head in a dependent position. Vertigo was frequently present, and the disposition to sleep was excessive, though, owing to the pain, could not be indulged in for more than a few minutes at a time. Nausea was occasionally a symptom, but never to the extent of being followed by vomiting.

On examining the spine of this gentleman, I found tenderness over the fourth and seventh cervical vertebræ. Two blisters were at once applied, and Aitken's syrup of the phosphate of iron, quinine, and strychnia, administered. From the first, improvement was manifested, and in less than a month the cure was complete.

Case III. *Irritation of the Dorsal Region of the Spinal Cord.*—Mrs. J. B., aged twenty-four, consulted me, March,

1868, for obstinate vomiting, and neuralgic pains in the left breast. She was thin, pale, and anæmic, and had suffered for over a year. She also complained of a dull, aching pain in the middle of the back, which was increased by even moderate physical exercise. The vomiting took place regularly after every meal, and even water was at once thrown up. She was under the impression that the disorder was the result of exposure for several hours to very severe cold while in an open boat.

Recognizing, at once, the fact that the main difficulty lay in the cord, I carefully examined the whole spine, and found excessive tenderness over the spinous processes of the sixth, seventh, and eighth dorsal vertebræ. There was also deep-seated spinal pain developed by percussion.

I ordered the application of a blister, and the internal use of small quantities, frequently repeated, of milk-punch (one ounce of brandy to three of milk). The first wineglassful was at once rejected, and so was a tablespoonful which she took half an hour subsequently. I then reduced the quantity to a teaspoonful every half hour. This was retained, and was the first nutriment of any kind which, for nearly eleven months, had not been rejected wholly or in part.

The next day I found that the blister had drawn well, and that the nausea and vomiting were greatly diminished, as were likewise the neuralgic pains. A teaspoonful of the following mixture was then directed to be taken three times a day, immediately after meals: ℞. Strychniæ sulph. gr. j, ferri pyrophosph., quiniæ sulph. āā. ʒss, acid phosph. dil., syrupus zingiberis āā. ℥ij. M. ft. mist. The milk-punch was still continued, but, in treble the dose, less frequently given.

Gradually all the symptoms decreased in violence, and at the end of two weeks she was enabled to retain a moderate quantity of food at each meal. Any excess was still, however, followed by vomiting. She had increased five

pounds in weight, and was greatly improved in personal appearance.

In two months she had gained twenty-one pounds, and was as well as she had ever been in her life. The spinal tenderness had entirely disappeared; seven blisters were applied in all.

Case IV. *Irritation of the Dorsal Region of the Spinal Cord.*—Mrs. W. had for more than three years suffered from spasmodic movements of the upper extremities, not distinguishable from those of true chorea, which occasionally were followed by contractions of the flexors of the wrists and fingers. There were also infra-mammary pain, eructations, and vomiting. When she came under my care, June 22, 1869, she was reduced to almost a skeleton, and was suffering, in addition to the symptoms above mentioned, from acute pain in the back. This pain she informed me had not been ordinarily very severe, but was, nevertheless, constantly present. On examination I found tenderness over the first, second, and third dorsal vertebræ. I at once applied the constant galvanic current in the manner already described, and continued it for five minutes, with the effect of mitigating the pain in the spine and the nausea. The ensuing day I repeated the application, and in addition prescribed the mixture given in Case III. She retained it on her stomach, as she did the food which she ate that day. Brandy in ounce-doses was given with her lunch and dinner. The galvanism was continued daily for eighteen days, at the end of which time she was free from pain, from the spasms, and from the vomiting. Her appearance was immensely improved, and she had increased seven pounds in weight. The galvanism was now discontinued, but the strychnia mixture and the brandy were persevered with for over a month longer. She was then well.

Case V. *Irritation of the Lumbar Region of the Spinal Cord.*—E. T., an unmarried lady, aged twenty-nine, consulted me, August, 1869, for paralysis of the lower extremi-

ties, attended with spinal tenderness and abdominal pains. She had been treated for inflammation of the spinal cord, had been cupped, leeched, and had had an issue made over the seat of the pain.

When I first saw her she was unable to walk, having been in this condition for several months. As she sat in her chair, she could readily move her legs in any desired direction, but to bear her weight upon them was an utter impossibility. There was no alteration of sensibility. Her general appearance was not anæmic, nor was she in the least degree hysterical. Upon careful examination, I was unable to find any reason to induce the belief that she was laboring under spinal congestion, meningitis, or myelitis, or that there was softening of, or pressure upon, the cord. I, however, discovered great tenderness over the first and second lumbar vertebræ, and found that strong pressure in this region induced deep-seated spinal pain and sharp neuralgic sensations along the course of the crural nerves.

Regarding the case as one of pure spinal irritation, I applied the constant galvanic current to the back every alternate day, and administered the following prescription: ℞. Zinci phosphidi, grs. iij, ext. nucis vom. grs. xv. M. ft. in pil. no. xxx. Dose, one three times a day. I likewise directed the application, to the painful part of the spine, of flannel, wrung out of spirits of turpentine, to be continued daily till redness and decided smarting were produced. A full and nutritious diet, with ale, was enjoined. Under this treatment she improved so rapidly in every respect that in twenty-three days she was able to walk with a cane, and in a few days more than a month was well, being in as good health, according to her own report, as she had ever enjoyed in her life.

ANÆMIA OF THE ANTERO-LATERAL COLUMNS OF THE CORD.

The phenomena which in my opinion are the result of an anæmic condition of the antero-lateral columns of the spinal

cord have hitherto been classed under the heads of spinal paresis, functional paralysis, reflex paralysis, inhibitory paralysis, paralysis from peripheral irritation, etc. Several of these names are applied with reference to the causes, others with reference to the symptoms, but none to the lesion.

Symptoms.—The most prominent symptom of anæmia of the antero-lateral columns of the spinal cord is paralysis of motion in those parts of the body which derive their nerves from the affected portion of the cord, and in many cases of those below the seat of the lesion. This paralysis is incomplete, the patient, if the lower extremities are affected, being able to walk, though he does so with difficulty. It is noticed, too, that some muscles are more apt to be paralyzed than others, the tibialis anticus and the peroneal group rarely escaping.

In the great majority of cases the paralysis is confined to the lower extremities, constituting paraplegia. The reason for this is, that the anæmic condition of the cord which causes the paralysis is more frequently excited by irritation transmitted from the genito-urinary and digestive organs than from any others.

Spasmodic contractions of the paralyzed muscles are not often met with, though occasionally there are slight twitchings, fibrillary in their character.

It is rarely the case that the paralysis extends, as it does in that which results from congestion of the cord. The affection usually supervenes suddenly, and is about as severe in the beginning as at any subsequent period.

The bladder and rectum are very rarely involved as a consequence of the spinal lesion, though disease of either of these organs often causes anæmia of the antero-lateral columns of the cord. In a few cases, however, I have witnessed both paralysis of the bladder and of the sphincter coming on late in the course of the disease, and evidently dependent on it.

Electro-muscular irritability is rarely impaired. Reflex

excitability is also generally unaffected. In the worst cases, tickling the sole of the foot will cause the leg to be drawn up, even against the volition of the patient.

Disorders of sensibility are not prominent features in anæmia of the antero-lateral columns of the spinal cord. Locally there is very rarely pain, and in the paralyzed parts there is neither anæsthesia, hyperæsthesia, nor abnormal sensations of any kind. There is never, in the uncomplicated affection, the sensation of constriction about any part of the body. The stomach and bowels are not often affected, unless there is at the same time some degree of anæmia of the posterior columns. But in one very interesting case, occurring in a lady of this city, and produced by exposure to extreme cold while crossing to Governor's Island in an open boat, there were vomiting every time food was taken into the stomach, and the most obstinate constipation I have ever witnessed. It very frequently happened that this lady had no operation from her bowels for over a month.

Causes.—Anæmia of the antero-lateral columns of the spinal cord may be produced by any cause capable of interrupting the flow of blood to the region in question, of lessening the calibre of its autocthonous arteries, or of so lowering the quality of the blood as to unfit it for the purposes of nutrition.

Thus it may be caused—though not without the implication of the posterior columns—by abdominal tumors compressing the aorta, or by disease of this vessel, leading to partial or complete obliteration ; by thrombosis or embolism of the spinal arteries ; or by direct loss of blood from vessels supplying the cord, or deriving their blood from the spinal vessels.

The calibre of the intra-spinal vessels may be lessened through the influence of extreme cold, and anæmia of the antero-lateral columns thus induced. Several cases of this kind have come under my care, in which paraplegia has supervened suddenly during or after exposure to very low

temperature, especially when combined with a moist state of the atmosphere. Lying on damp ground has caused it in a number of instances.

It not unfrequently follows exhausting diseases of various kinds. I have known it to supervene on dysentery, diarrhœa, cholera, typhoid fever, typhus, diphtheria, and several other affections.

But the most common cause of the disorder is undoubtedly peripheral irritation, and this is very frequently an affection of the genito-urinary organs. My friend Dr. S. Weir Mitchell[1] has written very exhaustively on this subject, and has shown the relation which exists between the different paralyses now usually called reflex, and injuries of nerves. Under the head of pathology I shall have occasion to return to Dr. Mitchell's valuable contributions.

Diagnosis.—Anæmia of the antero-lateral columns of the cord is distinguished from congestion by the facts that the symptoms are mitigated by the recumbent position instead of being increased in violence, as in the latter affection; that the paralysis shows no tendency to become more severe, and that, when the bladder or rectum is involved, the difficulty precedes the paralysis.

From anæmia of the posterior columns, it is diagnosticated by the fact that the more obvious symptoms are related to motility, sensibility not being involved, while in the former the reverse is the case.

The diagnosis from myelitis will be pointed out when inflammation of the cord is under consideration.

Prognosis.—The probability of a favorable termination is great. In fact, no affection of the cord is so susceptible of cure when there is no mechanical obstruction in the aorta or spinal arteries. But this opinion is expressed with the

[1] Circular No. 6, 1864, Surgeon-General's Office. Reflex Paralysis, by Drs. Mitchell, Morehouse, and Keen. Also Wounds and Injuries of Nerves by the same, Philadelphia, 1864. Also Paralysis from Peripheral Irritation, by Dr. Mitchell, New York Medical Journal, February, 1866.

understanding that the cause must first be removed. So long as this continues in action, anæmia of the antero-lateral columns of the cord is a very obstinate affection. When the arteries are obstructed, then, as in the brain under like conditions, softening of the cord may take place.

Morbid Anatomy and Pathology. — Post-mortem examination, of persons who have suffered with symptoms indicative of what I consider to be anæmia of the antero-lateral columns of the cord, does not reveal the existence of any material spinal lesion. The reason for this is that anæmia of the cord is, in the nature of things, a very difficult disease to detect, and cannot be definitely made out, unless the capillaries are measured under the microscope.

But it is this very absence of obvious lesions which indicates very positively the existence of anæmia, and the character of the symptoms shows that the antero-lateral columns are its seat.

Several varieties of paralysis result from anæmia of the antero-lateral columns. Classing these as Mitchell[1] has done, from their apparent causes, we find that there are—

1. Paralyses arising during disease of the genito-urinary organs.
2. Those which occur during or just after dysenteries, diarrhœas, super-purgation, or in connection with worms.
3. Such as arise during or after pneumonia or pleurisy.
4. Such as are seemingly brought on by dentition.
5. The paralysis of diphtheria, fevers, and eruptive disorders.
6. Such as seems to be occasioned by cold, or by cold and moisture.
7. Paralysis due to external injury.

To this list may be added—

8. Paralysis resulting from certain medicines and drugs.
9. Paralysis due to great emotional disturbance.

[1] Paralysis from Peripheral Irritation, with Reports of Cases, New York Medical Journal, February, 1866, p. 323.

Many cases of each of these varieties of paralysis have come under my notice, and there are few medical practitioners who have not witnessed instances referable to one or more of the foregoing categories. The principal theories of their immediate cause are—

1. That of Mr. Stanley,[1] by which certain varieties of paralysis are attributed to the transmissal of an irritation from a diseased organ to the spinal cord, whence it is reflected to the muscles as paralysis.

This is no explanation at all, and leaves the condition of the cord out of consideration. There is no proof whatever that an irritation can, without causing change in the structure of a nervous centre, induce either paralysis of motion or of sensation.

2. That of Dr. Brown-Séquard,[2] which ascribes the affections in question to a lesion of the cord, consisting in a spasm of the spinal vessels by which their calibre is diminished. This spasm is, according to this eminent neurologist, the result of a peripheral irritation transmitted through the nerves coming from a diseased organ or part of the body, to the vaso-motor nerves of the portion of the cord giving origin to these nerves.

This was, so far as I have been able to ascertain, the first attempt to designate the character of the lesion, which, as will be at once perceived, is anæmia. That anæmia can be induced by peripheral irritation is, I think, well established. But though this theory accounts for many cases of spinal paralysis, such as are now under notice, it will not embrace all, for we may have anæmia and consequent loss of motor power resulting from other causes than irritation. Moreover, Dr. Brown-Séquard did not fix the lesion in the antero-lateral columns, nor associate the symptoms with

[1] On Irritation of the Spinal Cord and its Nerves in Connection with Disease of the Kidneys, Medico-Chirurgical Transactions, vol. xviii., p. 260.

[2] Lectures on the Diagnosis and Treatment of the Principal Forms of Paralysis of the Lower Extremities, Philadelphia, 1861.

any derangement in the structure of this region of the cord.

3. Dr. Mitchell, in the paper to which I have already referred, divides the several kinds of paralysis mentioned into three classes: those which are asserted to be due to disease of the genito-urinary system, a cause which he denies *in toto;* those which are said to be produced by peripheral irritation of the intestinal canal, an influence which he also in great part denies; and those which follow wounds and injuries of nerves.

Dr. Mitchell rejects altogether the reflex theory of Dr. Brown-Séquard, and says:

"If I were now to sum up the probabilities in the way of causation of palsies peripherally induced, I should be disposed to refer some cases to exhaustion from too constant or excessive exercise of normal functions, and others to irritation from disease or injury, and to consequent exhaustion of the centres; while, as regards the intervention of vascular agency, I should reject the idea of prolonged vasal spasm, and consider it possible that in some instances over-excitation might result in dilatation of the vessels, in which case some material lesion would surely result if the condition in question were of long continuance."

While not prepared to accept Dr. Mitchell's views in their entirety, they are, in my opinion, perfectly in accordance with the doctrine of anæmia of the antero-lateral columns. As to whether this anæmia is the result of spasm of the spinal vessels, or exhaustion, is a question which, for the present at least, is not definitely settled. My own opinion is that paralyses of apparently peripheral origin are referable to anæmia, produced in some cases by vaso-motor spasm, and in others by nervous exhaustion.

The experiments of Küssmaul and Tenner [1] are perfectly conclusive as to the effects of cutting off the supply of blood

[1] The Nature and Origin of Epileptiform Convulsions caused by Profuse Bleeding, etc. New Sydenham Society Translations, London, 1859, p. 53, *et seq.*

to the spinal cord. These observers compressed the aorta in rabbits so completely that not a drop of blood could reach the spinal cord below the point of occlusion. The consequence was that there was complete paralysis of all the muscles receiving their nervous influence from the anæmic portion of the cord. The possibility, therefore, of spinal anæmia producing paralysis, is beyond doubt. In these experiments, however, the blood was of course shut off from both the anterior and posterior columns, and therefore the phenomena were not those of simple motor paralysis.

In practice, likewise, we often find that the anæmia is not restricted to either set of columns, and that the symptoms are accordingly those of motor paralysis, aberrations of sensibility, and functional disturbances in various organs, such as we have just considered as being caused by anæmia of the posterior columns.

Treatment.—The treatment is similar in general features to that applicable to anæmia of the posterior columns already considered, though there is not the same benefit to be derived from counter-irritation. The indications, therefore, are to remove the cause, to improve the general tone of the system, and to increase the amount of blood in the spinal vessels.

So far as the first indication is concerned, it very often happens that its fulfilment is sufficient for the entire removal of the anæmia, and the disappearance of the consequent paralysis. This is especially the case as regards those instances which are due to peripheral irritations of various kinds. Within the last few days a young lady, aged twelve, was brought to me by her mother to be treated for paraplegia, which had developed very suddenly. There was no evidence of serious organic difficulty, and no apparent cause of peripheral irritation. Her symptoms, however, all pointed to anæmia of the antero-lateral columns, and, on the principle of exclusion, I thought it probable there might be worms in the alimentary canal. I therefore administered

several doses of santonine, followed by castor-oil. A number of lumbrici were discharged, and the paralysis disappeared in the night as suddenly as it had arisen.

In another case, a gentleman was rendered paraplegic soon after contracting a catarrhal inflammation of the bladder. The bladder affection was disregarded by his physician, and energetic means were used against the paralysis, but without effect. I suggested the expediency of suspending the administration of the strychnia and the application of counter-irritants to the spine, and directing attention to the cure of the bladder difficulty. This was done, and, at the same rate as the inflammation yielded to the treatment, the paraplegia disappeared.

The general tone of the system is to be improved by such measures as were recommended for the accomplishment of the same end in anæmia of the posterior columns.

For fulfilling the third indication, strychnia and phosphorus are preferable to any internal remedies. I usually prescribe them together in doses of the tenth of a grain of the phosphide of zinc, with from a third to a half a grain of the extract of nux-vomica in pill, to be taken three times a day. Lately, however, I have pursued the practice of giving the strychnia in gradually-increasing doses till there is evidence of its characteristic physiological effects being produced. Two grains of the sulphate of strychnia are to be dissolved in an ounce of water, and ten minims, containing one twenty-fourth of a grain of strychnia, given three times a day; the next day eleven minims are administered for each dose, the next twelve, and so on till, as often happens, the paralysis yields, or till the reflex excitability of the legs is increased, or stiffness of their muscles or those of the nucha is induced. In either of these latter events the administration must be stopped for a day, and then the original dose of ten minims be given and increased as before. There is, according to my experience, no medication so effectual in all those forms of paralysis called reflex, inhibitory, func-

tional, etc., and which, in my opinion, result from anæmia of the antero-lateral columns of the cord, as this with strychnia. It requires care and prudence, and, if these qualities be exercised, is perfectly safe. It very generally happens that, before the patient reaches thirty minims (one-eighth of a grain) for a dose, the paralysis begins to yield. In one case, however, due to exposure to severe cold, I was obliged to carry the dose to sixty minims—equal to one-fourth of a grain of strychnia—before the excitability of the cord was increased, or any signs of the paralysis yielding were observed. The patient recovered after taking three-quarters of a grain of strychnia daily for over two weeks.

My notes show that in the last year I have treated, according to the method described, sixty-one cases of paralysis due to anæmia of the antero-lateral columns, and that all were cured. It is true, galvanism was used in some of them, and phosphorus in others, but the successful results were evidently mainly due to the strychnia.

The only local application which is decidedly beneficial is the constant galvanic current, which should be used in the manner recommended for anæmia of the posterior columns.

As regards the paralyzed muscles, the induced or faradaic current is useful in keeping them exercised, and thus preserving their nutrition. Friction and kneading exercise a like effect.

In those cases of spinal anæmia due to obstruction of the aorta, or occlusion of spinal vessels by emboli, no specific treatment is of any avail.

LEEDS SCHOOL OF MEDICINE

CHAPTER III.

SPINAL HÆMORRHAGE—SPINAL MENINGEAL HÆMORRHAGE.

These two conditions having a common cause, being often associated and having a general resemblance to each other, may properly be considered together.

Symptoms.—A hæmorrhage into the substance of the spinal cord is characterized by pain at the seat of the lesion, and by derangements of sensibility and of the power of motion in all those parts of the body below. These consist ordinarily of anæsthesia and loss of motility, but occasionally there are hyperæsthesia and spasms. In the great majority of cases the bladder and its sphincter and the sphincter ani are also paralyzed. Reflex excitability and electro-muscular contractility are soon impaired or altogether lost.

If the seat of the hæmorrhage be high up in the neck, death is almost instantaneous from the paralysis of the phrenic nerve.

When the lesion is meningeal, the symptoms are not generally so rapidly developed as when it is situated in the substance of the cord. The pain is greater and there is a more decided tendency to spasmodic jerkings in the limbs receiving their nerves from the part of the cord below the extravasation. Hyperæsthesia may alternate with anæsthesia, or this latter may alone be present.

The extent of motor paralysis is very variable, both as regards intensity and diffusion. Sometimes all the muscles below the seat of the lesion are more or less paralyzed; at

others, some muscles altogether escape. I have a patient at the present time under treatment who has, in consequence of a spinal hæmorrhage, probably meningeal, lost sensation in a small region of skin over the glutei muscles, and sensation and motion in all the tissues below both knees. Sensation and motion are intact in all other parts of the lower extremities. The bladder is unaffected, but there is very obstinate constipation.

Reflex excitability is often exaggerated, and the electro-muscular contractility increased in the early stage; but, if the patient survives the immediate effects of the lesion, both these faculties become impaired. Meningeal hæmorrhage taking place above the third cervical vertebra may be speedily fatal, from the interruption to respiration due to paralysis of the phrenic nerve.

Causes.—Spinal hæmorrhage, either in the substance of the cord or of the membranes, is almost invariably the result of injury. Thus it may be caused by blows on the vertebral column, by falls, or by gunshot, or by wounds with penetrating instruments. It may also be produced by tetanus and by the rupture of aneurisms, but is in either of these cases meningeal.

Diagnosis.—The diagnosis must mainly be determined by the history of the case, and by the facts that the symptoms come on suddenly and advance rapidly.

Prognosis.—Death is the almost invariable result. I have, however, known two instances of recovery. In one of these the patient, a boy of about fifteen, was thrown from his horse. Paralysis supervened immediately, and there was a severe pain at about the eleventh dorsal vertebra. The bladder was also paralyzed. For several weeks his life was despaired of, but he eventually recovered with the paraplegia remaining, and the necessity of drawing off the urine with a catheter. I saw him five years after the injury. He was still paraplegic, and the bladder was still paralyzed. Careful examination failed to show any displacement or

fracture of the vertebra, and I therefore felt warranted in concluding that there had been a spinal hæmorrhage, probably meningeal. The other case has been already cited. In this, the patient fell through a hatchway a distance of thirty feet, and struck on his back. Paralysis was almost immediate. He came under my care fifteen years after the event, and I diagnosticated a meningeal spinal hæmorrhage from the facts that there had been violent jerkings of the limbs and intense lumbar pain. There were no signs of fracture or displacement.

Morbid Anatomy and Pathology. — The extravasation in hæmorrhage into the substance of the cord is generally seated in the gray matter, and shows a greater tendency to extend longitudinally than laterally. The white matter is, however, occasionally involved.

The changes which ensue in the clot and in the limiting tissue are similar to those which take place in cerebral hæmorrhage.

In spinal meningeal hæmorrhage the blood is extravasated either between the bones and the dura mater, between the dura mater and the arachnoid, or between the arachnoid and the pia mater. The clot may be very small or very large. The latter is more frequently the case.

The symptoms which follow spinal hæmorrhage are the results of excitation and compression—the hyperæsthesia and the spasms being due to the former, and the anæsthesia and motor paralysis to the latter.

Treatment.—There is nothing to do in cases of spinal hæmorrhage but to maintain the patient in as quiet a condition as possible, and to keep ice constantly applied to the vertebral column. If there is time, ergot might, I think, be administered with advantage. In two cases which I have had the opportunity of observing from the first, both caused by falls from the loft of a stable, death took place within six hours; the symptoms gradually becoming more profound and advancing upward. After death, the hæmorrhage was

found to occupy the whole length of the spinal canal, and was seated between the bones and the dura mater. Of course, in cases like these, no therapeutical means can avail, and, even in slighter cases, treatment is of little if any service.

We may, however, by perfect rest, ice to the spine, leeches to the anus, and the administration of ergot, sometimes prevent hæmorrhage in cases of injuries of the cord which otherwise might be followed by extravasation.

CHAPTER IV.

SPINAL MENINGITIS.

Inflammation of the membranes of the spinal cord may be either acute or chronic.

ACUTE SPINAL MENINGITIS.

Acute inflammation may be seated either in the dura mater, the arachnoid, or the pia mater of the cord, or may simultaneously attack all three membranes.

Symptoms.—The symptoms indicating inflammation of the dura mater are not very decided, and beyond the occurrence of pain may not be observed at all. When combined with inflammation of the arachnoid and pia mater, the phenomena are more pronounced.

Acute inflammation of the arachnoid does not of itself give rise to characteristic symptoms, and it is rarely the case that it exists separately.

Acute inflammation of the pia mater can, however, be recognized without difficulty. It begins with a chill, as do others of the phlegmasiæ, and this is soon followed by febrile excitement. At the same time there is intense pain in the back, which is aggravated by every movement of the patient, but not by pressure on the part of the spine over the diseased portion of the membrane. Those nerves which have their origins from the affected region are the seat of severe pain, which is transmitted through their trunks and branches to distant parts of the body. Spasms of the muscles of the back are commonly present. These

are tonic in character, and may be so severe as to bend the body backward, producing an appearance like the opisthotonos of tetanus. At the same time the limbs below the seat of the lesion are strongly contracted. I have witnessed cases in which the knees were drawn up to the chin, and the heels to the buttocks.

At the same time there is impairment of motor power in all those parts of the body supplied by nerves coming from the cord below the diseased region, and in some cases voluntary control over the muscles is entirely lost.

So long as the affection is confined to the membranes of the lower portion of the cord, a fatal result may be deferred for some time, and the disease may become chronic; but, if it extends upward so as to involve the region from which the phrenic nerves arise, death very soon takes place by asphyxia.

So long as the spinal cord remains free from the disease, the reflex excitability and electro-muscular contractility remain unimpaired.

The bladder is not often involved, and the bowels may be obstinately constipated, or the fecal matters may be passed involuntarily.

CHRONIC SPINAL MENINGITIS.

This may arise in consequence of an acute attack, or it may be developed spontaneously. As in the acute form of the affection, pain constitutes a prominent feature, and is situated both in the spinal region and in other parts of the body. Spasms and contractions of the lower extremities, and spasms of the muscles of the back, are likewise prominent symptoms.

The pain in the spine is not increased by steady pressure over the vertebræ, but it is greatly aggravated by every movement of the body; for by such motion the nerves are compressed as they leave the spinal canal, and, as they are already in a condition of erethism, pressure cannot be borne.

The abnormalities of sensation are usually in the way of hyperæsthesia, which may sometimes be very acute.

The paralysis advances gradually, and rarely, at first, is very intense in any group of muscles. It is likewise subject to great variations in the degree of severity. Sometimes the patient finds that he walks tolerably well one day, while the next he can scarcely move a limb. These differences depend on the amount of fluid effused, which is subject to changes from day to day.

The bladder is sometimes paralyzed, the sphincter may be similarly affected, or this latter may be subject to repeated attacks of spasm, by which the evacuation of the urine is prevented.

The bowels, as in the acute form of the disease, may be either constipated, or the sphincter ani may be so paralyzed as to allow of the involuntary passage of the fecal matters.

Reflex excitability is rarely lessened, and is often considerably increased. In the case of a gentleman from Ohio who was recently under my charge for chronic spinal meningitis, the slightest touch on the sole of the foot was sufficient to cause the limb to be violently drawn up; and, in the case of a lady from New Orleans similarly affected, the contact of the bedclothes produced a like effect.

In several cases I have observed that any mental agitation, or even the attention directed to the affected limbs, was sufficient to cause violent spasmodic contractions.

Electro-muscular contractility is not generally impaired.

The symptoms are usually aggravated by the recumbent posture.

Bed-sores are a frequent accompaniment of chronic spinal meningitis.

Causes.—The most common cause of spinal meningitis, either acute or chronic, is exposure to cold and moisture. Several cases have come under my charge which clearly resulted from lying on the cold and damp earth, and from going to sleep in this situation. In one case which occurred

in a railway conductor, the train of which he had charge was obstructed in its passage by a heavy drift of snow. While workmen were cutting a way through it, he lay down on a pile of snow, and, being greatly exhausted, soon fell asleep. Soon after being awakened he had a slight chill and a mild fever, and the following day experienced severe pain in the back. This was soon followed by the other symptoms of spinal meningitis, not very intense in character, but persistent, for the affection passed into the chronic form. Two cases have come under my notice in which the affection was caused by the back being exposed to a strong and cold wind.

On account of this influence of cold in producing spinal meningitis, the disease is far more common in winter than in summer. Of thirty-nine cases that I have treated wholly or in part during the last six years, twenty-three occurred in the months from November to March inclusive.

Exposure to the direct rays of the sun is said to induce spinal meningitis, but I have never witnessed a case in which this cause could reasonably be inferred. I may make the same remarks in regard to the effects of strong muscular exercise.

It is, however, sometimes a consequence of wounds and injuries. Seven of the cases under my charge were due to traumatic causes.

Rheumatism is likewise an occasional cause.

Diagnosis.—The diagnostic phenomena of spinal meningitis, either of the acute or chronic form, are the pain in the back, increased on any movement of the spinal column; the pains in the course of the nerves having their origin from the diseased region; the tonic spasms of the muscles of the back, and of other parts of the body; the exaltation of reflex excitability; and the variations which take place in the extent and intensity of the paralysis.

Prognosis.—The course of spinal meningitis is generally progressively onward to a fatal termination—the patient

dying either by the gradual extension of the disease upward so as to involve more important nerves in the lesion, by the development of some intercurrent affection, or by exhaustion. I have, however, seen three cases in which the disease was arrested, and which will be more specifically referred to under the head of treatment. And Ollivier,[1] Brown-Séquard,[2] and Jaccoud,[3] admit the possibility of cure.

Morbid Anatomy and Pathology.—The lesions found after death from spinal meningitis may be confined to any one of the membranes, but more generally are restricted to the pia mater and the sub-arachnoid space. They consist in thickening of the membrane, spots of opacity, turgidity of the vessels, and the effusion of a large quantity of spinal fluid. This fluid is occasionally clear, but is more frequently full of flocculent matter, or is tinged with blood.

The alterations found in the arachnoid are of similar character, with the addition that there are numerous hard cartilaginous plates scattered through the diseased part of the membrane, which vary in size from that of a grain of wheat to a mustard-seed.

The dura mater, when it has been the seat of inflammation, becomes thickened and adherent to the bone. Occasionally it is perforated by the supervention of gangrene, and the pus collected between it and the vertebræ escapes into the space between the dura mater and arachnoid, and excites general meningitis. The theory of the symptoms observed in spinal meningitis is, that they are due to two immediate causes, excitation and pressure. The former is the result of the hyperæmia, the latter of the increased amount of spinal fluid causing pressure.

Treatment.—In the acute form of spinal meningitis, active measures are required. The application of leeches to the painful part of the spine, or of cups, so as to effect local depletion, will generally prove useful. Hydragogue cathartics

[1] Traité des Maladies de la Moelle Épinière, etc., Paris, 1827, t. ii., p. 295.
[2] Op. cit., p. 82. [3] Op. cit., p. 302.

are also beneficial, for by their action the vessels of the inflamed membranes are depleted of their blood, and the excessive amount of spinal fluid effused is in consequence more readily absorbed.

Mercury may also be advantageously administered either by inunctions with mercurial ointment or by calomel given internally, or by both these means. Calomel should be given in doses of from one to two grains every three or four hours, till the system is brought under its influence, as manifested by fetor of the breath.

The patient should be kept as quiet as possible, and should be enjoined not to lie on the back. For the relief of the dorsal and other pains, suppositories, containing each,half a grain of codeine, are often efficacious. They may be administered night and morning.

In the chronic form of the disease, depletion by bloodletting in any form is not so beneficial as in the acute variety or as in spinal congestion. Blisters are more admissible, and scarcely ever fail to do good. They should be applied on each side of the spinal column near the diseased region of the cord, and as soon as one heals another should take its place. The actual cautery I have never used, nor have I ever seen such good results from its employment as from the action of blisters. Purgatives are also useful for the same reasons which prevail in acute spinal meningitis.

Iodide of potassium is always a valuable agent, indeed more so than any other remedy employed in chronic spinal meningitis. I employ it in the form of a saturated solution, which contains about a grain to each drop. Of this, I administer the first day seven drops three times, preferably before meals; the next day eight drops to the dose, the next nine, and so on, till the patient takes from forty to sixty drops at the dose, according to circumstances. The iodide of potassium always acts best when largely diluted with water, so that, as the doses are increased, an additional quantity of water should be used.

I very often employ the corrosive chloride of mercury in combination with the iodide of potassium, in doses of the sixteenth of a grain with each dose of the iodide.

Diuretics may also frequently be given with advantage. Their object is the same as that which governs in the administration of purgatives.

In two of the cases cured, to which reference has been made, I derived the greatest benefit from repeated blisters, and the persistent use of iodide of potassium. The latter was carried to the extent of fifty grains three times a day in one of these cases, and sixty-five in the other.

At the same time the primary galvanic current was applied to the spine in the manner recommended for spinal congestion, and the induced current to the paralyzed limbs. I am very sure that electricity in both these forms should be used in most cases of chronic spinal meningitis. The following case, reported by J. Frank,[1] and quoted by Ollivier,[2] of acute spinal meningitis, is instructive:

"A captain, aged forty-two years, of sanguineo-bilious temperament, subject to rheumatic pains and hæmorrhoids, and addicted to the use of alcoholic liquors, was suddenly seized on the evening of the 2d of March, 1819, with a chill, which was soon succeeded by a burning fever, accompanied by pain in the lumbar region. During the night the pain increased, extended as high up as the occipital region, and gradually acquired great intensity. J. Frank was called in the morning at five o'clock, to see the patient, who was suffering acutely. He was uttering loud groans, was lying on his belly, with the superior and inferior extremities stretched out to their full length. To the questions put to him, the patient answered with great difficulty that he had pains all over his body, that he was unable to open his eyes, that his teeth were strongly clinched, and that a burning and pulsating pain extended from the occiput to the lower extremity

[1] Praxeos Med., etc., de rachialgite, tome vi., p. 76. Turin, 1822.

[2] Op. cit., p. 295.

of the vertebral column. The limbs, especially the inferior, were without sensation, but were agitated by occasional jerkings. There was such a constriction of the chest that breathing was scarcely possible, and the abdomen was likewise in a state of contraction. There were constipation, incontinence of urine, a pulse soft but 100 per minute, occasional palpitations of the heart, and a hot and dry skin.

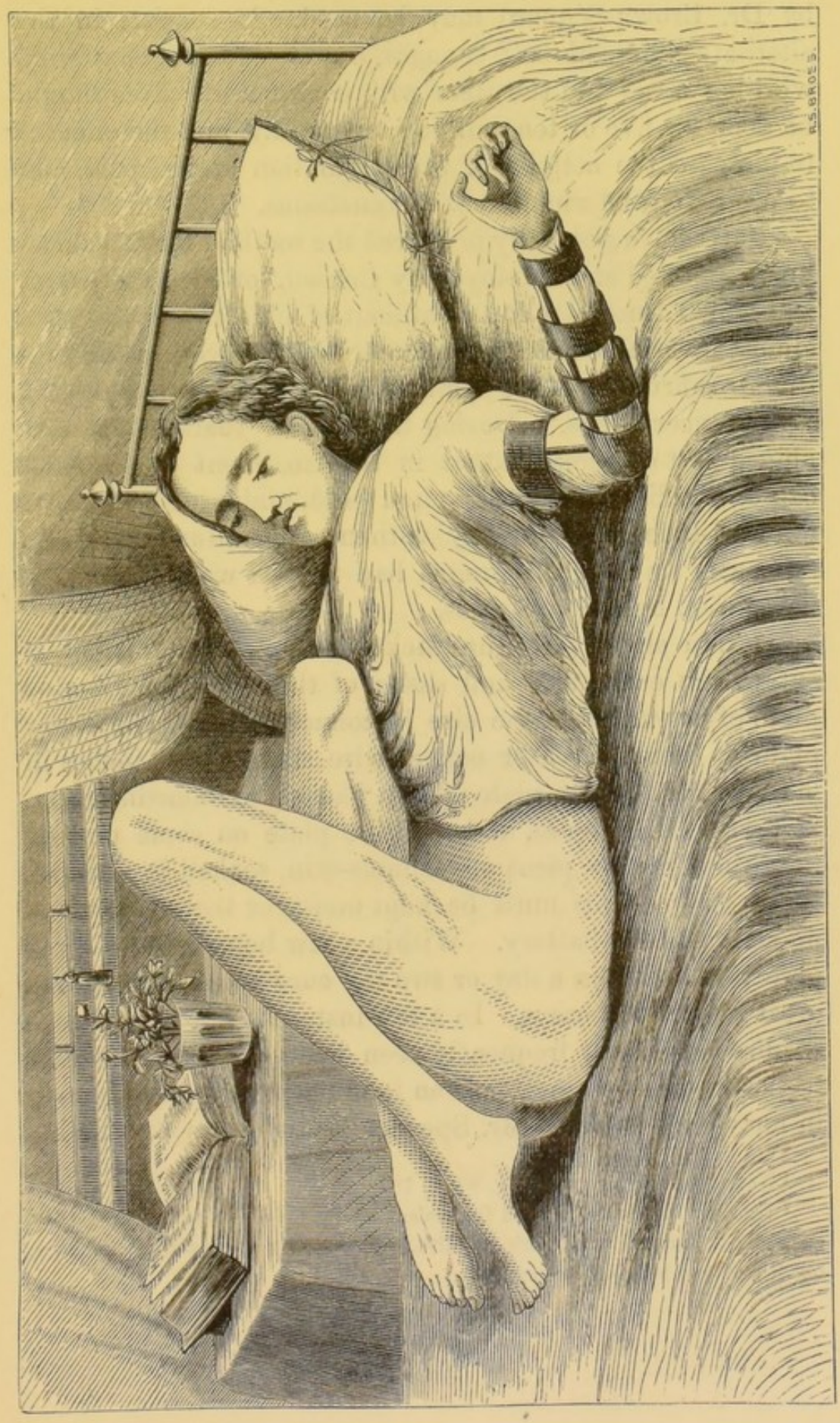
"Frank at once opened a vein in the foot, and abstracted sixteen ounces of blood. A dozen leeches were applied around the occiput, and as many scarified cups on each side of the spine. A decoction of tamarinds was given as a cathartic. These means were sufficient to restore the health of the patient in a few days. The bloodletting produced an almost immediate cessation of all the symptoms; for, a short time after its employment, the movement of the eyelids became easy, as well as that of the jaw; sensation reappeared in the extremities, and the dorsal pain diminished considerably in intensity."

As Ollivier remarks in regard to this case, several of the symptoms are those of spinal congestion. The sudden supervention of the disease, as well as its rapid disappearance, points to that affection. Nevertheless, its general features are those of acute spinal meningitis—an affection which, of course, cannot exist without congestion.

In a very interesting case under my charge several months ago, a cure of the spinal difficulty, which was chronic spinal meningitis probably of syphilitic origin, was accomplished by the use of the iodide of potassium and the corrosive chloride of mercury, as recommended on pages 449 and 450. In this case the affection had lasted for several years, and extended from the occiput to the lower extremity of the spinal cord. The limbs were constantly subject to violent spasmodic jerkings, and both legs and one arm were in a permanent state of contraction, which had existed for three years. Under the use of the iodide and the mercury, as mentioned, the

pain, which had been intense, ceased, the spasms of the limbs were stopped, the bladder regained its expulsive power, the bowels again began to act without purgatives or injections being required, and the limbs could be moved as extensively as the rigid contractions permitted. These had existed so long that the flexor muscles had become much shortened, and the skin in the groins and popliteal spaces was tense and unyielding. The accompanying woodcut (Fig. 21) shows the positions of the legs and arm at this time. Under these circumstances I requested the advice of my friend Prof. L. A. Sayre, and after consultation it was determined to divide the tendons of the tensor vaginæ femoris, the sartorius, the gracilis, and the biceps, on each side. When this was done by Prof. Sayre, the patient being under chloroform, careful but powerful efforts at extension were made, and the skin in the popliteal space on both sides was necessarily torn, owing to its contraction and inelasticity; the limbs were thus brought into a state of complete extension, and, by a system of weights and pulleys similar to that used in Buck's fracture apparatus, they were kept in this position. The patient was, however, too weak to endure the fatigue of the necessary extension and confinement. He took off the weights whenever they caused pain or great uneasiness. To add to the difficulties, a large bed-sore formed on the right buttock, and the strength of the patient declined so rapidly that, in order to save his life, the apparatus had to be entirely removed. He rapidly recovered, but, as cicatrization went on, the limbs again became contracted, and in the course of two or three months were as bad as ever. Pain in the back soon afterward supervened, the legs and one arm began to be affected with spasms, and the paralysis also returned. A renewal of the former medication again caused relief, and the patient has to this day remained free from any spinal difficulty, though his legs are still contracted. This is the third case of cure referred to as happening in my experience.

Fig. 21.



For the cure of the bed-sores the method recommended by Dr. Brown-Séquard may be used. It consists in the alternate application of sponges, one of which is saturated with hot water and the other with cold water. This should be done for five or ten minutes every day, and the effect is to increase the activity of the circulation of the part, and to promote the formation of granulations.

But I have generally preferred the method by galvanism first suggested and employed by Crussel,[1] of St. Petersburg, and which I used for the treatment of indolent ulcers with almost invariable success, in 1859, when surgeon to the Baltimore Infirmary. The method was also recommended by Mr. Spencer Wells.[2] During the last six years I have employed it to a great extent in the treatment of bed-sores caused by diseases of the spinal cord, and with scarcely a failure—indeed, I may say without any failure except in two cases where deep sinuses had formed which could not be reached by the apparatus.

A thin silver plate, no thicker than a sheet of paper, is cut to the exact size and shape of the bed-sore. A zinc plate of about the same size is connected with the silver plate by a fine silver or copper wire six or eight inches in length. The silver plate is then placed in immediate contact with the bed-sore, and the zinc plate on some part of the skin above—a piece of chamois-skin, soaked in vinegar, intervening. This must be kept moist, or there is little or no action of the battery. Within a few hours the effect is perceptible, and in a day or two the cure is complete in the great majority of cases. In a few instances a longer time is required. I have frequently seen bed-sores three or four inches in diameter, and half an inch deep, heal entirely over in forty-eight hours. Mr. Spencer Wells states that he has

[1] Neue Med.-Chirurg. Zeitung, No. 7, 1847, p. 235.

[2] Lectures on Electricity and Galvanism, by Dr. Golding Bird, London, 1849, appendix. There is an American edition of this very interesting little book, but it has long been out of print.

often witnessed large ulcers covered with granulations within twenty-four hours, and completely filled up and cicatrization begun in forty-eight hours. During his recent visit to this country I informed him of my experience, and he reiterated his opinion that it was the best of all methods for treating ulcers of indolent character and bed-sores.

Ergot is not so generally useful as in congestion, though I rarely fail to give it at some time or other in cases of chronic meningitis, with a view to the relief of the accompanying congestion. Strychnia is not at all admissible at any time. Reeves[1] recommends it in those cases in which pains, cramps, and contractions, are absent, but I have never seen such cases. Indeed, a case in which they were not prominent symptoms could scarcely be regarded as one of spinal meningitis.

[1] Diseases of the Spinal Cord and its Membranes, and the Various Forms of Paralysis arising therefrom. London, 1858, p. 55.

LEEDS SCHOOL OF MEDICINE

CHAPTER V.

ACUTE MYELITIS.

Acute inflammation of the substance of the spinal cord is not a very common affection, but it is so essentially distinct in its symptoms and results from chronic myelitis that it requires separate consideration.

Symptoms.—A chill is generally the first symptom observed, and this is followed immediately by high febrile excitement, during which the pulse often reaches a rapidity of 160 per minute. Alterations of sensibility and motility are noticed with the inception of the fever.

Among the first, pain in the back, at the seat of the lesion, is prominent. It is aggravated by percussion, and by the passage of a sponge saturated with hot water, or one with cold water, over the affected region. It is not, however, so intense in character as that attendant on meningitis.

The limbs below the seat of the inflammation are likewise affected with pains, which are mainly confined to the trunks of the nerves coming from the affected portion of the cord.

A pain is also experienced, in the great majority of cases, at the upper limit of the inflammation, and which extends around the body at that height. It is generally accompanied by a feeling of constriction similar to that which would be produced by a cord tightly tied around the body. It is probably caused by spasm of the abdominal or thoracic muscles.

None of these pains are increased by movements of the limbs or of the vertebral column, in which respects they differ from those met with in spinal meningitis.

In addition there are various derangements of the cutaneous sensibility in those parts of the skin below the seat of the disease, and especially in those parts supplied by nerves originating from the affected segments of the cord. These consist of formication, "pins and needles," a sensation as if water were trickling over the skin, as if the limb were asleep, and of sensations of cold or heat. Anæsthesia is the most common general condition of the skin, and it is often accompanied with cutaneous pains, which are the more intense the more profound is the anæsthesia. Thus, if we have ascertained that the cutaneous sensibility is very much impaired at a particular spot, we will frequently find this spot the seat of severe and spontaneous pains. In such cases, too, a prick with a pin is felt, but the ability to distinguish the two points of the æsthesiometer is lost, even when they are widely separated. Indeed, they may not be felt at all unless they are so used as to cause pain. I have several times observed patients whose tactile sensibility was almost entirely gone, but whose sensibility to pain was so great that they could not endure the contact of the bedclothes. The distinction, therefore, between insensibility to touch—generally called anæsthesia—and insensibility to pain—analgesia—must be clearly made.

Hyperæsthesia is occasionally present, but probably not unless there is meningitis associated with the myelitis.

Motility is affected at a very early period of the disease, and at first consists of simple twitchings of the muscles, and paralysis. The latter comes on with great rapidity, and may become complete in a few hours. Jaccoud[1] states that he has seen this result produced in thirty-six hours, and Ollivier[2] cites several cases to the same effect. The seat of

[1] Op. cit., page 318.

[2] Op. cit., chap. huitième, Myelite, ou Inflammation de la Moelle épinière.

the paralysis is in those parts of the body below the diseased portion of the cord. Thus, if the lower dorsal, or lumbar region be affected, the lower extremities, the sphincters of the bladder and rectum, and certain of the abdominal muscles, are involved; when the difficulty is in the upper dorsal or cervical region, the upper extremities, and, if the lesion is high enough, the diaphragm and other muscles of respiration, with those concerned in effecting deglutition, are paralyzed. Ocular troubles may also be present in cervico-dorsal myelitis, from irritation of the sympathetic nerve. These consist of dilatation of one or both pupils, and of exophthalmos.

It is not often the case that the homonymous muscles are equally paralyzed. Thus it is not uncommon to find one lower extremity entirely deprived of motility, and the other more or less possessed of its normal amount of power. The bladder is sometimes paralyzed, and sometimes not, and the same is true of the vesical and anal sphincters. Reflex power is generally exaggerated in the very first stage, and greatly diminished or altogether abolished subsequently. In a few cases I have seen tickling the sole of the foot fail to excite reflex movements in the lower extremity to which it belonged, but induce strong movements in the opposite leg. A similar fact has been observed as regards sensation; an impression made upon the skin of one foot, for instance, not being felt in that foot, but causing pain in the opposite foot.

Electro-muscular contractility is diminished unless, perhaps, in the very earliest stage of the affection, and there is almost invariably a tendency to rapid atrophy of the paralyzed muscles. Besides the foregoing symptoms, there are usually others referable to the viscera, and which differ according to the seat of the lesion. Thus, if the inflammation be seated high up in the cord, the actions of the respiratory muscles, of those concerned in deglutition, and of the heart, are affected. There may also be vomiting, derangement of

the liver, of the kidneys, intestines, and generative organs. The urine is often, if not invariably, alkaline, and there are frequent, and sometimes constant, painful erections.

The temperature of the paralyzed limbs always falls, and there is a strong tendency to the formation of sloughs and bed-sores, which frequently cannot be obviated. I have seen, in several instances, large sloughs form on each side of the vertebral column within twenty-four hours of the inception of the attack of acute myelitis.

Acute myelitis ordinarily runs its course in about three weeks, either, as is generally the case, terminating in death by asphyxia or exhaustion, or passing into the chronic form of the disease. It may, however, end fatally in a few days. In one case under my charge, occurring in a child ten years of age, and the result of pressure from caries and necrosis of the vertebræ, death took place in fifty hours. In another, due to a blow, a fatal termination was reached in forty-five hours.

Causes.—Acute myelitis is more frequently the result of injury than of any other cause. It is likewise a sequence of disease of the vertebræ, extending to the dura mater and other membranes, and of meningitis. It is also said to be produced by exposure to extreme heat or cold, by violent muscular efforts, and by venereal excesses. Seven cases have come under my observation. Of these, three were the result of wounds, two ensued on disease of the vertebræ, and two were caused by extension of acute meningitis.

Diagnosis.—The principal diagnostic marks of acute myelitis are the occurrence of the sensation of constriction around the body, the alkalinity of the urine, the rapid supervention of the paralysis, the great predisposition to sloughs wherever there is the least pressure, the excitation of pain in one part of the body by irritation applied to some other part, the causation of reflex movements in a similar way, and the marked depression of temperature in the paralyzed parts.

The absence of the characteristic symptoms of congestion and of meningitis will suffice likewise to separate it from these affections.

Prognosis.—The termination of acute myelitis is in death sooner or later. Even if it passes into the chronic stage, the alterations in the structure of the cord are so extensive as to be incompatible with the performance of its functions. Death was the result in all the cases that I have personally observed, and this event occurred in all within three weeks.

Morbid Anatomy and Pathology.—The morbid action in cases of acute myelitis may be limited to the white substance or to the gray substance, or may attack both these tissues. It may likewise affect the antero-lateral columns, the posterior, or extend to both. Undoubtedly, if we had sufficient opportunities to witness cases of spontaneous origin not the result of traumatic causes, or of the extension of other diseases, we should be enabled to distinguish by the symptoms which part of the cord histologically or topographically is affected. For there can be no doubt that, as in anæmia, or as we shall see hereafter in the chronic form of myelitis, the symptoms must be as characteristic as are the functions of the several histological and regional parts of the cord.

As regards the obvious morbid anatomical features, we find that when the lesion is situated in the white substance the membranes of the affected portion are congested, thickened, opaque in patches, and adherent to the cord. The cord is softened to a variable depth, and this portion is detached with the membranes if these be removed. This softened portion is in the early stage rose-colored and studded with red points, marking the situation of the enlarged blood-vessels. As the disease advances, the color deepens to a reddish brown, then begins to get lighter, and, passing through several shades of yellow, eventually becomes white.

When the gray substance is involved, the changes in its

physical appearance are similar; and, when both the white and the gray are the seat of the morbid process, it is impossible to distinguish the two substances from each other.

Microscopical examination shows the existence of congestion, and, as an essential feature, an increase in the amount of connective tissue or neuroglia of the cord. The evidences of this hypertrophy are seen in the increase of fusiform cells and in the production of multinuclear cells and free nuclei. These formations take place at the expense of the proper nervous tissue of the cord, the anatomical elements of which undergo atrophy and fatty degeneration. The nervous tubules are thus often disintegrated and their contents disseminated through the extraneous tissue. The axis cylinders are entirely surrounded by oil-globules, or are altogether broken up and rendered unrecognizable.

Should suppuration occur, the elements of pus are observed among those already described, and take their place to a considerable extent.

In case of the passage of acute myelitis into the chronic form, the centre of inflammation usually undergoes other changes, which, however, still maintain the general characteristic of hypertrophy of the neuroglia at the expense of the proper nervous tissue. Induration, or, as it is now generally called, sclerosis, is the result. Occasionally, however, the softening persists and becomes the permanent structural condition of the diseased portion of the cord.

When the lesion is in the gray substance, the microscope shows the nervous cells to be broken up, and the anatomical elements of the blood to be scattered through the tissue.

Treatment.—The treatment of acute myelitis offers no encouraging features. The most that can be done is to endeavor to prevent, as far as possible, the formation of sloughs, by placing the patient on a water-bed, and by sponging the parts exposed to pressure,with whiskey or with hot and cold water alternately applied. The treatment gen-

erally does not differ from that recommended in acute meningitis, the indications being almost identical. So far as my experience extends I have never found any means sufficient for cure, and the few successful instances that have been reported are doubtless, as Jaccoud suggests, cases of congestion or meningitis.

LEEDS SCHOOL OF MEDICINE

CHAPTER VI.

SPINAL SOFTENING.

Softening of the spinal cord is, as we have seen, the common termination of acute myelitis, in which connection it has been sufficiently considered; but it may originate primarily, and in that event possesses a clinical history very distinct from that of acute inflammatory softening.

Symptoms.—The first symptom usually noticed in softening of the spinal cord is numbness in those parts of the body below the seat of the lesion. Soon after the occurrence of this symptom there is weakness of the same parts, and then the deficiency of sensation and the feebleness of motor power advance together, both gradually becoming more and more strongly marked. There are no muscular twitchings, no contractions of the limbs, no pains either at the seat of the disease or in the paralyzed limbs.

The bladder very soon becomes involved, and the patient finds that, when he attempts to urinate, the stream is not so strong as it once was, and that he is obliged at times to use the expulsive force of the abdominal muscles in order to complete the evacuation of the bladder. Gradually the contractile power of this viscus becomes less, and finally is altogether lost.

The sphincter generally participates. The desire to urinate becomes more frequent, and when the inclination is felt the patient must at once yield to it. Eventually the bladder likewise becomes entirely paralyzed, and then there

is neither the ability to expel the urine nor to retain it, and consequently it dribbles away constantly.

Sometimes the first evidence of softening of the cord is perceived either in the bladder or its sphincter, and it may be restricted to these parts for a considerable period. I have a patient at the present time (February 9, 1871) under treatment for what I have no doubt is softening of the cord, and in whom the bladder-troubles were the only notable symptoms for over two years.

The intestines are similarly affected, and the bowels are either obstinately constipated or the sphincter ani is relaxed, leading to fecal evacuations as soon as the contents reach the rectum.

Reflex excitability is weakened from the first, and gradually disappears, unless, as is rarely the case, the gray matter be unaffected.

The progressive advance of the disease reduces the patient to a condition of utter helplessness. He is unable to walk, sensation is abolished in the paralyzed limbs, his urine and fæces are passed involuntarily, bed-sores occur, the venereal appetite is extinct, or, if it should remain, erections are impossible, and the parts of the body below the seat of the disease are to all intents and purposes cut off from communication with the parts above. This condition may last for years without a fatal termination ensuing, but intercurrent affections, especially resulting from the bladder-difficulties, may eventually cause death.

Such is the course of spinal softening when the lesion is low down and involves both antero-lateral and posterior columns. When it is higher up, the symptoms are also referable to the thoracic extremities, and to the muscles concerned in deglutition and respiration. There are likewise visceral disturbances.

When the lesion mainly affects, or is confined to the antero-lateral columns, the symptoms manifested are in intimate relation with the known physiological functions of the

region in question. Thus the power of motion in the limbs below the softened portion of the cord gradually becomes less, the gait is from the first staggering, and though even at a late stage the patient may be able to move his limbs while lying down or sitting, he cannot support the weight of his body upon them. When he tries to stand without extraneous aid, it is seen that he is especially weak in the knees and ankles. There is no more difficulty in standing or walking with the eyes shut than when they are open.

This paralysis of motion, in which the bladder generally participates, may be of the most profound degree, and yet sensibility be perfect. A gentleman was under my care in whom I diagnosticated softening of the cord in that part extending on the right side from the second dorsal vertebra downward probably as far as the fourth sacral, while on the left side it began at about the fourth lumbar and extended downward probably as low as the fourth sacral. I gave the lesion these topographical limits for the reason that on the right side the muscles supplied by the crural and sciatic nerves had lost their electro-muscular contractility, while it certainly did not extend above the origin of the ilio-hypogastric nerve, as the lower part of the rectus abdominis, which receives its motor power through this nerve, retained its contractile power. On the left side the muscles supplied by the crural were possessed of their normal motor power, while those supplied by the sciatic had lost their contractility. It was, therefore, very certain that on this side the lesion did not extend above the fourth lumbar, the lowest spinal nerve contributing to the formation of the crural.

I was able also to restrict the morbid process entirely to the antero-lateral columns, for in no part of the skin below the upper supposed limit of the lesion was there any loss of sensibility. The least impression made upon the skin was felt. Tickling the sole of the foot excited laughter, but no reflex movements. I was therefore able to determine that the gray matter was involved. The bladder was paralyzed,

and its sphincter likewise. The sphincter ani was also deprived of its contractile power to a great extent.

The patient died at Cape May, and I had no opportunity of making a post-mortem examination. There can be no doubt, however, that the lesion was essentially that which I have described. In all cases of spinal softening involving the antero-lateral columns, the electro-muscular contractility is soon lost, so that even the strongest induced or primary currents fail to cause contractions.

As regards the implication of the posterior columns, there is an equal facility for determining the fact from a consideration of the symptoms. The functions of these columns are intimately connected with sensation, and when such a morbid process as softening is set up in them the symptoms are those which indicate impairment of the cutaneous and muscular sensibility. Thus, in a gentleman now under my charge, there has been going on for several months a morbid action in the spinal cord unattended by any prominent symptoms except anæsthesia. There has never been pain or any derangement of motility, but simply a gradually-increasing loss of sensibility in both lower extremities and in all the other parts of the body below the upper limit of the seat of the lesion.

He is unable to walk in the dark or with his eyes shut, or to stand alone with his eyes closed and his feet close together, for he obtains no idea of his position unless he can have the aid of his eyes or hands.

He has full power over the bladder and voluntary control over its sphincter and that of the rectum, but he never experiences the desire to urinate, does not feel the flow of urine through the urethra, nor the passage of the fæces through the anus, and evacuates his bladder and bowels at stated periods merely from the knowledge acquired by experience that it is time to do so.

Examination with the æsthesiometer shows that the upper limit of the lesion on both sides is in that part of the

cord from which the second lumbar nerves are derived, for the loss of sensibility is apparent in all those parts supplied by the crural and sciatic nerves, both as regards the skin and the muscles. Very weak faradaic currents cause muscular contractions, but the strongest which it is possible to obtain from a powerful machine produce no pain.

In this case there is, I think, ample reason to diagnosticate a lesion of the posterior columns without any implication of the antero-lateral. The reasons for believing this lesion to be softening will be indicated under the head of diagnosis.

Causes.—The causes of spinal softening are not very clearly understood. Doubtless it arises as a consequence of acute myelitis, but it is often an independent and apparently a primary affection, being unpreceded by any obvious symptoms indicative of spinal derangement. Such influences as give rise to cerebral softening will, in all probability, cause spinal softening, and among them must be placed obliteration of blood-vessels from embolism and thrombosis. The actual occurrence of occlusion of spinal vessels from either of these causes has not, however, so far as I am aware, been demonstrated. The further etiology of spinal softening is not as yet a matter of any certainty, though I think several cases that have been under my observation could reasonably have their cause laid to excessive sexual indulgence.

Diagnosis.—The diagnostic marks of most value in cases of supposed spinal softening are the absence of sensory and motor excitement. Thus there are no pains referable to the back or other parts of the body, no hyperæsthesia, no twitchings, no spasms, no contractions, no exalted reflex actions. And this is the case in that form of the disease involving the whole thickness of the cord, or in either of those limited to the anterior or posterior columns. There is no other affection of the spinal cord which is not characterized, at some time or other of its progress, by irritation either of the sensory or motor nerves, or of both, excepting some cases

of spinal anæmia giving rise to the categories of symptoms previously considered. The clinical history of such cases, and the comparatively light character of the phenomena, will serve to distinguish them from those in which the lesion is softening.

Prognosis.—The prognosis is always unfavorable as regards recovery and complete restoration, but spinal softening is not necessarily a fatal disease. At least I have seen cases which had existed for many years, and which apparently had no elements of a fatal termination about them. But they were instances in which the seat of the disease was in the lower dorsal, or lumbar or sacral region of the cord. When it is higher up, the prospect of death ensuing is more probable. The restoration of the cord to its normal structure is impossible, and the patient lies paralyzed either in sensation or motion, or both, according to the situation and extent of the lesion, in a condition similar to that of a person who has received a wound inflicting irreparable injury on the cord. Such persons, as is well known, frequently live for many years afterward—then die of some entirely different disease. There is nothing about spinal softening calculated to produce exhaustion, unless it be the tendency which exists to cystitis from paralysis of the bladder, and the consequent inflammation liable to be set up from the action of the retained urine. Care, however, will very greatly diminish the danger from this source. I have had a number of patients under my charge who had not, for many years, had a passage of urine from the bladder which was not effected with the catheter, and they had, in all that time, suffered no marked inconvenience.

Morbid Anatomy and Pathology.—The appearance of a softened portion of the spinal cord to the naked eye has nothing very peculiar about it. When examined as to its consistence, it is seen to be sometimes as soft as cream, at others scarcely altered in the resistance which it offers to the touch. In the first instance, when the lesion involves

the gray and white matter together, section does not show the peculiar double cresentic arrangement of the former tissue, but it appears to be blended homogeneously with the white substance which surrounds it.

Microscopically it is seen that the nervous tubules constituting the essential anatomical elements of the white substance are broken up, and no vestige of them remaining in extreme cases—oil-globules and bodies called granule-masses, the constituent of which is fat, having taken their place. In the gray substance the nervous cells are destroyed, and oil and fat have made their appearance in large amount. Even the neuroglia or connective tissue of the cord exhibits a similar disintegration and regressive metamorphosis. These changes impair the functions of the cord, both as a nervous centre and as a structure serving for the transmission of sensory impressions to the brain, and of nervous force from it. When the disintegration is complete, the effect is the same as if the cord had been entirely divided by a cutting instrument.

Treatment.—There is nothing to be done which can by any possibility restore the integrity of the spinal cord after the process of softening has fairly entered upon its course. In the very early stages, if patients apply for treatment at these times, something may perhaps be accomplished by the use of phosphorus and strychnia, but the symptoms come on so insidiously and gradually that the subject of them rarely has his apprehensions excited till it is too late to do any thing toward arresting the disease. And even when we do see cases which in appearance exhibit the symptoms met with in spinal softening in its initial stage, and which recover under treatment, there must always be a doubt in regard to the accuracy of the diagnosis—for many cases of temporary anæsthesia and impairment of motility are due to anæmia of the cord, the result of the causes set forth in a previous chapter.

The patient, however, may be made comfortable to such

an extent as to materially prolong his life. Care should to this end be taken that he does not sustain a fall or suffer an injury whereby the diffluent portion of the cord would be disturbed in its anatomical relations, and the danger of an attack of acute meningitis or of myelitis incurred. Bed-sores should be prevented, or, if they occur, treated according to the methods previously mentioned, and full instructions should be given in regard to emptying the bladder with the catheter at regular times, and of going to stool at the same hour every day. Locomotion may be provided for by some one of the chairs devised for the use of paraplegics.

CHAPTER VII.

SCLEROSIS OF THE ANTERO-LATERAL COLUMNS OF THE SPINAL CORD.

Sclerosis affecting the antero-lateral columns of the spinal cord, whether it be diffused, multiple, or cortical, gives rise to symptoms which do not vary essentially with the form. It will therefore be advisable to consider them all under the present head, pointing out in the course of the description such distinctions as can be shown to exist.

Symptoms.—In cases of sclerosis limited to the antero-lateral columns, there are no aberrations of sensibility either in excess or diminution, but as it very generally happens that there is more or less meningitis, pain may be experienced, or there may be different degrees of anæsthesia from the posterior roots of the spinal nerves being implicated. From the same cause acting on the anterior roots, twitchings and jerkings of the muscles and limbs below the seat of the lesion will be produced. The existence of pain, or of muscular contractions, in the early period of the disease is, therefore, always indicative of the fact that the membranes are involved in the morbid process.

So far as the cord itself is concerned, the first symptom is loss of muscular power. At first the patient merely tires more readily, slight exertion fatigues him, and this is especially noticed in the muscles which flex the leg on the thigh, and the consequent sensation of weariness experienced in the popliteal space. Sometimes it is shown in the sudden relaxation of the extensor muscles of the leg, and the fall of the

patient thereby; at others, in the fact that the extensors of the foot become weak, allowing the toes to drop, and hence causing stumbling. The gait then becomes characteristic. Owing to the fact that the patient's extensor muscles are weak, he is unable to lift the feet high enough to cause them to clear the ground, and hence he throws them out by means of the abductor muscles of the thigh, and thus causes them to describe an arc of a circle. Then in putting them down the heel strikes the ground a longer time before the sole than it does in the natural gait, and hence the foot comes down with a jerking motion. This is the ordinary manner of walking practised by a person affected with the disease under notice. In another form of locomotion, the body is moved laterally on the thighs, first to one side and then to the other, in such a way as to cause the feet to be raised high enough without the complete action of the extensor muscles. The gait is therefore similar to that of a duck, or of a woman with a very wide pelvis. In a patient now in the New York State Hospital for Diseases of the Nervous System, this method of progression is very strongly marked. The motion of the body is almost serpentine, and the feet glide over the ground barely lifted high enough to avoid contact.

In both the methods of walking the patient requires support. At first a cane answers, then he comes to crutches, and eventually the assistance of an attendant becomes necessary.

As a consequence of the paralysis, the movements are often complicated and sometimes rendered impossible by the legs becoming interlocked at every attempt to walk. In a patient from Connecticut under my care, not long since, this difficulty was a very prominent feature, and though the muscles of flexion and extension were sufficiently strong to allow of his walking, those which abduct the thighs were so materially paralyzed as to produce the condition mentioned.

Reflex movements, so far from being lessened, are gener-

ally exalted, and this for the reason that sclerosis rarely affects to much extent the gray matter of the cord. Sometimes this exaltation is so notable a feature as to cause great inconvenience. A gentleman now under my charge, with sclerosis of the antero-lateral columns, has no contractions of the limbs or even twitchings of the muscles, unless through the influence of an irritation applied to them. He has no voluntary power whatever over the lower extremities, and yet the slightest touch on the sole of the foot or side of the ankle is sufficient to cause powerful reflex movements in either leg. In another, in which the disease is not confined to the anterior columns, but which likewise extends to the posterior, the mere contact of a straw with the sole of the foot—an irritation not felt, for there is plantar anæsthesia —causes contractions to such a degree as to flex both the leg and the thigh to the utmost possible extent.

The electro-muscular contractility is also usually increased. Under the influence of mild induced currents, every individual muscular fasiculus can be seen to contract, and, as the layer of fat under the skin is generally absorbed at an early period of the disease, the bundles of fibres can be very distinctly made out.

But, although in the early stages of the disease there are no muscular contractions unless there is irritation of the anterior roots of the spinal nerves coming from the affected portion of the cord, at a late period there may be involuntary movements, due entirely to the sclerosis. These are the result of the implication of the fibres of origin of the nerves in the morbid process, and consist of fibrillary contractions, twitchings of whole muscles, or jerkings of the limbs. They are manifested without being set into action by voluntary movements, and therefore differ from the twitchings observed in cases of cerebro-spinal sclerosis, and which are due to the association of the pons Varolii or other cerebral ganglia in the disease. Tremor is, therefore, never observed in spinal sclerosis of any form, diffused, multiple, or cortical,

unless the pons Varolii or superior ganglia of the brain are implicated. In the only case of this latter form published —that of Vulpian[1]—the sclerosis extended throughout the whole length of the cord, and likewise involved the pons Varolii, cerebellar, peduncles, and other intracranial organs, besides being accompanied by well-marked spinal meningitis. The tremor observed at a late period of the disease cannot, therefore, be ascribed to the lesion of the cord below the medulla oblongata.

The relations of tremor to the other forms of spinal sclerosis will be further considered under the head of pathology.

Another symptom, indicating the extension of the morbid action to the origins of the nerves, is atrophy of the muscles deriving their nervous influence from nerves originating at the diseased portions of the cord. In the cortical form of the disease, we should not expect this symptom to be present, for the lesion affects only the superficial layer of white substance, nor is it for the same reason apt to exist in cases of multiple sclerosis of the antero-lateral columns. It is—and the same may be said of muscular contractions—almost peculiar to the diffused form of the affection.

The bladder is often paralyzed, and hence the patient is obliged to use the catheter for the evacuation of the urine. The sphincter becomes involved at a later period.

But it frequently happens that the bladder and sphincter both escape. A number of such cases have been under my charge, and in one still being treated, which has lasted several years, and in which both lower extremities are entirely paralyzed, the bladder and sphincter retain full power. In those cases in which they are not implicated, the morbid action does not penetrate deep enough to involve the origins of the nerves.

The bowels are usually obstinately constipated at first,

[1] Note sur un cas de Méningite spinale et de Sclérose corticale annulaire de la Moelle Épinière, Archives de Physiologie, No. 2, 1869, p. 279.

and throughout the whole course of the disease till toward the close, when incontinence from paralysis of the sphincter ani takes place.

Sclerosis of the antero-lateral columns of the spinal cord is not a rapid affection, although its course is ordinarily progressively onward. Occasionally there are well-marked symptoms, and it often happens that medication produces, for a while, favorable results. The duration of the disease is from about two to five years. Cases have been reported in which death ensued within a few months, and I have a case now under my notice in which it has lasted twelve years, and in which, even now, the patient can walk tolerably well. Death takes place either from the extension of the morbid action, or from some intercurrent affection.

The symptoms, of course, vary with the position of the lesion. When the upper region of the spinal cord is affected, the superior extremities, and the parts of the body supplied with nerves from the diseased portion of the cord, participate. In a case which I first saw in consultation with my friend Dr. Walter F. Atlee, of Philadelphia, and which was subsequently for a long time under my immediate charge, the lesion was, in the beginning, confined to the very lowest part of the spinal cord. Gradually the disease extended upward, or new centres of sclerosis were formed, until at last, after three years, the muscles of deglutition and of respiration became implicated, and death took place. But for several months before this the patient was unable to use either legs or arms, or even to sit up. At no time, however, was the bladder deranged in any respect, and at no time were there pains or spasmodic action of the muscles. The cutaneous sensibility was scarcely affected, and there was no atrophy beyond that due to long-continued inaction of the muscles. No post-mortem examination was made, but there can be little if any doubt that the lesion was comparatively superficial, and that it was almost entirely confined to the antero-lateral columns of the cord.

In a similar case occurring in a distinguished legal gentleman of New Orleans, sent to me by my friend Dr. Cabell, of the University of Virginia, there is a gradual extension of the disease upward without any attendant pains, anæsthesia, or muscular contractions, except to a slight extent at first. In this instance, also, the bladder and rectum have escaped. That the lesion is superficial, and confined to the antero-lateral columns, I am very sure.

In another case, that of a gentleman from New Jersey, there is a similar condition, but with the additional phenomenon of muscular atrophy.

Such cases as the foregoing, and several others which have come under my notice, are doubtless to be classed with many of those placed under the head of what Duchenne[1] has called general spinal paralysis—while others have been referred to softening of the antero-lateral columns of the cord.

Causes.—Although sclerosis of the antero-lateral columns of the spinal cord may be due to an antecedent attack of acute myelitis, or one of meningitis, there is no doubt it is often a primary disease.

Sex is certainly a predisposing cause; probably, however, acting secondarily, from the fact that difference of sex almost necessarily implies difference in mode of life. Males are much more liable to the disease than females. Of the forty-eight cases that have been under my care, or in which I have been consulted during the past six years, forty-two were males.

Age is also influential. Of the forty-eight cases, all were over the age of twenty-five, and none were over sixty. One was over fifty-five, three between fifty and fifty-five, seven between forty-five and fifty, and all the rest under forty-five, when the disease first manifested itself.

[1] Paralysie générale spinale. De l'Electrisation localisée, etc. Paris, 1861, p. 258.

Hereditary influence is undoubted in many cases; so far, certainly, as it is exerted by the ancestors having suffered from some form of nervous disease. In other instances there is the transmission of a direct tendency to the disease in question by some no very distant ancestor having had the same affection.

Among the exciting causes are the excessive use of alcoholic liquors, inordinate sexual indulgence, exposure to severe cold, blows on the spine, and the gouty, scrofulous, or syphilitic diathesis. In the great majority of cases the cause is not apparent.

Diagnosis.—There is rarely much difficulty in recognizing sclerosis of the antero-lateral columns of the cord; for, as will have been perceived, the symptoms are well marked. The only affection with which it is really liable to be confounded is softening of the same region; and here the fact that sclerosis is almost always accompanied by meningitis, and that therefore there is in the first stage evidence of sensory and motor excitation, will generally suffice to render the diagnosis accurate. The gait of a person affected with sclerosis is, moreover, very different from that of one suffering from softening; the course of the disease is slower, and the paralysis rarely so profound. Other diagnostic marks will be perceived from a consideration of the symptoms of the two forms of chronic myelitis as I have described them.

Prognosis.—The prognosis is always grave; and, sooner or later, a fatal termination usually takes place. Still, great ameliorations are possible, and a cure is not impossible.

Morbid Anatomy and Pathology.—The essential feature of sclerosis occurring in the spinal cord is identical with that of the same condition affecting the cerebral hemispheres, and which has already been considered. It consists of hypertrophy of the connective tissue and atrophy of the proper nerve-substance. A part of the cord which has undergone

this change presents features which are easily recognizable. Its color is changed to a yellowish gray, and its consistence is much increased. At the same time there is a decided condensation, so that the circumference of the cord is lessened. As already stated, sclerosis appears in the spinal cord under three forms. In one of these, called diffused sclerosis—the *sclérose uniforme* of Jaccoud—the lesion occupies a considerable extent of the tissue, and may involve the whole thickness of the cord throughout its entire length. In another multiple or disseminated sclerosis—the *sclérose en plaques disséminées* of Charcot and the *sclérose diffuse* of Jaccoud—the lesions are several in number and are entirely isolated. In the third, cortical sclerosis—the *sclérose corticale annulaire* of Vulpian—the lesion is confined to the superficial layers of the cord. In whichever of these forms the lesions exist, they are marked by white striæ, which are the proper nerve-filaments, still presenting somewhat their characteristic color and consistence.

The membranes often exhibit evidences of inflammation, and are thickened, opaque in spots, or red in some cases, while in others they are adherent to each other and to the cord.

When submitted to microscopical examination, the sclerosed tissue is seen to consist mainly of an excessive amount of connective tissue—the neuroglia of Virchow. The cells are increased in size, and the nuclei are larger and much more numerous than in the normal condition. The capillaries are thickened, from the deposition on their walls of several layers of rounded cells.

The effect of this morbid process is to compress the nervous filaments and to cause their atrophy. The fluid portion undergoes fatty degeneration, and the axis cylinders become disintegrated.

In a case of sclerosis of the antero-lateral columns of the spinal cord, which was for a time under my care, I have

recently had the opportunity of making a careful examination of the diseased portions.

The patient, J. H., consulted me in the winter of 1869–'70. He was then unable to walk without a cane and the assistance of an attendant. He had previously been treated at a water-cure establishment, and more recently by the Swedish movement-cure, and of course without benefit. The symptoms were mainly connected with motility. Both lower extremities were paralyzed; the bladder was inactive, but not the sphincter, and there was obstinate constipation. There were occasional fibrillary contractions of the paralyzed muscles, and at times pain in the back and limbs—never, however, of any great degree of severity. There were no tremors, either with or without voluntary motions.

The patient obtained very little benefit from the treatment to which I subjected him, and I advised him to return to his home in Ohio. A few months afterward, he died.

The dorsal, lumbar, and sacral regions of the cord were sent to me for examination by his physicians, Drs. Ramsey and Bishop, of Delhi, Ohio. In a letter, the latter informed me that the vessels of the pia mater were injected.

The cord arrived in good condition, having been carefully preserved in strong alcohol. Upon inspection, the antero-lateral columns in the middle and lower dorsal regions to the extent of three and a half inches were seen to be of a grayish tint, which became deeper in shade from above downward. Below this, at the junction of the dorsal with the lumbar portion, was another patch two and a half inches in length, and also involving the whole superficies of the antero-lateral columns; and, separated from this by a portion of apparently healthy tissue, was another discolored, irregular patch, an inch and a half in length, along the left antero-lateral column; and, below this, a similar tract, two inches and an eighth long, involving the right antero-lateral column. The difference in consistence between these patches and the other parts of the cord was very decided,

and the white striæ were well marked. The sacral portion of the cord presented no abnormal appearance to the naked eye.

Sections of the cord were then made through the sclerosed portions; and it was seen that the gray matter was only involved where the horns approached the surface; and that, wherever a lesion existed, the normal contour of the sections was altered so as to make them sub-ovoidal, and thus to lessen the circumference. The greatest depth of any part of a sclerosed region was two-twelfths of an inch, and this was in the superior patch. The average thickness was about the one-twelfth of an inch.

The whole cord in my possession was then immersed in a solution of chromic acid in water, and left there for a month to harden. Immediately previous to examining with the microscope, the sections were colored by an ammoniacal solution of carmine. Under a twelfth-inch objective, it was seen that, throughout the whole extent of the sclerosed portion of any section, the nerve-tubes had entirely disappeared; and, wherever the gray substance was affected, the nerve-cells were diminished in number. In the place of these elements was connective tissue, a large quantity of molecules, and connective-tissue cells in great abundance.

In several sections taken from the dorsal, lumbar, and sacral regions, and which were apparently normal when viewed with the naked eye, the neuroglia was found to be in excess, and the nerve-tubes in a state of disintegration.

The gray matter, except in those sections made through the part where the sclerosed portion extended from the white matter to it, was uniformly healthy, and in no part were the posterior columns involved.

In this case there was no tremor, although it was clearly one of multiple sclerosis, probably entirely confined to the spinal cord. At no time had there been head-symptoms of

any kind. I think, therefore, that Dr. M. Clymer[1] is in error, where he states, under the head of "Disseminated Sclerosis of the Spinal Form," that "rhythmical spasms accompany any voluntary movement of the affected muscles." As previously stated, I am ot opinion that such movements are only present when the affection has also attacked the central ganglia—and, indeed, the cases cited by Dr. Clymer, in the appendix to his excellent memoir, abundantly sustain this view; for, of the sixteen cases tabulated by him, tremor is not stated to have occurred in any one in which the lesion was limited to the cord.

In the case of Dr. Pennock, reported by Drs. Morris and S. Weir Mitchell,[2] the sclerosed tissue was confined to the spinal cord and mainly to the lateral part of the antero-lateral columns. The posterior were involved to a very small extent. In this case there were partial anæsthesia, gradually advancing paralysis implicating all four extremities, and paralysis of the bladder. The intellectual faculties were never affected in the least. The course of the disease was progressively onward, and, though there was toward the last a total loss of voluntary power below the neck, reflex action remained unaffected. There were no tremors with or without voluntary movements. In regard to this case, Dr. Mitchell, who made the microscopical examination, remarks that there were:

"1. Integrity of mental and moral manifestations.

2. Absolute loss of voluntary motive power below the head, or rather below the neck.

3. Sensation nearly perfect.

4. Respiration good; reflex motion preserved and exhibited in the form of spasm or irritation of certain parts of the skin."

All of which are what we should expect to find in sclerosis almost entirely confined to the antero-lateral columns.

In the description of the disease as given by several au-

[1] Op. cit., p. 12. [2] American Journal of the Medical Sciences, July, 1868.

thors, great stress is laid on the occurrence of violent tonic contractions of the limbs. As the reader, however, will have inferred, these are not due to the sclerosis, but result from the meningitis which is often an attendant condition.

Treatment.—The indications are in the first stage to diminish the amount of blood in the vessels of the meninges and of the cord, and with this view, when I see a patient exhibiting the early symptoms of sclerosis, I prescribe large doses of ergot as recommended for spinal congestion. The results have been exceedingly favorable, and I think it very probable that by this treatment the advance of the disorder has been checked. But in those insidious forms of the affection not accompanied in the early stage by symptoms indicating hyperæmia, and in those cases in which the morbid process has apparently reached that stage in which there is proliferation of the neuroglia, ergot can be of no service. On the contrary, by lessening the amount of blood in the cord, and hence interfering with its nutrition, it is calculated to do harm. At this period I am inclined to think more benefit is to be derived from nitrate of silver, cod-liver oil, and the primary galvanic current, than from any other measures.

In Dr. Pennock's case it is stated that the water-cure was tried for a time with some temporary benefit, and Jaccoud declares that hydrotherapy and mineral waters are still the most efficacious remedies. My experience is not to this effect. Several of my patients had, before coming under my charge, made use of the means in question, but never with the least favorable result. Even the Turkish bath, which is so useful in some affections of the nervous system, does harm in this. I have in a number of instances seen all the symptoms aggravated by two or three such baths.

Counter-irritation I have employed repeatedly in all its forms, but never with the slightest benefit.

For the relief of any pain that may be present, codein in the dose of half a grain or more is to be preferred. Should

there be the least suspicion of syphilis, mercury and iodide of potassium, as recommended on page 322, should be used and continued for several weeks before it is deemed inefficacious.

The bladder should always be looked after if there is any paralysis, and the patient instructed to use the catheter at stated periods.

LEEDS SCHOOL OF MEDICINE

CHAPTER VIII.

SCLEROSIS OF THE POSTERIOR COLUMNS OF THE SPINAL CORD (LOCOMOTOR ATAXIA).

Although other writers, and especially Romberg,[1] had described a disease answering to that now generally known as locomotor ataxia, we are mainly indebted to Duchenne[2] for giving a full and distinct account of an affection which, before his studies, had scarcely attracted attention. In accordance with the plan pursued in the present work, of designating diseases as far as possible by the lesion, and not by the symptoms, I have decided to use the proper pathological term for the affection under question, even though it be not so familiar as the one usually employed.

Symptoms.—Posterior spinal sclerosis has no uniform set of initial symptoms. Sometimes it begins with dull, heavy pains in the small of the back or other part of the spinal column, which are very soon followed by sharp, electric-like pains, which shoot down the limbs along the course of the nerves, and which are very generally taken by the patient for twinges of neuralgia or rheumatism, or it may be first manifested by a feeling of constriction around the body like that which is so common in acute myelitis.

Again, the first symptoms are cerebral, and may consist of attacks of vertigo, epileptic fits, disturbances of vision, such as diplopia, ptosis, and defective accommodation.

[1] Lehrbuch der Nervenkrankheiten, Berlin, 1840; also Sydenham Society's Translation, London, 1853.

[2] De l'Ataxie locomotrice progressive, Arch. Gén. de Méd., 1858; also De l'Electrisation localisée, Paris, 1861.

At other times the stomach and bowels are disordered; vomiting is frequent, and there may be diarrhœa or obstinate constipation. Or, finally, the initial phenomena may be connected with the sensibility, giving rise to anæsthesia, and the various abnormal sensations connected therewith.

In whatever way it may begin, posterior spinal sclerosis is soon chiefly manifested by disorders of motility, but inquiry reveals the fact that these are in reality secondary, being dependent upon the diminished sensibility which always exists. As this is the essential feature of the disease, I propose to inquire into its characteristics at some length.

If the lesion, as it usually does, exists in the dorso-lumbar region of the cord, the first evidences of anæsthesia or of perverted sensibility are noticed in the feet. A common feeling is one as if the toes are too large for the shoe, or as if pieces of some plastic material are between them. Sometimes there are burning pains in the soles of the feet, and very generally "pins and needles" and other forms of numbness. A curious symptom is that, not only is the sensibility lessened, but the transmission of sensitive impressions to the brain does not take place with the normal degree of activity. I have noticed this phenomenon in rather more than half the cases that have come under my observation. In a lady, now a patient, a pin stuck into the calf of the leg is not felt for fourteen seconds on the right side and sixteen on the left. In a patient with posterior spinal sclerosis, under treatment in the New York State Hospital for Diseases of the Nervous System, if the feet are put into hot water the sensation is not felt for almost three minutes. As he said, "My feet might be scalded till the flesh dropped off and I would not know it till the mischief was done. Then I should feel it sharply." The explanation of this symptom is to be found in the fact that the conducting power of the posterior columns is lessened by the lesion, and hence the brain does not receive in the usual time the impressions made upon the nerves.

The ability to feel pain is therefore diminished, but there is, besides, a marked abatement of the tactile sensibility. The extent of this can only be accurately measured by the æsthesiometer. When this instrument is used, we find that the two points can be widely separated and a single impression only be felt on parts of the body which in the normal condition would give the sensation of two points at a much less distance apart. A gentleman from Virginia consulted me recently, in whom I diagnosticated posterior spinal sclerosis, and who, instead of being able to perceive the two points with the end of the index-finger, when the twelfth of an inch apart, could feel but one point, though the two were separated to the extent of an inch and a half.

This loss of sensibility gives rise to some curious sensations, especially in the soles of the feet. These are usually such as might be produced by the interposition of some substance between the foot and the shoe, or between the shoe and the ground. One patient feels as if he has cushions on the soles of his feet, another as if bladders of air are interposed, another as if he is constantly treading on sticks, or, if riding in an omnibus, as if the hem of a lady's dress had got under his feet, and one a short time since described the sensation to me as being like that which he thought he would feel if his feet had been dipped into tar, and then into sand.

In some cases the ability to distinguish differences of temperature, or to appreciate the sensations produced by the application of hot or cold bodies to the skin of the affected parts, remains, but this is not, as some authors assert, a constant phenomenon, for in the majority of cases the sensations produced by heat or cold are just as unappreciable as those caused by any means capable of giving rise to sensitive impressions.

But the symptoms by which a person with sclerosis of the posterior columns of the spinal cord is recognized most readily are those which relate to motility, and these phe-

nomena often make their appearance at a very early stage of the affection. At that time there is no loss of motor power, but there is an inability to coördinate the muscles—to bring them into harmonious action, and thus to execute with precision the various voluntary movements. Thus, in the act of standing, a great many muscles are simultaneously made to contract, and each one to just that necessary degree which is essential to maintaining the body in the erect posture. Very often the first evidence of any motor difficulty is experienced in regard to this faculty of standing. This difficulty is, however, not one of paralysis, for, if the patient looks at his feet, he has no more trouble in standing alone than a perfectly sound man.

A gentleman connected with the city government of Brooklyn consulted me a short time since for an affection which was very evidently posterior spinal sclerosis. The first indication of disease, as he informed me, was that it had been his habit, while at his morning ablutions, to shut his eyes, and he had noticed, about two months previously, that when he did so he could not maintain his equilibrium. When he visited me he was unable to stand with his eyes shut, and his gait was perfectly characteristic of posterior spinal sclerosis.

Before the locomotion of the patient becomes obviously affected, he experiences inconvenience in placing his feet upon small surfaces. Thus, when he attempts to enter a carriage, he finds it difficult to guide his foot to the step, and in mounting a horse he cannot readily hit the stirrup. A gentleman from Maryland, who is now a patient of mine, and who is affected with the disease in question, tells me that among the first symptoms which he noticed was the difficulty he experienced in putting his foot into the stirrup. He was obliged to use his hand as a guide. A like trouble is frequently experienced in ascending a staircase.

The gait of a person suffering from sclerosis of the posterior columns of the spinal cord is very much changed from

that which is natural. Instead of the foot being placed upon the ground with an easy motion, the heel a little in advance of the sole, and the latter gliding down gently, the leg is, as it were, jerked forward, the heel comes down suddenly, and the sole follows, at a considerable interval, with an abrupt flapping motion. In ordinary walking the placing of the foot on the ground consists of one movement—there being no stoppage between the touching of the ground by the heel and the planting of the sole of the foot; but, in the gait of a person affected with posterior spinal sclerosis, the foot is placed on the ground by two distinct movements, one for the heel and another for the sole of the foot.

But, besides these irregularities of the progressive movements, there are others which are likewise notable. The leg is not carried directly forward, but is thrown out a little from the median line, and this gives the patient a motion like that of one walking on a tight-rope, and balancing himself with a pole. The object of this movement is doubtless to widen the base, and thus to enable the patient to preserve more readily his centre of gravity within it. In standing, he, for the same reason, always separates the feet to a greater than normal distance.

In walking or standing, it will be observed that the patient affected with posterior spinal sclerosis keeps his eyes fixed on his feet, or on the ground a little distance in advance. He is obliged to do this for the reasons—which with others will be more fully considered under the head of pathology—that the sensibility of the soles of the feet being diminished, and the muscular sensibility being also lessened, he is deprived, to a great extent, of the chief means by which he was formerly enabled to recognize the position of his feet, and of the dynamic condition of his muscles. He hence is obliged to make use of another sense—his vision—in order to obtain the necessary information. Therefore, when he shuts his eyes, or is obliged to walk in the dark, he is deprived of the assistance of his eyesight, and, having only his

diminished tactile and muscular sensibility to guide him, moves in an exceedingly timid and disorderly manner, or else is unable to walk at all.

Under some circumstances he is unable to go forward, even with the assistance of his eyesight. Experience has taught him that he cannot rely on very important senses which formerly he implicitly trusted. He loses confidence in them, and is not reassured, even with vision to assist him. He therefore uses extraordinary caution in walking over a tiled floor, on the ice or snow, in descending a staircase, or in crossing a street crowded with vehicles. In a recent clinical lecture[1] delivered to the class of the Bellevue Hospital Medical College, I called special attention to this phenomenon of loss of confidence, and adduced several cases in illustration of this point.

That there is little paralysis of motion to account for these abnormalities, can be readily shown by a few inquiries and experiments. Thus it will ordinarily be found that the patient who is unable to stand with his eyes shut or take a step in the dark, can push strongly with his legs, or walk a short distance with a good deal of vigor. He is still good for a "spurt," but long-continued muscular effort fatigues him.

When the lesion is above the origin of the nerves which go to form the brachial plexus, the upper extremities are the seat of symptoms which are similar to those described as manifesting themselves in the legs. There are numbness and other indications of anæsthesia, together with more or less difficulty in coördinating the muscles into harmonious action. The patient finds that the ends of his fingers have lost, to some extent, their acute tactile sensibility, and that there is restraint in the management of the fingers. He experiences these difficulties in buttoning his clothes, in picking up a pin, in writing, and in other actions requiring

[1] Clinical Lectures on Diseases of the Nervous System. JOURNAL OF PSYCHOLOGICAL MEDICINE, January, 1871.

nice manipulation. If he attempts, for instance, to carry a glass of wine to his lips, he spills a portion of the contents; and, if told to place his finger on any particular part of his face, the movement is accomplished with a wabbling motion, and the finger is darted suddenly to the part as it approaches it. All persons possess a knowledge of where the different parts of their bodies are situated, which does not depend upon the sense of sight, although probably acquired by that sense and experience. There is such an intimate and exact relation between the ends of the fingers and the cutaneous surface of the body that, if a spot no bigger than the head of a pin be made with a pencil on the forehead, a person can close his eyes and touch it with the end of his finger without difficulty every time he makes the attempt. He can also, with the eyes shut, carry the end of his fingers straight to the tip of his ear, the middle of his eyebrow, or any other part of his body within reach. A person, however, laboring under sclerosis of the posterior columns of the spinal cord, cannot do any of these things. He loses, at a very early period of the disease, that intimate topographical relation which exists between the ends of the fingers and the rest of the body; and hence, when he closes his eyes, and attempts to put the tip of his index-finger on the end of his nose, he misses his aim, sometimes by as much as two or more inches.

As in the legs, when the lesion is so low down in the cord as only to affect them, there is no well-marked paralysis. The grip of the patient is strong, and the dynamometer shows the existence of considerable strength. He is, however, not capable of continued muscular effort; and, though he may be able to lift several hundred pounds, or to carry another person around the room, his muscles are exhausted with the gradual and regular expenditure of a much less amount of force.

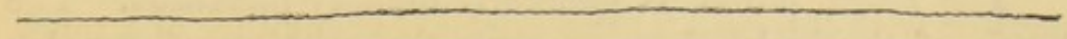
A phenomenon is often noticed as regards the upper extremities, which also exists with the lower, but which

cannot be so readily manifested—and that is, that the patient loses the ability to distinguish even considerable differences between weights. In the normal condition, if two weights differing in the ratio of thirty-nine to forty, are put one in one hand and one in the other, the difference is perceived without difficulty. The lower extremities, according to Jaccoud, are not so sensitive, and cannot distinguish a less difference than from about fifty to seventy grammes.

A person affected with posterior spinal sclerosis may have an ounce-weight put into his hand, and if in a few seconds it be removed, and one of half an ounce substituted, he will not be able to tell correctly which is the heavier. Or both hands may be extended, and the two weights placed simultaneously in them. The eyes should, of course, be closed. Sometimes less differences can be perceived, but ordinarily greater ones are not distinguished. In the case of a gentleman now under my charge, there is an impossibility of telling which of two pieces of lead, the one weighing one ounce and the other a pound, is the heavier. Späth [1] states that, in a case under his charge, the patient could not distinguish between two weights, which differed as one to one hundred.

No means for measuring the extent to which the patient is able to determine the state of muscular contraction is at all comparable to the dynamograph. The range of its usefulness is, however, limited—owing to the fact that posterior spinal sclerosis is not very frequently seated high enough in the cord to affect the muscles of the upper extremities. When the lesion is not above the origin of the nerves which go to form the brachial plexus, the line is straight, as in the accompanying figure:

FIG. 22.

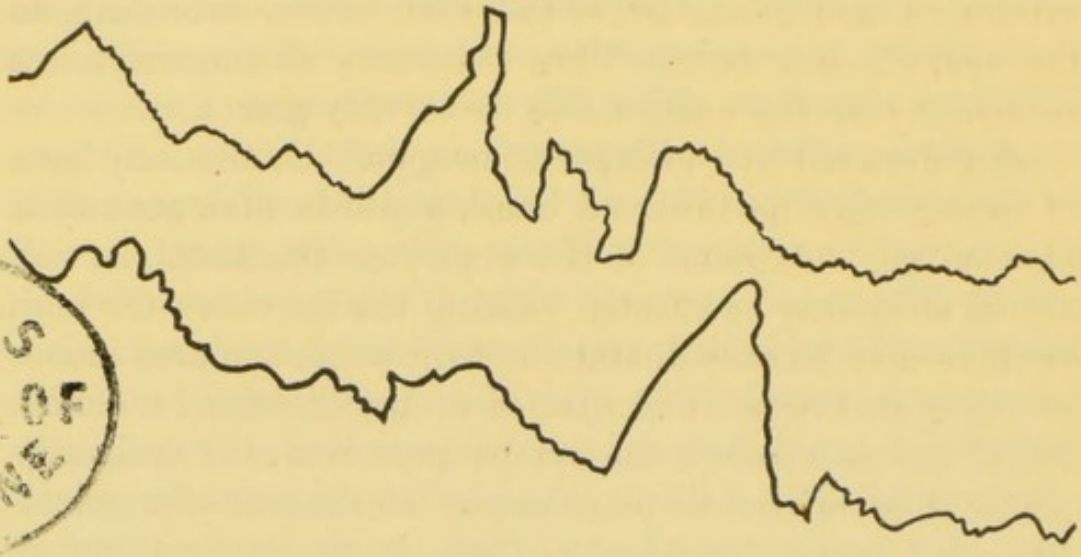


which represents the tracing made by a patient suffering from posterior spinal sclerosis of the lower dorsal region of

[1] Beiträge zur Lehre von der Tabes dorsualis. Tübingen, 1864.

the cord. But, when the seat of the disease in the cord is anywhere between the fifth cervical and first dorsal vertebræ, the ability to maintain a uniform degree of pressure is impaired, and lines resembling the following are produced:

Fig. 23.



EEDS
IOCLOF
DICINE

Both the above were made by the same patient, the upper with the right and the lower with the left hand. He was pefectly confident, till I showed him the tracings, that he had exerted a uniform pressure while the paper was traversing the pencil.

Under the name of baræsthesiometer, Eulenberg[1] has recently described an instrument for estimating the sense of pressure, by means of which very accurate determinations can be made for different parts of the body. He succeeded in demonstrating a considerable impairment of the sense of weight in the great majority of cases of locomotor ataxia examined, even when sensibility to pain, tickling, or electric irritation, was but slightly affected, and the sense of temperature was normal.

The reflex power is generally notably increased. The touch of the bedclothes, or even the rubbing of one leg against the other, is sufficient to cause powerful contractions.

[1] Allg. Med. Cent. Zeitung, No. 93, 1869. Also Journal of Psychological Medicine, October, 1870, p. 622.

Involuntary movements of the limbs, independent of those due to reflex excitations, are rarely met with.

The electro-muscular contractility is always increased.

It has already been mentioned that there are frequently ocular troubles. These generally occur among the first symptoms, and relate either to vision, to the movements of the eyeball, or to both. Thus there may be amaurosis due to atrophy of the optic nerve, or of the disk, a condition readily detected by the ophthalmoscope. Or the third pair of nerves may be involved, causing ptosis, divergent strabismus, and dilatation of the pupil; or the sixth pair of nerves alone may be affected, causing convergent strabismus; or there may be only dilatation of the pupil and prominence of the eyeball from the irritation propagated from the cilio-spinal centre through the sympathetic nerves. These ocular troubles never take place in posterior spinal sclerosis existing below the cilio-spinal centre—the upper dorsal region of the cord. The disturbances in the healthy action of the stomach and intestines, which have already been alluded to as common initial symptoms, are sometimes very distressing. As the pains in the limbs are often taken for evidences of neuralgia or rheumatism, so these gastric and intestinal troubles are frequently regarded as indicating the existence of dyspepsia. I have had a number of patients under my charge who, with double vision, ptosis, incoördination, and the other symptoms of posterior spinal sclerosis, had been told that "it was all dyspepsia," because vomiting and gastric pain were prominent features of the disease. These symptoms are also due to the relations of the sympathetic nerves with the spinal cord, and are not present in cases where the lesion is low down in the lumbar region.

When, however, this part of the cord is involved, there are very remarkable disorders of the genital system. These consist of frequent nocturnal emissions with or without erections, or of an inordinate desire for sexual intercourse. A gentleman who consulted me a few weeks ago, and who was

affected with the disease in question, informed me that he had several times had as many as eight seminal emissions in one night, and that his sexual desire was almost inextinguishable.

Paralysis of the bladder is a common circumstance, and the sphincter is not infrequently likewise affected. The bowels are usually obstinately constipated.

The feeling of constriction around the body which is so common a symptom in acute myelitis, and which is met with in other organic affections of the cord, is rarely absent in cases of sclerosis of the posterior columns.

Although the course of the disease in the great majority of cases is onward to a fatal termination, there are often periods of remission as in other spinal affections, and it rarely happens that the duration is not several years. A gentleman from Westchester County, in this State, has been affected for over twenty years, and still walks tolerably well. Another from Boston, sent to me by my friend Dr. Van Buren, had been subject to the disease for over twelve years. When I saw him he could not stand with his eyes shut, had the characteristic ataxic gait, was subject to genital and urinary difficulties, but yet was no worse than he had been six years previously. Another, from Pittsburg, has been in a stationary condition for several years; and another, from Binghamton, in this State, remains about as he was three years ago. I could easily cite twenty others whom I occasionally see professionally, who hold their own, and who have been affected for from five to ten years. Romberg gives the average duration at from ten to fifteen, Jaccoud at from six to eight, and all authors agree that the course is slow. Of ninety-one patients affected with sclerosis of the posterior columns of the spinal cord who have been under my charge during the last six years, three only have as yet died, so far as I am aware. Of these, one had been affected seven years, one eight years, and one eight and a half years. There are several cases now under my charge in

which the affection has existed longer than either of these terms.

The advance of the disease in the cord causes an aggravation of all the symptoms, and the appearance of others not previously noticed. The loss of motor power is now a prominent feature, the muscles become atrophied, bed-sores make their appearance, there is anasarca, and inflammations of the joints may occur. These latter are not common, but they are interesting as showing how disease of the cord may interfere with the due nutrition of the various parts of the body.

Their connection with posterior spinal sclerosis was first pointed out by Charcot.[1] Previous to his observations, they had been noticed, but they were ascribed to an intercurrent rheumatism, and, many years before locomotor ataxia was recognized as an independent disease, the association of spinal disease with inflammation of the joints was pointed out by Prof. J. K. Mitchell,[2] of Philadelphia, and his son, Dr. S. Weir Mitchell, with Drs. Morehouse and Keen,[3] had also related cases in which wounds of the spine had been followed by arthritis. Since Charcot's paper was published, Dr. Benjamin Bell [4] has cited cases of like affections coexisting with posterior spinal sclerosis.

In the cases in question there is no fever, redness, or pain. The swelling is due to the accumulation of liquids in the synovial cavity, and this affection is the result of defective nutrition of the bony, cartilaginous, and soft parts connected with the joint.

These accidents make their appearance usually in the interval between the occurrence of the shooting-pains so

[1] Sur quelques Arthropathies qui paraissent dépendre de une lésion du Cerveau ou de la Moelle Épinière, Archives de Physiologie, No. 1, January, 1868, p. 161.

[2] American Journal of the Medical Sciences, vol. viii., 1831, p. 55.

[3] Gunshot Wounds and other Injuries of Nerves, Philadelphia, 1864.

[4] On Diseases of the Joints connected with Locomotor Ataxy, Medical Times and Gazette, October 31, 1868.

characteristic of the first stage and the motor difficulties which mark the beginning of the second stage.

Of the ninety-one cases of posterior spinal sclerosis which have come under my observation, in five only were there any difficulties of the joints.

Death may take place either as the direct consequence of the lesion of the spine, or as the result of some intercurrent affection such as pneumonia, dysentery, phthisis, or cystitis.

Causes.—I have been very unsuccessful in my efforts to ascertain the cause in the greater number of persons, affected with sclerosis of the posterior columns of the spinal cord, who have been under my observation. The opinion is very prevalent that it is generally the result of excessive venereal indulgence; and, although this is undoubtedly sometimes a cause, it certainly is not so common a one as is generally supposed. I have carefully inquired into the etiology of all the cases I have seen, and have only been able to assign inordinate sexual indulgence as the cause in seven. Injuries were apparently the cause in four cases, standing in a constrained position in three, the excessive use of alcoholic liquors in three, a syphilitic taint in three, undue mental exertion and anxiety in two, and in the remainder there was nothing that could be assigned as an exciting cause. So far as we know, it would appear that the etiology is identical with that of antero-lateral spinal sclerosis. As regards the predisposing causes of age, sex, and hereditary influence, the similarity is equally well marked. Of the ninety-one cases observed by me, two only were in women.

Diagnosis.—A consideration of the symptoms detailed in the foregoing pages will prevent posterior spinal sclerosis from being confounded with any other affection of the spinal cord. It may, however, be difficult at times to discriminate between it and lesions of the cerebellum, and the distinction has frequently not been made by very skilful

diagnosticians. At one time Duchenne held the view that locomotor ataxia was really the result of a lesion of the cerebellum, but he subsequently[1] retracted this opinion, and now believes that the spinal cord is the seat of the disorder.

In a recent memoir[2] I have endeavored to point out the differences between cerebellar disease and the affection now called posterior cerebral sclerosis. In that essay I have said: "Derangement of locomotion certainly does result from injury or disease of the cerebellum. Experimental physiology, as well as pathology, proves this. Beyond a doubt the disorder is, however, clearly due to vertigo. There are, moreover, headache, vomiting, and eventually in some cases hemiplegia, generally of the opposite side to that of the cerebellar lesion, a fact at variance with Larrey's assertion. The gait of a person thus affected is exactly similar to that of a drunken man. As Carre says, the movements are not abrupt, jerking, and exaggerated, as they are in locomotor ataxia. They are more uncertain, and do not depend upon any defect of coördination, but upon weakness of the voluntary power.

"When either of the peduncles of the cerebellum is affected there is an irresistible impulse to go sideways, and sometimes gyratory movements are produced."

The characteristic symptom of cerebellar lesion is vertigo; and, although this is sometimes met with in posterior spinal sclerosis, it is not a prominent feature, and is rarely present at all except in the very earliest stage.

In cerebellar lesions the cutaneous sensibility is unimpaired, whereas in posterior spinal sclerosis it is always diminished.

A patient with disease of the cerebellum can stand and

[1] Diagnostic différential des affections cérébelleuses et de l'ataxie locomotrice progressive. Gazette Hebdomadaire, 1866.

[2] The Physiology and Pathology of the Cerebellum. JOURNAL OF PSYCHOLOGICAL MEDICINE, April, 1869.

walk better with his eyes shut than with them open, for the vertigo is not in the former condition felt to the same extent. The reverse is true of posterior spinal sclerosis. The history of the case will also serve as a good guide to the diagnosis. In the latter or even in the developed stage of posterior spinal sclerosis it would be difficult to mistake it for any other affection.

Prognosis.—The prognosis is no more favorable than that of antero-lateral spinal sclerosis. A few cases are cured, more are relieved, but the great majority go on unchecked. Of the ninety-one cases upon which this chapter is based, five were cured. Of these, two were probably of syphilitic origin, but in the other three no such cause was at all probable. One of them was a woman.

The cases in which amelioration has been produced are more numerous. In fact, it is not at all uncommon to succeed in retarding the onward progress of the disease, and of thus prolonging the life of the patient.

Morbid Anatomy.—Sclerosis of the posterior columns, as of the anterior, may be either diffused, multiple, or cortical. It may be restricted to the white substance of one or both posterior columns, or may also involve the gray matter. The posterior roots of the spinal nerves may also be implicated in the lesion.

The situations of the lesions and their character were well known to Romberg[1] before the researches of Duchenne, Charcot, and others. Thus he states that he was present at the post-mortem examination of the cord of a former patient. The organ was reduced one-third in diameter, and the atrophy was confined to the lower part of the posterior columns. The posterior nerve-roots were also involved, but the anterior columns were healthy.

I have stated under the head of symptoms that posterior spinal sclerosis is often accompanied by "head-symptoms," and may be first manifested by an epileptic convulsion. So

[1] Op. cit., p. 399.

generally is it the case that there are cerebral difficulties that I hesitated for some time whether the disease should not be classed among those affecting the cerebro-spinal system.

The lesions found in the brain never involve primarily the hemispheres. To be sure, it is sometimes the case that there are mental difficulties, but these come on toward the close, and are probably the result of defective nutrition.

The cerebral lesions are in very intimate relation with the posterior spinal columns. They are therefore met with in the lower cerebellar peduncles, in the restiform bodies, and in the optic thalami, and consist of atrophy with degeneration. The optic nerve is apt to participate, and hence the principal ophthalmoscopic appearances to which attention has already been called.

The other nerves which are often affected are the auditory, the third, and the sixth.

Romberg[1] was well acquainted with the fact that the cerebral nerves are frequently atrophied in the disease under consideration.

Although it is probable that the sympathetic is atrophied in some part of its extent, in many cases of posterior spinal sclerosis, the fact has not been demonstrated, except as regards one instance reported by Donnezan, in which a filament from the superior cervical ganglion was found atrophied. The ganglion itself was healthy.

In the later stages of the affection the muscles may exhibit a condition of atrophy. In such cases their tissue will be found on microscopical examination to have undergone fatty degeneration and substitution to a greater or less extent.

Pathology.—The theory of posterior spinal sclerosis which is generally held is, that the lesion impairs a faculty by which the muscles are brought into harmonious action—a faculty of coördination. According to this view, the first

[1] Op. cit., p. 399.

thing to be done was to locate this faculty in an organ, and Duchenne, with whom it originated, and who still holds it, adopting the ideas of Flourens and others, placed this faculty in the cerebellum, and therefore regarded what he designated progressive locomotor ataxia as a disease of the cerebellum.[1] Thus he says:

"In conclusion, regarding the order of appearance, and the habitual progress of the symptoms which mark the three periods of progressive locomotor ataxia, we find that the central morbid action which produces the phenomena symptomatic of this disease begins in general in the motor nerves of the eye, and in the tubercular quadrigemina, extending thence to the superior and inferior cerebellar peduncles and finally to the cerebellum."

As already stated, Duchenne has since abandoned this view of the location, and now assigns its seat to the posterior columns of the cord, but, in order to make the morbid anatomy agree with the theory of the disease which he holds, he places his faculty of coördination in the cord. But, although it has been established by numerous post-mortem examinations that the cerebellum is not the seat of lesion in cases of locomotor ataxia, and although the differential diagnosis between diseases of the cerebellum and posterior spinal sclerosis has been very clearly made out, there are some who still hold the view that, although the cerebellum shows no traces of disease, and that, although the posterior columns of the spinal cord may be in a state of sclerosis, the symptoms are the result of an interruption to the passage, from the cerebellum through the posterior columns to the spinal nerves, of that force which coördinates the muscles into harmonious action. In the memoir to which reference has already been made, I have entered at length into the consideration of the question of the location of a coördinating faculty in the cerebellum, and have, I think, adduced sufficient facts and arguments to show that coördination is

[1] De l'Électrisation localisée, deuxième édition, Paris, 1861, p. 611.

not one of its functions. Without going into a full account of the subject, a synopsis of the conclusions arrived at will probably not be deemed out of place:

1. The consequences of removal of the cerebellum, if the animal survives the immediate effects of the injury, are not enduring. This conclusion is supported by experiments by Flourens,[1] Harting,[2] Wagner,[3] Dalton,[4] myself,[5] and others. The physiological inference, of course, is, that, if the faculty of coördination resided in the cerebellum, it ought to be permanently removed with the ablation of the organ.

2. The entire removal of the cerebellum from some animals does not apparently interfere in the slightest degree even for a moment with the regularity and order of their movements. I have performed a number of experiments with reference to this point, on different classes of animals. They prove very clearly that the cerebellum is not the generator of coördinating power in all animals that have it: a fact in comparative physiology which is fatal to the hypothesis that this is its function in man.

3. The disorder of movements which results in birds and mammals immediately after injury of the cerebellum is not due to any loss of coördinating power, but is the result of vertigo.

If the cerebellum be removed from a pigeon it exhibits disorder in its movements, but a careful examination of the phenomena exhibited, shows that it is suffering from a vertiginous sensation. Even when placed upon its breast

[1] Recherches expérimentales sur les propriétés et les fonctions du système nerveux, Paris, 1842.

[2] Experimenta quædam de affectibus læsionum in partibus encephale, 1826.

[3] Nachrichten von der Universität und der Königl Gesellschaft der Wissenschaften zu Göttengen; also Journal de la physiologie de l'homme et des animaux, Avril, 1861.

[4] American Journal Medical Sciences, January, 1861, p. 83; also Treatise on Human Physiology, 4th edition, 1867, p. 416.

[5] Op. cit., p. 24.

and allowed to remain at rest, there is a trembling and swaying of the body, such as is produced by alcoholic intoxication. Exactly such symptoms can be caused by giving pigeons bread soaked in alcohol.

4. The phenomena of cerebellar disease or injury, as exhibited in man, are not such as show any derangement of the coördinating power.

Many cases are on record which support this proposition. Andral[1] states that, of ninety-three cases of cerebellar disease which he has studied, only one appeared to support the theory which locates the coördinating power in the cerebellum.

Many special instances might be brought forward, and several have occurred in my own practice. The case of Alexandrine Labrosse, reported by Combette,[2] is, however, worth referring to more specifically. His paper is entitled "Case of a young girl who died in her eleventh year, in whom there was complete absence of the cerebellum, of the posterior peduncles and of the annular protuberance." Magendie examined the brain after her death, and satisfied himself that the defect was congenital. As M. Combette remarks in regard to this case, Alexandrine Labrosse had been able to walk for several years, but always in an uncertain manner. Gradually her legs lost their strength, and she became paraplegic. She preserved the use of her upper extremities to the last. It is very evident, therefore, that the weakness of her legs was due to paralysis, for, had it been the result of incoördination, the arms must necessarily have participated.

For these reasons, I think, it cannot be considered with any degree of probability that the cerebellum has any thing whatever to do with the symptoms manifested in sclerosis of the posterior columns of the cord. Neither is it, in my

[1] Clinique Médicale, seconde édition, tome v., p. 735.

[2] Journal de Physiologie éxpérimentale et pathologique, par F. Magendie, tome xi., Paris, 1831, p. 27.

opinion, necessary to assume the existence of an organ whose office it is to exercise a coördinating power.

Other authors have ascribed the incoördination which is so prominent a phenomenon of posterior spinal sclerosis to the loss of what they call the muscular sense.

Sir Charles Bell[1] has argued strongly in support of the existence of such a sense. He enunciates his theory in the following sentence:

"Between the brain and the muscles there is a circle of nerves; one nerve conveys the influence from the brain to the muscle, another gives the sense of the condition of the muscle to the brain."

It is by this connection that we are enabled, according to Sir Charles Bell and other physiologists, to form an idea of the state of contraction of a muscle, and to lessen or increase the contraction as may be necessary. According to some writers, in posterior spinal sclerosis the patient loses this muscular sense, or is unable to exert it, for the reason that the spinal columns through which the perception reaches the brain are, by disease, rendered incapable of transmitting it.

In my opinion—and I shall endeavor to support it presently—there is no such a perception as that referred to, and its existence is certainly not established by the case reported by Dr. Ley to Sir Charles Bell, and which is incorrectly quoted by Trousseau; for it proves nothing more than that defective sensibility existed, and that the sense of sight had to be used in order to obtain a correct idea of what the insensible muscles were doing.

A lady having been recently delivered, and having suffered severe hæmorrhage, was seized soon afterward with headache and numbness. Dr. Ley was called to see her.

"I found her," he says, "laboring under severe head-

[1] On the Nervous Circle which connects the Voluntary Muscles with the Brain. Philosophical Transactions. Also, The Nervous System of the Human Body. London, 1830, p. 225.

ache, not confined to, but infinitely more violent upon one side than the other, and occupying the region of the temporal and occipital bones above the mastoid process, and attended with considerable pulsation.

"Upon one side of the body there was such defective sensibility, without, however, corresponding diminution of power in the muscles of volition, that she could hold her child in the arm of that side so long as her attention was directed to it; but, if surrounding objects withdrew her from the notice of the state of her arm, the flexors gradually relaxed, and the child was in hazard of falling. The breast, too, upon that side, partook of the insensibility, although the secretion of milk was as copious as in the other. She could see the child sucking and swallowing, but she had no consciousness from feeling that the child was so occupied. Turgescence of that breast produced no suffering, and she was unconscious of what is termed the *draught* on that side, although that sensation was strongly marked in the other breast.

"Upon the opposite side of the body there was defective power of motion, without, however, any diminution of sensibility. The arm was incapable of supporting the child, the hand was powerless in its grip, and the leg was moved with difficulty and with the ordinary rotatory movement of a paralytic patient, but the power of sensation was so far from being impaired that she constantly complained of an uncomfortable sense of heat, a painful tingling, and more than the usual degree of uneasiness from pressure or other modes of slight mechanical violence."

After a few months she died; having, in the mean time, received no improvement from the active treatment employed, and having also become pregnant again. On post-mortem examination there were found evidences of chronic inflammation of the membranes of the brain. The cord was not examined. Certainly this case presents nothing which may not be met with in any patient who has anæs-

thesia on one side and paralysis of motion on the other. I have observed a number of similar cases, and they neither prove the existence of a muscular sense, nor do they have any special bearing on posterior spinal sclerosis beyond the fact that they exhibit deficient sensibility.

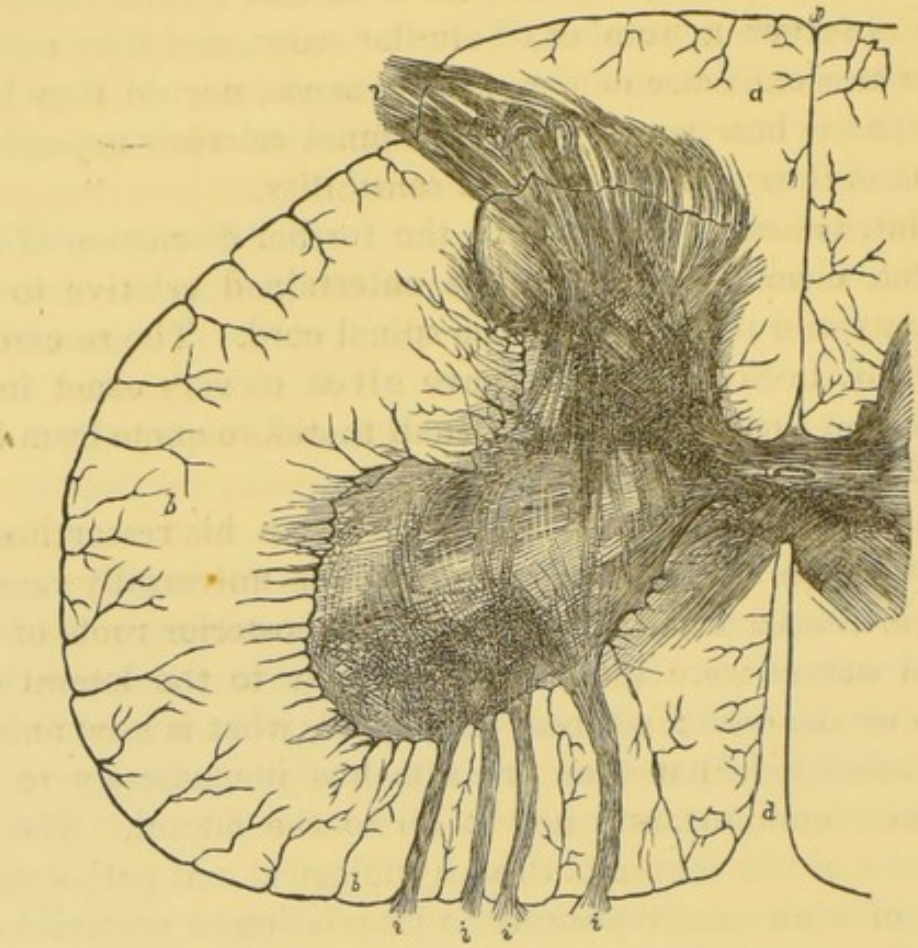
But, before proceeding to the further discussion of this subject, clear ideas should be entertained relative to the anatomy and physiology of the spinal cord. The researches of Dr. J. Lockhart Clarke have given us very exact information on these points, and I shall therefore quote from him in full.[1]

As Dr. Clarke states, before he began his researches on the structure of the spinal cord, it was universally taught, both in England and abroad, that the posterior roots of the spinal nerves were attached exclusively to the lateral columns of the cord; whereas he showed, what is now universally admitted, that they are attached immediately to the posterior columns, and not at all to the lateral. The importance of this fact in both a physiological and pathological point of view, and especially in its relation to posterior spinal sclerosis, will presently appear.

In Fig. 24, which represents a transverse section of the left lateral half of the lumbar enlargement of the cord, the posterior nerve-roots (*l*) are seen to enter through nearly the entire breadth of the posterior column (*a*); and in Fig. 25, which represents a longitudinal section of the cervical enlargement of the cord, we see the course of the roots of four consecutive nerves (P, P, P, P) within the cord. These roots are of three kinds: The first kind (*a*, *a*, *a*, *a*) enter the cord transversely, and pursue a very remarkable course. Each bundle, after traversing the longitudinal fibres of the posterior column (P C) in a compact form, and at a right angle, continues in the same direction to a considerable but

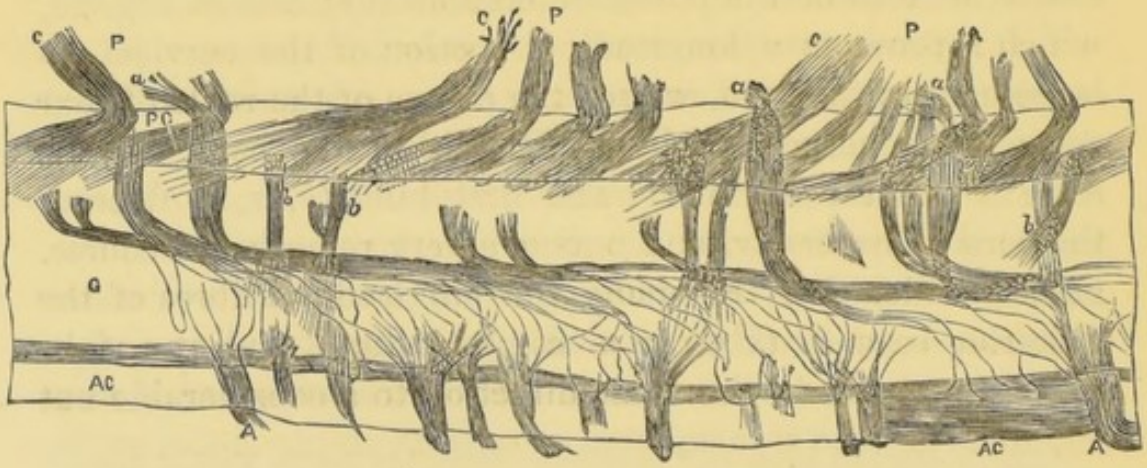
[1] See Dr. Clarke's paper on "Locomotor Ataxy," in British Medical Journal, September 25, 1869, p. 344, from which I take this account and the accompanying woodcuts.

Fig. 24.



variable depth within the gray substance (G), dilating and again contracting, so as to assume a fusiform appearance.

Fig. 25.



It there bends round upon itself, at a right or more obtuse angle, and, running for a considerable distance in a longitu-

dinal direction down the middle of the cord, sends forward, at short intervals, into the anterior gray substance, a series of fibres, some of which mingle with those of the anterior roots (A), while others enter the anterior white column, as at AC, AC, in which they run longitudinally, both upward and downward.

The second kind of posterior roots (*b*, *b*, *b*) also traverse the posterior column transversely, but sometimes a little obliquely from without inward. Their component fibres are finer than those of the other bundles, measuring about the $\frac{1}{7000}$th of an inch in diameter. Some of these fibres cross over transversely to the gray substance of the opposite side through the posterior commissure behind the canal. Others extend into the posterior and lateral white columns of the same side, while the rest may be traced deeply into the anterior gray substance (G, Fig. 23), where they diverge in different directions, and are ultimately lost to view.

The bundles forming the third kind of posterior roots (*c*, *c*, *c*, Fig. 23) enter the end obliquely. A few of their fibres proceed near the surface of the posterior column both upward and downward, and pass out again with roots above and below them. The rest cross the posterior column obliquely and chiefly upward, a small number only running downward. Interlacing at the same time with each other and with the roots already described, these fibres diverge, and for the most part reach the gray substance at points successively more distant from their entrance into the cord in proportion to the obliquity of their course, the most divergent and superficial taking a longitudinal course at least for some distance, with the fibres of the posterior column, among which they are lost. From these investigations (*Philosophical Transactions*, 1853), Dr. Clarke inferred that the posterior white columns of the cord cannot be the only channels for the transmission of sensory impressions, an inference which was verified two years later by the experiments of Brown-Séquard (*Gazette Médicale*, 1855).

Such being the anatomical connection of the posterior nerve-roots with the posterior columns of the cord, it is evident that scarcely any part of the length of those columns can be damaged either by injury or disease without involving destruction of a corresponding number of nerve-roots; and, since reflex action of the cord requires that impressions be conveyed by nerve-roots to the gray substance, the diminution of reflex action in cases of injury to the posterior columns is thus readily explained.

Dr. Clarke, in the anatomy and physiology of the posterior nerve-roots and their relations to the cord, which he has thus so satisfactorily elucidated, presents a theory of the phenomena of incoördination met with in posterior spinal sclerosis, to which I will allude more specifically directly. In the mean time a few words in reference to the "muscular sense" are necessary to the understanding of the whole subject.

Landry[1] declares that, whenever a muscle is caused to contract, the brain perceives the seat and the extent of the contraction. I am very sure that no sensation starts from the muscle which can give the brain any idea on the subject. As Trousseau[2] remarks:

"An important distinction must be drawn between the consciousness of a movement which has been executed, and the consciousness of the muscular contraction which performs the movement. When after shutting our eyes we execute without effort a pretty extensive movement, we are unable, even on paying the strictest attention, to feel the contraction of our muscles, although we may feel the movement communicated to the lever by the contracted muscles. This fact is so true, that, when we ask an intelligent person, who knows nothing of anatomy and physiology, which is the seat of the movements through which the fingers are flexed or extended, he immediately points to the hand, and

[1] Mémoire sur la paralysie du sentiment d'activité musculaire, Paris, 1855.

[2] Op. cit., p. 159.

never to the forearm. It is only when the muscular effect is considerable and kept up for a long time, that it is perceived where the contraction really occurs. Normally, then, we have no consciousness of muscular activity, but merely the consciousness of the movement itself, which is a perfect ly different thing."

In a very thorough essay on the subject, Dr. Bastian[1] has discussed the whole subject of the "muscular sense." He denies—and I think with good reason—the existence of any such special sense. In his opinion, there is no consciousness of the state of muscular contraction, and that the estimations by which we regulate the extent to which it is necessary, for instance, to contract the muscles of the upper extremity to sustain a certain weight in the hand, are "inferences based upon previous sensory impressions of the passive kind, upon impressions emanating from the skin, from the joints, and from the muscles themselves, so that in my opinion there are *no* conscious impressions derivable through the 'muscular sense.'" This, as I think, is not to be considered as an appanage of the intellect, but rather as an unconscious organic guide in the performance of voluntary movements. Why, it may be asked, do I not, as Trousseau has already done, deny its existence altogether? And to this I should reply by saying: "Although there is no evidence to lead us to believe that we derive any conscious impressions through the intervention of this so-called 'muscular sense,' there *is* evidence to show that the brain is assisted in the execution of voluntary movements by guiding impressions of some kind, which, while they differ from the impressions producible by means of the ordinary cutaneous and deep sensibility, may differ still further from these, owing to the fact of their not being revealed in consciousness at all."

This impresses me as being a very philosophical view of this rather difficult question, and it is in part sufficient to ex-

[1] Remarks on the "Muscular Sense" and on the Physiology of Thinking. British Medical Journal, May 1, 1869, and subsequent numbers.

plain the incoördination existing in cases of posterior spinal sclerosis; but, for the full understanding of the subject, it appears to me we must bring forward another fact in the physiology of the spinal cord which has not hitherto, so far as I know, been made applicable. The spinal cord serves two distinct purposes in the economy. It transmits nervous force to and from the brain, and it is a centre which generates nervous force. Referring now to the anatomical details given by Dr. Lockhart Clarke, we find that the posterior nerve-roots not only reach the white substance of the posterior and antero-lateral columns, but that they are in intimate relation with the gray matter. Now, the white substance simply serves for the transmission of nervous force, the gray elaborates it. Hence a great many of the muscular actions which we perform are done through the agency of this gray matter of the cord, and are without the intervention of the brain, and the brain can only be brought to bear upon them through the agency of the white substance. The states of muscular contraction are, therefore, in all probability, perceived by the gray substance of the cord, and, as the brain has no consciousness of the perceptions of the cord, we are not made aware of the states of muscular contraction. The muscular sense, therefore, does not exist, at least in the same manner as do the other senses.

In sclerosis of the posterior columns of the spinal cord the lesion generally involves the posterior nerve-roots, the posterior white substance, and the posterior cornua of gray substance. Hence the cord loses both in the ability to transmit and to generate nervous force. Those unconscious acts of muscular coördination which are regulated by the gray substance of the spinal cord can no longer be perfectly accomplished, and the brain is brought to assist in the determination through the sense of sight. The patient cannot stand well with his eyes shut, or walk in the dark, or determine differences of weight, because he is relying altogether on the perceptive faculty of the spinal cord, and this organ

is not in a condition to perform its work with precision; and his movements and muscular contractions are rendered still more uncertain from the fact that the cutaneous sensibility is diminished.

In the normal condition we frequently—in determining weights, for instance—are greatly assisted by the sense of sight, and there is nothing surprising in the fact that, in a disease like posterior spinal sclerosis, the spinal cord should be unable to perceive states of muscular contraction without the assistance of the brain. And, as the conducting power of the cord is also lessened, the brain cannot act with its full power; and, therefore, even with all the assistance to be derived from the chief generator of nervous force in the body, the patient's muscles are not so well coördinated as in health.

Dr. Lockhart Clarke, in the memoir already cited, explains the incoördination upon another principle, which, although, as I think, not sufficient to account for the phenomenon, may, and probably does, exercise some influence. His view is, that there is a physiological state of the muscles dependent on reflex action, that is absolutely essential to the proper coördination of voluntary movements, and that is, their tonicity, or that moderate but constant state of contraction which keeps the antagonist muscles, or those that are variously opposed to each other, in equilibrium or static tension. In the performance of voluntary movements a constantly-varying number of muscles, each of which differs more or less in force and in the particular direction which it gives to the limb or part, are associated together in action in an endless variety of ways. Each of the muscles that compose these varying groups must contract either simultaneously or successively to a certain particular extent, with a certain degree of force, and with a certain degree of rapidity, in relation to the actions of the others, according to the resultant direction desired in the voluntary effort; and this endless variety of ways, in which a constantly-varying number of muscles are balanced against each other in contrac-

tion for the performance of constantly-varying and complicated voluntary movements, affords the most exquisite and beautiful example of what, in physical science, is termed the composition of forces. In this balancing of muscular force we have to learn, by experience, and to remember the exact voluntary effort required to contract each muscle to its proper extent, with its proper force, and with its proper degree of rapidity, in relation to the action of the others that complete the group employed. Now, it is evident that if some of the muscles of the group employed have lost their normal tension or tone, they will not properly respond to the intentions of the voluntary stimulus, and will fail to perform their proper part in balancing the effects of the other muscles of the group that retain their tension, in the execution of any given movement. In proportion, therefore, to the exact amount of tension lost by any muscle or muscles of the group, and the number of muscles that have lost that tension, there must necessarily be a proportionate amount of disorderly movement of incoördination. But it appears to be satisfactorily proved, by the experiments of Brondigeest, Rosenthal, and others, that this constant tension or tone of the voluntary muscles is due to a constant reflex action of the cerebro-spinal centres, and is immediately dependent on impressions conveyed from the muscles to those centres by the posterior roots of the nerves. Now, Dr. Clarke has shown how these posterior spinal roots are spread out through the posterior columns of the cord; how impossible it is for these columns to be destroyed to any great extent without involving destruction of the nerve-roots; and how, consequently, the columns are so destroyed in locomotor ataxia. But, except in the very last stage of this malady, all the posterior roots are not injured by disintegration, and some of them are still competent to carry impressions to the gray substance of the cord; so that some of the muscles retain their tone, while others lose it to a greater or less extent.

This is Dr. Clarke's explanation of the peculiar feature of posterior spinal sclerosis, in almost his own words. That it is ingenious and plausible, all physiologists and pathologists will admit.

Treatment.—A great many medicines have been employed in the treatment of sclerosis of the posterior columns of the spinal cord, but few have been productive of any material benefit. Leaving out of mention those which my experience has taught me are inefficacious, I will specifically refer only to those which I have seen produce some amelioration of the symptoms.

As in the corresponding affection of the antero-lateral columns, ergot in large doses is often beneficial in the early stages of the disease, and the bromide of potassium may be combined with it in doses of from thirty grains to a drachm three times a day. Cod-liver oil is always advantageous, and the primary galvanic current applied as recommended for antero-lateral sclerosis is a main feature of the treatment.

I ordinarily begin the treatment of every case of posterior spinal sclerosis by these means if the disease has not yet passed beyond the first stage—that which is characterized by the presence of the shooting electric pains previously described—employing at the same time measures directed to the relief of particular difficulties. Thus, for gastric derangements, bismuth will often prove of service, or the constant galvanic current may be passed through the pneumogastric nerve, the positive pole being placed over the nerve in the neck, and the negative rubbed over the epigastric region, or, what is usually still more efficacious, Boudault's pepsin may be given in doses of fifteen or twenty grains with each meal. I have frequently had the most satisfactory results from this agent when all others have failed.

The pains in the back and around the abdominal or thoracic regions are best combated with codeine in doses of

from half a grain to one or two grains, according to circumstances.

If the case comes under observation when the motorial difficulties are well marked, or, if, after having used it for a month, no decidedly beneficial effect follows the treatment just specified, I omit the ergot, and use instead, the nitrate of silver in doses of the quarter of a grain three times a day. This remedy has apparently proved serviceable in several cases which were well advanced, but I am not able to speak definitely on the subject, for the reason that with it bromide of potassium, and especially galvanism, were used. Two cases were cured by the combined remedies—one of them was that of a distinguished journalist, who, in the first place, was treated with ergot, and subsequently, when this medicine appeared to be of no further effect, with the nitrate of silver. At the present time this gentleman is well, free from pains, able to coördinate, and with no symptom of the affection remaining. The disease was first manifested by an epileptic paroxysm, and soon afterward ocular troubles made their appearance. He was under treatment for about four months. The other case was that of a lady of this city. The disease, in her, began with pain in the back, and electric pains in the lower extremities. Ptosis, dilatation of the right pupil, and diplopia followed, and then gradual loss of sensibility in the soles of the feet, and difficulty in coördinating the muscles of the legs. The disease had lasted two years and a half when the patient came under my charge. She was treated with the nitrate of silver and the other remedies mentioned for nearly a year, and throughout the whole period gradually improved till her recovery was complete. The nitrate of silver was suspended for a week after each month of its administration.

In a third case ergot and nitrate of silver were given together without the bromide of potassium. This case was that of a gentleman, a merchant of this city, residing in Bridgeport, Connecticut. He had had ocular troubles, and

was suffering from pains, incoördination, plantar anæsthesia, paralysis of the bladder, and swelling of the right knee, when he came under my charge, being sent to me by my friend Dr. Hubbard, of Bridgeport. The disease had then lasted only a few months. With the medicines, the constant galvanic current to the spine and spinal nerves was employed. He was entirely cured in less than three months. In all cases inquiry should be made with reference to the existence of a syphilitic taint. If affirmative results follow the investigation, the iodide of potassium should be administered in gradually-increasing doses as recommended for acute spinal meningitis, or in combination with corrosive sublimate, according to the formula given on page 322, recollecting that galvanism is likewise to be used, and such other treatment as the special symptoms may seem to require. Two cases were cured by this treatment; one of them was that of a gentleman from the West—a fully-developed case—who had been treated by my friend Dr. Bumstead for other syphilitic troubles, and who sent him to me for his spinal difficulty. The incoördination, plantar anæsthesia, pain in the lumbar region, and the electric pains, were all present, together with slight diplopia. He was under treatment for about ten months. I met him a few weeks since in a railway-car, the picture of health, and, as he told me, perfectly well.

The other case occurred in the person of a gentleman of this city, and was similar in general features to the preceding. A cure was obtained, after like medication, in six months.

In several cases I have obtained ameliorations by the use of phosphoric acid, phosphorus, and chloride of barium. My experience with the latter medicine is not as yet sufficient to enable me to say that it has effected a cure, but the therapeutical promise is good. I have administered it in eight cases with decided benefit. Five of these are still under treatment with it. If the vesical sphincter be paralyzed,

belladonna may be used with advantage. Hydro-therapeutics in all forms, counter-irritation of all kinds, and faradisation, have never, according to my experience, been of the slightest benefit, except as regards the use of the latter to the affected muscles. The ether-spray recommended by Jaccoud has been entirely inefficacious in my hands, and the same may be said of all plasters and embrocations.

One auxiliary means of treatment I have lately employed with advantage, and that is, keeping the patient as much as possible from using the groups of muscles which have lost their coördinating power, and requiring him, when he walks, to employ crutches to assist him. By systematically carrying out this plan the nervous force of the patient is not wasted, and a diseased organ, such as is his spinal cord, is not overtasked.

Without making a separate chapter for the subject, it may be well to mention here the fact that chronic myelitis may affect a lateral half of the cord embracing in the lesion one antero-lateral and one posterior column, or any other combination of columns. Several such have come under my observation, and an interesting case of lesion of an antero-lateral and posterior column of one side is reported in a clinical lecture on Spinal Paralysis,[1] delivered by me at the Bellevue Hospital Medical College. The patient is still at the New York State Hospital for Diseases of the Nervous System, and is steadily improving.

[1] Journal of Psychological Medicine, January, 1871.

LEEDS SCHOOL OF MEDICINE

CHAPTER IX.

TUMORS OF THE SPINAL CORD.

Following the example of Jaccoud, I shall consider under one head, tumors of the cord, of the membranes, and those which, growing from the interior surfaces of the vertebræ, may compress the cord, and thus interfere with its functions by deranging its structure. In the present state of our knowledge, we have no sufficiently exact data by which to discriminate between these several growths.

Symptoms.—The phenomena which result from intra-spinal tumors, like those due to congestion, are of two categories, resulting as they do either from irritation or compression. Under the first head are embraced pain in the back, in the limbs, and in the viscera, if the posterior columns are mainly the seat of the lesion or subjected to the pressure of a vertebral tumor, and twitchings of the muscles, and contractions of the limbs, if the antero-lateral columns are principally involved. When both sets of columns—as is generally the case—are affected, the troubles of sensibility and of motility are both present.

If the tumor is situated in the cervical or upper dorsal region, there is generally tonic contraction of the muscles of the neck, by which the head is thrown backward, causing the patient to present the appearance of a person affected with the opisthotonos of tetanuos. There are in such a case usually ocular troubles, such as those previously mentioned, and more or less gastric derangement. The symptoms, so far as the limbs and viscera are concerned, vary

in their extent according to the situation of the morbid growth.

The symptoms of compression are anæsthesia and motor paralysis. These may or may not be accompanied with muscular atrophy. Reflex excitability and electro-muscular contractility are generally at first increased, or at least not lessened, but, as the pressure increases and the structure of the cord becomes more disorganized, they are lessened.

Many cases, of what may with Drs. Charcot and Brown-Séquard be called hemi-paraplegia, are due to spinal tumors. It often happens that these are small and compress a lateral half of the cord, leaving the other affected only by the transmitted pressure. A very remarkable case has been reported by Charcot,[1] in which the left inferior extremity was completely paralyzed, while the right was simply weak without having lost the power of contraction in any of its muscles. On the other hand, sensibility was greatly lessened in the right limb, while it was exalted in the left. There was paralysis of the bladder, but no atrophy of either limb. Finally, anasarca and bed-sores appeared, and the patient gradually sank. On post-mortem examination, a tumor was found growing from the dura mater on the anterior face of the cord and compressing its left lateral half. The accompanying woodcuts (Figs. 26 and 27), reduced from Charcot's lithographic representations, show the situation and relations of this tumor. Fig. 26 shows the growth *in situ*, and Fig. 27 the parts as they appeared when the tumor was pushed aside so as to allow the cavity to be seen in which it was lodged.[2]

Recollecting the facts that the fibres of the anterior or motor columns of the cord decussate at the medulla oblon-

[1] Archives de Physiologie, No. 2, p. 291.

[2] This case is quoted at length by Dr. Brown-Séquard in the *Lancet* of September 25, 1869, p. 429. In previous and subsequent numbers of this journal Brown-Séquard has contributed much valuable information on the subject of hemi-paraplegia.

gata, while those of the posterior or sensory columns cross over soon after they enter the cord from the posterior roots of the spinal nerves, we can understand why, when the paralysis of motion is confined to one side, or is greater on that side, that the lesion is on the corresponding side of the cord,

Fig. 26.

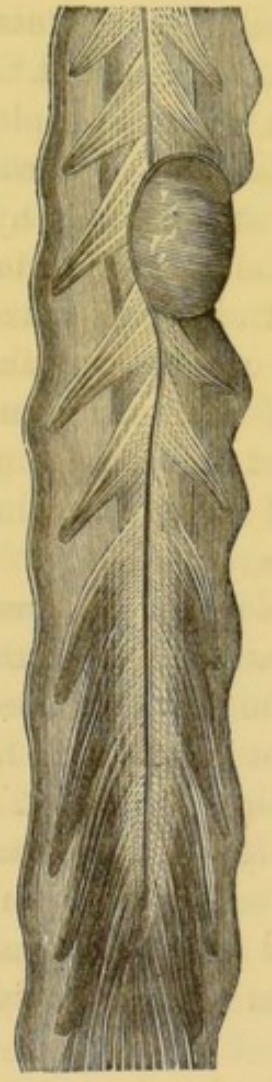
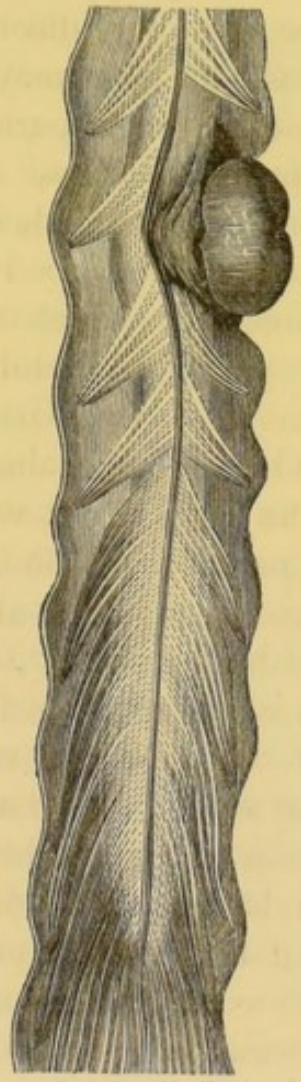


Fig. 27.



and that this loss of motility should be accompanied with anæsthesia of the opposite side of the body.

Under the name of painful paraplegia (paraplégie douloureuse), Cruveilhier referred to a form of spinal disease which has been subsequently described more fully by Charcot. This latter author has observed six cases, in all of which

there was cancer of the mammary gland. In three of these he had the opportunity of making post-mortem examinations, and discovered carcinoma of a lumbar vertebra in each, to which the irritation and compression of the cord were due. According to him, "the skin, especially during the paroxysms of pain, is often very sensitive to the touch. At the same time walking becomes troublesome, and later the patient cannot walk without help; finally, muscular atrophy ensues, and the patient loses the power to stand."

Simon,[1] from whom I quote these details, under the head of "paraplegia dolorosa," describes a case which came under his own observation, in which, during life, symptoms similar to those mentioned by Charcot, were noticed, and in which, after death, a cancerous tumor was found growing from the first lumbar vertebra and compressing the posterior columns of the cord. Other lesions were present in the posterior columns both above and below the tumor; they were apparently of the nature of sclerosis. Similar cases have been described by other authors.

Although it is rendered certain that cancerous tumors of the vertebræ may give rise to paraplegia characterized by great pain, it must be borne in mind that these symptoms are not a necessary accompaniment of the lesion, and that they are met with in other affections of the cord.

Causes.—Nothing is known relative to the etiology of intra-spinal tumors beyond the fact that they may result from the syphilitic, scrofulous, and cancerous diatheses.

Diagnosis.—There are no certain marks by which we can determine with any great degree of certainty that a tumor is compressing the spinal cord. We may suspect such to be the case when the motor paralysis is more marked on one side of the body than the other, and the anæsthesia exists to a greater extent on the opposite side. The existence of

[1] Berliner Klinische Wochenschrift, Hefte 35 and 36, 1870; also JOURNAL OF PSYCHOLOGICAL MEDICINE, January, 1871, p. 125, translation and abstract by Dr. D. F. Lincoln.

either syphilis, scrofula, or cancer, in connection with spinal difficulties not clearly referable to some other disease, may likewise excite the suspicion that a tumor exists.

Prognosis.—This is always unfavorable. It is less so when a syphilitic origin can be made out. No others recover.

Morbid Anatomy and Pathology.—The most common intra-spinal morbid growths are those which are developed from the vertebræ, and they include many syphilitic, scrofulous, and cancerous tumors. They originate either from the bones or from the periosteum. Formations resulting from either of these diatheses may also grow from the meninges or the substance of the cord.

Parasitic tumors due to either the echinococcus or the cysticercus may also be developed within the spinal canal. Their usual seat is in the membranes; and, according to Ollivier,[1] the echinococcus is found in the spinal cavity of women only.

Aneurismal tumors occasionally form in the intra-spinal arteries, and may compress the cord. Aneurisms of the thoracic or abdominal aorta may, by pressure, cause absorption of the vertebræ, and may thus eventually subject the cord to their influence.

Treatment.—The attempt should always be made, whenever the existence of a tumor of the spinal cord is suspected, to effect its removal by anti-syphilitic treatment, with iodide of potassium and mercury. The following case will show the advantages of following this course:

In the summer of 1869 I was requested to visit a gentleman who I was informed was paraplegic and subject to paroxysms of great suffering. On making my examination I found his limbs contracted, his reflex excitability augmented, and motor paralysis and anæsthesia of both lower extremities. There were intense pain in the lower dorsal region, and violent spasms of the sphincter vesicæ, alternat-

[1] Traité des Maladies de la Moelle Épinière, Paris, 1837, t. ii., p. 549.

ing with paralysis of it and the bladder. There were also paroxysms of severe pain in the head, and occasional attacks of delirium. He denied any syphilitic infection, but, on examining his head with my hands, I found a gummy tumor of the scalp over the right occipital region. Further inquiry and examination revealed the existence of a similar tumor over the left radius. I inferred that there might be one or more like growths within the spinal canal, and I administered the iodide of potassium in gradually-increasing doses, with the bichloride of mercury in doses of the sixteenth of a grain three times a day. In less than two months every symptom of disease, except a general weakness, had disappeared. The tumor of the scalp went during the first month; that of the arm a week later. The iodide of potassium was carried up to fifty grains three times a day. This patient continues in good health up to the present time. Even if there was not sufficient reason to diagnosticate the existence of an intra-spinal syphilitic tumor, the success of the treatment can scarcely leave a doubt on the subject.

LEEDS SCHOOL OF MEDICINE

CHAPTER X.

SECONDARY DEGENERATIONS OF THE SPINAL CORD.[1]

It is a well-recognized fact that disease of an organ promotes its atrophy and degeneration. A muscle, which from any cause is rendered incapable of contracting, becomes smaller, and its fibrillæ undergo conversion into fat. The same law applies to other organs, and among them the spinal cord. Whatever interrupts the passage of the normal excitations through its columns causes degeneration. Thus, if there be a cerebral hæmorrhage, preventing the action of the brain on the muscles, the anterior columns of the cord, not being stimulated by their accustomed excitation, undergo the change mentioned. If the cord itself be the seat of a lesion, or the posterior nerve-roots, and perhaps even the nerves or muscles, the posterior columns above, no longer being required to convey impressions to the brain, suffer atrophy and degeneration. To this alteration, which is not itself a primary disease, but which is always, in its very nature, consecutive to lesions in superior or inferior parts of the nervous system, the term secondary degeneration has been applied.

The fact that the spinal cord is affected by lesions of the brain was observed by Cruveilhier,[2] who, however, failed to

[1] This chapter is mainly a condensation of the admirable memoir on the same subject, by M. Ch. Bouchard, published in the Archives Gén. de Méd., 1866. This memoir has been translated by Dr. E. R. Hun, of Albany.

[2] Anatomie Pathologique, liv. xxxii., p. 15.

notice any consecutive change in the cord below the decussation of the pyramids.

L. Türck was the first specially to inquire into this important subject, and, in a series of memoirs extending through the years from 1851 to 1855, he showed that the cord underwent secondary degeneration, both from lesions of the brain and of its own substance. Since these memoirs, other pathologists, among whom MM. Charcot, Turner, Rokitansky, Vulpian, Cornil, and Lancereaux, may be mentioned, reported cases, but no one has investigated the subject with so much thoroughness as M. Bouchard.

Symptoms.—The most important symptoms referable to secondary degeneration of the cord from cerebral lesions are muscular contractions. These are not the contractions which sometimes exist from the very inception of a hæmorrhage, for instance, but those which come on at a later period of the disease, and which, like the first, have generally been thought the consequence of irritation existing about the cicatrix. Bouchard, however, shows very clearly that they are the result of secondary changes taking place in the spinal cord, and the clinical history of which has not hitherto been carefully studied. They are very frequent. Of thirty-two cases of old hemiplegia analyzed by Bouchard, they were present in all but one. From my own experience I think it is safe to say that it is very rare to meet with a case of hemiplegia of over a year's duration in which they do not exist.

In examining a patient suffering from an old hemiplegia, it is common to find the forearm of the paralyzed side flexed to some extent on the arm. Frequently, also, the fingers are bent into the palm of the hand, the hand flexed on the forearm, and the whole member carried across the front of the body, and held firmly against it by the contraction of the pectoralis major muscle. In such a case we find the muscles atrophied, hard, and stretched to an extreme degree of tension. Rectification of the position is, to a great extent,

impossible by the voluntary efforts of the patient. He may be able to accomplish a little motion, and to do still more by using the sound hand to extend the affected arm; but, if the hemiplegia has been of considerable duration, the range of his motility, with or without assistance, is very small, and is sometimes nothing. I have found that the electric contractility of such muscles is diminished in some of their fibres, unaffected in others, and exalted in others, so that, when the electrical stimulus is applied, a hard, irregular, and knotty contraction is obtained.

This condition is much more common in the muscles of the upper extremity than in those of any other part of the body. The muscles of the trunk are never involved, and, unless, as Bouchard appears to think, the muscles of the face are occasionally affected, the difficulty is entirely confined to the extremities. Of these, the upper are much more frequently its seat. Thus, of the thirty-one cases of rigidity with contraction, studied by Bouchard, the upper extremity was implicated in all, and the lower but in fourteen. In none of his cases was the lower extremity affected without the upper also participating, and he lays this down as an invariable occurrence. I have, however, a patient now under my charge, a gentleman from the West, who five years ago had an attack of cerebral hæmorrhage which rendered him hemiplegic on the left side. There is not the slightest contraction of the muscles of the left upper extremity, but the toes of the left foot are strongly flexed, and the sole of the foot turned inward by the contraction of the flexor longus digitorum, and the tibialis posticus.

In a case also now under my charge, the upper extremity is not carried across the front of the body, but is drawn backward by the contraction of the latissimus dorsi.

The period at which these secondary contractions begin in cases of hemiplegia has been carefully studied by Charcot, and he has ascertained that they habitually make their appearance during the second month. The fingers are usu-

ally the first to be affected from the contraction of the flexor muscles in the forearm. A symptom mentioned by Bouchard, as sometimes occurring, a trembling in the arm when it is raised, I have witnessed several times.

When the cord itself is the seat of primary disease, the anterior columns below undergo degeneration, and muscles become permanently contracted. Many cases of distortion which ensue on sclerosis, tumors, and other lesions, are the result of this secondary degeneration. M. Charcot is of the opinion that the epileptiform attacks sometimes met with in hemiplegics may result from these secondary descending degenerations affecting the peduncles, the pons, and the medulla oblongata.

No symptoms referable to ascending secondary degenerations—those of the posterior columns—have been recognized.

Causes.—Secondary degeneration of the spinal cord may result from primary lesions of the cerebral hemispheres, of the cerebral peduncles, of the pons Varolii, of the medulla oblongata, of the spinal cord itself, and of the posterior roots of the spinal nerves. The immediate causes are the loss of the due supply of arterial blood, and the arrest of nutritive action from deficient nervous influence.

The **Diagnosis** calls for no special consideration.

Prognosis.—This is not so unfavorable as might at first sight be supposed. Bouchard concludes that a cure is possible even in severe cases. In five cases which came under his observation, and in which there was complete paraplegia due to the compression of the cord in Pott's disease, complete cures were obtained in four, and a partial cure in the other. In the four entirely successful cases as regards the restoration of sensibility, and the power of motion to the paralyzed limbs, there were contractions. He therefore concludes that the nerve-fibres of the cord, like those of the peripheral nerves, may be regenerated.

My own experience is to the same effect. In cases of

muscular contractions resulting from cerebral hæmorrhage, and secondary degeneration of the cord, and in like difficulties due to primary lesion of the cord itself, followed by secondary degenerations, I have several times succeeded in effecting the complete relaxation of the contracted muscles, and the entire restoration of sensibility and the power of motion to the paralyzed limbs.

Morbid Anatomy and Pathology.—Secondary degeneration is only found in the white substance, the gray being always unaffected. This might certainly have been expected, owing to the fact that it is the conducting power of the cord only that is lessened, and, as this power resides entirely in the fasciculi of the white substance in the antero-lateral and posterior columns, it is here that we find the lesions. When a fibre belonging to the white substance is injured, either in the cord or in its intra-cranial prolongations, the secondary degeneration ensues either above or below the seat of the primary lesion, but it extends through the entire length of this portion to its central or peripheral extremity, according as it involves sensory or motor filaments. To these two varieties, the terms ascending and descending degeneration are applied. The affected fibres alone are changed, and the alteration extends throughout their whole length. But, as the white fibres are constantly receiving other fibres which have had no initial injury, the secondary degeneration becomes relatively less the greater the distance is from the seat of the primary lesion.

The morbid condition depends upon three processes: atheroma of the capillaries and the formation of granular corpuscles in the degenerated tissue; the degeneration and atrophy of a greater or less number of nervous filaments; the proliferation of connective tissue which takes the place of the nerve-tubes. These changes are similar to those which occur in the several forms of sclerosis, to which attention has already been directed.

Treatment.—Nothing is said by Bouchard relative to the

treatment. I have obtained the best results from the use of the primary galvanic current to the cord, the same or the induced current to the muscles, forcible extension and flexion of the contracted limbs, and the internal administration of nitrate of silver, chloride of barium, and cod-liver oil. It will generally be found that the opposing muscles are more or less paralyzed, and that great good may be effected by stimulating them with the primary or induced currents. The division of tendons is never necessary, unless for the rectification of distortions of the toes or fingers. Sometimes the toes are strongly flexed against the sole of the foot, rendering it almost impossible to walk, from the pain produced by the dorsal surface being brought in contact with the ground, and hence obliged to bear the weight of the body. In such cases the tendons may with propriety be divided, unless the toes can be kept extended by some convenient prothetic apparatus.

LEEDS SCHOOL OF MEDICINE

CHAPTER XI.

TETANUS.

Two varieties of tetanus are generally described by systematic writers—the idiopathic and the traumatic; but, as they are characterized by similar phenomena, differing mainly as to their modes of origination and severity of their symptoms, there would be no advantage in considering them separately.

Symptoms.—The first symptom to make its appearance in cases of tetanus is a feeling of pain or oppression in the epigastric region. In the beginning it does not attract much attention, but, as the disease advances, it becomes exceedingly severe, and adds greatly to the discomfort of the patient.

Soon after the occurrence of this pain, uneasiness is generally observed about the throat. This is, perhaps, no more than a sense of stiffness of the muscles concerned in deglutition, but it is not long before swallowing is impeded to a considerable extent. With these symptoms there are ordinarily mental and physical depression, sensations of chilliness, and a general feeling of *malaise*.

The foregoing constitute a prodromatic or formative stage, which may last a few hours or several days, and which is occasionally overlooked when the disease is intense and rapid in character.

In the next stage the epigastric pain is still a prominent symptom. It is seated just below the sternum, and generally extends backward to the spinal column. It appears to

be due to spasm of the diaphragm, so that this muscle is among the first, if not the very first, to be affected in the vast majority of cases. The difficulty of swallowing increases, and then the muscles of the jaws become contracted, constituting the condition known as trismus or lockjaw. At first there is only stiffness of these muscles with those of the neck, but gradually they become rigid, and the patient experiences difficulty, if not impossibility, in opening the mouth. The facial muscles do not escape, and an expression like the risus sardonicus is produced from the retraction of the angles of the mouth, the elevation of the alæ nasi, and the expansion of the nostrils. At the same time the eyes are staring, the brows corrugated, and the countenance anxious or wearied in appearance.

Sometimes gradually, at others suddenly, the morbid action extends to other muscles. Generally it passes to those of the neck, the back, and the loins, causing violent contraction, and bending the body backward. This state is called opisthotonos. The contraction of the powerful muscles referred to is so great as to cause the body to assume the form of an arch, the head being thrown far back, the abdomen protruded, and thus, if the patient were placed on his back, only the occiput and heels would touch the bed. Opisthotonos is the usual variety of spasm.

Two other forms are occasionally met with. In one of these—emprosthotonos—the body is bent forward from the contraction of the thoracic, abdominal, and pelvic muscles. In the other—pleurosthotonos—it is bent laterally. This latter may be met with in opisthotonos, owing to the muscles on one side being more strongly affected than on the other. Both emprosthotonos and pleurosthotonos are rare. Of very many cases of tetanus that have been under my observation, I have only seen the former four and the latter three times. The spasms characteristic of the disease are tonic; but, though they do not entirely relax, they are marked by more or less exacerbation, according to

the severity of the attack, and the care taken of the patient. Any cause calculated to excite reflex action will induce an accession. Thus the contact of the bedclothes with the body—the legs especially—the touch of the hand, the forcible shutting of a door, the rumbling of carriages in the street, even the blowing of a breath of air on the skin, may produce an aggravation of the spasm. Even without any apparent excitation these fits occur. They are marked by great pain, and may be so violent as to break the teeth, and the bones of the legs, and tear the large muscles of the thighs. During their continuance, and often when they are not present, the pain at the pit of the stomach becomes unendurable, and the patient may lose consciousness through its intensity. I have several times seen this event occur.

The tonic rigidity of the muscles of respiration induces difficulty of breathing, and the same result may ensue from spasmodic closure of the glottis. Death has frequently taken place suddenly from one or other of these causes.

With all this muscular excitement and mental disturbance there is rarely any fever. The skin is hot, and the thermometer often ranges from 105° to 110° Fahr., but the pulse is frequently small and weak.

Owing to the difficulty of swallowing, the patient suffers from hunger and thirst, and thus the powers of the system are still further reduced. The bowels are always obstinately constipated.

Wakefulness is generally present from the first. When the patient does sleep, it usually happens that the muscles are relaxed, to be again suddenly affected with spasm as soon as he awakes.

The mind is clear throughout, even in the most severe cases. When loss of consciousness occurs from extreme pain, it is from syncope, and not from any implication of the brain in the essential nature of the disease. Death usually takes place by apnœa. It may, however, result from

exhaustion, and, according to some authorities, from the spasmodic action attacking the heart.

The duration of the disease is very variable. The shortest case on record is one observed by Prof. Robinson, of Edinburgh. The patient, a negro waiter, cut his finger with a piece of broken china. He was immediately seized with tetanus, and died within fifteen minutes. Mr. Poland quotes a case in which death took place in five hours; in a case cited by Lepelletier in a few hours; in one by Dr. Jackson in twelve; in one by Dr. Leith in eighteen; and in one observed by Mr. Curling in nineteen.[1]

The shortest duration in any case I have witnessed was twenty-six hours, though I believe there were several much shorter, which occurred during the recent war in this country.

The average period of duration in fatal cases is from the third to the fifth day. Instances in which it has been prolonged far beyond this limit are not uncommon. Hennen[2] reports a case in which it lasted six weeks, and then the patient died of another disease. He reports another case in which it lasted seven weeks, and ended in recovery. I have seen three cases in which it extended to the fifth week.

The period which elapses between the reception of the cause and the beginning of the symptoms is also subject to great variation. In a case already cited it was only fifteen minutes; in another, quoted from Dr. Randolph by Reeves,[3] the spasms ensued immediately after the patient was stung by a bee; and in another, which occurred in his own experience they came on in a sensitive female immediately after running a needle into her finger. There is doubt, however, as to such cases really being tetanus. In the last one cited it

[1] All the above instances are quoted from Reeves's Diseases of the Spinal Cord and its Membranes, London, 1858, p. 387, *et seq.*

[2] Observations on some Important Points in the Practice of Military Surgery, etc. Edinburgh, 1818, p. 263.

[3] Op. cit., p. 377.

is stated that "the body and extremities were rigid, mouth closed, and the jaws fixed, the eyes the same. At short intervals the whole body was affected with convulsive shocks; the administration of a dose of chloroform removed them, but the back and neck remained rigid for three days." This attack was probably a manifestation of hysteria. In eighty-one cases collected by Mr. Curling, the disease began between the fourth and fourteenth days, both inclusive, and in nineteen on the tenth day. The following table from Reeves shows the period of the occurrence of the disease in three hundred and forty-three cases:

Within six, twelve, eighteen, or twenty-four hours...	12
From 1 to 2 days.....	12
" 3 to 5 "	37
" 6 to 8 "	94
" 9 to 12 "	77
" 12 to 14 "	52
" 15 to 17 "	25
" 18 to 20 "	9
" 21 to 23 "	9
" 24 to 26 "	6
" 27 to 29 "	9
" 30 to 32 "	1
	343

Causes.—The most common cause of tetanus is bodily injury of any kind, from the slightest to the most severe, and of any part of the body, although wounds of some parts, as of the thumb and great toe, are more apt to be followed by the disease than those of other regions. It has been known to result from the bite of a tame sparrow, from the sticking of a small fish-bone in the pharynx, from a seton in the thorax, from the stroke of a cane across the back of the neck, from the blow of a whip-lash, from fractured bones, and from every other imaginable wound or injury. In a case under my charge in this city, it was caused by a splinter of wood slightly scratching the palm of the

hand, in another a slight punctured wound of the foot produced it.

Next in frequency to wounds, tetanus is induced by exposure to cold and damp. This is the exciting cause in the great majority of cases of idiopathic tetanus, and it increases liability in those who have suffered from wounds. It was not uncommon, during the recent war, for the number of cases of tetanus to be very much increased immediately after a sudden change of the weather from dry and mild to wet and cold.

It has also apparently been caused by worms, by abortion and labor, and by diseases of the womb. Terror has the reputation of having induced tetanus in one case reported by Dr. Willan, and in others observed by Hennen.

In the form occurring in very young children, and known as trismus nascentium, it appears to be induced by inattention to the cut umbilical cord.

The tendency to tetanus, especially among soldiers and others who have been wounded, is increased by poor diet, confinement in ill-ventilated hospitals, inattention to cleanliness, and neglect to give proper care to the wounds they may have received.

Diagnosis.—The only affections with which tetanus is liable to be confounded, by any but the most ignorant, are the hysterical simulated affection, and the condition induced by poisoning with strychnia and other substances of its class.

That hysteria can simulate tetanus, as well as almost all other diseases, we have abundant evidence. A case has already been referred to in this chapter which was evidently hysterical, and several others have come under my observation. A lady now under my charge has repeated attacks of hysterical spasms, during which her jaws are tightly closed, she is unable to swallow, and her body is bent backward so as to assume the position of opisthotonos.

Such seizures are readily distinguished from tetanus by

the facts that they are unaccompanied by pain or real distress, are of very transient duration, and are accompanied by other manifestations of hysteria.

From the artificial tetanus caused by strychnia, the diagnosis is more difficult; for, so far as the more obvious symptoms go, there is such a great similarity that even the most skilful diagnosticians might be, for a time, undecided. It is well known that strychnia is not unfrequently used for the purpose of committing murder or suicide, and it is possible so to employ it for either of these purposes as to cause its effects to extend over a long period of time, and thus to add to the difficulties attending the discrimination. Even in such a case, however, the diagnosis can be made if due care and a thorough inquiry into the history of the case be made.

In the first place, the tetanus of strychnia always shows itself in the lower extremities before trismus ensues. The legs are stretched widely apart, and the hands are generally involved. In natural tetanus, trismus precedes spasm in the extremities; indeed, the lower extremities are rarely affected to any great extent. The arms generally escape altogether.

The epigastric pain, which constitutes so prominent a feature of true tetanus, is not present in the toxic variety. I have witnessed three cases of poisoning by strychnia, and this pain was not complained of in either of them.

In the tetanus of strychnia, the symptoms are developed with great rapidity, and death takes place generally within a half an hour, although life may be prolonged, in exceptional cases, somewhat beyond this peried. In true tetanus it is very rarely the case that death takes place within twelve hours, and ordinarily not till several days have elapsed.

In those cases of poisoning by strychnia in which the doses have been small, and administered at comparatively long intervals, the symptoms are mitigated in violence, and consequently one of the distinguishing features of the two

affections is lost. Still, the general character and sequence of the phenomenon are the same, and it is not probable that careful observation and inquiry will fail to elicit the true nature of the case.

Prognosis.—The longer the time that has elapsed between the reception of the injury or subjection to other cause, the greater is the probability of a favorable termination. When the paroxysms are slight, and the intervals between them long, the prognosis is also more favorable. The duration of the disease is likewise an important element in the prognosis; and, when it has lasted over a week, death does not often take place. Cases are, however, on record in which a fatal result has supervened after the affection has existed for several weeks.

Tetanus is, nevertheless, one of the most fatal of maladies. Dr. O'Beirne[1] witnessed two hundred cases without a single recovery. Hennen[2] never saw a case of acute symptomatic tetanus recover. McLeod[3] has collected and analyzed twenty-three cases which occurred in the British army in the Crimea, of which but two recovered. Demme[4] refers to eighty-six cases in the hospitals in Italy during the campaign of 1859, of which six were cured; and Hamilton[5] has observed eight cases, of which three recovered.

Nine cases have been under my immediate care, of which there were three recoveries. Of the many cases which I observed in the course of my inspections of camps and hospitals in the army during the recent war, I do not know how many terminated favorably. I am disposed, however, to believe that the number was not great. Hamilton states that his information leads him to think that, of

[1] Dublin Hospital Reports, vol. iii., pp. 343, 378. [2] Op. cit., p. 262.

[3] Notes on the Surgery of the War in the Crimea, London, 1858, p. 153, *et seq.* Also table, p. 439.

[4] Militar-Chirurgische Studien, Würzburg, 1861.

[5] A Treatise on Military Surgery and Hygiene, New York, 1866, p. 595.

one hundred and fifty cases which occurred during the war, the recoveries were few.

Morbid Anatomy and Pathology.—The results of post-mortem examination of patients who have died of tetanus are very unsatisfactory. Rokitansky,[1] in chronic cases, has found a proliferation of connective tissue in the spinal cord. Wedl,[2] in one case, discovered increased redness of a portion of the spinal cord. Curling[3] declares that serous effusion with increased vascularity is generally observed in the membranes investing the medulla spinalis, and also a turgid state of the blood-vessels above the origin of the nerves; and Lockhart Clarke[4] regards the constant lesion as consisting of a granular degeneration of the cells of the cord.

On the other hand, it often happens, especially in very rapid cases, that nothing is found which can fairly be regarded as constituting the essential feature of the disease. Billroth[5] affirms that his examinations of the spine and nerves, in cases of tetanus, have thus far given only negative results, and this is in accordance with the observations of the great majority of pathologists. While, therefore, there appears to be no doubt that the disorder is dependent upon some lesion of the spinal cord, and probably of the gray matter, our examinations have not as yet enabled us to determine the nature of the morbid process.

It is contended by some authors that tetanus, like hydrophobia, is due to blood-poisoning. The fact, that a condition, so nearly resembling it as to be with difficulty diagnosticated from it, may be caused by the injection of strychnia into the blood, appears to favor this view. However this

[1] Beiträge zur Pathologie des Tetanus. Virchow's Archiv, t. xxvi., 1862.

[2] Rudiments of Pathological Histology. Sydenham Society Translation. London, 1855, p. 276.

[3] A Treatise on Tetanus, etc., London, 1836.

[4] Lancet, 1864, and Medical Times and Gazette, 1865.

[5] General Surgical Pathology and Therapeutics, in Fifty Lectures. Hackley's Translation. New York: D. Appleton & Co., 1871, p. 353.

may be, the character of the symptoms indicates the spinal cord to be the seat of the disease.

The spinal cord is both an organ for the generation of nerve-force, and for conducting impressions to and from the brain. In tetanus it is this first-named function which is deranged, and this is shown by the great exaltation of reflex excitability which exists. Every thing capable of causing a reflex movement of the slightest kind, and even excitations which in health would be altogether unperceived by the cord, augment its intrinsic action to a great extent where tetanus exists.

Now, we are able to produce a similar increase of reflex action by strychnia; and, in those cases of disease in which the amount of blood in the cord is increased, very small quantities of strychnia produce the characteristic phenomena of stiffness in certain muscles, and of augmented reflex excitability. The condition is aggravated by the medicine; and, if we had no other facts to support the theory, we should be warranted in concluding that, in cases of strychnia-poisoning, the amount of blood in the cord and the excitability of the organ are both increased. From a consideration of all the points bearing on the subject, we are warranted in concluding that tetanus essentially consists in a morbid exaltation of the functions of the spinal cord as a nerve-centre.

Bernard[1] has investigated this matter with his usual exactness. He says:

"Strychnia produces convulsions by exaggerating the sensibility of certain parts; it also causes reflex movements. We have seen that the point of departure is in the sensitive system; for, where the posterior roots of the nerves are cut, the animal dies without convulsions."

An experiment performed by myself and my friend and

[1] Leçons sur les Effets des Substances toxiques et médicamenteuses, Paris, 1857, p. 386.

collaborator, Dr. S. Weir Mitchell,[1] shows that the action of strychnia is to destroy the nervous excitability from the centre to the periphery. Its influence, therefore, must first be exerted on the spinal cord.

"Under the skin of a large frog, whose left sciatic nerve was previously divided, a few drops of a strong solution of strychnia were introduced. Tetanic spasms ensued in two minutes. After forty-five minutes the nerves were irritated by galvanism. That of the left side, which had been cut, responded energetically, while no motions could be produced through the uncut nerve. The former remained excitable for two hours later."

Bernard[2] asserts that the action of strychnia extends no farther than the spinal cord; and any one who has seen a frog under the influence of this substance cannot have failed to notice that all the symptoms indicate exalted spinal action.

We are therefore led by observation and experiment to the conclusion that tetanus is seated in the spinal cord, and that, although we cannot at present affirm an identity of lesions, in each case we shall probably eventually be able to define them with as much accuracy as we do those of other spinal diseases which a few years since were equally obscure.

Treatment.—There is scarcely a sedative or stimulant remedy in the pharmacopœia which has not been employed and recommended in tetanus. Aconite, ether, belladonna, chloroform, cannabis Indica, conium, opium, tobacco, Calabar bean, ice, counter-irritants, alcohol, and many other substances, have been used, and cases reported which have apparently recovered under their administration. Then, of surgical means, excision of the injured nerve and amputa-

[1] Experimental Researches relative to Corroval and Vao; two new Varieties of Woorara, the South American Arrow-Poison. American Journal of the Medical Sciences, July, 1859. Also Physiological Memoirs, Philadelphia, 1863, p. 181, *et seq.*

[2] Op. cit., p. 359.

tion of the wounded member have also been recommended, but are not, I believe, practised now. Latterly the bromide of potassium and hydrate of chloral have been employed with favorable results.

A case in which the latter agent was successfully used in tetanus is reported by Dr. Wirth,[1] of Columbus, Ohio. In about a month the patient took nine ounces and two drachms, in doses of from thirty to forty grains, at times as often as every one and a half hour. In this case opium in large doses had been administered without effect. A number of other cases in which chloral was administered are cited in the same number of the New York Medical Journal in which Dr. Wirth's case appears, in several of which it was successful.

A very thorough analysis by my friend Dr. D. W. Yandell,[2] of Louisville, of an unpublished report on tetanus, by Dr. R. O. Cowling, embraces so much valuable information on the subject that I quote the summary entire. The term *acute* is applied to tetanus occurring within nine days of the injury, and chronic to cases ensuing after nine days:

"*Calabar bean* was given in thirty-nine cases, with thirty-nine per cent. of recoveries. Of these reported cures, but one was of acute tetanus; five others were in cases which recovered before the expiration of fourteen days. *Per contra*, there were ten deaths from chronic tetanus.

"*Indian hemp* used in twenty-five cases, with sixty-four per cent. of recoveries, of which three cases were acute, and six recovered before the symptoms lasted fourteen days.

"*Chloroform* relieved seventy per cent. of thirty-five cases, nine of which were acute, and eight recovered before fourteen days. Three chronic cases died, and two after symptoms lasted fourteen days.

"*Ether.*—Sixty per cent. of fifteen cases recovered; five

[1] New York Medical Journal, November, 1870, p. 419.

[2] American Practitioner, September, 1870, p. 152.

acute; seven inside of fourteen days. One chronic case died.

"*Opium.*—Fifty-seven per cent. of one hundred and sixty-five cases recovered; twenty-two acute; twenty-nine before the fourteenth day. Twenty-six chronic cases were lost, and four after the disease had continued fourteen days.

"*Tobacco* relieved fifty per cent. of forty-one cases; six acute; six before fourteen days of the disease. Four chronic cases died, and one after fourteen days.

"*Quinine.*—Seventy-three per cent. of fifteen cases recovered; one acute; three before fourteen days. Three chronic cases ended fatally, and one after fourteen days' duration.

"*Aconite.*—Eight per cent. of fourteen cases recovered; none acute; none recovered before fourteen days. Death in one chronic case.

"*Stimulants.*—Eighty per cent. of thirty-three cases recovered; four acute; six within fourteen days. Six chronic cases died, and three after fourteen days.

"*Mercury.*—Fifty-seven per cent. of seventy-five cases got well; twelve before fourteen days. Seventeen chronic cases were lost, and two after fourteen days.

"*Bleeding.*—Fifty-five per cent. of fifty-eight cases recovered; nine acute; ten before the fourteenth day. Seven chronic cases were lost, and two after fourteen days.

"*Cold Affusion.*—Seventy-three per cent. of eleven cases recovered; three acute; three before fourteen days. Two chronic cases died.

"*Ice-bags.*—Seventy-seven per cent. of nine cases recovered; one acute; two in less than fourteen days.

"*Amputation.*—Sixty per cent. of seventeen cases recovered; four acute; four in less than fourteen days. Three chronic cases died, and one after fourteen days.

"*Division of nerve* relieved seventy-five per cent. of three cases; one acute; one before the fourteenth day. One chronic case died.

"*Purgatives.*—Sixty-six per cent. of seventy-four cases recovered; thirteen acute; twelve before fourteen days. Ten chronic cases died, and three after fourteen days.

"*Turpentine* relieved seventy per cent. of sixteen cases; six acute; four before fourteen days. Five chronic cases died, and two after fourteen days."

Among the conclusions arrived at by Dr. Yandell from these data are, that "recoveries from traumatic tetanus have been usually in cases in which the disease occurs subsequent to nine days after the injury; that when the symptoms last fourteen days recovery is the rule, and death the exception, apparently independent of the treatment; that chloroform, up to this time, has yielded the largest per centage of cures in acute tetanus; that the true test of a remedy for tetanus is its influence on the history of the disease: does it cure cases in which the disease has set in previous to the ninth day? does it fail in cases whose duration exceeds fourteen days? and that no agent, tried by these tests, has yet established its claims as a true remedy for tetanus."

It is, perhaps, scarcely necessary to say that I fully accord with these opinions.

Judging from its effects upon the spinal cord, it was supposed by Mr. Morgan that woorara injected into the blood might prove efficacious in tetanus. Experience, however, has not confirmed this view; and the researches of Dr. Cowling show that it is one of the most inefficient of remedies.

In a case which was under my charge ten years ago, when I was one of the surgeons of the Baltimore Infirmary, I injected corroval—a remedy which the investigations of Dr. Mitchell and myself had proved to be antagonistic to strychnia—into the blood. The patient, a colored boy, became affected with tetanus two days after his arm had been amputated by my friend and colleague Prof. Nathan R. Smith. Cannabis Indica, morphia, and chloroform, had been used without effect, when at my request Prof. Smith turned the case over to me, in order that corroval might be admin-

istered. Two drops of a strong solution of the substance in water were injected into the cellular tissue of the forearm. At the time the pulse was 160, and the respirations about 75. There was very decided opisthotonos. In three minutes the pulse had fallen to 152. Two more drops were then injected, and the pulse fell to 144. As it soon rose again, two more drops were injected, when it fell to 132, and the respirations to 64. The spasms still continuing, two more drops were injected. In five minutes the pulse began to decline rapidly, and in ten minutes had fallen to 90. At this time the patient had a violent tetanic spasm, and during its continuance the pulse became intermittent. It then rapidly went down to 40, then to 30, and during a violent spasm the patient died. From this record it will be seen that at no time did the corroval exercise the least effect over the disease.[1]

As I have stated, three successful cases have occurred in my practice. One of these I saw in consultation with Dr. J. Lewis Smith, of this city. It was traumatic, and had ensued two weeks after a wound of the foot by a nail. The patient was treated by cannabis Indica, and the persistent application of ice to the spine. The spasms were greatly lessened in force and frequency, and recovery took place within two weeks. Another, which was also traumatic and acute—that is, making its appearance within nine days after the injury—was treated according to the same plan, and recovered in sixteen days, though the jaws remained stiff for several weeks afterward. The wound was caused by an ice-pick being accidentally thrust through the hand. The third case was that of an eminent musician of this city, who, while drilling with the regiment to which he belonged, injured his thumb with a splinter from the stock of his rifle. The first evidence of tetanus appeared on the twelfth day.

[1] Traumatic Tetanus. Inoculation with Corroval. Death. By Edward Milholland, M. D., Resident Physician at the Baltimore Infirmary. In Maryland and Virginia Medical Journal, January, 1861, p. 13.

The attack was not very severe. I administered the extract of cannabis Indica (Squires's) in doses of half a grain every two hours, and kept up the application of ice to the spine continuously for six days. There were several violent spasms during this period, and the opisthotonos was well marked. At the end of a week the cannabis Indica was omitted for a day, but, the spasms becoming more frequent and severe, it was resumed as before, and continued with tolerable regularity for ten days longer. During this period there were but two spasms, and the opisthotonos became less. It was then gradually diminished, and on the twenty-fifth day was left off altogether, the patient being convalescent.

I am disposed to think that, whatever internal medication be adopted, the application of ice to the spine is a measure which should always form a feature of the treatment.

LEEDS SCHOOL OF MEDICINE

SECTION III.

CEREBRO-SPINAL DISEASES.

CHAPTER I.

HYDROPHOBIA.

Although there are objections to the name employed to designate the terrible disease I now propose to consider, the same is true of all other terms which have been applied to it, and the present has the advantage of being well known. So long as we are obliged, through ignorance of pathology and morbid anatomy, to use a nomenclature based on symptoms, we must expect to be inexact. The name hydrophobia is as old as Galen, and still retains its preëminence, notwithstanding the fact that the symptom on which it is based is sometimes absent.

Symptoms.—Beginning with the reception of the injury by which the body has been inoculated, we find that it heals in the ordinary way, and that there are no immediate signs of infection. At a period which varies greatly in different cases, pain or a sensation of uneasiness is usually experienced at the seat of the wound. This, however, is rarely of such intensity as to cause suffering, and probably would generally be overlooked or disregarded but for the apprehension which the patient has, and which directs his attention to every sensation which can be attributed to the wound. But there

may be absolutely no pain or uneasiness other than such as are met with in all wounds till the phenomena of the affection are manifested. The period between the reception of the injury and the beginning of the symptoms of hydrophobia is known as the stage of incubation.

The duration of this stage is variable. It is rarely shorter than a month, and probably never longer than two years. Instances are on record, however, in which the disease has been developed within ten days, and others, about which, however, there is much doubt, in which the latent period has reached to ten years and longer. The vast majority of cases occur within seven months after the reception of the wound. In four cases which have been under my observation, the period of incubation varied from sixty-two days to four months and a half.

During this period of incubation there are not often any indications of what is going to take place except in those cases in which there are abnormal sensations in the cicatrix or its neighborhood. Sometimes there are depression of spirits, anxiety, and derangement of the digestive functions, but these symptoms may fairly be attributed to the peculiar circumstances of the case, aside from any toxic influence due to infection.

The first symptoms which generally appear are directly connected with the cicatrix, which, if it has previously been free from abnormal appearances and sensations, now becomes subject to both. But there is no constancy even in these phenomena. They were altogether absent in one of my cases, and very slightly manifested in one other, if they were present at all. In this case, which I saw in consultation with Dr. S. G. Cook,[1] of this city, the patient, after other symptoms had appeared, occasionally clutched the place where he had been bitten, but denied, on being asked, that there was any pain at the spot.

[1] A case of Hydrophobia. JOURNAL OF PSYCHOLOGICAL MEDICINE, January, 1870, p. 80.

But, though there may be no symptoms of swelling, redness, or pain about the cicatrix, there are abnormal sensations in the nerves which radiate from it. Thus, if the injury has been in the leg, pains are felt along the courses of the sciatic and crural nerves; if in the hand, similar sensations are experienced in the radial, ulnar, median, and other nerves of the upper extremity. Occasionally the pain is felt in the epigastric region, and in any situation is ordinarily accompanied by headache. At about the same time the respiration becomes sighing and irregular, there is a feeling of oppression or constriction in the chest, the pulse loses its force and uniformity, and there is an indefinable sense of anxiety. The sleep is scarcely ever natural. Either there is insomnia or drowsiness, and sleep, when obtained, is disturbed by frightful dreams, and is unrefreshing. The bowels are constipated, the skin is dry, and there are alternate chills and flushes of heat. The duration of this stage is from two to four days.

And then the period of full development begins; characterized, at first, by an increase in the symptoms just mentioned, and subsequently by the appearance of others not previously present. A peculiar sense of uneasiness is felt at the epigastrium, and a pain and constriction of the throat, which add greatly to the distress. The tongue becomes stiff and painful, and articulation is thereby rendered indistinct; the respiration increases in irregularity, and becomes noisy and oppressed; the rigidity of the muscles of the throat prevents or impedes deglutition, and the patient dreads attempting to swallow, from the experience he soon acquires that his efforts in this direction are attended with pain and spasm, which greatly increase his sufferings. Sometimes the convulsion of the pharyngeal muscles is so great that substances are thrown with great force out of the mouth. This was the case in two of the instances I witnessed. At the same time the spasm extends to other parts of the body, and occasionally becomes general. It is accompanied by

pain in the epigastrium, and sometimes in the spine. Solids are swallowed with much more ease than liquids. Indeed, so great is the difference that the patient cannot even entertain the idea of swallowing any fluid without being thrown into spasms. The sound of water splashing or trickling, the sight of it, the thought of it, and even an impression remotely connected with water, such as that produced by the reflection of rays of sunlight on the face by a mirror, will bring on a paroxysm of convulsions. With the spasm there are sobbings, trembling, and then a condition of exhaustion, during which the patient is bathed in perspiration.

The following day the phenomena are still more strongly marked. The mouth is dry and parched, and yet the patient dare not attempt to quench his thirst; vomiting ensues, the pulse becomes rapid and small, the pain in the pit of the stomach still increases, the headache is intense, and the countenance expresses terror, anxiety, and suffering. The pain in the spine augments and extends to the muscles of the neck and abdomen. The secretions of the mouth are altered, and the saliva is mixed with a frothy, tenacious mucus, which the patient is constantly attempting to eject, but which collects as fast as he can spit it out. The mouth and fauces are dry and painful, articulation is almost impossible, and every attempt to relieve the distress by a few drops of water induces a return of the spasms and convulsions. Finally every reflex excitation reaches the muscles of the throat; the contact of the bedclothes, the jarring of the bed by persons walking in the room, the rustling of window-curtains—any thing capable of acting on the hearing, the eyesight, or the touch, may cause the spasms.

As the disease advances, all the symptoms increase in violence, and still others make their appearance. The urine and fæces are often passed involuntarily, the skin becomes exquisitely sensitive, the body is in a constant state of agitation and tremor, alternating with spasms, and the tough,

stringy, tenacious mucus collects in the throat and impedes respiration.

Thus far the mental symptoms have scarcely been considered, but they are present almost from the first. Indeed, they may be among the very first indications of disorder. They consist of emotional disturbances of various kinds, and sometimes radical changes of character and disposition.

It has been alleged by some authors that the dreams, at a very early period after inoculation, are connected with the animal giving the wound. I have never met with this symptom, but in the case previously cited, and which I saw twice in consultation, a circumstance still more remarkable is related by Dr. Cook. The patient, a child three years old, was bitten by a bitch in heat on or about August 20, 1870. On November 15th the mother noticed that he slept badly; on the 16th, among other manifestations, he "was cranky all day." On the 17th he was seen by Dr. Cook.[1]

"On entering the room," says the doctor, in his report of the case, "and seeing several children, and not noticing any thing wrong with any of them, I very naturally inquired which was the patient. I was pointed to a little boy sitting at a table in a high chair. On approaching him, he turned his face toward me, revealing the most peculiar-looking eyes I have ever seen. They were not like those seen in persons suffering from delirium in prolonged fevers, nor yet like those we see in the second stage of cerebral meningitis, although somewhat resembling both of these conditions, but more like the eyes of a person in a fit of violent anger, slightly combined with a feeling of fear.

"When I reached out my hand to touch his, he shrank from me as from a blow, at the same time making a desperate effort to catch his breath, precisely as a naked person might if a pail of cold water was unexpectedly poured over him. This I understood to be a laryngeal spasm. It was

[1] Op. cit., p. 81.

very brief, lasting but the fraction of a minute, probably not more than ten seconds. I took a seat at a little distance from him, where I could see his every motion, and regarded him attentively for a long time.

"He seemed an unusually intelligent child, for one of his age, speaking very distinctly with a clear, ringing voice, which his parents informed me was a little unnatural, as it 'seemed strained.' He had at times a disposition to stammer, which was also unnatural. For one hour after my observation commenced he talked almost incessantly of dogs, and repeated very few sentences a second time. He seemed familiar with all the most common breeds, relating some anecdote of the bull-dog, the mastiff, the bird-dog, the spaniel, the coach-dog, and the poodle.

"Connected with all his narratives was a tragic or gloomy termination. The mastiff, after carrying him an incredible distance about the city, finally disappeared through a bottomless hole in the street, he only escaping a similar fate by suddenly dismounting. The bull-dog, after bringing for his admiration and pleasure a great variety of puppies, suddenly turned cannibal, and swallowed the whole lot. The spaniel, after having been his playmate for a very long time, finally took it into his head one day to get on to a coffin that was being carried through the streets, and ride away to reappear no more."

There were no other evidences of disordered mental action in this child, and he died, perfectly conscious to the last.

Usually, however, this is not the case, and various morbid desires are entertained by the patient. Thus, in a case which I saw in this city in 1865, there was an impulse to strike those near, and an intense dislike of certain persons who had always been intimate friends of the patient. In both the other cases there were paroxysms of previous delirium, during which the sufferers bit and struck at all within their reach, and of which hallucinations and delu-

sions constituted marked features. In the case of the boy just cited, the stories of dogs which he related were evidently delusions which he accepted as realities.

Death usually takes place on the third day after the accession of the symptoms indicating the full development of the disease. The chief of these is laryngeal spasm. A fatal termination is rarely delayed till after the third day, though cases are not uncommon in which it has ensued on the first or second day. In all the cases, except one, which have been under my observation, the third was the fatal day. In Dr. Cook's, the latest I have seen, the disease may be considered as having been fairly developed on the 17th of November, the first day in which any spasm of the throat was witnessed. Death resulted on the evening of the 18th.

Generally death takes place during a spasm. This was the result in three of my cases. In the other—the latest—the child died quietly. In the former condition apnœa is probably the immediate cause of death; in the latter, exhaustion. In all cases, the powers of life, from the violent convulsions, the loss of sleep, and the deprivation of food and drink, are drained away to the utmost.

Causes.—It has generally been supposed that hydrophobia has but one source in the human subject, and that is, inoculation by the saliva of an animal affected with rabies. It cannot be communicated to one individual by the saliva of another affected with hydrophobia, although there is no doubt that, under certain circumstances, the saliva of man, as well as the milk and other secretions, may become poisonous. Neither can dogs or other animals be infected by inoculation with the saliva of a hydrophobic man. Magendie's[1] experiment, the only one of the kind which has ever succeeded, is of exceedingly doubtful import, as hydrophobia was prevailing among dogs at the time, and the animal may have been previously bitten.

[1] Dictionnaire des Sciences Méd. Art. Rage, t. 47, p. 46. Also Journal de Physiologie, t. i., p. 47.

But it is very probable that the saliva of healthy animals, the dog especially, is capable of producing hydrophobia in man and other animals. A case of the kind is recorded in *Hufeland's Journal* of December, 1839, and similar ones are frequently met with. In none of the cases I have witnessed was the dog which had inflicted the wound supposed to have been rabid. In one case which I saw in this city, with a physician whose name I cannot recall, the patient, a stableman, was bitten by a dog that was to all appearance in perfect health. In the case reported by Dr. Cook, the animal, a bitch, was being led quietly through the passage-way of the house, when the child became entangled in the chain, fell against the dog, and was bitten apparently in anger. The animal was well known, and was not even suspected of being hydrophobic. She was in heat; and Dr. Cook raises, for the first time to my knowledge, the question whether this circumstance renders the saliva of the animal capable of inducing hydrophobia in the human subject. With a view of throwing as much light as possible on the subject, he consulted the records of Bellevue Hospital, in order to ascertain the facts in relation to a man who died of what was supposed to be hydrophobia from the bite of a bitch in heat. The results of his inquiries were to show very certainly that the man did die of hydrophobia; that the animal was not rabid, and that she was in heat.

It would appear that the saliva is the only means of communication. Dupuytren, Breschet, and Magendie, endeavored to convey the disease by injecting the blood of dogs, suffering from rabies, into the veins of healthy dogs, but always unsuccessfully. The flesh, milk, semen, and abdominal secretions, were likewise found not to be media for transmission.

No other animals than those of the genera *canis* and *felis* have been clearly shown to be capable of communicating the disease. The power has been claimed for the rat, but on insufficient evidence. The wolf is said to be the

most dangerous of all in this respect, for the reason probably that it seizes the neck or face, parts not fully protected by clothing, and thus the saliva is not so apt to be rubbed off as when the leg, for instance, is the part attacked.

The slightest abrasion of the skin coming in contact with the saliva may be sufficient for inoculation. Cases are recorded in which the disease has resulted from dogs licking the hand or face on which there were pimples or sores.

Diagnosis.—That protean disease, hysteria, occasionally puts on the semblance of hydrophobia. Several cases of the kind have occurred to me, and in all the symptoms were in general character very much like those which are exhibited by genuine hydrophobia, though in some respects, perhaps, a little exaggerated. It will in these and similar cases—the result of fright and imagination—often be found that the patient has been bitten by a dog not long before. There is a want of consistency about the symptoms which of itself is sufficient to excite suspicion as to the real character of the phenomena. Thus, although at times the attempt to swallow will excite laryngeal and other spasms, these do not always occur under similar circumstances, and are not induced by those secondary and more refined influences, such as the sound of falling water, bright lights in the face, excitations applied to the skin, seeing others drink, etc., which so generally cause them in the real disease. There are not the same anxiety and depression in the simulated disease as in the real, though the apparent emotional disturbance is much greater. The hysterical patient is loud in the expression of apprehensions, while the real hydrophobic one, though intensely anxious and terrified, endeavors to prevent others perceiving the state of his mind.

The history of the case, the existence of the hysterical diathesis, and the fact that the symptoms come on soon after the bite without any period of incubation, will further aid in establishing the diagnosis between the false and the real disease.

Hydrophobia has been confounded with tetanus, and some writers have regarded it as a modified form of this affection. The distinction is, however, so well marked that it scarcely seems necessary to dwell upon it. The facts that in tetanus the spasms are tonic, while in hydrophobia they are clonic; that in the first-named they are mainly shown as regards the jaws and back, while in the latter they radiate from the throat; that in tetanus the mind is clear throughout, while in hydrophobia more or less mental implication is always present, will suffice to render any mistake in the diagnosis of the two diseases impossible.

Prognosis.—There is no authentic instance on record of a cure of hydrophobia. Several such have been reported, but inquiry has always shown misstatement or error somewhere. The fact, that the hysterical counterpart has several times been regarded as the real disease, is the main support for the opinion of some authors that the affection is curable.

Several years ago Dr. Ligget,[1] of Maryland, reported a case of hydrophobia cured by calomel. A careful examination of the details of this case excites very grave doubts in my mind in regard to its really being an instance of the disease in question.

The subject was a negro-woman who had been bitten about two weeks before any symptoms were manifested. The dog was lying quietly in the yard, and bit her in the great toe as she was teasing him with her foot. The animal was at once chained up, and died in two or three days with "all the symptoms of *rabies canina* in its most virulent form." It does not appear that the doctor saw the dog, and it is very probable that the rigid confinement would have caused the animal to exhibit symptoms which would easily be mistaken by laymen for those of hydrophobia.

Again, the period of incubation was unusually short, and the symptoms, as detailed by Dr. Ligget, are clearly

[1] Case of Hydrophobia successfully treated with Drachm Doses of Calomel, Am. Jour. Med. Science, January, 1860, p. 96.

not those of hydrophobia. Thus, although he repeatedly states that there was inability to swallow liquids, there is no distinct mention made of the pathognomonic laryngeal and pharyngeal spasms which occur in hydrophobia, and which are so frightful in character. The convulsions all appear to have been general, and there was a "horror" of water, which is not a phenomenon of the true disease. For these reasons I am constrained to believe that the disease treated by drachm-doses of calomel was in reality one of hysteria which assumed the form of hydrophobia. In this opinion I am sustained by an eminent medical gentleman residing in Dr. Ligget's neighborhood, who, as the latter admits, declared the affection to be "a case of that protean disease, hysteria, simulating hydrophobia." Calomel has been repeatedly tried before and since Dr. Ligget's case, but without effect.

But, although the prognosis is so hopeless in the developed disease, it is much more favorable as regards the supervention of hydrophobia from the bites of rabid animals, for, of those bitten by dogs unmistakably affected with the disease, not more than one in fifteen become successfully inoculated. This liability differs greatly according to the circumstance of the part being covered or not. The wounds of the face, neck, or hands, are much more likely to be followed by hydrophobia than those inflicted on the legs or feet, where the virus is rubbed off by the clothing before the teeth reach the flesh. The bite of a rabid wolf is more apt to be followed by the disease than the bite of a dog, for the reason that the first-named generally seizes the throat or face. Thus, Trolliet states that at Brives, in France, seventeen persons were bitten by a rabid wolf, of whom ten died of hydrophobia; and, of twenty-three bitten by another, thirteen died. On the other hand, Hunter states that on one occasion a dog bit twenty persons, of whom only one was inoculated. Those first bitten by a rabid animal are more liable to have hydrophobia than those bitten subsequently, when the poison is in a measure exhausted. Prob-

ably the most dangerous wounds are those which barely penetrate the epidermis, and in which, therefore, the venom is not washed away by any flow of blood.

Morbid Anatomy.—There are no post-mortem appearances which can be regarded as peculiar to hydrophobia. The brain and its membranes are generally congested, as is also the upper part of the spinal cord with its membranes, but these changes are met with in other diseases, having no affinity by their symptoms with hydrophobia.

Sometimes the nerves at the wound are found inflamed, but this is not a uniform occurrence. The eighth pair has been found to present a pinkish appearance in some cases. In four cases in which the blood was examined by Schivardi,[1] infusoria of the genera *bacterium*, *monas*, *vibrio* and *torula* were found.

The fauces, pharynx, larynx, trachea, and lungs, are generally found reddened and congested, as much from the asphyxia as from any specific influence of the disease.

Doubtless with our present knowledge of the intimate structure of the nervous tissues, and the perfection of our means of observation, we shall, ere long, be enabled to detect the nature of the changes which take place in hydrophobia. Now we know nothing of any importance on the subject.

Pathology.—Hydrophobia, if we may judge from the symptoms, essentially consists in a hyperæsthetic condition of the hemispheres, the medulla oblongata, and the upper part of the spinal cord. The hallucinations and other mental phenomena point to the hemispheres, the irregular actions of the respiratory muscles, and the heart, together with the gastric derangement and pharyngeal convulsions, indicate the implication of the pneumogastric nerves, and the spasms of the larynx point to the origin of the spinal accessory nerves in the spinal cord.

The nature of the virus is unknown. It is probably of

[1] Observations nouvelles sur la rage. Besançon, 1868, p. 22.

the nature of a ferment, but this cannot be regarded as satisfactorily proved.

In 1820, Dr. Marochetti observed, in the Ukraine, that during the formative period of hydrophobia small vesicles or pustules formed under the tongue, and that, if these were opened and cauterized, the further development of the disease was prevented. I have never been able to find these formations, but they were recognized, two years after Marochetti published his account, by Magistral, in France. This latter opened and cauterized them in the manner recommended by Marochetti in ten cases, in five of which, nevertheless, the affection went on to full development, and the patients died. I am not aware that any one else has discovered these pustules.

For full details relative to hydrophobia as it appears in dogs, I must refer the reader to the late Mr. Youatt's excellent book on canine madness. I may, however, state that it is very clearly established that canine rabies is not so frequent in very hot as it is in temperate or cold weather; that it is not induced by thirst or improper food, or by preventing copulation.

Treatment.—The measures of treatment relate to those proper immediately after the infliction of the wound, with the view of preventing the development of the disease, and those advisable after the affection is unmistakably manifested.

Under the first category comes excision, which should be performed as soon as possible, and which is probably the best of all prophylactics. The operation should not be done with a niggardly hand, but every part with which the teeth of the animal have come in contact should be removed, as well as the tissue into which the poison may have become infiltrated. Previous to the operation, in fact as soon as the wound has been received, a tight ligature should be bound around the limb immediately above the injury, and, after the knife has done its work, cupping-glasses should be

applied over the spot till the tissues in the vicinity are thoroughly drained of blood. I have performed excision, for the wounds received from dogs certainly rabid, six times, and always with the effect of preventing hydrophobia.

Cauterization may be performed instead of excision, and is preferred by some practitioners. Mr. Youatt used it with over four hundred persons bitten by rabid animals, and never unsuccessfully. Four times he employed it on himself, but there is a strong probability that the practice at last failed with Mr. Youatt himself, for he committed suicide while supposed to be suffering from the initial symptoms of hydrophobia.

He preferred the nitrate of silver as an escharotic. Others have made use of the actual cautery, caustic alkalies, the mineral acids, arsenic,chloride of zinc, and carbolic acid. I have employed cauterization four times upon persons bitten by rabid dogs, and always with success.

Mr. Youatt at one time had faith that the scutellaria lateriflora, or scullcap, was a preventive. He moistened three pieces of tape with the saliva of a rabid dog, and inserted them as rowels into the skin of three dogs. To two of these he gave scutellaria combined with belladonna, while the third was left to itself. On the twenty-ninth day after the inoculation this latter became rabid, while the others, several months afterward, were alive and well.

Notwithstanding this experience, it would not be justifiable in the physician to neglect performing either excision or cauterization as soon as possible after the reception of the bite. Even if several weeks or months have elapsed, one or the other—preferably excision—should be performed.

As to the treatment of the fully-developed disease, there is nothing in my opinion which has hitherto succeeded in arresting its onward course. Cases of cure have been reported, but, as already stated, they are open to the suspicion of not being true instances of the disease. Excessive bloodletting has been reported as a successful remedy; injection

of warm water into the veins dissipated the paroxysms in a case reported by Magendie, the patient, however, dying; and nearly every stimulant, narcotic and sedative, in the materia medica, has been used. In the case which I saw with Dr. Cook, and which has already been cited, the hydrate of chloral was administered. The effect certainly was to mitigate the severity and frequency of the spasms, but it was, as Dr. Cook states, given too late in the course of the disease to produce any permanently curative result. In the present state of our knowledge, I should be more disposed to rely on the hot-air bath at a temperature of about 200° Fahr., and the administration of hydrate of chloral in large doses frequently repeated, than on any other plan of treatment. In Dr. Cook's case the Turkish bath was proposed, but the parents of the child would not consent to its use.

Before concluding my remarks on this disease, it is proper to allude to the attempts of Dr. Schivardi,[1] of Milan, to cure the disease by the primary galvanic current. In one case the current was feeble, and was continued for nineteen hours. Great improvement ensued; the oppression disappeared, and the dysphagia was entirely relieved. Through some misunderstanding, advantage was not taken of these ameliorations, and the patient was allowed to die.

In the other case, which was one of undoubted hydrophobia, occurring in a girl nine years old, the current from twenty-two Daniell's cells was employed. The current was passed from the soles of the feet to the forehead for fifty-eight hours almost continuously, and the duration of the disease prolonged to seven days and seven hours, when the patient died. During the last two days there were no hydrophobic symptoms.

Further trials are necessary before the therapeutical value of galvanism in hydrophobia can be ascertained.

[1] Observations nouvelles sur la rage.

CHAPTER II.

EPILEPSY.

Epilepsy, although only a symptom of a morbid condition, must for the present be considered as a disease, for the reason that we are not able to designate with certainty its exact seat, or the nature of the lesion which exists. It is characterized by paroxysms of more or less frequency and severity, during which consciousness is lost, and which may or may not be marked by slight spasm, or partial or general convulsions, or mental aberration, or by all of these circumstances collectively. The essential element of the epileptic paroxysm is loss of consciousness. Without that there is no true, fully-formed epileptic paroxysm.

Symptoms.—Although in many cases there are no precursory phenomena, it often happens that there are indications of an approaching attack. These are exceedingly variable in character and situation. They may consist of pain in the head, a sensation of constriction or fulness, vertigo, noises in the ears, a feeling as if the ears are stopped with cotton or water, flashes of light, or sudden blindness, illusions or hallucinations of any of the senses—irritability of temper, extraordinary cheerfulness, difficulties of speech, pains in various parts of the body, especially in the stomach, bowels, or ovaries, sensations of numbness or of tingling, or of an indescribable character, which begin in an extremity or in some other region, and appear to pass rapidly to the head—a feeling of constriction in the throat, vomiting, sudden evacuation of the bladder or rectum, erections of the

penis, with or without the sexual orgasm, and discharge of semen, with many others of almost every possible description.

The prodromata may precede the attack by a considerable period, but usually are only a few moments in advance of it. Indeed, often the interval is so short that they may be regarded as a part of the paroxysm.

The sensations of numbness, or of tingling, or of an electric shock, as they are differently described by patients, or of pain which originate in some distant part of the body, and seem to run rapidly toward the head, are called the aura. This aura is usually of the same character in every attack of the same patient, though occasionally it varies.

Delasiauve,[1] of two hundred and sixty-four cases, found the paroxysms unannounced in one hundred and one, and with precursory phenomena in one hundred and eighty-three. The prodromata were immediate in one hundred and fifty cases. These he divides into seven categories, as follows. It is to be recollected that cases may appear under one or more categories, according as the prodromata, as is often the case, are met with simultaneously in different parts of the body:

First Series.—*Precursory Signs in the Head.*—Seventy-five cases.

Vertigo, flashes of light	23
Headache, weight in the head	15
Heat of face	3
Various localized sensations	13
Indefinite sensations	1
Illusions, hallucinations, and other sensorial aberrations	9
Rotation of the head or of the eyes	5
Grinding of the teeth, derangement of the motility of the tongue	2
Tendency to sleep	1
Constriction of the throat	3

[1] Traité de l'Épilepsie—Histoire—Traitement—Médecine Légale, Paris, 1854, p. 47.

Second Series.—*Precursory Signs in the Thorax.*—Twenty-two cases.

Oppression of the chest and sense of suffocation	9
Sensation of a ball or of motion in the pectoral region	2
Shivering sensation of cold or of an aura	5
Pain or heat	4
Palpitations, spasms	2

Third Series.—*Precursory Signs in the Abdomen.*—Thirty-two cases.

Pain with or without oppression, eructations, vomiting	13
Intestinal or uterine colic	3
Sensation of a ball	3
Sensation of cold, of a vapor, etc.	6
Stomachal heat	1
Undefinable sensations	6

Fourth Series.—*Precursory Signs in the Extremities.*—Ninety-four cases.

Numbness, contractions, jerkings, retractions, cramps, formications, etc.	36
Pain with or without spasms	13
Tremblings	10
Aura or phenomena approaching thereto	20
Undefinable sensations	15

Fifth Series.—*Precursory Signs, consisting of General and Undefinable Sensations.*—Twenty-two cases.

General agitation or rotation of the body	8
Condition of discomfort, fainting, etc.	6
Vague sensations	7
Moroseness	1

Sixth Series.—*Precursory Signs situated in the Genital Organs.*—Five cases, such as retraction of the testicles, aura starting from the testicles and spermatic cords, sensations located in the uterus, etc.

Seventh Series.—*Exceptional Cases.*—Desire to defecate, to urinate, profuse perspiration, etc.

Of two hundred and eighty-six cases of epilepsy which have come under my observation, and in which inquiry was made as to the occurrence of prodromata, I ascertained that they existed in one hundred and twenty-eight. They did not differ in general character from those specified by Delasiauve.

The Paroxysm.—Great differences are observed in the character and severity of the paroxysm. Ordinarily two varieties are recognized, the *petit mal* or slight attack, and the *grand mal* or severe seizure. The first is unattended by marked spasm or agitation; the latter is characterized by more or less violent tonic and clonic convulsions. These divisions are, however, not regarded as sufficiently precise by those who have studied the disease in question with care and precision, and more minute classifications of the phenomena of the epileptic paroxysm have accordingly been made. The one which I have used in my lectures at the Bellevue Hospital Medical College for several years past is less complex than some others, and embraces all the known varieties. It is as follows:

1. Momentary unconsciousness without marked spasm.
2. Unconsciousness with evident though local spasm.
3. Unconsciousness with general tonic and clonic convulsions.
4. Irregular or aborted paroxysms.

Besides these several varieties, there are certain accompaniments, such as mania and paralysis, which will require consideration.

1. *Momentary Unconsciousness without Evident Spasm.*—The patient is perhaps standing, engaged in conversation, when a momentary blank in his mental processes occurs. It probably does not attract attention; it is instantaneous, disappears, leaving no feeling of discomfort after it, and there is an almost immediate continuance of his thoughts and speech.

Or he may be walking in the street when the accession

occurs. He loses himself for an instant, but he continues to walk, and does not even stagger.

In somewhat more severe seizures, if conversing, he stops suddenly, stares vacantly but fixedly for a moment, and may drop any thing which he has in his hand.

If walking, his steps are arrested for an instant, he staggers and would fall but for the quick return of consciousness.

Such is the general character of these absences, faints, spells, etc., as they are popularly called; varying, however, according to the circumstances of the moment and the condition of the patient. They frequently exist for a long time without the patient paying much attention to them. In a gentleman now under my charge they occurred several times in the course of the day when walking, riding on horseback, sitting quietly in his library, engaged in conversation, or eating. He did not consider them of much importance, and was surprised when I informed him they were epileptic. The continuity of his acts was scarcely interrupted, and those about him never noticed that any thing was wrong.

In the case of a young lady they occur generally at the dinner-table. She drops her knife and fork, looks steadily to the front, ceases to eat, and in about two seconds resumes her occupation with a long-drawn inspiration. Those near her observe that her countenance becomes very pale, and that she does not hear or see.

Sometimes these attacks, slight as they are, are followed by pain in the head, vertigo, confusion of ideas, numbness, and other evidences of nervous derangement, which may last for several hours, and which become more pronounced as the epileptic condition becomes more confirmed.

2. *Unconsciousness, with Evident though Local Spasm.*— In this variety the loss of consciousness is of longer duration than in the preceding, and is attended with convulsions light in character, but yet apparent to those around. The

eyes are fixed, as in the first variety, the mind becomes a blank, and there is a sensation of vertigo immediately before the loss of consciousness, and at the time of its restoration. The face usually becomes pale first and then red, or either of these conditions may occur without the other being observed.

The spasms may be very slight. Sometimes there is momentary strabismus, at others retraction of the angles of the mouth on one or both sides, rotation of the head or a sudden drawing of it backward, or the tongue is thrust forward and the jaws close on it, inflicting slight injury. Again, the chair in which the patient may be sitting is pushed back with some force, and the body is bent forward, or the muscles of the neck may be affected, and the circulation thus interrupted in the veins of the neck, causing a dark hue of the complexion.

Sometimes the spasms have an appearance of being volitional. A patient under my charge tugs violently at his hand; another walks about the room, but without taking any determinate course; a young lady leaves her chair and stands upon another one at some distance from her, and another talks all kinds of gibberish. My experience of such cases is in accordance with that of Reynolds,[1] to the effect that there is no recollection of these acts. These attacks are often preceded by prodromata of various kinds. The duration never exceeds a minute, and is generally much less.

3. *Unconsciousness, with General Tonic and Clonic Convulsions.*—Prodromata may or not be present. In any event the paroxysm occurs suddenly. The first circumstance may be a cry of a very peculiar character, somewhat resembling the bleating of a young lamb. The eyes become fixed, and the patient falls to the ground, usually with a bound, as if he is shot. The loss of consciousness occurs with the cry or the fixedness of the gaze.

[1] System of Medicine, vol. ii., p. 261, Art. Epilepsy.

The muscles are now thrown into a state of tonic contraction; the respiration is impeded, or altogether arrested; the face, if at first pale, becomes dark; the pupils are dilated, and sensibility is entirely abolished.

Careful examination of a patient in this stage of the paroxysm reveals some important features: the body is rigid, but is usually inclined more to one side than the other, in the position of a tetanic patient with pleurosthotonos; the eyes are open, and are twisted to one side; the face is likewise more retracted on one side than the other; the sterno-cleido-mastoid muscles, and others of the neck, stand out like thick cords; the carotids throb with force; the veins of the head and neck are turgid with black blood, and the pulse is usually weak and fluttering.

After this stage has lasted for a period varying from two or three seconds to half a minute, a great change ensues. The unconsciousness continues, but the general tonic spasm relaxes, and clonic convulsions take its place. These are general, but are ordinarily more strongly marked on one side of the body than on the other. The muscles of the face are alternately contracted and relaxed; the tongue is often thrust between the teeth, and, the jaws being closed upon it, it is terribly injured; the upper and lower extremities are in a state of continued agitation, and the contents of the bladder, rectum, and vesiculæ seminales, may be evacuated.

The respiration is forced and irregular, froth issues from the mouth, and, if the tongue has been bitten, it is colored with blood.

The muscles of the neck do not relax to any considerable extent; consequently the veins remain distended, and the face continues to be livid. The pupils oscillate, sometimes being dilated and then contracted, or one may be contracted and the other dilated. The heart beats with great irregularity, both as to force and frequency.

This stage may last from a few seconds to five minutes.

Cases of longer duration are on record, but they are exceedingly rare.

The third stage of the paroxysm is characterized by the gradual return of consciousness. The patient, though still somewhat convulsed, looks around him and gives evidence of returning sensibility in other ways. The pupils cease their disorderly movements, and are contracted; the respiration and pulse become more regular, and he may even attempt to speak. It often happens that little spots of extravasated blood make their appearance under the skin of the forehead, eyelids, cheeks, and sometimes on the neck and breast. These disappear in a few days.

The duration of this stage is from a few seconds to four or five minutes, and it is often so slightly marked as to escape observation.

With the cessation of the convulsive movements the stage of stupor usually supervenes, though it may be entirely absent, especially in old cases of epilepsy. During this stage there are sometimes clonic spasms of no great degree of severity. It may last a few minutes or several hours. When the patient arouses from it, he generally has headache, and a feeling of lassitude and soreness of the muscles, from the violent contractions they have undergone.

4. *Irregular or Aborted Paroxysms.*—In these it may happen that the loss of consciousness is not complete, or that the patient has convulsive movements partial in character and accompanied simply by vertigo, or he may have unconsciousness lasting for an hour or more, during which he performs automatic acts, of which he has no recollection, but which are not accompanied by any movements that can properly be called spasmodic.

In his interesting lecture on "Apoplectiform Cerebral Congestion," Trousseau [1] cites a number of cases which were clearly instances of irregular or aborted epileptic paroxysms. Among them is that of a magistrate whose sister was an

[1] Op. cit., Bazire's Translation, p. 19, *et seq.*

inmate of a lunatic asylum. He was president of a provincial tribunal. One day he got up all of a sudden, muttered a few unintelligible words, and went to the deliberating-room. The usher followed him, and saw him make water in a corner. A few minutes afterward he returned to his seat, and again listened with intelligence and attention to the pleadings momentarily interrupted. He had no recollection of the incredibly incongruous act he had committed. This gentleman belonged to a literary society, which held its meetings at the Hôtel de Ville, of Paris. At one of these, during the discussion of an important historical point, he was seized with vertigo. He ran quickly down to the Place de Hôtel de Ville, and walked about for a few minutes on the quays, avoiding with success both carriages and the passers-by. On recovering himself he perceived that he had come out without his great-coat and his hat. He therefore returned to the meeting, and resumed with a perfectly-lucid mind the historical discussion in which he had already taken a very active part. He retained no recollection whatever of what had occurred between the beginning of the attack and the moment he recovered himself.

Many cases similar to these might be cited from other authors. From a number which have happened in my own experience I adduce the following :

J. H. consulted me for epilepsy in the summer of 1869. His ordinary attacks were of the fully-developed form ; but upon two occasions they were different from any with which he had previously been affected. On one of these, while overlooking some workmen, he was observed to put his hand to his head, and then suddenly to run toward a fence, which he speedily climbed. Jumping down into the back-yard of the adjoining house, he seized a stick of wood near by, and made a furious onslaught on the door and windows. While thus engaged he was seized by several men, and forcibly held notwithstanding his struggles. While thus being restrained he recovered his consciousness, but had no

recollection of any thing which had taken place after he had put his hand to his head, which action he said was due to severe pain with vertigo. The duration of the attack was not over three minutes.

On the other occasion he was seized with pain and vertigo while engaged in paying a bill at a coal-yard. He rushed into the street, and began to turn rapidly round. He was seized and held till he recovered his consciousness. This attack lasted about four minutes.

Subsequently he had a similar paroxysm in my consulting-room. His face suddenly became very pale, his eyes were fixed, and his pupils oscillated. Suddenly he rose from the chair, grasped the mantel-piece for an instant, and then rushed violently around the room, throwing his arms about, and uttering a peculiar inarticulate cry. I made no attempt to restrain him, and in about two minutes he became calm. During the whole paroxysm his face was pale, and at its close the pupils were dilated. He had no recollection of any thing which had occurred after he rose from the chair, but was conscious then of vertigo.

Another case is that of a girl brought to my clinic at the Bellevue Hospital Medical College during the summer of 1869. She had been severely injured in the skull by a fall against a mass of old iron. Necrosis subsequently ensued, and several large pieces of the external table were exfoliated. While before the class, she started to her feet, and walked several times around the closed area. She was unconscious, and to all appearance insensible. When the paroxysm was over, she returned to her seat. The duration did not exceed a minute, and there was no excitement or delirium.

Cases such as these are sometimes classed as epileptic mania, but it is better to restrict this term to those paroxysms of mental aberration which come on after a true epileptic attack.

Epileptic fits may take place at night during sleep, and the patient be unaware of their existence, unless he in-

flicts some injury on himself, such as biting his tongue, or is told of their occurrence by persons who may be in the same room with him. In two hundred and six of my cases the period of access is noted, and of these forty-seven were nocturnal, and one hundred and fifty-nine diurnal.

In the intervals between the paroxysms epileptics often exhibit certain evidences of disordered mental, sensorial, and motor functions. Thus, as regards the first category, the memory may be impaired, and there may be diminished mental power. There are, however, many exceptions to this rule; and, even where there have been a great many attacks, the mind may preserve its normal degree of integrity. As Reynolds remarks, in regard to this point: "A patient may be epileptic and a lunatic; he may be epileptic and asthmatic, but there are some epileptics whose minds are as healthy as their lungs; and, so far as the natural history of epilepsy is concerned, it is a mistake to derive it from complicated cases." Still, in the majority of cases, it will be found that the mind sooner or later becomes involved, and it sometimes happens that a single attack causes marked intellectual deterioration.

Derangements of sensibility are common from the beginning. Headache, a feeling of constriction around the forehead, and occasionally a pain at the back of the head, are noticed. Vertigo is also frequently present, as are also sensations of numbness in different parts of the body. The pupils are almost invariably dilated.

The motor power of the patient is generally weakened without there being any decided paralysis. Twitchings of the muscles are not uncommon, and there is often a general excitability of the reflex faculty of the spinal cord, by which jerkings of the limbs are produced by slight excitations.

In examining with the ophthalmoscope the fundus of the eye in epileptics, we can often detect evidences either of cerebral congestion or of anæmia, and thus obtain valuable indications for treatment. During the last two years, in my

lectures at the Bellevue Hospital Medical College, I have constantly insisted on this point, and in my cliniques have exhibited several cases in which I had been guided to successful treatment by the ophthalmoscope. Drs. Köstle and Niemetshek,[1] of Prague, consider that the brain in epileptics is always anæmic, and that this condition is invariably found by ophthalmoscopic examination. According to these observers, the venous pulse is produced when the eye is made anæmic, and they assert that the retina is anæmic, and that there is consequently venous pulsation in every case of epilepsy. That this opinion is erroneous, both as to facts and inferences, I am very sure. Venous pulsation, so far from being indicative of anæmia, really shows the existence of the very opposite condition. My observations are, however, to the effect that venous pulsation is present in many cases of epilepsy, and that it accompanies dilatation of the veins.

There is no invariable rule as regards the occurrence of any particular form of epilepsy in the same person. It thus often happens that all the varieties of paroxysm mentioned, except the irregular or aborted form, which is more rare, are met with in one individual. The more severe form may occur at longer intervals, and the milder forms more frequently. As regards frequency, there are great variations. Some patients go a year or more without attacks, while others have several every day. It generally happens that the intervals become progressively shorter. As a rule, attacks of the milder forms are more frequent than the fully-developed paroxysm, and attacks of the latter are milder, as they are more frequent.

Mania is sometimes a consequence of epilepsy. It comes on after the attack, and is rarely of more than a few minutes' duration. Those cases in which it precedes the paroxysm, and lasts several hours or days, are cases of

[1] Prager Vierteljahrschrift, H. 106, 107, 1870, and Quarterly Journal of Psychological Medicine, January, 1871, p. 128.

mania conjoined with epilepsy—a combination which, as every insane asylum shows, is not uncommon. The mania of epilepsy is usually of a very exalted character, and during its existence the subject may commit homicide or other crimes.

The mental state of epilepsy has been well studied by Falret,[1] and a very interesting case has been recently reported by Dr. Thorne,[2] in a paper entitled "Masked Epilepsy." In this instance the patient often returned to his home without being able to give any account of what he had been doing or where he had been. During these attacks he was frequently the subject of that form of mental derangement called kleptomania. Generally they ensued on paroxysms either of the *grand* or *petit mal*, but sometimes they were substituted for the regular seizures. He had no recollection of what transpired during the attacks. Sometimes he was furiously excited in them, and would endeavor to injure himself and others in his blind rage.

The medico-legal relations of epilepsy do not, however, come within the scope of the present treatise.

Paralysis may follow epilepsy, but, unless the case is complicated with some organic difficulty of the brain or spinal cord, the loss of power is temporary.

Causes.—Among the predisposing causes of epilepsy hereditary tendency stands first. Reynolds[3] states that, in about one-third of the cases under his observation, hereditary taint existed. He does not, by this statement, however, mean to assert that epilepsy existed in one-third of the parents, but that some disease of the nervous system, more or less closely allied to epilepsy, was present in either the parents, the grandparents, the aunts, uncles, brothers, or sisters. Only twelve per cent. of his cases

[1] De l'État Mental des Épileptiques. Archiv. Gén. de Méd., Décembre, 1860, et Avril, et Octobre, 1861.

[2] St. Bartholomew's Hospital Reports, 1870.

[3] Op. cit., p. 253.

gave a distinct history of epilepsy in either branch of their families.

Herpin,[1] of sixty-eight cases, found that ten were descended from epileptic ancestors.

Delasiauve,[2] of three hundred cases, found decided evidence of hereditary tendency in thirty-three. In one hundred and sixty-seven there were no data, and in one hundred and twenty hereditary taint was denied. Of the thirty-three cases, five were descended from epileptic ancestors.

Sieveking[3] found that hereditary influence was present in 11.1 per cent. of his cases.

In my own experience I have notes in regard to this point in one hundred and seventy-one cases. Of these, twenty-one had epileptic fathers, mothers, grandparents, uncles, aunts, brothers, or sisters, and twenty-four had relatives insane, hysterical, cataleptic, affected with severe neuralgia, or of remarkably irritable nervous systems.

Sex does not appear to exercise any appreciable influence as a predisposing cause. Of two hundred and six cases noted by myself, one hundred and ten were in males and ninety-six in females. Other authors have, however, had directly opposite experience.

Age has a very decided influence. Reynolds gives the following table of one hundred and seventy-two cases collected by himself:

Age at Commencement.	Males.	Females.	Total.
Under 10 years	10	9	19
Between 10 and 20 years	66	40	106
Between 20 and 44 years	25	20	45
Over 45 years	1	1	2
Total	102	70	172

[1] Du Pronostic et du Traitement Curatif de l'Épilepsie, Paris, 1852, p. 325.

[2] Op. cit., p. 189.

[3] On Epilepsy, etc., London, 1858, p. 74.

My own cases were as follows:

Age at Commencement.	Males.	Females.	Total.
Under 10 years	12	13	25
Between 10 and 20 years	62	45	107
Between 20 and 45 years	29	32	61
Over 45 years	7	6	13
Total	110	96	206

It is thus seen that the period of life between ten and twenty years is that at which epilepsy is most apt to occur. The experience of others is to the same effect. The influence of temperament has been thought important by some writers. But, aside from the different opinions entertained relative to the characteristics of the temperaments, it is by no means established that, even when strictly defined, temperament exercises any effect as a predisposing cause. I have no accurate records on this point, though so far as my memory serves me I have observed no marked predominance of epileptics with any temperament.

The exciting causes may very properly be classified as psychical, eccentric, general organic changes, and physical influences. Relative to the influences of these causes, Reynolds gives the following table:

Nature of Cause.	No. of Cases.
I. Psychical—such as fright, grief, worry, overwork	29
II. Eccentric irritation—dentition, indigestion, venereal excesses, dysentery, etc.	16
III. General organic changes—fatigue, pregnancy, miscarriages, rheumatic fever, scarlet fever, diphtheria, pneumonia	9
IV. Physical influences—blows on head, falls, insolation, cuts	9

In my own cases no cause could be assigned in one hundred and four. The remaining one hundred and two cases were, according to the evidence received, caused as follows:

Fright	5
Anxiety	4
Grief	6
Over-mental exertion	17
Dentition	11
Indigestion	11
Venereal excesses	15
Menstrual derangement	10
Blows on head	7
Falls	3
Sunstroke	2
Scarlet fever	2
Measles	1
Diphtheria	2
Pregnancy	3
Syphilis	3
	102

Diagnosis.—The diagnosis of epilepsy presents no difficulties to the careful observer. It may, however, be confounded with several conditions, the principal of which are cerebral congestion, cerebral hæmorrhage, hysteria, the convulsions of infancy and of Bright's disease, poisoning by opium and alcohol, syncope, and with the convulsions of epileptiform character which occur in the course of certain organic diseases of the brain.

The diagnosis from cerebral congestion and cerebral hæmorrhage has already been given in the chapters treating of these affections. In hysteria, the convulsions, which are sometimes epileptiform in character, are preceded or accompanied by other evidences of the hysterical state. Consciousness is rarely entirely lost, the tongue is not bitten, and there is no subsequent stage of stupor.

The convulsions of infancy not epileptic are not repeated but from a readily-ascertained exciting cause, such as dentition, indigestion, falls, etc. So far as the paroxysm is concerned, I know of no specific points of difference; but it must be recollected that the paroxysm is not the only fea-

ture of epilepsy, and that it is the only feature of infantile convulsions. These latter may pass into epilepsy; but, if they do not, I have never been able to find a single case in my experience in which epilepsy ensuing in adult life has been preceded by the ordinary infantile convulsions. In Bright's disease, though the convulsions may be epileptiform in character, coma is the principal feature, and the history of the case will further serve to render the diagnosis exact. The same remarks are applicable to poisoning by opium and alcohol.

From syncope epilepsy is distinguished by the facts that the loss of consciousness is sudden and complete, that the pulse is not feeble, and that recovery is rapid. These remarks apply to the milder attacks without convulsions. From the more severe forms of the paroxysm the distinction is too obvious to require amplification.

In organic diseases of the brain, such as tumors, softening, sclerosis, etc., the accompanying symptoms, pain, paralysis, tremor, imbecility, difficulties of speech, and derangements of the special senses, will serve to distinguish them from epilepsy.

Epilepsy is often assumed by designing persons for purposes of fraud. In such cases the pretender usually overacts his part; his sensibility is not abolished, as may readily be ascertained by putting the end of the finger on the conjunctiva, and the size of the pupils is not altered.

Prognosis.—The prognosis depends to a great extent on the duration of the disease. Recent cases can often be cured, but those which have lasted for several years are rarely brought to a favorable termination. Among the other unfavorable elements are the existence of hereditary influence, the beginning of the disease late in life, the presence of material mental weakness, and the existence of long intervals between the attacks.

As regards the probability of the supervention of any form of intellectual derangement or debility, the most im-

portant ascertained point is that the mild paroxysms unattended by convulsions are more productive of mental decay than the severe form of seizure. The occurrence of the first attack late in life is likewise a predisponent to dementia.

I have never, in my own experience, known death to take place during a paroxysm of true epilepsy; such cases, however, do occur. Usually, however, some intercurrent affection carries the patient off, though even with this liability life is sometimes astonishingly prolonged. I am acquainted with the case of a lady who is now sixty-five years of age, and who, since her tenth year, has averaged six paroxysms daily, all of the severest character. Her mind is almost entirely gone, but physically her health is excellent, and to all appearance she may live twenty years longer.

I am not aware of any exact observations tending to show the relative danger to life of attacks of the milder and severer forms; though it is reasonable to suppose that, so far as regards the occurrence of death during the paroxysm, the convulsive form is more fatal.

Morbid Anatomy.—In post-mortem examinations of persons dying epileptic, abnormal conditions are found in every part of the brain and spinal cord. Some of these lesions are undoubtedly secondary, others unessential, while those which may be considered primary vary in their seat and character. In a great many cases, perhaps the majority, no lesions are discoverable.

No one has been more thorough in the search for the essential cause of epilepsy than Schroeder van der Kolk;[1] though his observations can scarcely be regarded as yielding conclusive results, they serve to show, when taken in connection with the pathology of the disease in question, that its seat is mainly in the medulla oblongata, with secondary im-

[1] On the Minute Structure and Functions of the Medulla Oblongata, and on the Proximate Causes and Rational Treatment of Epilepsy. New Sydenham Soc. Translation, London, 1859.

plication of other parts of the cerebro-spinal nervous system. Oftentimes, in accordance with other pathologists, he found nothing to account for the affection, but at others he found hardening and contraction of the medulla oblongata, and again degeneration of the brain either as a consequence or cause of the disease. Microscopical examination sometimes showed him the medulla indurated, sometimes softened, and, as a constant phenomenon, "whether the patient died in or out of the fit, great redness and vascular tension in the fourth ventricle, penetrating into the medulla oblongata sometimes to a considerable depth." These appearances were due to enlargement of the blood-vessels, as was shown by microscopical measurements. It is probable, however, as Schroeder van der Kolk asserts, that the lesions in question are the results, and not the causes, of the paroxysms. Still they suffice to indicate the main seat of the disease to be the medulla oblongata.

Other observers have not so uniformly found this enlargement of the blood-vessels of the medulla. In three cases of death occurring in epileptics, in which I have had the opportunity of making post-mortem examinations, they certainly did not exist, nor was there any other lesion detected by the most careful microscopical exploration. In one other case the vessels of the medulla oblongata were enlarged, and there was amyloid degeneration of the pituitary body.

Pathology.—The points which may be considered as to some extent established relative to the pathology of epilepsy are briefly summarized as follows by Reynolds:[1]

"1. That the seat of primary derangement is the medulla oblongata and upper portion of the spinal cord.

"2. That the derangement consists in an increased and perverted readiness of action in these organs, the result of

[1] Op. cit., p. 275, and more fully stated in his Treatise on Epilepsy, its Symptoms, Treatment, and Relations to other Chronic Convulsive Diseases, London, 1861, chapter v., p. 238.

such action being the induction of spasm in the contractile fibres of the vessels supplying the brain, and in those of the muscles of the face, pharynx, larynx, respiratory apparatus, and limbs generally.

"By contraction of the vessels the brain is deprived of blood, and consciousness is arrested; the face is or may be deprived of blood, and there is pallor; by contraction of the vessels which have been mentioned, there is arrest of respiration, the chest walls are fixed, and the other phenomena of the first stage of the attack are brought about.

"3. That the arrest of breathing leads to the special convulsions of asphyxia, and that the amount of these is in direct proportion to the perfection and continuance of the asphyxia.

"4. That the subsequent phenomena are those of poisoned blood, i. e., of blood poisoned by the retention of carbonic acid, and altered by the absence of a due amount of oxygen.

"5. That the primary nutrition-change, which is the starting-point of epilepsy, may exist alone, and epilepsy be an idiopathic disease, i. e., a *morbus per se*.

"6. That this change may be transmitted hereditarily.

"7. That it may be induced by conditions acting upon the nervous centres directly, such as mechanical injuries, overwork, insolation, emotional disturbances, excessive venery, etc.

"8. That the nutrition-changes of epilepsy may be a part of some general metamorphosis, such as that present in the several cachexiæ—rheumatism, gout, syphilis, scrofula, and the like.

"9. That it may be induced by some unknown circumstances determining a relative excess of change in the medulla during the general excess and perversion of organic change occurring at the periods of puberty, of pregnancy, and of dentition.

"10. That it may be due to diseased action, extending

from contiguous portions of the nervous centres or their appendages.

"11. That the so-called epileptic aura is a condition of sensation or of motion, dependent upon some change in the central nervous system, and is, like the paroxysm, a peripheral expression of the disease, and not its cause."

While admitting the correctness of these conclusions, they do not, in my opinion, tell the whole story of the theory of epilepsy. In very many memoirs Dr. Brown-Séquard has pointed out the dependence of the affection upon injuries of the upper part of the spinal cord, and upon irritations existing in various parts of the body. His researches, and facts observed every day by physicians who see many cases of epilepsy, show very conclusively that the starting-point is often in the sympathetic nerve—the nerve by which the calibre of the blood-vessels is regulated.

Neither can I accept the view that the first intra-cranial condition producing a paroxysm is in all cases spasm of the blood-vessels and the consequent deprivation of the blood-supply to the brain. On the contrary, I am very sure that the primary state is often paralysis of the cerebral blood-vessels and resulting hyperæmia. By this condition the medulla oblongata is thrown into a state of over-excitation, giving rise to convulsions, and consciousness is lost from the fact that the hemispheres participate. That convulsions epileptiform in character may be produced both by cerebral anæmia and cerebral hyperæmia, when either condition involves the medulla oblongata, is a fact which experiment has abundantly established, and that loss of consciousness follows either condition involving the hemispheres is equally certain. We have, consequently, two kinds of epilepsy—the one due to anæmia, the other to congestion—and it is to this fact that is due the circumstance that sometimes the paroxysms are prevented by measures which tend to increase the amount of blood in the brain, and at others by remedies which exercise a contrary influence. The ex-

istence of the two species of epilepsy is likewise shown by ophthalmoscopic examination—a point upon which I have already insisted.

During natural sleep the amount of blood is, as I have elsewhere shown, decreased from the quantity which circulates in the cerebral blood-vessels during wakefulness. Epilepsy occurring during sleep is therefore of the anæmic variety. But it often happens that sleep passes gradually into stupor, from the fact that causes tending to increase the flow of blood to the brain, or to arrest its passage from this organ, are in operation. In such cases epilepsy of the congestive variety may be induced.

In those cases in which the tongue is bitten the medulla oblongata is probably always in a condition of hyperæmia; and this state, as Schroeder van der Kolk has very conclusively shown, is mainly in the course of the roots of the hypoglossal nerve. The intermissions between the attacks are ingeniously explained by the same able observer, by likening the cells of the medulla oblongata to Leyden jars charged with electricity, or to the electrical organs of the conger-eel and torpedo. After being discharged, time is necessary for the reaccumulation of sufficient electricity to discharge them again; and, when the cells of the medulla have once discharged themselves in an epileptic convulsion, a period must elapse before another access can take place.

The foregoing remarks apply in the main to that form of epileptic seizure characterized by convulsion. In the imperfectly-developed attacks the implication of the medulla oblongata must be very slight, the hemispheres being the organs mainly affected, and the condition being sometimes anæmic, at others hyperæmic.

It must not be supposed, from what has been said, that simple cerebral anæmia and simple cerebral congestion, attended with epileptiform convulsions, are identical with the anæmia and congestion of epilepsy. This disease is cerebral anæmia or congestion with another element, the exact na-

ture of which we do not understand, but which is certainly of such a character as to constitute the main differential point between epilepsy and any other affection.

Treatment.—To attempt the consideration of all the means which have been employed in the treatment of epilepsy would be a hopeless task. Their mere mention would require several pages of this treatise. I shall therefore content myself with detailing the methods which I have found most useful.

First among remedies are the bromides of potassium, sodium, or lithium. The first should be given to adults in doses of at least fifteen grains three times a day, and is generally required in larger quantity. It acts very much better when largely diluted with water. A saturated solution contains about thirty grains to the drachm, and a half a teaspoonful or more should be mixed in at least a gill of water. Latterly I have used the bromide of sodium in doses a little smaller. It is, I think, more readily assimilated, and is less apt to produce gastric irritation, but, in other respects, its action is similar to that of the corresponding potassium salt. The bromide of lithium has a more immediate action; but I have not succeeded in obtaining any continuous effects from it in epilepsy which I could not derive from the potassium or sodium salt, and its great cost is a bar to its lengthened administration.

It must be clearly understood that the bromide is to be taken for at least a year, and in most cases longer, before its administration is stopped. After the initial dose has been given for about two months, if there are no symptoms indicating bromism, I increase the doses by one-half, if there has been no paroxysm in the mean time. If there have been paroxysms, I increase by one-half after each paroxysm until they are arrested, or until I am convinced that the bromide is inefficacious or injurious. In the case of a gentleman from Cincinnati, I began with twenty grains three times a day; he still had attacks; I increased the

doses to thirty grains, with little or no effect; then to forty-five grains, and, as he still had an occasional fit, I increased the doses to four a day. He therefore took one hundred and eighty grains a day, and then his paroxysms ceased. I have never given beyond this quantity, and, if it had not proved successful, I should have abandoned the idea of arresting the disease with the bromide of potassium. The gentleman in question has had but one attack during the last three years, and this was the result of his suddenly omitting to take his medicine for several days. When he was thoroughly under the influence of the drug, I reduced the doses, and he now takes thirty grains three times a day. He had been previously treated with smaller doses, with good effect at first, but for some time before he came under my care they had lost their influence.

With either bromide I usually conjoin the oxide of zinc in doses of two grains three times a day. In several cases in which large doses of the bromide had failed, the paroxysms were arrested when the oxide of zinc was given in addition. In one case in which the bromide aggravated the disease, and in which the oxide of zinc was ineffectual, the patient, a young lady of this city, was cured after taking the lactate of zinc in doses of four grains three times a day. In no other case has this salt produced the least effect in my hands.

I rarely continue the oxide of zinc for a longer period than two months, for the reason that it appears, after that period, to produce a cachexia, manifested by loss of appetite, anæmia, and general debility.

With the bromide I generally administer strychnia in doses of the thirty-second to the twenty-fourth of a grain, for the purposes of a tonic, and for counteracting, to some extent, the debilitation produced by the bromide.

When the opportunity affords, I always make use of the constant galvanic current, applying it to the brain and sympathetic nerve. There should ordinarily be three *séances* a

week, each of about ten minutes. For one-third of this time I pass the current antero-posteriorly, one pole being placed on the back of the neck, and the other on the forehead; for another third, one pole is placed on each mastoid process, and for the other, one on the sympathetic nerve in the neck, and the other on the spinal column at about the first dorsal vertebra. The current should be derived from ten or fifteen Smee's or Daniell's elements.

Occasionally I have employed setons—a skein of silk, or a piece of thin india-rubber, being passed through the skin of the nucha.

The results obtained are shown in detail in the following synopsis:

One or other of the bromides was given in two hundred and eighty-six cases.

The condition of the patient was aggravated in nineteen cases; all were of the nocturnal form, and one has been reported in detail.[1]

No appreciable effect was produced from doses of one hundred and twenty grains a day in twenty cases, and the administration was stopped. Eleven of these were nocturnal.

No appreciable effect from doses of one hundred and sixty grains a day in four cases, and the medicine was accordingly discontinued.

The severity and frequency of the fits were diminished, but they were not arrested, with doses ranging from forty-five to one hundred and sixty grains daily, in one hundred and fifty-two cases.

No fits occurred while the bromide was being taken, in ninety-one cases. Of these latter the bromide has been stopped for over six months, and there has been no return of the fits in sixty-five cases.

In the remaining twenty-six cases the bromide has to

[1] A Case of Epilepsy due to Cerebral Anæmia, JOURNAL OF PSYCHOLOGICAL MEDICINE, April, 1868, p. 368.

be continued in order to prevent the recurrence of the paroxysms.

Of the two hundred and eighty-six cases in which the bromide was administered, the seizures were therefore entirely arrested or lessened in frequency or severity in two hundred and forty-three, while in forty-three it was either positively injurious or without effect.

Of the two hundred and forty-three completely or partially successful cases, the bromide was combined with oxide of zinc in seventy-four.

Of these seventy-four cases, fifty-two were, of the sixty-five, thoroughly successful cases.

The primary galvanic current was used to the brain, medulla oblongata, spinal cord, and sympathetic nerve, in one hundred and thirty cases.

Of these one hundred and thirty cases, fifty-nine were among the entirely successful cases.

Setons were employed in fifteen cases.

Of these fifteen cases, three were among the completely successful cases.

These data go to show that the treatment which in my hands has been productive of the best results, is that in which one of the bromides, oxide of zinc, and the primary galvanic current, were conjointly used.

In forty-three cases the bromide was productive either of an injurious effect, or was without influence. The majority of these were nocturnal.

Five of these unsuccessful cases were subsequently treated with other remedies. One was cured by the lactate of zinc; one by changing the time for sleeping, and by the administration of iron, quinine, and porter; and three by strychnia, in small doses, as recommended by Mr. Tyrrel.

Of the one hundred and fifty-two cases in which the bromide was only partially successful, four were cured by strychnia.

I have not considered a case as fully cured till the pa-

tient has been for six months without a fit after ceasing to take medicine. In all, out of the two hundred and eighty-six cases cited, seventy-four were of this category. Thus far there have been, so far as my knowledge extends, but seven relapses. In two of these the fits recurred ten months after ceasing to take the medicines; in two fourteen months; and in three fifteen months. In all of these the medicines were at once resumed, and they are now under treatment. None of these relapses have as yet been from the strychnia treatment. My experience with this agent, though thus far satisfactory, is not yet sufficiently extensive to warrant the expression of a decided opinion as to its value when compared with the bromides. I am inclined, however, to think that it is more efficacious in the nocturnal form of epilepsy than in the diurnal, and in the non-convulsive rather than in the convulsive varieties. A point connected with the treatment with the bromides must not be overlooked, and that is the cachexia which so generally attends their administration in large doses. In a memoir,[1] published over two years ago, and which has been cited in another connection, I brought forward several cases in which this cachexia had been produced. Greatly-increased experience has confirmed the opinion there expressed, that it never causes any permanently ill effects, though I have frequently seen great constitutional disturbance induced. In three cases large carbuncles were caused, and in a few I have been obliged to suspend for a time the administration of the medicine.

I am very sure that bromic cachexia is favorable to the eradication of the epileptic tendency, and I therefore endeavor to produce it as soon as possible. It appears in many cases to alter the whole organism of the patient to such an extent as to leave him, when it disappears, with his nutritive processes and his proclivities so modified that epilepsy is no longer possible. The physician will require all his firmness

[1] On Some of the Effects of the Bromide of Potassium when administered in Large Doses, JOURNAL OF PSYCHOLOGICAL MEDICINE, January, 1869, p. 46.

and courage to persevere in those cases in which the bromism is extreme. But he should not yield unless the phenomena are so intense, and the strength of the patient so reduced, as to excite his gravest apprehensions.

Five cases of epileptiform seizures, not included among the cases cited, have been under my charge, in which there was a decided syphilitic taint present. In these the bromide of potassium was not administered to any considerable extent, reliance being mainly placed on the iodide, given in gradually-increased doses. In three cases the result is unknown; in two the treatment was entirely successful.

My experience with trephining is limited to one case, in which the operation was performed by my friend Dr. Van Buren. It was unsuccessful, although there had been an injury of the skull, to which we thought the paroxysms might be due. In this case the bromide was only partially successful. It is included among the cases previously cited. The operation is, I think, entirely proper in cases apparently the result of injury to the cranium, and several successful instances are on record.

But, before resorting to any specific treatment for epilepsy, diligent search should be made for the cause, and this should be removed, if possible, without the least delay. Often an eccentric irritation, such as worms in the intestinal canal, implication of a nerve in an injury, disorders of menstruation, etc., can be discovered, without the removal of which a permanent cure is impossible. In several of the cases cited, success in the treatment was in a great measure due to acting on this principle.

The hygienic management of the patient is important. A large portion of the day should be passed in the open air, bodily exercise should be regular, but not excessive—the food should be nutritious, but neither exciting nor indigestible. The importance of avoiding every alimentary substance, calculated to cause gastric or intestinal irritation, cannot be overestimated. I have frequently seen parox-

ysms directly caused by nuts, dried fruits, pastry, heavy and badly-baked bread, excess in the use of alcoholic liquors, confectionery, and the like. The bowels must be kept regular. Baths should be frequently taken, but should not be so cold as to cause severe shock or physical depression. Turkish baths, I am inclined to think, are useful in many cases, particularly in those occurring in persons of full and gross habit of body.

Overheated and ill-ventilated apartments should be avoided. The clothing should be warm in winter and cool in summer. The mind should not be overtasked and the emotions must not be unduly excited.

Individual attacks may sometimes be prevented. One gentleman under my charge assures me that he can often dissipate the premonitory symptoms, and thus stop the development of the paroxysm by a strong exertion of the will. Another can arrest them sometimes by changing the position of his body. If standing, he lies down; if lying down, he rises suddenly and paces the room violently. Another stops them by putting salt in his mouth, and two can frequently prevent them by tightening straps which I have instructed them to keep constantly around the wrist. In all these cases there is an aura, and in the two latter it appears to start from the hand.

A short time since I instituted, at the New York State Hospital for Diseases of the Nervous System, a series of experiments with the chloride of sodium in the treatment of epilepsy, but, although the salt was given in large doses, it produced no marked effect. I was led to these investigations by the facts that the bromide of potassium, the bromide of sodium, and the chloride of potassium, had been employed with success, and I thought there was sufficient analogy to warrant the hope that the chloride of sodium might prove beneficial.

As regards other remedies for epilepsy, I have but little to say. Belladonna has never in my hands produced the

least effect, neither has digitalis, nor indigo, nor cotyledon umbilicus, nor any of the salts of copper. I might say the same thing of the other so-called remedies. Hydrate of chloral in three cases mitigated the frequency of the paroxysms, but only for a short time. In several other cases it was without effect; Calabar bean was slightly beneficial in one case.

The treatment during the paroxysm remains to be considered. It is simple, and, beyond a few obvious measures, consists in letting the patient alone. The head should be elevated, the collar and cravat loosened, a piece of soft wood put between the teeth so as to prevent injury to the tongue, and the patient so placed that he cannot fall or otherwise injure himself in his struggles. During the subsequent stupor he should be kept quiet. Bloodletting is never necessary, although it is recommended as proper in certain cases by Jaccoud.

CHAPTER III.

CATALEPSY.

Although there are no post-mortem appearances characteristic of catalepsy, the phenomena of the disease observed during life point to its seat in the brain and spinal cord. Like epilepsy, therefore, it is a symptom representing an unknown morbid change in the nervous centres.

Symptoms.—Catalepsy is an affection marked by the occurrence of peculiar paroxysms at regular or irregular periods. The seizures usually come on with suddenness, and are characterized by more or less complete suspension of mental action and of sensibility, and by the supervention of muscular rigidity, causing the limbs to retain, for a long time, any position in which they may be placed. The phenomena, therefore, relate to the mind, to sensation, and to motion.

The suspension of mental action is, in general, complete, but in some cases there are an imperfect consciousness and an ability to appreciate strong sensorial impressions. Thus, in a case quoted by Dr. Chambers from Dr. Jebb—which, however, was clearly a case of catalepsy complicated with hysteria—the patient, before emerging from the paroxysm, sang "three plaintive songs in a tone of voice so elegantly expressive, and with such affecting modulation, as evidently pointed out how much the most powerful passion of the mind was concerned in the production of her disorder, as indeed her history confirmed."[1]

[1] Art. Catalepsy, in Reynolds's System of Medicine, vol. ii., p. 100.

The aspect of a cataleptic patient is very striking. The eyelids are sometimes wide open, at others gently closed; the pupils are dilated, and do not respond to strong light; the respiration is slow, regular, but generally so feeble as to be perceived with difficulty; the pulse is usually almost imperceptible, but is rhythmical and sluggish; the face is pale, the mouth is half open, and the rigidity of the body, and the coldness of the extremities, add to the death-like appearance which impresses all beholders.

The cutaneous sensibility is ordinarily completely abolished. Pins may be stuck into the skin and they are not felt; but, owing to the abolition of the power of motion and of reflex action, it is possible that in some cases, at least, the patients would give evidence of sensation if they could. Cases are on record in which tears have been caused by excessive emotional disturbance excited by the words or actions of persons surrounding the patients, thus showing that the senses of sight and hearing were capable of being exercised. Such instances are, however, rare, and are probably imperfectly-developed paroxysms, or those complicated with hysteria or ecstasy.

The symptoms relating to the muscles are very remarkable. Coming on, as the paroxysm usually does, without warning of any kind, the patient is at once arrested in any act which is being performed, and the whole body assumes a condition of extreme rigidity. The power of the will over the muscles is lost, and the limbs preserve any position in which they may be placed by the by-standers. Thus, if the arm be raised from the side, it remains extended, and may keep this position for an hour or longer before it sinks slowly back to its original situation. No matter how awkward or irksome the position may be, it is retained till the exalted irritability of the muscles becomes thoroughly exhausted.

The ability to swallow is not lost, and the electric contractility of the muscles is not perceptibly affected one way or the other.

The paroxysm may last a few minutes or hours, or may be prolonged to several days.

The temperature of the body, in all the cases that have come under my observation, was reduced from two to four degrees below the normal standard, and in the extremities much more than this.

The paroxysm generally disappears with as much abruptness as marked its accession. A few deep inspirations are taken, the eyes are opened, or lose their fixedness, the muscles relax, and consciousness is restored. In fully-developed seizures the patient has no knowledge of what has transpired during the attack.

Seven cases of true catalepsy, uncomplicated either with hysteria or ecstasy, have been under my professional care. In two of these the seizures were more or less imperfectly developed, and strong sensorial excitations were, in a measure, perceived and recollected after emergence from the attack. But in every instance the character of the impression was misinterpreted. A bright light thrown upon the eyes with a mirror was spoken of as an "angel's wing which brushed across my face," and the scratch of a pin was remembered as "a piece of ice being drawn over the skin."

In these cases there was the consciousness of mental action during the paroxysm, but it was difficult for the patients to describe the thoughts which took place. They appeared to be somewhat of the nature of dreams. In both cases the muscular rigidity was well marked, but was not excessive, and appeared to be mainly manifested in the extensors. It was not difficult to extend the arm or the leg, but flexion required the exertion of a good deal of strength.

In the other five cases the paroxysms were completely formed. Consciousness was entirely abolished; there was no sensibility anywhere, and no reflex actions could be excited except those of deglutition. In one of these cases, seizures several times occurred in my consulting-room, and I had the opportunity of ascertaining the effect of electricity.

If the arm was extended, the strongest induced current I could apply to the biceps, though causing contraction, failed to procure flexion, but relaxation of the extensors was at once produced by the application to them of the primary current.

I likewise, in this case, repeatedly examined the fundus of the eye with the ophthalmoscope, and invariably found the choroids pale, and the retinal vessels straight and attenuated.

In none of these cases was there any knowledge of what passed during the paroxysms, and no consciousness of there having been any mental activity.

Cataleptic persons are usually of dull and sluggish mental and physical organization. Such has certainly been the case in all the instances that have come under my observation. The disease does not ordinarily show any decided tendency to become worse, either as regards the severity or frequency of the paroxysms, provided the exciting causes be avoided. On the contrary, there is often a well-marked natural tendency to spontaneous cure, or, at least, to a cure through the influence of purely hygienic influences, moral as well as physical.

In the majority of cases catalepsy is complicated with hysteria or ecstasy, and sometimes with epilepsy. Of this latter combination I have seen two cases, and in one of these ecstasy was also a feature. This case I have recently alluded to in another communication.[1] The patient was a young girl, was cataleptic on an average once a week, and epileptic twice or three times in the intervals. Five years previously she had spent six months in France, but had not acquired more than a very slight knowledge of the language—scarcely, in fact, sufficient to enable her to ask for what she wanted at her meals. Immediately before her cataleptic seizures, she went into a state of ecstasy, during which she recited poetry in French, and delivered harangues

[1] The Physics and Physiology of Spiritualism. New York, 1871, p. 55.

about virtue and godliness in the same language. She pronounced at these times exceedingly well, and seemed never at a loss for a word. To all surrounding influences she was apparently dead; but she sat bolt upright in her chair, staring at vacancy, and her organs of speech in constant action. Gradually, she passed into the cataleptic paroxysm, in which she usually remained for from one to three hours. Many cases of the combination of catalepsy with hysteria and ecstasy have become celebrated in other relations than those of true science.

Causes.—Among the predisposing causes, sex is the most efficient. All my cases were in females, and the instances of the disease occurring in males are very rare. Hereditary influence is likewise generally apparent. Of the seven uncomplicated cases under my observation, all had relatives affected with some well-marked disease of the nervous system. In two cases, there were near relatives insane; in two, the mothers were hysterical; in one, a brother was epileptic; in one, the father was similarly affected; and, in one, a sister was cataleptic. It rarely begins after the age of twenty-five. Of exciting causes, emotional disturbance stands first. Four of my cases were directly the result —one of fright, one of anger, one of grief, and one of the shock caused by a boy starting out suddenly from behind a door where he had been concealed. In one other case, the cause was worms in the intestinal canal; and, in the other two, I could not ascertain with certainty what the cause was, though I had strong reasons for suspecting it to be masturbation.

The **diagnosis** is not a matter of the least difficulty to any one who has even an imperfect knowledge of the phenomena, except, perhaps, as regards its discrimination from hysteria, that simulator of almost every nervous disease. In those cases complicated with hysteria, the distinction is of no importance; in others, the uniformity of the characteristics which indicate catalepsy, with a consideration of

the general history of the case, will serve to make the diagnosis sufficiently precise. It must, however, be borne in mind that the two diseases are near of kin, and that the discrimination is important more as a matter of abstract science than as one of any bearing on the therapeutics. It is, however, sometimes a matter of moment to distinguish between the cataleptic paroxysm and death. In former times, instances were not uncommon in which the mistake was made, to be discovered after life had really become extinct in the coffin. Such fatal errors would probably be impossible now with the stethoscope for examining the heart, the thermometer for determining the temperature, electricity for acting on the muscles, and, above all, the ability to place the limbs in positions which they maintain against the laws of gravity. Moreover, our knowledge of diseases in general is such as to enable us to determine with great certainty the course they are liable to take, and the manner in which death occurs in each.

Prognosis.—This is usually favorable, even in severe cases. All my patients recovered under the treatment to be presently mentioned.

There is scarcely any thing to say under the head of **morbid anatomy.** It has been stated[1] that, in the few cases in which post-mortem examinations have been made of persons dying while cataleptic or subject to seizures, the Pacchionian bodies were found enlarged, but I have been unable to trace the assertion to its source.

The **pathology** of catalepsy is very imperfectly known. The symptoms show that the brain and spinal cord are involved, and there is some evidence to show that they are in a state of anæmia. But there is a condition induced in these organs which is the essential feature of the disease, and of this we know nothing. There is a possibility that the affection may be a masked form of epilepsy, and this

[1] Art. "Catalepsie" in Nouveau Dictionnaire de Médecine et de Chirurgie pratiques, t. sixième, p. 456. Paris, 1867.

view is borne out by the fact that the treatment which is most successful in this latter disease is most efficacious in catalepsy.

Treatment.—The bromide of potassium, or one of the other bromides previously mentioned under the head of epilepsy, is the most efficient agent in the treatment of catalepsy. I have never yet failed to cure the disease with this remedy, combined with the oxide of zinc, and with the simultaneous use of strychnia and other tonics. I have never, however, had occasion to give it in larger doses than twenty grains, three times a day, or to continue it beyond eight months.

In no disease of the nervous system, not even excepting hysteria, is it more necessary that the mind should be brought under proper discipline and kept as far as possible from the operation of all causes calculated to promote emotional excitement. At the same time a well-regulated system of hygiene, as regards all the physical requirements of the body, is indispensable.

CHAPTER IV.

ECSTASY.

Though closely allied to catalepsy, ecstasy differs from it in several important particulars. One of the main points of difference is that the patient recollects the train of thought which has been going on during the seizure, and this of itself is sufficient to warrant their being separately considered. It often happens, however, that the two diseases alternate or coexist.

Symptoms.—In ecstasy there is muscular immobility rather than rigidity; the eyes are open, the lips parted; the face is turned upward, the hands are often outstretched; the body is erect and raised to its utmost height. A peculiar radiant smile illumes the countenance, and the whole aspect and attitude is that of intense mental exaltation.

The mind is so filled with some particular train of thought, that excitations of the senses, if of moderate intensity, are not perceived. We meet with this fact often in normal conditions, when the mind is deeply engaged in reflection, or when it is engrossed with some powerful emotion.

Most of the religious impostors who have at various times made their appearance, and many very sincere and devout persons, have been ecstatics.

In its combinations with catalepsy, chorea, and hysteria, ecstasy has frequently played an important part in the history of the civilized world—at one time, leading to a belief in witchcraft; at another, to demoniac and angelic pos-

session; at another to mesmerism and clairvoyance; and, in our day, to spiritualism. The consideration of these follies, though interesting, scarcely comes within the scope of the present treatise.

Causes.—Ecstasy, though not entirely confined to the female sex, is very much more common in women than in men. It appears to be produced in those who are of delicate and sensitive nervous organizations by intense mental concentration on some one particular subject—generally, one connected with religion, or some other abstract train of thought. It was formerly quite common among the inmates of convents, and is now not unfrequently met with at camp-meetings and spiritualistic gatherings.

There are no points about the **diagnosis** requiring special consideration, and the **prognosis** is always favorable, if the subject can be submitted to proper moral and physical treatment. As the disease is never fatal *per se*, we know nothing of its **morbid anatomy**. The pathology, as indicated by the symptoms, points to the implication of both the brain and spinal cord, but there is no satisfactory theory of the disorder other than that which refers it to cerebral and spinal preoccupation—a kind of setting of the current in one direction, whereby all other occupation is for the time prevented.

Treatment.—The means of treatment, though not differing essentially from those proper for catalepsy, require, nevertheless, special mention of some particulars. The influence of moral force in preventing and curing ecstasy is well marked, and many instances are on record in which epidemics of it have been arrested by arguments addressed to the fears of the subjects. I have several times aborted and prevented ecstatic manifestations by making preparations to cauterize the region of the spine with a red-hot iron.

A great deal can be done by giving as little notoriety to ecstatics as possible. They glory in the idea that they are

of sufficient importance to excite attention and discussion, and they are accordingly stimulated to continue their performances so long as they are noticed, and an air of mystery is attached to them.

Removal from all associations calculated to continue the exciting and morbid train of thought which has developed the disease under notice should, of course, be a point in the treatment.

Electricity, and the other measures of treatment recommended for catalepsy, will prove serviceable in ecstasy. By galvanization of the sympathetic nerve, I, on one occasion, immediately cut short a paroxysm of ecstasy, and, by continuing the practice every alternate day for about six weeks, effectually cured the patient, who for several years had been subject to seizures every two or three days.

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CHAPTER V.

CHOREA.

Although it is quite certain that several distinct affections are included under the term "chorea," these are analogous to each other, and, as we know little about the essential anatomical features of these disorders, and as they are allied by their symptoms, it will be advisable, for the present, to consider them together.

Symptoms.—Even in simple typical and uncomplicated cases of chorea, the symptoms exhibit great variety. They are connected mainly with the mind, with motility, and with sensibility, though, at the same time, the functions of organic life are generally more or less deranged.

Among the earliest symptoms of chorea are those referable to disordered brain-action. The character and disposition of the patient undergo a marked change, and there is, besides, from the first, a very decided impairment of mental vigor. The emotions are easily excited, and the temper becomes fretful and irritable. Hallucinations are not uncommon, and these are generally connected either with the sight or hearing. Sometimes both these senses are involved.

The sleep is generally disturbed by disagreeable dreams, sometimes reaching to the intensity of nightmare, and these are so vivid that the patient often considers them realities.

In a few cases, there is decided mania, but this is not of a very aggravated form, and is of temporary duration. Three such instances have recently been under my care, all

occurring in young girls of about the age of puberty, and exhibiting in all other respects the typical characteristics of chorea.

In two cases under my observation, the first notable event in the course of the disease was an epileptic paroxysm, which, however, was not repeated in either case.

The most prominent symptoms of the disease are, in the great majority of cases, exhibited in the irregular and disorderly muscular contractions which make their appearance at a very early period, and which have given it a name in nearly every language of the civilized world. Thus, we have the terms chorea (*χορεία*, a dance), St. Vitus's dance, St. Guy's dance, etc.

In the beginning the foot of one side drags a little, and soon afterward the corresponding upper extremity becomes affected with the choreic movements. These are manifested in the fingers, in the flexion, extension, and rotation of the wrist, and in the movements of the elbow and shoulder. No matter where the hand be placed, it cannot be kept steady, but it and the whole extremity are in a constant state of agitation. Before long the muscles of the neck and face participate, the head is jerked from side to side, and a continual series of grimaces is the result of the actions in the facial muscles.

In some cases the involuntary movements are confined to one lateral half of the body, constituting the form known as hemichorea. This is the case in about one-fourth of the cases. Thus, of two hundred and thirty-five cases cited by Sée,[1] the phenomena in sixty-four were limited to one side. This limitation has not, as was formerly supposed, any relation with hemiplegia, but is solely the result of the suspension of the progress of the disease.

At first the movements are moderate, but they go on, be-

[1] De la chorée et des affections nerveuses en général, avec leurs rapports avec les diathèses, et principalement avec le rheumatisme. Mém. de l'Académie de Médecine, 1850, t. xiv., p. 343, *et seq.*

coming more and more severe, until, in extreme cases, the condition of the patient becomes exceedingly pitiable. The arms, the legs, the face, and head, are in almost constant action. Every attempt to perform a voluntary movement excites still more the disorderly actions, and thus the patient is unable to feed or dress himself, and sometimes even walking becomes impossible.

In one type of cases the convulsive movements come on paroxysmally, and are often of the most astonishing character. The patient is, perhaps, lying quietly on the bed, when suddenly the head is thrown backward, the limbs set in involuntary motion, and the muscles of the trunk contract so violently as to throw the sufferer forcibly to the floor. Again, a series of gyratory motions ensues, and the patient turns round on one foot until complete exhaustion follows; or there may be leaps and contortions of various kinds. Sometimes the movements are rhythmical. A lady, who a short time since was under my charge, was suddenly seized with an irresistible impulse to bend the left elbow. The arm continued in motion for half an hour, and then the right arm began a like movement. In a few minutes the head began to nod, then the left knee was alternately flexed and extended, and finally the right knee became similarly affected. For over an hour these movements continued, and then a regular alternation ensued—first the left arm, then the right, then the head, next the left leg, and finally the right leg. These actions were perfectly timed, and were all performed in exactly ten seconds, as I ascertained by determinations made on several occasions. As she sat in a chair, or lay on a bed, she was a curious sight. Though she was good-tempered with it all, her emotional system was in a state of great exaltation. She recovered in a few weeks.

In another case a lady from New Jersey was affected in a still more extraordinary manner. While sitting sewing one day, after having been greatly fatigued the previous

night, her leg began to tremble violently. In a few minutes the arm of the same side became involved, and very soon the other limbs and the head were affected. She was now in a state of general tremor, and, on attempting to rise, fell to the floor. She was then seized with another kind of movement. Her legs were drawn up forcibly, and then suddenly extended, and this with inconceivable rapidity. She was placed on a bed, but was unable to stay there unless held by several persons, so strong were the contractions which took place. On one occasion she was thrown over five feet, her body coming to the floor with great violence.

The following day a fresh series of phenomena ensued. She began to turn somersets, and continued these actions for several hours without appearing to be greatly exhausted. Then she jumped suddenly to her feet, and rushed round in a circle with such swiftness that she could not direct her steps, and she several times knocked her body with great force against the walls and furniture. Then she danced for several hours, and toward evening became tolerably quiet, though there was still involuntary twitching of nearly all the muscles. In all the various movements she went through, every attempt to hold her only made her worse, and she begged that she might be let alone, as the effort to control her by physical force made her head swim, and gave her a severe headache. At night the paroxysms ceased, but they were renewed as soon as she awoke in the morning, and continued with but little intermission, and in every possible form, till she went to sleep.

On the third day I visited her, and found her in the midst of a series of movements such as I have described. Her pulse was irregular, her respiration hurried, and her countenance evinced great anxiety. There was no evidence of any hysterical complication.

I at once proceeded to administer chloroform by inhalation, and in a few moments she was completely under its influence. The paroxysms ceased soon after the inhalation

was begun. I kept her in a state of anæsthesia for half an hour. When she recovered consciousness she was perfectly composed, and remained so all the rest of that day. I left directions that the inhalation was to be repeated if there should be any return of the choreic paroxysms, but there were none. She slept well all night, and the following morning was quiet till about eleven o'clock, when a slight tremor began, which was at once quieted by the chloroform. I saw her again that day, and began a treatment consisting mainly of strychnia in gradually-increasing doses, and renewed my directions in regard to the chloroform. After this she had a few attempts at paroxysms, but they were always stopped by the inhalation of the chloroform, and in a few weeks she was well.

Chorea of rhythmical or uniform character has often prevailed epidemically. The most authentic recorded visitation of the kind was one which occurred at Aix-la-Chapelle in 1374. This was in the form of a dancing mania, and is fully described by Hecker[1] under the name of St. John's Dance. The men and women subject to it met in the streets and churches, where "they formed circles hand-in-hand, and, appearing to have lost all control over their senses, continued dancing, regardless of the by-standers, for hours together in wild delirium, until at length they fell to the ground in a state of exhaustion. They then complained of extreme oppression, and groaned as if in the agonies of death, until they were swathed in cloths bound tightly around their waists, upon which they again recovered, and remained free from complaint until the next attack. This practice of swathing was resorted to on account of the tympany which followed these spasmodic ravings, but the by-standers frequently relieved patients in a less artificial manner, by thumping and trampling upon the parts affected. While dancing they neither saw nor heard, being insensible to external impressions through the senses, but were haunt-

[1] Epidemics of the Middle Ages. Sydenham Society Translation, 1844, p. 87.

ed by visions—their fancies conjuring up spirits, whose names they shrieked out; and some of them afterward asserted that they felt as if they had been immersed in a stream of blood, which obliged them to leap so high. Others, during the paroxysm, saw the heavens open and the Saviour enthroned with the Virgin Mary, according as the religious notions of the age were strangely and variously reflected in their imaginations."

In the most fully-developed and best-marked instances of the disease, it was often ushered in by an attack of epileptic convulsions.

The affection spread like wild-fire—being fed by that principle of imitation which appears to be so powerful an influence in causing the propagation of this and analogous disorders of the nervous system. Those affected were generally regarded as being possessed by evil demons, and consequently only to be cured by the exorcisms of the clergy.

In 1418 it broke out in Strasbourg, and there received the name of St. Vitus's dance, from the fact that the most efficacious means of cure was thought to consist in the intercession of this saint.

Similar attacks of dancing mania had occurred before that of St. John, but the details are more or less obscure, and several have occurred since. Among these latter must be placed the tarentism which overran Italy, and various more restricted epidemics of like disorders. In our own country we have had the Jumpers, and we still have the Shakers. In addition to these are many of the manifestations of witchcraft, which were choreic, and of which this country has had its full share, and of spiritualism, which it enjoys the doubtful honor of having initiated.[1]

In chorea, even of the ordinary simple kind, the speech is imperfect, owing to the incoördination of the muscles directly concerned in articulation, and those which effect

[1] See the author's "Physics and Physiology of Spiritualism" for more complete details on this and analogous subjects, and for accounts of other examples.

respiration. There are therefore stuttering and stammering, and at times a peculiar difficulty of speaking, owing to the attempt being made when the chest is empty; that is, when expiration has first been accomplished. The tongue and lips rarely escape being involved to a considerable extent.

The muscles of mastication and deglutition are generally affected, and hence the food is imperfectly chewed, and often causes choking from difficulty of swallowing it.

In some cases chorea is accompanied with paralysis—the chorea paralytica of authors. This loss of the power of voluntary motion is usually hemiplegic, and involves the same muscles, which are the seat of the irregular movements. Occasionally there are contractions of the limbs, but not to any great degree.

Chorea is sometimes of very limited extent. It may be only shown in the hand or foot, but more frequently, when restricted in its topography, it is manifested in the head or face. There may be only a little twitching of the muscles at the angles of the mouth, or of those which raise the upper lip, or of the orbicularis palpebrarum, by which the eyelids are closed, or of the levator palpebræ superioris, or of the corrugator supercilii, or occipito-frontalis. Sometimes the head is rotated suddenly, or twitched to one side, or there is a shrugging of the shoulders.

In several cases that have been under my care, the abnormal manifestations were entirely confined to the organs of voice or speech. In one instance—that of a young girl from Illinois—while there was a general hyperæsthesia of the whole nervous system, there were no choreic movements except of the respiratory and laryngeal muscles. The respiration was therefore exceedingly irregular, and at times inarticulate sounds were made, which were involuntary. Articulate speech was lost from inability to coördinate the muscles, but there was no paralysis, for the tongue could be moved freely in all directions, and the lips were as mobile

as ever, except when the patient made an effort to speak. After a few weeks the sound from the larynx was made regularly at each expiration. There were no sounds during sleep.

In this case there was a strong hysterical element present. The affection resisted all treatment, and finally I sent the patient home, scarcely improved except in her general health. One morning she awoke, began to speak, and there was no resumption of the laryngeal sounds. She has continued well ever since, now over two years.

Again, there may be an irregular action of the muscles of speech, and in consequence words are uttered against the will of the patient, and often without any previous knowledge of what is going to be said. Several such cases have been under my observation, and I have alluded to two of them in a recent lecture[1] on chorea. Since then another remarkable case of the kind has come under my care. In this instance there is scarcely a minute during the day that the speech is not going on, and this without the least power on the part of the patient to arrest or direct it. If he is asked a question, he can only use a few apposite words, the others being altogether without relation to the subject about which he wishes to speak.

The convulsive movements in chorea almost invariably stop during sleep. They are also sometimes temporarily arrested by intense mental occupation, but are always rendered worse by emotional disturbance or physical fatigue. On the contrary, they are diminished by mental and emotional quietude.

Strange as it may appear, the sensation of being tired is scarcely ever experienced by choreic patients. Generally, however, there are wandering pains in the limbs, headache, and pain in the back. The cutaneous sensibility is usually increased, but in some cases it is greatly lessened, and may be abolished altogether in some parts of the body.

[1] Journal of Psychological Medicine, January, 1871, p. 51.

The functions of the several viscera are ordinarily more or less deranged. There are paroxysms of palpitation of the heart, and the action of this organ is to some extent irregular during the whole course of the disease. Endocardial murmurs are often present, either systolic or diastolic, but are the result of the anæmia which is so prominent a feature of chorea. Respiration is imperfect; the stomach does not digest well; and there are nausea and vomiting. The bowels are constipated; the urine is loaded with phosphates, and is of diminished quantity; and the menstrual function in girls is imperfectly performed, either as regards quantity or quality. The skin is dry and harsh, the hair loses its gloss, the complexion is pale, the lips bloodless, the pupils dilated, and the sclerotic coat of the eye of more than normal whiteness.

The tendency of chorea is to increase to a certain point, and then to gradually diminish. In favorable cases occurring in children, it runs its course in about three months. This period can be materially shortened by appropriate treatment. Sometimes, it ceases very suddenly, and, in others, passes into a chronic condition, which may last for years or during the life of the patient. Occasionally, it terminates in death, either directly or in consequence of the supervention of some intercurrent affection. Three fatal cases have come under my observation. One of these I saw several times in consultation with my friend Dr. T. G. Thomas. The patient was a young lady about twenty years of age, and her paroxysms were of the most violent character, sometimes being so strong as to cause her to throw herself off the bed, or to dash about the room with great force. No treatment appeared to exercise any restraining effect, and, after about two years, she died of an abdominal affection. There was no post-mortem examination. In the other two cases, death ensued from exhaustion.

Relapses are common in chorea, especially in children,

and sometimes as many as half a dozen attacks occur. Subsequent seizures are usually less severe than the first.

Chorea is often complicated with hysteria—a combination which will be described hereafter. It may also exist in conjunction with rheumatism and malarial fevers, and the exanthemata.

Causes.—Chief among the predisposing causes of chorea is age. It is more frequent during the period extending from six to fifteen years than during all the rest of life. Sée, of five hundred and thirty-one cases, found four hundred and fifty-three of ages ranging from six to fifteen years.

Of eighty-two cases, occurring in my own experience, sixty-seven were of ages between six and fifteen years. Under the age of six, the disease is less frequent as we go toward birth. Cases have been met with in infants at the breast of six months old. The youngest case I have had was a girl of eighteen months.

After fifteen, the disease, unless it occurs as an epidemic, is not very common. Cases are, however, met with in adults, and even in very old persons. I have seen four cases in individuals over thirty and three in persons between the ages of twenty and thirty. Of course, I refer to the origination of the disease at these ages: instances of its beginning in childhood, becoming chronic, and lasting through life, are not so rare. In those cases reported by authors of the affection originating very late in life, we have every reason to conclude that they were instances of organic lesions of the brain or spinal cord—probably, sclerosis—giving rise to rhythmical movements or paralytic tremor.

The female sex is much more liable to chorea than the male. Of Sée's five hundred and thirty-one cases, three hundred and ninety-three were girls and one hundred and thirty-eight boys.

Of my eighty-two cases, seventy were females and twelve males. Rheumatism has been supposed to be a predispos-

ing cause of chorea. Of one hundred and twenty-eight cases, Sée found sixty-one in association with rheumatism, but, when we come to inquire further, we find that only thirty-two of these were articular rheumatism, while the rest were cases in which there were wandering pains which may have been, and probably were, without the least affinity with true rheumatism.

While it is certainly the case that chorea sometimes follows or exists coincidentally with rheumatism, I doubt if its influence is any more than that of a depressing agent to the organism. Of the eighty-two cases observed by myself, only sixteen were connected with rheumatism, while eighteen were just as intimately related to other diseases.

The affection appears to be more common in winter than in summer. Of my cases, fifty-four occurred in the six months from October to March, and twenty-eight in the other six months of the year.

Among the exciting causes, those connected with the emotions occupy the first place. Twenty-seven of my cases were directly the result of fright, apprehension, anxiety, mental excitement, or some other cause of the kind. In eight it was induced by intense study at school, and in four from imitating others similarly affected. This latter factor is not of so general application as in former times, when social life was different. To it is doubtless to be ascribed the spread of choreiform movements through certain localities, and especially convents, such as occurred in the thirteenth, fourteenth, and fifteenth centuries, to some of which reference has already been made.

Among other causes, bad hygienic influences and exhausting diseases generally are to be mentioned.

Pregnancy is also asserted to be a cause, and cases are on record in which the fœtus has been born choreic of a choreic mother.

Diagnosis.—There is not much danger at the present day that chorea will be confounded with many of the diseases

from which, not long ago, it was not clearly disassociated. Thus from paralysis agitans, epilepsy, locomotor ataxia, cerebral and cerebro-spinal sclerosis, the fuller acquaintance which we have in recent years acquired of these maladies prevents the necessity of dwelling on their characteristics as distinguished from those of chorea. The course of the latter disease and the symptoms, other than those connected with motility, are in the others so different that no one who has studied their phenomena could fail in making a correct diagnosis.

With hysteria, some of the forms of chorea may be confounded, and the two affections are not infrequently blended in the same person. It must be confessed, too, that there are cases in which the diagnosis cannot be clearly made out, So far as the patient is concerned, the difficulty of forming a correct opinion in such cases is not a matter of much moment.

The great majority of cases of chorea, such as are met with in children, are readily distinguished from hysteria. The facts of the disease occurring before puberty in so large a proportion of instances, that the emotional system is rarely disturbed as in hysteria, that the affection is not so paroxysmal, and that the accessions of hysteria are more sudden, will be sufficient to render the diagnosis accurate.

Prognosis.—This is usually favorable in those cases which occur before puberty. The chorea of adults is, however, in most instances, a very unmanageable affection, and generally either terminates in death or becomes permanent. Cases in which death has ensued have been reported by various authors—among them, Dr. John W. Ogle,[1] Dr. J. Hughlings Jackson,[2] and Dr. G. Sée.[3] As already stated,

[1] Remarks on Chorea Sancti Viti, including the History, Course, and Termination of Sixteen Fatal Cases, etc. British and Foreign Medico-Chirurgical Review, January, 1868, p. 208.

[2] The Physiology and Pathology of Hemi-Chorea. Edinburgh Medical Journal, October, 1868.

[3] Op. cit.

three fatal cases have occurred in my own experience. The tendency, however, in the chorea of young persons is decidedly toward recovery, even under unfavorable circumstances as regards hygiene or medical treatment.

Morbid Anatomy and Pathology.—In many cases of persons dying, either from chorea or from intercurrent affection, no changes have been found which could, with probability, be regarded as constituting the disease. In other cases, morbid alterations from the healthy state have been found. The idea has, therefore, prevailed that there are two kinds of chorea—one which is entirely functional, belonging to the so-called neuroses, the other the result of organic disease of the brain or spinal cord, or both. In Ogle's sixteen fatal cases, congestion of the brain and its membranes was found in some, while in others the difficulty existed in the spinal cord.

In an analysis of one hundred cases of chorea, Dr. Hughes[1] cites fourteen fatal cases. In all but four of these there was intra-cranial congestion with other structural changes, such as softening, opacities and adhesions. The spinal cord was not examined in six cases. Of the remaining eight, it was healthy in three, and congested, softened, or with adhesions or opacities of the membranes in the remaining five.

In seven fatal cases, collected by Romberg,[2] there were softening and degeneration of different parts of the brain and of the spinal cord.

Other similar cases have been reported, and in the majority there were fibrinous concretions on some portion of the heart's valves or lining membrane.

In 1850 and 1863, Dr. Senhouse Kirkes[3] published the details of a number of cases which went to show the asso-

[1] Digest of One Hundred Cases of Chorea. Guy's Hospital Reports, vol. iv., 1846, p. 360.

[2] Lehrbuch der Nervenkrankheiten, B. ii.

[3] London Medical Gazette, 1850, and Medical Times and Gazette, 1863.

ciation between chorea and rheumatism, and he made the prediction that "future experience will still more positively demonstrate that an affection of the left valves of the heart, with the presence of granular degeneration upon them, is an almost invariable attendant upon chorea, under whatever circumstances the chorea may be developed." The relation is also insisted upon by Sée and other authors, and such cases as those of Ogle are cited in its support. But the doctrine is only applicable, with any probability, to the fatal cases, and, in those of Ogle, rheumatism was not always an antecedent. In regard to this point, I am entirely in accord with the views expressed by Dr. Ogle in the following extract, which I make from his valuable paper:

"Again it might be asked, if there was merely a mechanical cause (which, of course, would be constant in operation), such as embolism, why should the movements be so decidedly and universally interrupted during quiet sleep? Or, why should certain peculiarities as to age or sex be considered as predisposing influences? Recognizing the frequent existence of these fibrinous deposits, or granulations on the heart's valves in chorea, I should be much inclined to look upon these post-mortem appearances rather as results of some antecedent condition of the blood, common also to the choreic condition. It is very freely recognized that this affection is frequently in some way or other connected with that condition of blood which obtains in what we call anæmia, or that existing in rheumatic constitutions. In both of these states we know that the fibrine of the blood is much in excess (as also it is in pregnancy and other conditions looked upon as obnoxious to chorea), and in these states we know that the fibrine (with which the blood is surcharged) is very prone to be readily precipitated, either owing to its superabundance or from other obscure and acquired properties (possibly also from some interference with the relation of the fibrine and the other constituents of the blood), upon the heart's walls or

valves. May not this hyperinosis be the explanation of the coincidence alluded to? In most cases, the deposit is probably very slight, and, in many cases, so slight as to require search for it. May it not infrequently be that it is often only found in quite the dying state? Speculation might suggest that the fibrinous deposits arise from some interference with the degree of solubility of the fibrine, induced by the presence of some ununited elements within the blood (some result of tissue-metamorphosis) produced by the excessive muscular action and other functional disturbance which exists in the choreic state, thus being not in any way related to this state as a cause, but as a consequence."

In the paper to which reference has already been made, Dr. Hughlings Jackson associates hemi-chorea with the plugging by emboli of the vessels of the corpus striatum of one side, and, in a recent valuable paper, Dr. Charlton Bastian [1] says:

"I need only hint at the important bearing which the possibility of the occurrence of minute embolisms of this kind may have in the elucidation of previously-obscure forms of so-called functional disease of the nervous system, as I hope shortly to publish the details of a fatal case of chorea, in which such embolisms led to ruptures and obliterations of small vessels throughout the corpora striata and in the course of the middle cerebral arteries generally—this being a case of bilateral chorea in which delirium was also present."

As the result of our present knowledge of the morbid anatomy of chorea, while it cannot be said that we are able to define its seat with accuracy, we have strong evidence to support the view that it is not a neurosis or functional affection—if, indeed, there are any such—and that it is the result of changes taking place in the cerebro-spinal system.

[1] On the Plugging of Minute Vessels in the Gray Matter of the Brain, etc. British Medical Journal, January 30, 1869, p. 96.

As previously stated, I am inclined to think that there are at least two distinct diseases—one due to spinal and the other to cerebral lesion—the latter probably consisting of several forms—but that it is advisable to consider them as one disease of various types, until further investigation enables us to speak with certainty on the subject, and to classify them according to the morbid anatomical condition of each.

Treatment.—Diseases which are almost certain to terminate fatally, and those which ordinarily recover without medical treatment, are very sure to have a great many medicines used in their therapeutics. Chorea belonging, as it does, to this latter category, has a medical armamentarium almost equalling that of hydrophobia. I shall, of course, not even pretend to mention all these measures, but will merely cite those which the weight of evidence, and especially that derived from my own experience, indicates as the most effectual. Of the benefit to be derived from proper medical treatment in shortening the duration of the disease, and preventing chronicity, I have no doubt.

In this country zinc is probably more used in chorea than any other single remedy. I have employed it in many cases, and sometimes with good results. My preference is for the sulphate, which I give in gradually-increasing doses, from two or three grains up to twenty or thirty three times a day, dissolved in a sufficient quantity of water, to prevent gastric irritation. When the choreic symptoms begin to disappear, the doses should be diminished in the same gradual manner in which they were increased.

Iron is also frequently administered as a sole remedy, and still more generally as an adjuvant. Indeed, no matter what special treatment may be adopted, iron is generally indicated to improve the quality of the blood. I rarely use it unless for this latter purpose.

Arsenic enjoys a high reputation in the treatment of chorea, and by some is regarded as almost a specific. Al-

though I have several times given it with great advantage, I have repeatedly had it fail in my hands. I have administered it twice hypodermically for choreic movements involving the muscles of the neck, as recommended and successfully used by Dr. Radcliffe. In one of these it failed, but in the other it was thoroughly effectual. Five minims of Fowler's solution, diluted with an equal quantity of water, were injected into the cellular tissue immediately over the belly of the left sterno-cleido-mastoid muscle, the muscle which was affected. The following day six minims were injected, and so on till the quantity reached ten minims. By this time the jactitations had nearly ceased, and a few more injections of ten minims each were sufficient to render the cure complete. In this case zinc, electricity in the forms of the primary galvanic, and induced currents, iron, morphia, and several other measures, had failed.

Tartarized antimony, copper, sulphate of aniline, Calabar bean, and various other substances, have been employed with more or less success, according to reports, but I have no personal experience of their value.

I have used both the primary galvanic and induced currents in sixty cases. In my opinion they are inefficacious except in that form in which there is distinct paralysis.

Without stopping to detail other means, I will describe the modes of treatment which my experience has convinced me are most efficacious. As one of the remedies, I usually administer the bromide of potassium or sodium in moderate doses, so as to render the sleep sounder. I do not regard this as an essential part of the treatment, and, if the patient is exceedingly anæmic, I do not urge it.

My main reliance is on strychnia, which I think should be given in gradually-increasing doses, somewhat after the manner recommended by Trousseau. Two grains of the sulphate of strychnia are dissolved in an ounce of water, and for a child of from ten to fifteen years of age five minims should be given three times a day. This quantity rep-

resents the one forty-eighth of a grain of the salt. The following day six minims are administered at each dose, the next seven, the next eight, and so on till the physiological effects of the medicine, as evidenced by stiffness of the legs and neck, are obtained. Sometimes these are not perceived till twenty or twenty-five minims are taken at a dose. In other cases they follow on doses of ten minims. When they take place, the doses should be at once reduced to the original five minims, and the increase carried on as before. This plan of treatment certainly shortens the duration of the disease very materially, and causes great improvement in the general health of the patient. Sometimes the effect is so well marked, and is so immediate, that it is not necessary to increase the doses to the extent of causing muscular cramps, but generally the full therapeutical effect of the drug is not obtained till the calf of the leg, or the nucha, has slight tonic spasm. I have never seen the slightest ill consequence follow this mode of treatment, and the doses are increased so gradually that with careful watching danger need never be apprehended. I have carried it out in thirty-two cases occurring in children under the age of fifteen, and in three cases in persons of adult years, without a single failure.

In one of the latter the affection was limited to the speech, there being an inability to utter words in accordance with the ideas. In this case the dose was increased to thirty-five minims before any rigidity of the legs was perceived, and then the command over the language began to appear, and by continuing the doses at thirty-five minims the patient was entirely cured within a month. In this case the initial dose was ten minims.

Quite recently I have made use of the ether-spray to the spine as employed by Lubilski, Zimberlin, and others, and my success has been unequivocal. The whole spine is exposed, and the ether is thrown upon it from the occiput to the sacrum for about ten minutes every day, or every alternate day, according to the severity of the attack. Ten

applications are the maximum number I have found it necessary to make, and thus a cure has always been obtained within two weeks. I have employed this means in thirteen cases in my private practice, and in three in the New York State Hospital for Diseases of the Nervous System. Strychnia has been given at the same time, but undoubtedly the beneficial results are mainly to be attributed to the ether.

In the paroxysmal forms of chorea, ether or chloroform by inhalation is often necessary to cut short or prevent an immediate seizure, but in other respects the treatment mentioned is entirely applicable.

In all cases hygienic measures are of the utmost importance. Exercise in the open air is indispensable; the food should be of the most nutritious character; the bedroom should be well ventilated; bathing should be frequent; the bowels should be kept well regulated, and the child, if at school, should be at once removed, and all study for the time be interdicted. Ridicule or threats, so often indulged in toward choreic children, generally do harm, but at the same time they should be encouraged to use all reasonable effort to prevent a bad habit being formed. In the epidemic variety of the disorder, threats, and even strong repressive measures, are, on the contrary, decidedly beneficial in curing and arresting the further progress of the disease.

CHAPTER VI.

HYSTERIA.

A LARGE volume might be written on hysteria—and many such have been published—and there would still be points in its clinical history unconsidered. It is difficult, therefore, in a general treatise like the present, to give a full view of a disease which plays so important a part in nervous pathology, and which is so varied in its manifestations. The most that I can hope to do is to lay down certain broad principles and features, and leave the recognition of details to the intelligence and discrimination of those who read this work.

Symptoms.—The phenomena of hysteria may be manifested as regards the mind, sensibility, motility, and visceral action, separately or in any possible combination. Thus it is not uncommon to meet with cases in which the only evidence of the disease is seen in abnormal mental action; others are characterized solely by derangements of sensibility, such as hyperæsthesia or anæsthesia; others by aberration of the faculty of motion, such as paralysis, spasms, contractions. Again, all of these categories may be witnessed in the same person, giving rise, among other phenomena, to coma and convulsions; and again, some one or more of the viscera may be deranged in their functions, and thus the appearance of organic disease be simulated.

As there is such a marked want of uniformity in the character of hysteria as it affects different persons, I will

not endeavor to present a typical case of the disorder, but will consider separately the principal phenomena which may have an hysterical origin. But, in setting out to make the attempt, I am reminded of Dante's despair at the thought of his inability to describe the horrors of the ninth gulf:

"Chi poria mai pur con parole sciolte
Dicer del sangue, e delle piaghe appieno,
Ch'io ora vidi, per narrar pici volte?
Ogni lingua per certo verria meno,
Per la nostra sermone, e per la mente,
C'hanno a tanto comprender poco seno."

The Hysterical Diathesis.—Though it is very common to hear the hysterical diathesis or temperament mentioned by medical authors, I have never been able to recognize its existence by any external traits. The fact that it has been so very differently described by writers, from Hippocrates and Galen, to our own day, is good evidence that it is not readily detected.

Thus, Hippocrates and Galen recognized the existence of the hysterical temperament, but each gave it different characteristics. Louyer-Villermy[1] had very decided views of its features, and he described it as follows:

"Every hysterical woman is stout, short, dark, plethoric, full of life and of health. The complexion is brunette and ruddy, the eyes black and sparkling, the mouth large, the teeth white, the lips of a carnation red, the hair luxuriant and of the color of jet, the sexual organs well developed, and the spermatic liquid abundant."

Aside from his physiological error relative to the spermatic liquid, these are the characteristics of the women of the south of Europe. If he had lived in the north, where hysteria is fully as common, he would have found that his description of the hysterical temperament would not have

[1] Quoted by Briquet, Traité Clinique et Thérapeutique de l'Hystérie, Paris, 1859, p. 91.

held good. Indeed, Sydenham, Whyte, Copland, and other English authors, represent the hysterical predisposition with almost the very opposite characteristics. As Briquet[1] remarks, there is no hysterical constitution appreciable by the study of external appearances. The disease takes women as it finds them, blondes, brunettes, stout, thin, strong, weak, ruddy, or pale, there is no choice. Some hysterical women have delicate figures, and intelligent minds, but there are others whose dull, stolid faces give evidence of their stupidity; and others, again, whose thin, fleshless, and wan faces, tell us that the Greek type of female beauty is not to be regarded as predisposing to the development of hysteria.

While, therefore, admitting the existence of the hysterical diathesis, I know of no marks by which its presence can be determined, other than the acts of the patient, which go to make up the clinical history.

Mental Symptoms.—These are very various, but generally consist in emotional disturbance, an inability or indisposition to exert the will, and in the existence of illusions, hallucinations, or delusions. Attacks are often characterized by no other prominent symptoms than those connected with mental action, and they may assume every possible character. At times, the patient is depressed in spirits, and sheds tears profusely; a few minutes afterward, she has forgotten her grief, and laughs immoderately, without adequate cause. Sometimes she laughs and cries at the same time.

Or, there may be a total insusceptibility to any emotion, a listless *insouciance*, which contrasts strongly with her natural disposition. Or, again, an emotion the exact opposite of the proper one is excited. This is quite a common form of manifestation. A mother, for instance, is informed that her daughter has contracted an improper marriage, and is immediately seized with immoderate laughter, and

[1] Op. cit., p. 92.

shows every expression of pleasure, when the rest of the family are overwhelmed with grief and shame. Another draws the chief prize in a lottery, and begins at once to cry and wring her hands. A third, hearing that burglars have entered the house and have stolen all her jewelry and silver, sits stolidly in her chair, her hands folded in her lap, and her whole expression indicating the most complete indifference. During either of these conditions, she may be entirely silent, or excessively voluble, or she may exhibit other hysterical phenomena.

As regards the will, the manifestations of disorder are sometimes very remarkable. That the patient is, for the time being, unable to exert it, is evident, but, under the influence of some strong exciting cause, she frequently astonishes those about her by suddenly reacquiring her lost volitional power.

A young lady came under my charge for what was supposed to be a disease of the spinal cord. She had taken to her bed suddenly, soon after striking her back rather gently against the edge of a table, declaring that she could not walk. On examination, I was convinced that there was no disease whatever of the spine, other than that of a purely hysterical character, and I so expressed myself to her. She, nevertheless, insisted upon it that her spine was seriously injured, and she continued to keep her bed, lamenting daily her sad fate at being compelled to pass so long a time shut out from the enjoyments of life. There was no paralysis or even simulation of it, for she moved her legs about freely enough in the bed. But, one evening, her brother, who had long been absent, returned home. She heard the bustle in the house attendant upon his arrival, but all were too busy to pay any attention to her in her chamber up-stairs. Suddenly exclaiming, "I can stand this no longer," she sprang from her bed, rang for her maid, and, hurrying on her clothes, proceeded down-stairs and entered the drawing-room, to the great surprise of all the family.

In another case, a lady closed her eyes, and declared that she could not open them. She was brought to me as a case of double ptosis. There was no spasm of the orbicularis palpebrarum on either side, and I had no difficulty in opening the eyes by gently raising the lids. The pupils were normal; there was no diplopia, and there were no evidences of such cerebral lesions as are generally met with as causes of ptosis. Moreover, she was subject to paroxysms of hysterical syncope. Under the circumstances, I had no hesitation in expressing my opinion to her friends that the case was one of hysteria. I advised the use of the induced current to the eyes, and she found this so disagreeable, not to say painful, that two applications were sufficient to restore her volitional power, so that she opened her eyes without difficulty.

In my remarks on aphasia, I have cited a case (p. 166) in which the power to speak suddenly returned under the influence of excitement, and was as suddenly lost again, to be gradually recovered.

Many cases of this loss of volition in hysteria have been under my care, and most physicians have witnessed similar instances.

Illusions are very common phenomena of hysteria, and these may be connected with any or all of the senses. A ball rolling over the floor is taken for a rat; the sound of rain falling on the roof is mistaken for the noise of burglars in the next room; the knives used at table all smell "fishy;" every thing tastes sour or bitter or sweet, as the case may be, and a draught of cold air on the hand is supposed to be the touch of a person or a spirit.

Hallucinations of various kinds are equally frequent. Images are seen where there is nothing; voices are heard where there is absolute silence; odors are smelt where there is nothing to smell; and strange tastes are perceived when the mouth is empty.

Thus one patient sees angels, another demons, another

animals of various kinds. One hears voices whispering to her, another musical sounds, and another noises like the breaking of glass or dishes. Another is constantly sensible of a smell as if something is burning, and another always has a taste of turpentine in her mouth.

It is not often the case that these erroneous perceptions impose on the intellect, but sometimes they do, and then delusions are entertained, or these may, as in cases of absolute insanity, be formed without the intervention of the deranged perceptive faculties. They differ, however, from the delusions of insanity, such as have been already described, in the facts that they do not last long, and that they rarely exercise any very powerful influence over the actions of the patient.

Besides these mental phenomena indicative of cerebral disturbance, there are, sometimes, an extraordinary acuteness of understanding and readiness at reasoning and speech quite beyond the natural powers of the patient. At other times, on the contrary, the intellect is dulled, and the conversational power reduced to a low point.

Sensibility.—This may be affected so as to result in the production either of *hyperæsthesia* or *anæsthesia.*

Hyperæsthesia, caused by hysteria, is characterized by the facts that it is never permanently fixed in one place, that it is generally excessively acute, and that it is unaccompanied by evidences of serious disease of the nervous centres or the nerves. A common seat is the skin, and its favorite regions are the trunk, especially the skin over the mammary glands, and that covering the labia majora. Another situation frequently affected is the skin of the face.

Cutaneous hyperæsthesia may consist either of spontaneous pain or of tenderness to impressions made upon the surface of the body. Muscular hyperæsthesia, or myalgia, is likewise common. Dr. Inman[1] has investigated this branch of the subject very carefully, and has ascertained

[1] On Myalgia : its Nature, Causes, and Treatment, etc. London, 1860.

that the painful spots correspond to the origins and insertions of the muscles.

Muscular pains due to hysteria are often mistaken for pains of the viscera. Thus the headache which is so frequent a phenomenon of the hysterical condition is very seldom located within the cranium. It may be of very limited extent, constituting the form known as the *clavus hystericus*, or may be of more extensive limits. Its ordinary situations are the frontal regions, occupying, in this case, the occipito-frontalis and corrugator supercilii muscles; the temporal regions, being then located in the temporal muscles; the vertex, being then seated in the tendon of the occipito-frontalis muscle; and the occipital region, in the occipito-frontalis, trapezius, splenius and complexus. Briquet states that, of three hundred and fifty-six hysterical patients whom he questioned on the subject, three hundred were constantly subject to headache. I have very rarely met with a case of hysteria in which it was not constantly present, and never one in which it was not a symptom at some time or other.

Pains are often felt in the muscles of the chest, abdomen, and back. This latter is a favorite situation, especially in the region between the shoulders, and in the muscles on each side of the vertebral column in the lumbar region.

Pains in the joints are common manifestations of hysteria, and they are often mistaken for serious organic disease. When, as is sometimes the case, they are accompanied with contractions of the muscles, the liability to error on the part of the practitioner is increased. Sir Benjamin Brodie,[1] several years ago, pointed out the true nature of certain affections of the joints occurring in hysterical women, and, since his time, others, among whom Barlow[2] and Skey[3]

[1] Illustrations of Certain Local Nervous Affections. London, 1837.

[2] A Treatise on Diseases of the Joints. London.

[3] Hysteria, etc. Six Lectures delivered to the Students of St. Bartholomew's Hospital, 1866. London, 1867.

are to be mentioned, have called special attention to the subject The pain may be attended with swelling, but there is no accumulation of fluid in the cavity of the synovial membrane. The knee is more frequently affected than any other joint.

Neuralgia often has an hysterical origin, and may be in the form of toothache, pleurodynia, sciatica, or pain in the course of any other nerve. The viscera are likewise frequently hyperæsthetic; the stomach, bowels, the kidneys, bladder, uterus, and ovaries, are the organs most frequently affected.

The organs of the special senses rarely escape having their sensibility exalted, and, consequently, there are increased power of vision, morbid acuteness of hearing, and an abnormal sensitiveness of the smell and taste. Sometimes with these hyperæsthetic conditions there is pain.

Anæsthesia.—Though not so common as hyperæsthesia, anæsthesia is frequently a manifestation of hysteria. Its most common seat is in the skin. In the days of witchcraft, many an hysterical woman, with anæsthetic spots on her skin, went to the gallows or the stake on suspicion of being leagued with the devil. The belief was that, wherever the hand of the arch-fiend or his assistants touched the skin, the spot at once lost its sensibility.

Two patients are now under my charge in whom there is hemi-anæsthesia, paroxysmal in its character. When it is at its height, no irritation applied to the skin is felt, not even the wire brush of a powerful induction-coil. In neither case are the attacks preceded or accompanied by numbness.

Sometimes the location is very limited, and the loss of sensibility may be partial or complete. In the former case, there is numbness, and the full extent can only be exactly ascertained by the æsthesiometer.

The mucous membranes may become anæsthetic. The one most frequently affected is that which lines the genital

canal. In such a case, the sexual passion is entirely extinguished, coition is unattended with pleasure, and may even excite disgust.

The organs of the special senses may be the seat of anæsthesia, and thus blindness, deafness, loss of the senses of smell and of taste, may be caused, more or less complete in character, in different cases.

Anæsthesia of the muscles is occasionally met with, and has, at times, been the occasion of much discussion in medical and theological circles. Many of the phenomena observed in the Jansenist *convulsionnaires* were the result of muscular anæsthesia. In an essay[1] recently published, I have called attention to the symptoms, and have adduced several cases from the records of my own experience. The extent of the anæsthesia is sometimes remarkable. In some of the cases that have been under my care, the most powerful induced currents which it was safe to use failed to cause pain in the muscles to which they were applied.

Alterations of Motility.—These may be evidenced in the way of paralysis or of clonic or tonic spasm.

Hysterical paralysis has long been known, and is quite a common manifestation of the affection. It may appear in the character of hemiplegia, paraplegia, or of much more restricted extent. I have a case, now under care, in which it is limited to the index-finger, and I have had several in which a single muscle of the eyeball, or in which the levator palpebræ superioris, was alone affected.

Hysterical aphonia is due to paralysis of one or more muscles of the larynx. Like the loss of power in other muscles from a similar cause, it often comes on very suddenly, and as suddenly disappears.

Paraplegia, hysterical in its character, may be partial or complete as regards a muscle, group of muscles, or a limb. When incomplete, the patient, if it involves the lower extremities, drags her limbs sluggishly along, or shuffles

[1] The Physics and Physiology of Spiritualism. New York, 1871.

her foot over the floor, using a cane or crutches, or holding on to articles of furniture that may be in the room. There is nothing about the gait like that of locomotor ataxia or, in fact, of any other of the diseases of the cord already considered; and careful observation will generally reveal the fact that, during one interview and examination, the patient walks very unequally, according to the state of her mind at the time, or the influences which act upon her.

Spasms may be either tonic or clonic, and may affect any muscle of the body. In the pharynx, tonic spasm causes the sensation to which the term *globus hystericus* is applied, and which gives rise to the sensation of a ball in the throat. In the œsophagus, spasm may continue for a long time, and may thus simulate stricture. It may also be seated in the stomach, intestines, or bladder.

In the limbs spasm of the tonic character causes contraction, and thus, especially when combined with paralysis, may give the appearance of organic lesion. I have frequently known hysterical contractions to last several months at a time, and have had many cases of the kind under my charge in which the actual cautery had been applied to the back for supposed inflammation of the cord.

Clonic spasms simulate chorea. They are especially common among the women who attend spiritualistic gatherings, and indeed I have seen several cases at such places among the weak-minded men who believe in the nonsense called spiritualism.

The functional actions of the viscera are exceedingly liable to derangement in hysteria. Any organ of the body may be affected, but the stomach appears to be the favorite one. There may be obstinate vomiting, or persistent flatulence, or acidity, or indigestion in some other form; or the bowels may be the seat, giving rise to intestinal indigestion, diarrhœa, or obstinate costiveness; or the kidneys may be involved, and there may be an enormous secretion of pale, limpid urine, or the quantity may be reduced to a

minimum; or the uterus or the ovaries may be the seat. Not infrequently organic disease of the heart is simulated, there being palpitation and general irregular action of this organ.

Besides these several manifestations of hysteria, there are paroxysms of the disease, characterized by emotional disturbance, spasm, convulsions, partial loss of consciousness, and sometimes coma. All these phenomena may be manifested during an attack, or a seizure may consist of any one or more of them. The convulsions sometimes bear a resemblance to epilepsy, sometimes to tetanus, sometimes to hydrophobia, sometimes to catalepsy, sometimes to chorea. But, though simulating these diseases, the hysterical paroxysm can be readily distinguished from either of them, mainly by the facts of its lack of consistency, the absence of the constitutional disturbance which attends the others, and by the presence of emotional excitement, and the consequent irrational laughing or crying.

Mania may be simulated, but the false can scarcely be mistaken for the real disease by any practitioner with his wits about him.

Causes.—Of the predisposing causes, sex stands first. During the last six years three hundred and thirty-two cases of hysterical disease have been under my charge or been seen by me in consultation. Of these, three hundred and twenty-nine were females. Of the three males, one was a young gentleman, the son of a distinguished citizen of Virginia, in whom the affection was induced by excessive study. One was a lawyer in this city, the disease in him simulating epilepsy; and the third was a shop-keeper from New Jersey, who had tetanoid paroxysms attended with fits of sobbing, crying, and laughing, and in whom it was excited by masturbation.

But, while there is this great predominance of females as the subjects of hysteria, I do not believe that the fact is due to any particular influence of the uterus or other gen-

erative organs. It is probably the result of the delicacy of organization, and the greater development of the emotional system, acted upon by the exciting causes to be presently mentioned.

Age is another predisposing cause. The period of life at which hysteria is most common is that extending from sixteen to twenty-five. After the latter age there is a gradual decline until the age is reached at which the menstrual function begins to become irregular, and then the number of cases increases.

The civil condition, as regards marriage or celibacy, is to be taken into consideration among the predisposing causes. Undoubtedly the disease is much more frequent among the single than the married, but it is by no means confined to them. In my opinion the increased proclivity of single women to hysteria is not to be attributed to ungratified sexual desires, or even to the non-fulfilment of the functions of the generative organs, but rather to that lack of aims in life, and the consequent reflection of the thoughts and emotions upon self, which are so inseparably connected with the present condition of single women. Certainly those celibates who have made for themselves objects in existence are no more subject to hysteria, in my experience, than married women. Want of occupation is one of the powerful predisposing causes of hysteria, and it is to a great extent through the direct influence of this factor acting upon a more impressionable organization that, in my opinion, hysteria is more common in women than in men. In those savage and semi-savage countries where women work, hysteria is unheard of. It used to be almost unknown among the negro women in the South, but since their emancipation, if my inquiries have ascertained the truth, it is becoming quite common among them.

Hereditary influence is undoubtedly an important predisposing cause of hysteria. My own statistics are not complete on this point, but of the two hundred and nine

instances in which I have made the inquiry, one hundred and thirty-one had either hysterical mothers, aunts, or grandmothers, and many of the others had relatives affected with other nervous diseases. Briquet speaks very emphatically of the decided influence of hereditary tendency as deduced from his inquiries.

The luxurious habits of life attendant upon refinement and education conduce to the development of hysteria. Attendance at theatres and operas, the cultivation of music, the reading of poetry and novels, the study of art, and any other influence capable of developing the emotional system at the expense of the purely physical or intellectual, favor the growth of hysterical tendencies.

Of exciting causes, sudden emotional disturbance ranks first. Anxiety, grief, disappointment, the intense desire of self-gratification, a fit of ill-temper, with other similar factors, often induce paroxysms of the disease. Mental or physical fatigue, menstrual derangement, or uterine or ovarian difficulties, may also act as exciting causes.

But probably, above all these, is the contagion set in action by the contact with an hysterical person. I have seen a whole hospital ward of women thrown into paroxysms of hysteria by one patient suffering from an attack.

Diagnosis.—To detail the diagnostic marks which distinguish hysteria from other diseases would require more space than is proper in a work like the present, and would, moreover, be rather a work of supererogation. The physician has simply to recollect that all hysterical affections have a family resemblance, and that, although almost every known disease may be simulated, yet that the counterfeit is never a good one. Attention to the symptoms of the several diseases already and to be described, with a careful observation of the case, and due inquiry into the antecedents of the patient, will prevent a mistake being made.

He must also recollect that the hysterical patient always tries to impress others with the belief that she is very ill.

She craves sympathy, and feeds on it with the effect of nourishing her disease. If she can deceive her medical attendant by appealing to his kindly emotions, she will do it, but failing in this she will try her power over his fears, and will leave no stone unturned to deceive him. Careful watching, with thorough skepticism, will either result in her detection, or in her defeat from sheer weariness.

Prognosis.—As regards the prospect of recovery from any particular manifestation of hysteria, or from a paroxysm of any kind, the prognosis is favorable, provided proper treatment be employed, but, as regards the liability to further attacks, much depends on the circumstances which surround the patient and the time during which she has been subject to the affection. If she can be submitted to proper treatment, without the interference of herself or her friends, the prospect of recovery, even in bad cases, is good; but if she is to be allowed to do as she pleases, or if injudicious friends are constantly lavishing the sympathy and mistaken kindness which keep her disease alive, there is not much use in medicine or hygiene, and, as Reynolds says, the "case is hopeless, and might as well be left alone."

Morbid Anatomy and Pathology.—Hysteria contributes absolutely nothing to the science of morbid anatomy. The brain, the spinal cord, the sympathetic nerve, give no evidence of its former presence. It is true, hysteria very rarely causes death, but hysterical patients have died of intercurrent affections, and post-mortem examinations have been made, and nothing which could reasonably be regarded as the essential cause of the disease has been found. Several of the older writers imagined that they had discovered the lesion in the genital organs, in the stomach and intestines, in the brain, and even in the spleen; but modern research teaches us differently. At present, then, we are in total ignorance of the character of the lesion. From the symptoms, which are so obviously indicative of disordered

brain and spinal cord, I have felt myself justified in classing it provisionally at least among the cerebro-spinal diseases.

The pathology or morbid physiology of hysteria is beginning to be better understood as our knowledge of the cerebral and spinal actions becomes more complete. Looking at the brain as a complex organ evolving a complex force—the mind—we can understand the possibility of certain parts of it becoming disordered, as regards excess, diminution, or quality, in the results of their actions. We have seen, under the head of insanity, that the mind is made up of certain sub-forces—the perception, the intellect, the emotions, and the will—and that these, when disordered, constitute varieties of insanity, which are easily recognized.

Hysteria essentially consists in the predominance of the emotions over the intellect, and especially over the will, and this exaltation may be so intense as to interfere with the sensibility of various parts of the body, or to derange the contractility of muscles.

At the same time, in the paroxysms of the disease, the reflex and automatic functions of the spinal cord are involved to a great extent.

We daily witness examples of the influence of emotions on sensibility and motility. Fear renders the sensibility more acute and produces trembling, which is simply clonic spasm; grief causes tonic contractions of the muscles; surprise, terror, or horror, paralyzes them; joy or anger destroys sensibility to pain, and so on.

At the same time that there is this exaltation of emotional power in hysteria, the power of the will is not only relatively but is absolutely diminished. The two factors, acting together steadily and persistently, induce many of the manifestations of hysteria. The disease is, therefore, a partial insanity—an insanity, however, in which the patient does not entirely lose the power of control, and which is capable of being overcome by the voluntary effort of the patient, provided a sufficient stimulus to normal volition

be brought to bear. It thus happens that, through the influence of such stimulus, every symptom of hysteria disappears as if by magic.

The spinal cord is often secondarily affected, and it is likewise frequently primarily involved. The gray or the white substance, the posterior or the antero-lateral columns may be implicated, the symptoms varying accordingly. Through the spinal cord, in its abnormal condition, we have the convulsions of various kinds, the spasms, contractions, and the paraplegic phenomena connected with motion and sensation.

As to the influence of the vaso-motor system, though I admit its existence, I am convinced that it it is simply a link in the chain, and is secondary to the emotional disturbance already mentioned.

Treatment.—No cases are so well calculated to test the patience and tact of the physician as those of hysteria. For he has an affection to deal with which not only requires proper medical treatment, but in which he must often exert the highest mental qualities, in order to cure the disease. A great deal, therefore, depends on the knowledge of human nature and the force of character of the physician; and it is doubtless owing to this fact that some physicians, with all their medical knowledge, fail in curing hysterical affections, while others, with no superior science, succeed at once.

The first thing to be done is to gain the confidence and, what is of still greater importance, the respect of the patient. Having done this, any treatment, moral or medical, calculated to relieve her, will be much more apt to produce the desired effect.

During the period between the paroxysms, the treatment must be directed mainly against symptoms. If the patient can be made to believe that her case is thoroughly understood, that she is not suspected of shamming, and that with her assistance the hyperæsthesia, or anæsthesia, or paralysis, will be removed, the effect which is desired will

probably be produced. For putting an hysterical patient into a proper frame of mind, I know of nothing equal to the bromides, of either potassium or sodium, given in large doses, repeated three or four times a day, till the full effect is obtained. This, of itself, will generally relieve hyperæsthesia wherever it may be seated, and the influence over the mental phenomena of the disease is usually very decidedly shown.

If anæsthesia be the prominent condition, electricity is to be used, and it is almost a specific. I have never seen a case of hysterical anæsthesia resist it. A few days ago, I was consulted by a young lady who was entirely anæsthetic over the whole of the surface of one side of the body, and who had suffered for several weeks. Three applications of the induced current through the wire brush, which was passed, at each *séance*, over the whole anæsthetic region, entirely cured her.

For hysterical paralysis, strychnia and phosphorus are the best internal remedies. They may be taken together in the form recommended on page 58, and rarely fail to produce a cure. Their effect is, however, greatly increased by the use of electricity, both of the primary and induced forms—the first being applied to the spine, and the latter to the paralyzed muscles.

In cases of spasm, I prefer the bromides, internally, and the primary galvanic current, applied to the contracted muscles.

Visceral derangements are best treated by strychnia and phosphorus, as recommended for paralysis. Counter-irritation, in the form of blisters, is almost always of service. For gastric difficulties, the subcarbonate of bismuth, in doses of fifteen or twenty grains, after each meal, will generally prove of service. In a very obstinate case of hysterical vomiting, recently under my charge, every thing failed but hydrocyanic acid.

Hysterical paroxysms are best treated with ether or chlo-

roform, administered by inhalation. Recently I have repeatedly used the hydrate of chloral, but it has not in my hands been as speedy or as effectual in its action as either of the other agents. I give them to the extent of producing complete insensibility, and repeat them again and again, if there are any evidences of a return of the seizure. Whether in the purely emotional paroxysms or those characterized by muscular spasms of various kinds, or any possible combination, nothing is equal, according to my experience, to ether or chloroform by inhalation. I have tried every other known means, from cold water, dashed in the face, to moral suasion, and none of them are comparable to ether or chloroform.

But, for the dissipation of the hysterical tendency, long-continued treatment is necessary. Medicines which are ordinarily regarded as antispasmodics, such as valerian, assafœtida, musk, and the like, I have never seen produce any benefit in any form of hysteria, and, for the purpose of causing any radical change in the organism, they are worse than useless. As medicines for this object, I know of nothing superior to phosphorus, in some one of its forms, and strychnia. They should be taken for months in small doses, and should be supported by all hygienic measures calculated to improve the tone of the system. Travel is of inestimable advantage, and, above all, association with persons of both sexes, whose intellects control their emotions, and who are endowed with sound common-sense and that tact and knowledge of human nature which, for the purposes of every-day life, are of more value than many other qualities often ranked above them.

It is, perhaps, scarcely necessary to state that the society of other hysterical persons must be rigidly eschewed, and that even the casual meeting with such individuals is dangerous.

CHAPTER VII.

MULTIPLE CEREBRO-SPINAL SCLEROSIS.

We have already considered the subject of sclerosis as it affects the brain and spinal cord separately. We have still to treat of it as existing in these nervous centres simultaneously. Although recognized, over thirty-five years ago, by Cruveilhier and Carswell, it is only recently, mainly through the observations of Charcot and Vulpian, that attention has been again directed to sclerosis of the cerebro-spinal variety, a form which differs from those already described in this treatise, both in its extent and in the symptoms by which it is characterized.

Symptoms.—The initial symptoms vary according as the morbid process begins in the brain or spinal cord. In the former case, the first prominent manifestation of disease may be an epileptic fit. In other cases, there are headache, vertigo, ocular troubles, such as ptosis, diplopia, or amblyopia, failure of the hearing, and, very often, defective articulation. The mind does not participate to any considerable extent, unless the hemispheres be involved in the lesion.

Or, there may be hemiplegia as a consequence of cerebral congestion, and even mania, from a like cause. These attacks are sometimes frequent, and usually leave more or less mental weakness after them.

Tremor is often first seen in the tongue, more frequently in the eyeball, of one or both sides, which oscillates when the patient is told to turn it inward or outward, but which is steady when he looks directly to the front. This tremor

is called nystagmus, and is, as we have already seen, met with in other diseases of the nervous system.

Tremor is indicative of loss of power, and it gradually becomes more strongly marked and extends to other muscles of the body as other parts of the cerebro-spinal system become involved. It is never, however, a constant phenomenon in any form of sclerosis affecting the spinal cord alone. Its presence is peculiar either to cerebral disease or to lesions occurring at the same time in the brain and spinal cord.

After a time, which is subject to great variation in different cases, the loss of power extends to the limbs, and this feature is often accompanied with aberrations of sensibility. If, as is generally the case, the membranes of the cord are congested or inflamed, there are spasmodic jerkings or twitchings of the limbs, but in some cases these are never observed. In the case of a gentleman from South Carolina, who consulted me at the instance of my friend Dr. Darby, of that State, and who was obviously affected with multiple cerebro-spinal sclerosis, there had never been the slightest involuntary movement, independent of the peculiar form of tremor in the limbs which constitutes so prominent a feature of the disease.

The lower extremities are generally very much more paralyzed than the upper, and, when they become involved, festination often makes its appearance. The gait of the patient, thus, becomes similar to that of a person in whom the lesion is limited to the brain.

If the sclerosis begins in the brain before attacking the spinal cord, tremor precedes the paralysis—the affection being then entirely cerebral in character; but, when, as is generally the case, the lesion appears primarily in the spinal cord, paralysis is noticed before the tremor. In fact, there is never, as previously insisted on in my remarks on multiple cerebral sclerosis, any tremor, unless the superior ganglia of the cerebro-spinal system are involved. The fact that it is

only shown when a voluntary movement is made also assists us to distinguish it from the tremor of multiple cerebral sclerosis, as well as from that of paralysis agitans. In the cerebro-spinal form of the disease, therefore, the patient remains without tremor so long as he is quiescent. But, if he attempts to cross one leg over the other, or to carry a glass of water to his lips, the extremity executing the movement is at once seized with tremor, and the act is performed with great difficulty.

The ability to place the fingers on any part of the body, unassisted by the eyesight, is impaired, as in the cerebral form of the disease, and in sclerosis affecting the posterior columns of the spinal cord.

As the disease advances, the paralysis becomes more strongly marked; the limbs are permanently contracted; the bladder loses its expulsive force; its sphincter no longer completely closes the orifice; the bowels become obstinately constipated, and there is a strong tendency developed to the formation of bed-sores. The head-symptoms likewise increase in intensity, but the mind remains clear to the last in the great majority of cases. Indeed, my observation of many cases has convinced me that in the cerebro-spinal form of sclerosis the hemispheres are not often involved, even when the disease has lasted several years.

The difficulties of articulation notably increase, and the muscles of deglutition likewise become involved. In consequence, the saliva is not swallowed as often as it should be, and it therefore dribbles from the mouth. Mastication is difficult, and the facial muscles gradually become involved. The countenance of the patient at this period is not unlike that of a person suffering from glosso-labio-laryngeal paralysis, as in fact might be expected, the same nerves and muscles being involved. Finally, the patient dies from exhaustion, or from some intercurrent disease.

Few diseases are so irregular and ununiform in their phenomena as the cerebro-spinal form of sclerosis. This

is due to the fact that the organs liable to be the seat of the disease are numerous and of varied functions. The essential features of the affection are tremor occurring generally after paralysis, and only manifested during the performance of voluntary movements. It is not always necessary, however, that the movements should be of the partially-paralyzed limbs, for I have seen cases in which tremor was excited in a paretic leg by the act of executing voluntary movements with a sound hand.

The following histories will contribute to a fuller understanding of the subject:

Cruveilhier[1] reports the case of a cook, aged thirty-seven, who six years before coming under observation noticed that he was losing power in the left leg, so that he nearly fell in the street. Three months subsequently the right leg became similarly affected, and then the superior extremities followed. They were tremulous and weak, but the patient was still able to use them to some extent. The sensibility remained intact, and the reflex faculty of the cord was unimpaired. In other respects the patient was condemned to immobility. There were no spasmodic retractions of the limbs, and no painful contractions. The articulation was imperfect, but the intelligence was unaffected. There appear to have been no marked head-symptoms in this case. "Point de cephalalgie jamais de cephalalgie, le malade entendait à merveille." After death there was found gray degeneration of the spinal cord, of the medulla oblongata, of the pons Varolii, of the right cerebral peduncle, of the right optic thalamus, of the corpora callosa, and of the fornix. The hemispheres were not involved.

Two other cases, similar in general character to the foregoing, are given, in neither of which were the hemispheres involved.

Another case, that of Josephine Pajet, is cited by Cru-

[1] Anatomie pathologique du corps humain, Paris, 1835, 1842, t. ii., liv. xxxii., Fig. 4, Pl. II.

veilhier.[1] In this there was almost complete insensibility of the inferior extremities, though the patient was able to move the toes, the feet, and the legs. There were no cramps and no contractions. There was also diminished sensibility of the superior extremities. All the limbs were weak, and the arms were affected with tremor. The patient could walk and sew when first seen. The right hand was stronger than the left. There was a sensation of a tight band around the abdomen. After death there was gray degeneration of the cord, and of the pons Varolii.

In none of these cases were there spasmodic jerkings or tonic contractions of the limbs. Two cases have been reported by Friedreich.[2] In one of these a man, aged twenty-one, was the subject. Among the first symptoms were mental excitement, vertigo, pain in the head, and weakness of the lower extremities. The gait was unsteady, and there was tremor upon any emotional excitement, or on the attempt to execute movements. This affected the upper and lower extremities, the head and the eyeballs. After death, patches of sclerosed tissue were found on the tubercula mammillaria, the cerebral peduncles, the pons Varolii, and the medulla oblongata.

The other case was that of a woman, aged twenty, who was attacked, when seventeen years of age, with weakness of the right leg. Soon afterward the left became affected, and subsequently the arms. These latter were rendered tremulous at every attempt to move them. The speech was implicated, and there was nystagmus. The mind was weakened, and the sensibility was impaired.

In the first of these cases the disease appears to have begun in the brain; in the second, in the spinal cord.

Vulpian,[3] under a title which goes to show how even the

[1] Op. cit., liv. xxxviii., Fig. 1, Pl. V. [2] Deutsche Klinik, No. 14, 1856.

[3] Note sur la Sclerose en Plaques de la Moelle épinière. L'Union Médicale, No. 70, Juin 14, 1866, p. 507. Like other writers, Vulpian, in this paper, brings together cases which have no affinity except as regards the general character of the lesion.

best authorities have confused the whole subject of sclerosis, describes an interesting case communicated by Charcot. In this instance a woman aged forty-three, of nervous temperament, had been subject to frequent attacks of facial neuralgia, and had often suffered from vague pains without determinate seat. In 1856 she suffered from attacks of vertigo, which, from being rare at first, subsequently came on five or six times a day. Sometimes she fell, but never lost consciousness, or had any convulsive movement.

Shortly afterward, during the night, she was seized with vomiting, cramps in her limbs, and a numbness of the right side. In the morning she was hemiplegic. Fifteen days afterward motion reappeared in the arm, but the leg remained paralyzed. In 1859 she had another attack of hemiplegia, and this time was deprived of speech for fifteen days. After this seizure, there were contractions of the flexors of the fingers, and of the forearm of the right side. In 1861 she had a third attack.

In 1862 (January 1st) she came under M. Charcot's care.

The intellectual faculties were not involved. The right superior extremity was almost entirely paralyzed, and was in a state of rigidity and contraction. The lower extremities were permanently extended, and could not be flexed but by great effort. Sensibility was perfect throughout, and reflex movements could still be excited. She died February 9th.

On post-mortem examination, patches of sclerosed tissue were found in the right middle cerebral peduncle, the pons Varolii, the medulla oblongata, and the cervical region of the spinal cord. The hemispheres were perfectly healthy.

In this case, it is probable that the contractions were mainly due to secondary degeneration of the cord, a condition which, as we have seen, is analogous to sclerosis. It will be observed that there were no tremors, either with or without voluntary motions.

Another important case has been reported by M. Magnan:[1]

[1] Mémoires de la Société de Biologie, Paris, 1869.

A woman, aged thirty-four, came under observation in July, 1869. In 1848, when thirteen years of age, she had an attack of typhoid fever, from which she lost her sight. The first symptom of her disease occurred in 1867, and consisted of trembling of the hands and arms whenever she endeavored to execute any difficult movement. Before long, the tremor involved the lower extremities; but there was no paralysis till about eight months previous to her admission to the hospital. At this time, every effort at motion caused tremor. The hands, arms, legs, eyeballs, and even the muscles of the trunk were involved. The articulation was defective, and there were various painful sensations in different parts of the body. Ophthalmoscopic examination showed atrophy of the optic disks and nerves.

The diagnosis in this case was multiple cerebro-spinal sclerosis—an opinion which I do not think is warranted by the facts. The lesion was probably entirely confined to the brain. The main reason which leads me to entertain this view is, that the tremor appeared before the paralysis. I cite the case for the purpose of showing how little accord there is among authors relative to the association of symptoms with lesions in the several forms of sclerosis.

Eleven cases of the cerebro-spinal form of sclerosis have been under my care; and, though I have not had the opportunity of verifying my diagnosis in a single instance, I think the symptoms have been of such a character as to indicate the existence of the lesion so graphically described by Charcot, Friedreich, and Bourneville and Guérard.[1] The fact, that several of the histories were written out before Charcot's investigations gave me a clew to their real import, will tend, I think, to increase their value.

Mr. M., a gentleman fifty-three years of age, consulted me April 8, 1865, at the instance of my friend Prof. Fordyce Barker, M. D., for partial paralysis with tremor, mainly

[1] De la Sclérose en Plaques disseminées. Nouvelle Étude sur quelques Points de la Sclérose en Plaques disseminées. Bourneville, Paris, 1869.

affecting the right arm and leg. Two years previously he had suffered from vertigo and headache, which were followed by a slight attack of hemiplegia of the right side, unattended by loss of consciousness. He gradually recovered from this, but, about six months before he came under my observation, he noticed that his right leg began to drag, and, soon afterward, that the arm of the same side became weak. About the same time he had headache, vertigo, and weakness of sight. A short time subsequently—about a month as well as he could recollect—the arm was seized with tremor while attempting to carry a glass of wine to his lips. The agitation continued to grow more violent on any voluntary movement of the arm, and gradually his speech became involved.

When I saw him he was still suffering from occasional attacks of vertigo and headache; the lips were agitated whenever he attempted to move them, the tongue was tremulous, and his speech was consequently halting and jerking. There was also nystagmus, a symptom which he had not noticed.

The right arm was unaffected with tremor so long as he allowed it to rest on his knee or to hang by his side; but, in the act of moving it, the whole extremity was agitated by a series of short, vibratory motions, consisting of flexions and extensions, which continued so long as he persevered in the movement, or kept the arm in any position requiring muscular exertion. The right leg was weak, and dragged so that he struck his foot against any slight obstruction. There was a little tremor in it when he attempted to cross it over the other as he sat in a chair.

I treated him solely with the primary galvanic current, which I passed through the brain and spinal cord—the first time such an operation was ever performed in this country for the treatment of disease. My diagnosis was incipient softening of the ganglia at the base of the brain and of the upper portion of the spinal cord. My opinion was, that the

hemispheres were not involved, as there were no symptoms indicating mental weakness or disturbance.

I made an application of about fifteen minutes' duration every day. He gradually but rapidly improved, and to such an extent that on the 19th of April he wrote to me as follows:

"Yesterday must be marked with a white stone as the best day yet. Foot active, hand and arm steady, and spirits good. If we can manage to fix these good effects, cure is certain.

"I hope the magic pile will be ready to repeat its good work on Saturday next."

He continued to improve for several weeks, then gradually went back to his former condition, and from that rapidly grew worse. The paralysis invaded the other side, then tremor followed, the speech became much more difficult, and he died in the country two years subsequently.

Miss H., of Connecticut, aged thirty-five, consulted me January 20, 1870, for paralysis and tremor. About two years previously, she had noticed a weakness of the right arm, which had been preceded by occasional attacks of not very severe headache and vertigo. The arm gradually became weaker, and in the course of a few months began to shake whenever she attempted to use it. Before the year had expired, the right leg began to drag a little, and lost a good deal of its natural strength. Her speech also became difficult, not from any failure to remember words, but from tremor of the tongue and weakness, with a little rigidity of the lips.

When I saw her, the articulation was halting and syllabic; there was nystagmus in both eyes; the right arm was very weak; she could only move the index of my dynamometer four degrees, equivalent to a pressure of two pounds and a half, while with the left hand she could move it twenty-eight degrees. Every attempt to move the arm caused trembling of the whole extremity. So long as she

refrained from any exertion of voluntary power, it remained free from agitation. She could not write, owing to the tremor which the effort to do so excited. There was slight tremor in the leg, when she slowly raised the foot from the ground.

The mind was perfectly intact, and she was entirely free from any emotional weakness.

In this lady's case I diagnosticated multiple cerebro-spinal sclerosis—the "sclérose en plaques disseminées" of Charcot.

I treated her with the chloride of barium and the primary galvanic current. By the following autumn she had improved so much that she could walk several miles without fatigue, lifted her foot clear of the ground, could move the index of the dynamometer to thirty degrees, was free from tremor, except when she attempted to write, and then it was only manifested to a slight extent. I now ceased using the galvanism, but continued the chloride of barium. On the 28th of January, 1871, she paid me a visit. She was then walking well, but there was still a very slight tremor when she attempted to execute delicate or difficult movements with the right arm. I directed the continuance of the barium.

Mr. H., of South Carolina, a highly-educated and intelligent gentleman, consulted me, September 12, 1870, for paralysis and tremor. As he entered my consulting-room, the tendency to festination was exceedingly well marked. On examination, I found his mind perfectly clear. There were nystagmus and syllabic articulation. On moving the left arm or left or right leg, the limb became tremulous. There had never been any head-symptoms.

On the 19th, at my request, he wrote a short account of his disease, which I here transcribe:

"I was never robust in health, but, on the other hand, I have never had, since childhood, a serious spell of sickness. My manner of life has been sedentary—that of a student.

I was always careful not to overtask myself until I became engaged, in the year 1864, in a mathematical research. I was for a considerable length of time very much absorbed in this work, and allowed it to encroach seriously upon my hours of recreation and sleep.

"In the fall of 1865, after having accomplished the above work, I observed a slight lameness in my left foot—a tendency to strike the toe against the inequalities of the ground—an inability to raise quickly enough the front part of the foot.

"After my return home, summer of 1866, from Europe, where I had spent five or six years, the lameness in my foot increased rapidly, and in the winter of 1866–'67 a lameness in my left hand was very perceptible—an inability to move the fingers quickly, and a tremor, particularly of the thumb, when I attempted to do so.

"The above symptoms have gradually grown worse, and within the last year the right leg has become involved, to the extent that it begins to shake when I stand upon it, and it shakes even while sitting, when I am under excitement, or when I execute difficult voluntary motions with my hands.

"The disease seems to make greater progress in hot weather. I have at no time suffered pain, my appetite and digestion are good, and I generally sleep well."

This gentleman improved greatly through the use of the primary galvanic current, chloride of barium, and tincture of hyoscyamus, during the two weeks that he remained in New York under my care. On his return to South Carolina he took a primary-cell battery with him.

On the 11th of January, 1871, he wrote to me as follows:

"Sometimes I thought I was improving slowly, or at any rate not losing ground, and then again, for several days together, I would feel confident that I was falling back. But now I think I can certainly say I am growing worse.

All my symptoms have been worse—lamer, more nervous, and the disease more general in its effects. My right hand, which has heretofore been comparatively unaffected, is now seriously implicated, and yet I still manage to write after a fashion. I find it very difficult to dress myself, and I must make several attempts before I can get up from a sitting or a lying posture.

"What could have caused the improvement that took place while I was under your immediate treatment?"

In this case I diagnosticated multiple cerebro-spinal sclerosis, and I think those acquainted with the disease will agree with me in my view of the case; and yet there was as strongly-marked festination as I have ever seen. The gentleman could trot well, could mount a staircase without much difficulty, but walking slowly, or descending stairs, troubled him greatly. According to some authors, this symptom would, of itself, have been sufficient to contra-indicate the existence of sclerosis, and to have placed the disease among the neuroses. My views on this point have already been expressed under the head of multiple cerebral sclerosis.

J. F., a gentleman of this city, forty-two years of age, consulted me November 29, 1870. On the 4th of July previously he had indulged rather freely in champagne, and the following morning awoke with severe headache, vertigo, and nausea. Although he recovered from this attack, he never felt quite as well as before, and was frequently subject to headache and vertigo—symptomatic, as he thought, of gastric disorder. About a month after his first symptoms he was suddenly conscious of a singular sensation about his left eye, and on looking in the glass discovered that the upper lid had dropped, and that he could not raise it. This was about five o'clock in the afternoon, and by ten that night the lid entirely covered the pupil. The following morning it was not so low, but he found that he saw double. He continued to attribute all his troubles to the

stomach, and began taking some quack remedy recommended to him for dyspepsia.

In the course of a few days, feeling no better, he went to the sea-shore, and while there noticed that his right arm became weak, and that he frequently let things drop from his hand. He had difficulty in shaving and in dressing himself, from inability to coördinate the muscles, and there was numbness of the ends of the fingers. During all this time he had suffered more or less from headache, vertigo, and double vision, and the ptosis still continued. Gradually the left arm became involved, and, by the time the paresis in this extremity was well established, the right arm was affected with tremor, but only when he attempted to execute movements with it. Thus, as he said, he could place the hand on a table and it would continue perfectly quiet; but, as soon as he took a pen to write, or even endeavored to raise the hand from the table, it was seized with tremor. The left arm soon became similarly affected, and eventually the left leg lost strength and was rendered tremulous by any attempt at muscular exertion. He noticed also, what, as I afterward learned, his friends had perceived several weeks before, that his articulation was imperfect, and that it was necessary for him to make a mental effort to talk distinctly.

He returned to the city about the middle of October, and employed a "rubber" to restore, as he said, the circulation to his limbs. Continuing to get worse, he consulted me.

At this time there was festination. The speech was syllabic and accentuated, the tongue and lips were paretic and tremulous, there was nystagmus in both eyes, ptosis and diplopia from paralysis of the left sixth nerve, and dilated pupil of the right eye. There were also occasional headache and vertigo, but not to the same extent as at first.

Both arms and the left leg were partially paralyzed. He could not raise either upper extremity out from the side,

owing to the complete paralysis of the deltoids, but he could flex both forearms, and move his hands and fingers tolerably well. There was no tremor while he refrained from using them, but the least attempt at voluntary motion excited them to agitation. The same was true of the left leg. Examination with the ophthalmoscope showed both optic disks to be white, and the retinal vessels small and straight.

With the dynamometer he could only exert a pressure of nine degrees with the right hand and eleven with the left. The line made with the dynamograph was descending, showing his inability to maintain, even for a short time, a uniform muscular contraction.

There was no loss of sensibility, except in the upper extremities. He had occasionally suffered from pains in the back, about the region of the shoulders.

The power over the sphincters was intact.

This gentleman could stand and walk as well with his eyes shut as with them open. On rising from his chair, which he did with difficulty, he always felt impelled to take a few steps forward, which were a stagger rather than a voluntary movement. In walking, the body was inclined forward, and he went in a kind of jog-trot.

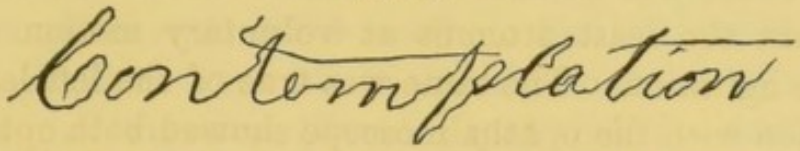
He attributed his disease to dissipation of all kinds, in which opinion I expressed my concurrence.

Under treatment with galvanism, hyoscyamus, and chloride of barium, this patient has improved, but not as yet sufficiently to warrant any strong hope of a permanent cure.

A gentleman from the northern part of the State of New York consulted me in January, 1871, and again in March. His symptoms, though decided, were not very severe in character. Gradually, however, there had been for two years a loss of power supervening in the muscles of the right side of the body, and lately ocular troubles had made their appearance. Tremor, on making any voluntary movement, was just beginning to appear when I last saw him.

Its influence over his handwriting is seen in the following *fac simile:*

FIG. 28.



One patient, with multiple cerebro-spinal sclerosis, attends the out-door department of the New York State Hospital for Diseases of the Nervous System. He has marked head-symptoms. And another, from Philadelphia, who was supposed to be suffering from cerebral disease, consulted me a few days ago. In this case the affection probably resulted from a fall.

The remaining four cases, two of which are now under treatment, do not present any such peculiar phenomena as to warrant their histories being given in detail.

Causes.—Nothing very definite is known of the etiology of the affection in question. It probably is induced by such causes as give rise to the purely cerebral form of the disease. Age does not, however, appear to exercise so important an influence. Four of my cases were over fifty years, and one of them, the gentleman from Philadelphia, was over sixty; six were over forty and under fifty, and one was between thirty and forty. All were males but one.

In two cases, it was apparently caused by excessive mental application, in one by anxiety, in one by a fall, in two by dissipation. In the remaining five cases I could discover no obvious cause. In none of them were there rheumatic, syphilitic, or other morbid diathesis.

Diagnosis.—The facts of the tremor making its appearance after the paralysis, and of its only—or, at least, with rare exceptions, and then only in the latter stages of the disease—being manifested when voluntary movements are being made, will suffice to distinguish the cerebro-spinal form of sclerosis from either of the other varieties. The points to recollect are these: that, in simple cerebral scle-

rosis, the tremor appears before the paralysis, and does not depend on the voluntary contraction of muscles for its excitation; in simple spinal sclerosis there is no tremor at all. I have already insisted on these distinctions in my remarks on the other forms of sclerosis of the nervous centres.

Prognosis.—This is very generally unfavorable. In only one case have I had reason to expect a cure. It often happens that amendment very decided in its character takes place soon after the beginning of the treatment with galvanism and barium. This has been the case in every instance of the disease that has been under my charge; but in only one has it been permanent. In those now under treatment, there has as yet been no relapse; but the time is too short to speak with any confidence in regard to the ultimate result.

Morbid Anatomy and Pathology.—The remarks made under this head, when the cerebral and spinal forms of sclerosis were being considered, apply to the cerebro-spinal variety. Charcot[1] has considered the subject of sclerosis mainly in its histological relations. The main points are—and these have already been stated several times—that the morbid process essentially consists in hypertrophy of the neuroglia at the expense of the proper nerve-substance, and that this is a consequence of inflammatory action. In the present form of the disease, the sclerosed tissue appears in the form of plates or nodules in different parts of the brain and spinal cord.

Treatment.—The means which I have found most efficacious have been stated in the histories of the several cases cited. They consist of galvanism, chloride of barium, and hyoscyamus. The galvanism should be passed through the brain and sympathetic nerve as recommended under the head of multiple cerebral sclerosis (p. 298). In addition, a strong current—that from sixty cells—should be passed

[1] Gazette des Hôpitaux, Nos. 102, 103, 140, 141, 143, 1868.

through the spinal cord, as recommended for antero-lateral and posterior spinal sclerosis.

The chloride of barium should be given in solution in water, in doses of a grain three times a day—the hyoscyamus in doses of from one to two teaspoonfuls of the tincture.

I have sometimes given the nitrate of silver in fourth-of-a-grain doses, three times a day, and very generally recommend cod-liver oil with each meal. Occasionally I have administered iodide of potassium and the bichloride of mercury, with the view of counteracting a possible syphilitic diathesis.

Whatever measures are adopted should be continued for several months at least, and, if the improvement persists, for a much longer period.

CHAPTER VIII.

ATHETOSIS.

Under the name of athetosis (*Ἄθετος*, without fixed position), I propose to describe an affection which, so far as I know, has not heretofore attracted the attention of medical writers, and of which two cases have come to my knowledge. It is mainly characterized by an inability to retain the fingers and toes in any position in which they may be placed, and by their continual motion. From these phenomena, I have applied the term athetosis to the disease, having as yet had no opportunity of ascertaining by post-mortem examination the nature of the lesion to which the symptoms are due.

These symptoms will be evident from the following histories:

J. P. R.,[1] aged thirty-three, a native of Holland, consulted me September 13, 1869. His occupation was bookbinding, and he had the reputation, previous to his present illness, of being a first-class workman. He was of intemperate habits. In 1860 he had an epileptic paroxysm, and, since that time to the date of his first visit to me, had had a fit about once in every six weeks. In 1865 he had an attack of delirium tremens, and for six weeks thereafter was unconscious, being more or less delirious during the whole period.

[1] This patient was several times at my cliniques before the class of the Bellevue Hospital Medical College, first in the autumn of 1869 and last in January, 1871.

Soon after recovering his intelligence, he noticed a slight sensation of numbness in the whole of the right upper extremity, and in the toes of the same side. At the same time severe pain appeared in these parts, and complex involuntary movements ensued in the fingers and toes of the same side.

At first the movements of the fingers were to some extent under the control of his will, especially when this was strongly exerted, and assisted by his eyesight, and he could, by placing his hand behind him, restrain them to a still greater degree. He soon, however, found that his labor was very much impeded, and he had gradually been reduced, from time to time, to work requiring less care than the finishing, at which he had been very expert.

The right forearm, from the continual action of the muscles, was much larger than the other; and the muscles were hard and developed, like those of a gymnast.

When told to close his hand, he held it out at arm's length, clasped the wrist with the other hand, and, then exerting all his power, succeeded, after at least half a minute, in flexing the fingers, but instantaneously they opened again and resumed their movements.

I treated him with galvanism, primary and induced, for four months, without notable result. His fits were, however, arrested with bromide of potassium.

His memory began to be impaired soon after his attack of delirium tremens, and his intellect was manifestly weakened when I first saw him.

January 17, 1871, he entered the New York State Hospital for Diseases of the Nervous System, when the following points, which I cite from the report of Dr. Cross, the Resident Physician, were noted:

The head is symmetrical, but is peculiar in shape—the posterior portion rising to a much higher point than the anterior, while the latter slopes downward and forward, giving the cranium the form of that of a Flathead Indian.

The special senses are normal. The intellect is somewhat impaired, and his ideas are not so vivid at one time as at another. His memory is much enfeebled. There is slight tremor of both upper extremities, but there is no paralysis of any part of his body. There are, however, involuntary grotesque muscular movements of the fingers and toes of the right side, and these are not those of simple flexion and extension, but of more complicated form. They occur, not only when he is awake, but also when he is asleep, and are only restrained by certain positions, and by extraordinary efforts of the will. Thus those of the fingers are arrested when the wrist is firmly grasped by a strong hand, or when it is less forcibly held in a vertical position. But, if the arm be extended horizontally, the fingers at once begin their movements. During their continuance the arm is hard and rigid, and the calf of the leg is also in the same state of tonic spasm while the toes are in motion. The movements are somewhat paroxysmal, being worse at times than at others. During the remissions the power of the will over the muscles is more effective than when the paroxysms are at their height.

Sensibility to touch, pain, tickling, and temperature, are normal in all other parts of the body. There is slight tremulousness of the tongue, but no difficulty of articulation. There are no oscillatory movements of the eye-balls (nystagmus).

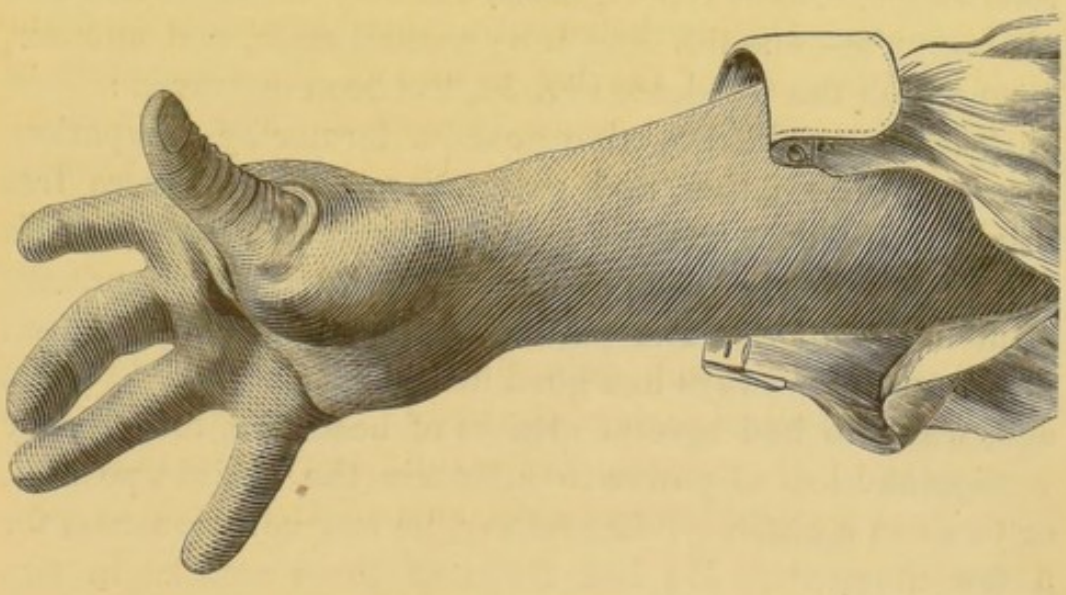
The involuntary contractions of the fingers and toes do not take place quickly, but slowly, apparently as if with deliberation and with great force. The numbness and pain in the arm, hand, leg, and foot, have increased in proportion to the increase in the contractions.

The toes are not involved to the same degree as the fingers. Position does not, however, afford the same relief to them as to the fingers, and the spasms are more tonic in character. The muscular development is greater in the right arm and leg, from the almost continuous muscular

action. The toes are kept restrained to some extent by the boot, but as soon as it is removed they become flexed and take on their peculiar movements.

When, by a strong effort of the will, he succeeds for an instant in arresting the movements in the hand, the little finger at once becomes strongly abducted, the third finger participates to some extent, the second finger is slightly flexed, the index-finger is extended, and the thumb is extended to its very utmost. These are the positions in all cases in which he succeeds in quieting the actions, and they are well shown in the accompanying woodcut (Fig. 29) taken from a photograph.

Fig. 29.



On account of the severe pain in the whole arm, caused by the spasms in the muscles, the patient is at times unable to go to sleep until quite exhausted. On awaking, however, after a few hours' repose, although the actions have continued during his sleep, they are not so severe as at any other time through the day or night. This state of comparative repose lasts for about half an hour.

His habits are bad. He boasts that he has often drunk as many as sixty glasses of gin in a day, and it is therefore doubtful whether the tremulousness observed in the tongue

LEEDS SCHOOL

and the muscles generally is the effect of the disease, or of drink, or of both combined. I have never, however, seen him drunk, or even under the influence of liquor. His mental faculties are decidedly more obtuse than when he first came under my observation.

Under the use of the primary galvanic current to his brain, spinal cord, and affected muscles, and the internal use of chloride of barium, he is certainly improving, but I have little hope of any permanent result being obtained. His epileptic paroxysms are kept down with bromide of potassium.

The second case occurred in the practice of Dr. J. C. Hubbard, of Ashtabula, Ohio, who forwarded to me the following excellent report, dated January 11, 1870, and two photographs—one full length on a small scale, and another, from which the woodcut, Fig. 30, has been engraved:

"H. S., aged thirty-nine years, a farmer by occupation, married. His father and paternal grandfather were free drinkers of ardent spirits. His only brother died of phthisis pulmonalis, and I think he inherits a tubercular tendency from his mother. The patient is short, muscular, is well made, and has always had good health till about eight years ago, when he had several attacks of headache, followed by vertigo and loss of power to maintain the upright posture, or to sit in a chair. After falling, he lost consciousness for a few moments. He had three of these attacks in two months.

"Three years after the last one, being five years and a half ago, while at work on a hot day in the open air, he lost consciousness and fell to the ground. This attack was more severe than the preceding ones, and he was confined to his bed three days. The headache was very severe, and continued a week after he left his bed. Aphasia, and the incoördination now affecting his right forearm and right leg, were the sequence of this stroke. His powers of speech were gradually reëstablished in the course of six weeks, but

the impediment to normal voluntary muscular motion has remained to this day.

"In June last [1869] he applied to me for relief from cephalalgia, pain in the right side of the chest, cough, and dyspnœa. He complained also of vertigo and of flashes of light before his eyes. His memory and judgment were slightly impaired, and he was gloomy and irritable.

"His utterance of most words was perfect, but he stammered over at least one word in each sentence. It required a good deal of effort for him to connect his ideas and his sentences. He stumbled at monosyllabic words, such as *and*, *then*, *to*, *at*, and other conjunctions, but in a moment, after considerable effort, he could speak these words and conjoin his sentences correctly.

"On examining his right foot, I found that he had lost the normal antagonizing force between the flexors and extensors of the toes. The toes were ordinarily in a state of flexion, so as to present their ends to the floor. He could restore the balance in muscular action by a strong effort of the will, pressing at the same time the sole hard upon the ground, and drawing the foot backward a little. Soon, however, the extensors would be wearied by their extra work, and the toes would resume their abnormal position. The foot is slightly inverted at every step, and it is not exactly guided by the will. His gait is awkward—the foot being set down with a kind of pawing motion, as in talipes varus.

"A similar incoördination is observable in the right hand and fingers. He cannot flex his fingers without the aid of the opposite hand, but when it is closed the grasp is as strong as ever. By an intense action of the will he can keep his fist closed for a few moments, till the apparently tired flexors give way. The little and ring fingers are but partially extended, and are strongly abducted. The abductor minimi digiti, and the flexor brevis minimi digiti, are hypertrophied, firm, hard, and in a state of contraction

most of the time, and the affected hand measures three-fourths of an inch more around the palm than its fellow. Tactile sensibility is as perfect in the affected limbs as in the others. His muscular powers are good, and he thinks he can walk twenty-five miles without injurious fatigue. The temperature of the affected limbs is slightly lower than that of the opposite ones. Has slight headache frequently, generally at evening; sleep relieves it. He sleeps well when undisturbed by pains in his limbs. Tongue clean and tremulous. Has slow, moving pains, from the hand and foot up to the body. They often last half a day, and are worse at night. Has no pain, tenderness, or feeling of weakness, in any part of the spine.

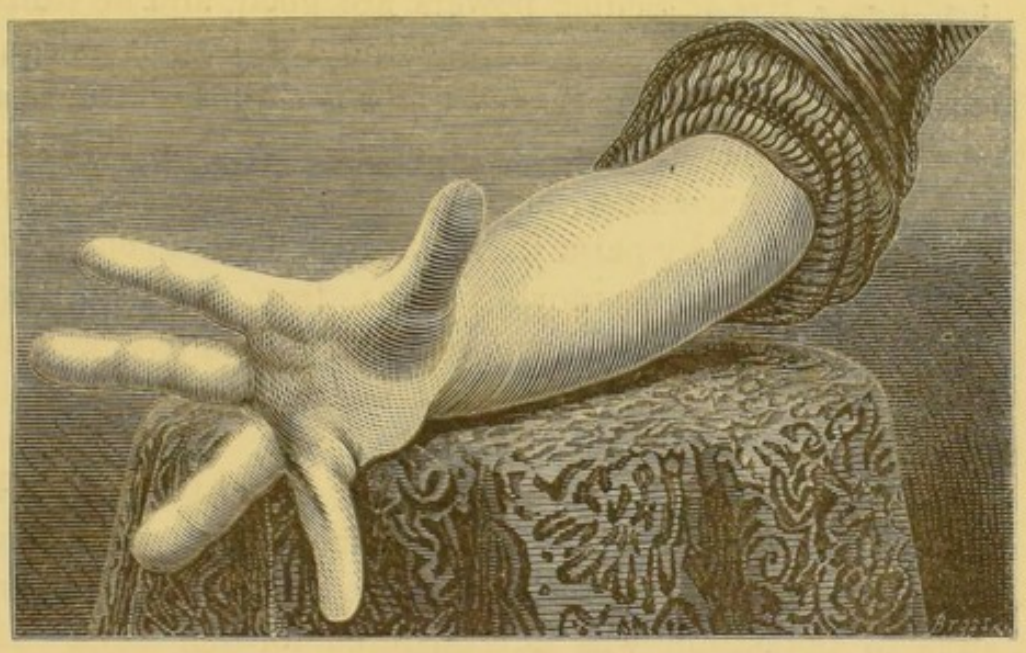
"He had no systematic treatment till last June. The chest-symptoms referred to were owing to subacute bronchitis. A seton was inserted between the shoulders, and iodide of potassium was administered for ten days. His lungs being then better, phosphoric acid, cerium, cannabis Indica, sulphate of quinine, and sulphate of iron, were given till the 1st of December following. He then felt so much better that he discontinued the medicines. The seton continued to discharge till the date of this communication [January 11, 1870], and he presents at this time a very marked improvement. His headache is not severe, he has less pain in his limbs, and he speaks without hesitation. By a strong effort of the will he can close his hand without assistance. He came five miles on foot, in a driving snow-storm, to see me to-day."

The accompanying woodcut (Fig. 30) is from one of Dr. Hubbard's photographs. The resemblance to the condition shown in Fig. 29 is very striking, and the histories of the two cases are so nearly identical, in regard to all essential points, as to leave no doubt that they describe instances of the same disease. Dr. Hubbard's case was probably, when he wrote the history, in a more advanced state than is mine at the present time. The distortion of the hand is certainly

SCHOOL OF MEDICINE

greater. In the other photograph, which is indistinct, the toes are seen fully flexed.

Fig. 30.



The symptoms of athetosis are clearly indicated in the foregoing histories. Both cases came on with epileptic paroxysms—a feature accompanying other organic diseases of the brain and spinal cord. In both there are similar head-symptoms, tremulousness of the tongue, numbness on the affected side, pains in the spasmodically-affected muscles, and especially complex movements of the fingers and toes, with a tendency to distortion. In neither case is there any paralysis. Relative to the character of the lesion producing these symptoms, and its exact seat, I am not yet prepared to speak with any degree of certainty. The phenomena indicate the implication of intra-cranial ganglia, and the upper part of the spinal cord. The analogies of the affection are with chorea and cerebro-spinal sclerosis, but it is clearly neither of these diseases. One probable seat of the morbid process is the corpus striatum.

I should not have incorporated these cases and remarks in the present treatise, but with the hope of calling out the

experience of others on the subject, by directing attention to an affection which has probably heretofore been overlooked or confounded with some other.[1]

[1] Since the foregoing chapter was written, my friend and colleague, Prof. Fordyce Barker, to whom I showed the cuts and described the cases, has informed me that several years ago he had an exactly similar case in his practice.

SECTION IV.

DISEASES OF NERVE-CELLS.

Under this section I propose to consider certain diseases which are due to degeneration and atrophy of the cells in intimate relation with nerve-roots, and which immediately preside over the functions of the nerves arising from them.

CHAPTER I.

ATROPHY AND DISAPPEARANCE OF TROPHIC NERVE-CELLS (PROGRESSIVE MUSCULAR ATROPHY).

Although cases of progressive muscular atrophy were noticed by the older writers, the first systematic account of the disease was given by Duchenne,[1] in 1849. In 1850 M. Aran[2] published his memoir, in which he gives the histories of eleven cases; and three years subsequently Cruveilhier[3] read a paper on the same subject before the Académie de Médecine. About the same time other memoirs were published on the subject.

[1] Atrophie musculaire avec transformation graisseuse. Mémoires de l'Académie des Sciences, 1849.

[2] Recherches sur une Maladie non encore décrite du Système musculaire. Arch. Gén. de Méd., 1850.

[3] Sur la Paralysie musculaire progressive atrophique. Arch. Gén. de Méd., 1853.

But, although Cruveilhier was not the first to write upon the affection in question, he was the first to describe it, and Duchenne and Aran were aware that he had done so in his lectures for several years. The disease is therefore sometimes called Cruveilhier's atrophy.

Symptoms.—The first symptom observed in the majority of cases is loss of strength and dexterity in certain muscles of the body. If these are in the lower extremities, the patient finds that he tires in walking sooner than he used to do. If in the upper extremities, he experiences weakness in the shoulder, arm, or hand, according to the muscles affected.

Soon afterward pains simulating those of neuralgia are felt in the paretic muscles, and in the majority of cases—according to my experience in all—fibrillary contractions are perceived. Thus, of twenty-nine cases of progressive muscular atrophy which have been under my charge during the past six years, these contractions formed a prominent feature in every one. They consist of slight twitchings of separate bundles of muscular fibres, and give the sensation of something alive being under the skin. They can often be seen, especially when superficial fibres are involved, and they are generally the *avant courriers* indicating the extension of the disorder.

The loss of strength attracts the attention of the patient to his limbs, and then he finds that the weakness is accompanied by atrophy. If, as is usually the case, the disease begins in one of the upper extremities, the thenar and hypothenar eminences very commonly give the first evidence of atrophy. The ball of the thumb disappears, and the muscles filling the space between the first and second metacarpal bones—the adductor pollicis and the first interosseous—likewise shrink away. The whole outline of the metacarpal bone of the thumb can thus very soon easily be made out.

The ball of the thumb is often the starting-point of the disease, and, when this is not the case, it generally becomes

involved at some time or other in the course of the affection. Of the twenty-nine cases occurring in my experience, the disease appeared first in the ball of the thumb in eight, and eventually attacked this part in thirteen others. The upper extremities were the original seat of the disease in seventeen cases, the trunk in four, and the lower extremities in eight. Whether the affection begins in an upper or lower extremity, the tendency is for the opposite member to be next involved.

The physiognomy of progress in muscular atrophy is very striking, particularly when the face or the hand is its seat. No very well marked case of the former has come under my observation, but it can readily be understood that the change effected by the disappearance of the facial muscles must be very evident. In the hand, the atrophy of the muscles which give this member its plumpness, and enable it to perform the complex movements of which the fingers are capable, causes appearances which are easily recognizable. By the disappearance of the thenar and hypothenar eminences, the skin over them hangs in loose folds, the thumb falls by its own weight, and cannot be brought into apposition with the index-finger—the palm of the hand is hollowed out, and the metacarpal bones can be distinctly seen and felt.

In the forearm, the situation of the disease can be readily ascertained by the flattening produced by the disappearance of the affected muscles, and in the arm and shoulder the effects of the disease are still more evident. In two cases, one of them sent to me by my friend Prof. Van Buren, the disease had begun in the right deltoid, and had not extended beyond this muscle when the patients came under my charge. In both, the shoulder was flattened, and the head of the humerus and the acromion process could be distinctly seen. In another case it was limited to the trapezius and scapular muscles of both sides.

In the lower extremity, the changes in the foot are not so remarkable as the corresponding ones in the hand, but the

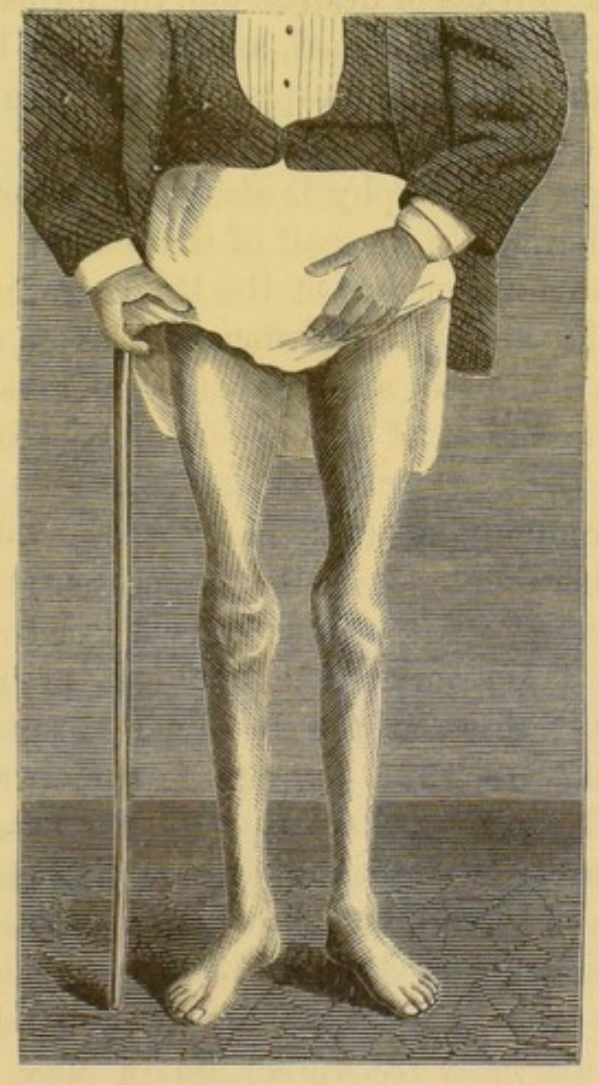
effects produced by the atrophy of the peroneal muscles, the tibialis anticus, and those forming the calf of the leg, are very striking. In the one case, the foot drops, and the patient is obliged to bend the knee to a greater extent than usual in order to make the toes clear the ground; in the other, the heel cannot be raised, and the ankle gives way with the weight of the body. When the muscles on the anterior face of the leg are in process of destruction, the forms of the tibia and fibula can be distinguished, and the space between the two bones is unfilled. The disappearance of the calf makes the posterior aspect of the leg flat.

In the thighs the atrophy is also readily perceived, and modifies very materially the gait of the patient. When the extensors on the anterior face of the thigh are involved, the leg cannot be thrown forward; when the flexors are the seat, the leg cannot be raised, and the whole member has to be lifted up by the action of the flexors of the thigh on the pelvis.

In the accompanying woodcut (Fig. 31), taken from a photograph, there is an excellent representation of the lower extremities of a patient affected with progressive muscular atrophy. He formed the subject of my clinical lecture on this disease at the Bellevue Hospital Medical College, February 18, 1871, having the same day been admitted to the New York State Hospital for Diseases of the Nervous System. The affection began with electric pains in the legs, weakness, and head-symptoms, consisting of confusion of ideas, vertigo, dimness of vision, headache, etc. There was also numbness in both the lower and upper extremities. He partially recovered, but in May, 1867, there was a return of the head-symptoms, the electric pains, and numbness, to which were superadded cramps, fibrillary contractions in both hands and legs, with tingling and twitching. In the course of three weeks he was obliged to use crutches. From this time, he noticed the atrophy of the muscles of both legs, and it has gradually extended till it

has involved the muscles of the lower third of both thighs.[1] In the legs, the extensors are almost entirely destroyed, as are also the gastrocnemii and solei. The figure, owing to the position of the patient when the photograph was taken, does not show very well the effects of the disease in the legs, but the atrophy in the thighs is distinctly indicated.

FIG. 31.



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Besides the paralysis, which it must be clearly understood results from the atrophy, and is directly proportional to its extent, there may be contractions. These, when present, are due to the fact that the atrophy has not attacked all the muscles of an extremity simultaneously, or to a like

[1] From notes prepared by Dr. Cross, Resident Physician of the hospital.

degree, and consequently, the normal antagonism being destroyed, distortions take place. When these occur in the hand, they produce the *main en griffe* of Duchenne. Of the twenty-nine cases occurring in my experience, seven only had any distortions. In infantile paralysis, which is similar in several respects to progressive muscular atrophy, contractions and distortions are much more common.

The reflex movements are generally diminished, except in the early stages, while the fibrillary contractions are present, and the electric contractility diminishes *pari passu* with the muscular tissue. The temperature of the affected parts is always lowered several degrees, and the capillary circulation is languid.

The pupils are sometimes contracted from the implication of nerve-cells in the cilio-spinal region of the cord. This was the case in one or both eyes in four of my cases.

The course of the affection is slow, but in the great majority of cases it advances to a fatal termination. Death takes place from the muscles of respiration becoming involved, from exhaustion, or from some intercurrent affection. Several of my cases have lasted over ten years.

In a recently-published memoir, MM. Duchenne and Joffroy[1] have shown that glosso-labio-laryngeal paralysis is sometimes complicated with progressive muscular atrophy, and that this latter affection, implicating the muscles of the tongue, the lips, and the veil of the palate, has hitherto been confounded with the first-named disease. It differs from it, however, in the essential fact, which is applicable to the disorder appearing in other parts of the body, that the loss of power is not the initial symptom, but results directly from the diminution in the size of the muscles.

Causes.—Progressive muscular atrophy is not a disease of old age. Only one of my cases was in a person over fifty; two were between forty and fifty, and twenty-six were under

[1] De l'Atrophie aiguë et chronique des Cellules nerveuses de la Moélle et du Bulbe rachidien. Arch. de Phys., No. 4, 1870, p. 499.

forty. Of these latter, two were between fifteen and twenty, and two between eight and ten. The period of life at which it appears to be most common is that extending from twenty-five to thirty-five.

Sex is a strong predisposing cause. All of my cases were in males. Roberts[1] states that, of ninety-nine cases, eighty-four were males, and only fifteen females. Other authors have noted the great proclivity of males. The difference appears to be due to the greater severity of muscular exertion required in many of the occupations of men.

Hereditary influence is a well-recognized predisposing cause. Two of my cases sent to me by Dr. Lincoln, of Washington City, were brothers, and nine others had relatives affected with the disease.

The exciting cause is often impossible of detection. This was the case in eighteen of the instances that have come under my observation. Of the remaining eleven, injuries of the spine were the cause in two; exposure to cold and dampness in three, and excessive muscular exertion in six. Of these latter cases, one occurred in the person of a ballet-dancer, the disease making its appearance first in both gastrocnemii muscles simultaneously; one in a gentleman who had overtasked the muscles of the upper extremities by severe and long-continued exertion in rowing, the muscles about the shoulders being affected; in one, the muscles of the right hand were first attacked, as the result of excessive use of the pen in writing; in one, it was induced by the occupation, that of a bricklayer, requiring the patient to bear the weight of his body, during his work, mainly on one leg—the one attacked; in one, it was apparently induced by running a long distance; and in one, it attacked the muscles of the hand and forearm, beginning in the ball of the thumb in a man whose occupation—faro-dealer—required him to use his thumb and fore-finger in a peculiar way for many hours at a time.

[1] An Essay on Wasting Palsy. London, 1858, p. 135.

Diagnosis.—The sharp, shooting, electric pains may, in the beginning, cause progressive muscular atrophy to be mistaken for posterior spinal sclerosis, but the subsequent symptoms are so very obvious, that the error cannot be of long duration, and are of such a character as to render the recognition of the affection a matter of no difficulty.

Prognosis.—From what has been said, it will readily be apprehended that progressive muscular atrophy is a very serious disease; indeed, it is one of the most progressive of all the affections to which the term has been applied.

In only two cases have I succeeded in arresting the course of the disease, and in restoring the atrophied muscles. One of these was that of a highly-intelligent gentleman, formerly an officer of the navy, but now a resident of this city, whose case has already been referred to as having been induced by rowing; the other, was that of the patient sent to me by Prof. Van Buren, also previously mentioned, in whom the affection was induced by cold, and which began in the right deltoid muscle. Both of these patients were entirely cured, regaining full muscular power.

In three other cases, which I saw before the disease had advanced to a great extent, its progress was arrested, but there has as yet been no restoration of the wasted muscles; in neither of these was there any probable cause of the affection.

The existence of an hereditary tendency renders the prognosis much more grave; and the fact of the disease having lasted a long time is also of unfavorable import.

Morbid Anatomy.—Investigations in regard to the morbid anatomy of progressive muscular atrophy relate to the condition of the muscles, of the nerves supplying them, and of the centres from which the nerves are derived.

The atrophy of the muscles is due to the degeneration and ultimate disappearance of the fibrillæ. To the naked eye they appear pale and attenuated. By microscopical examination, it is seen that the transverse striæ of the fibril-

læ are in course of disappearance, and as the disease advances they are perceived to fade away altogether. Eventually, the longitudinal striæ also disappear. At the same time, the muscular fibrillæ break up into granules, and then undergo regressive metamorphosis into fat. It is not uncommon to see a bundle of fibrillæ, in one part of which the transverse striæ only have vanished; in another, the longitudinal; in another, the process of disintegration complete; and in another, oil-globules occupying their place. Fat-corpuscles are frequently found deposited between the bundles of fibrillæ. After a time the fat disappears, and nothing is left of the muscle but a cord of connective tissue made up of the myolemma.

By means of the little trocar described in the introduction, I have frequently removed small pieces of atrophied muscles during life, and submitted them to microscopical examination.

The anterior roots of the spinal nerves ultimately distributed to the affected muscles are generally found atrophied, from the disappearance of a certain number of nerve-tubes. This feature was first observed by Cruveilhier.

The spinal cord has been examined in cases of progressive muscular atrophy by Bergmann, Meryon, Gull, Luys, Lockhart Clarke, and others, with very different results; some of these observers finding no change whatever, and others detecting notable variations from the normal structure. In three cases examined by Clarke,[1] disorganization of the spinal cord, especially of the gray matter, was found, with, in one case, deposit of amyloid corpuscles.

More recently Hayem,[2] and Charcot and Joffroy,[3] have

[1] Beale's Archives of Medicine, vol. iii., 1861; also, same, vol. iv.; also, British and Foreign Medico-Chirurgical Review, vol. xxx., 1862.

[2] Note sur un cas d'Atrophie musculaire progressive, avec Lésions de la Moélle. Archives de Physiologie, No. 2, 1869, p. 221, and No. 3, 1861, p. 391.

[3] Deux Cas d'Atrophie musculaire progressive, avec Lésions de la Substance grise et du Faisceaux antéro-latéraux de la Moélle épinière. Arch. de Phys., Nos. 3 and 5, 1869.

studied the morbid anatomy of progressive muscular atrophy with great care. In Hayem's case, the disease affected the muscles of the upper extremities to such an extent as to render them powerless from the shoulders down. The patient died from paralysis of the diaphragm and of pneumonia.

On post-mortem examination, the spinal cord appeared healthy to the naked eye. The anterior roots of the cervical nerves were, however, notably atrophied. The most attenuated were those of the second, third, fourth, and fifth pairs. The sympathetic was healthy. On microscopic examination of the cord, the most marked characteristic was atrophy and disappearance of the nerve-cells. In some portions there were none to be seen, but there were large numbers of free nuclei, and of cells containing many nuclei. The atrophy of the nerve-cells, and of the anterior cornua of gray substance, was greatest at the level of the second and third cervical nerves, and extended as low as the fifth cervical. This region was that from which the nerves supplying the atrophied muscles were derived. In the dorsal and lumbar regions there was no atrophy of nerve-cells or of nerve roots.

A consideration of this case shows, as Hayem remarks, that it is one which, during life, exhibited the usual symptoms of progressive muscular atrophy, and that, at the post-mortem examination, lesions were found in the muscles in the anterior roots of the nerve, and, above all, in the spinal cord. The alterations from the healthy structure of the cord consisted of

1. Abnormal vascularization with dilatation, and sclerosis of the arterioles, and of the larger capillaries.

2. A more or less abundant exudation surrounding the blood-vessels.

3. Multiplication of the elements of the interstitial tissue (the neuroglia), and, finally, atrophy, and disappearance of a very great number of the nerve-cells.

These facts point to the existence of chronic inflammation of the gray substance of the cord.

The two cases of MM. Charcot and Joffroy have also been very carefully and thoroughly studied.

The chief features of the first case were, progressive muscular atrophy, especially marked in the superior extremities; atrophy of the muscles of the tongue and of the orbicularis oris, and paralysis with rigidity of the inferior extremities. The patient was a woman, and, becoming suddenly very weak, died asphyxiated.

At the autopsy, the anterior roots, especially those of the cervical region, were found greatly atrophied and discolored. The cord appeared healthy to the naked eye, except that at the dorso-lumbar enlargement it was softened. On microscopical examination, however, the nerve-tubes of the anterior columns were discovered to be atrophied, a great number being only represented by the axis cylinder while the connective tissue was very much increased. The posterior columns were not involved in the least.

In examining the gray substance of the cervical region, the authors were struck with the extreme degree of atrophy which the cells of the anterior cornua had undergone; a large proportion of them had entirely disappeared, leaving no trace behind them. The posterior cornua appeared to exhibit all the qualities of the normal condition.

The alterations in the other regions of the cord were not directly connected with the progressive muscular atrophy, except as regards the medulla oblongata where the cells of the nuclei of origin of the hypoglossal were found to be atrophied, and even completely destroyed. In the second case, similar structural changes were found.

The essential points in the morbid anatomy of progressive muscular atrophy are no longer a matter of doubt. The bearing of these points on the real nature of the disease is next to be investigated.

Pathology.—At the outset of the inquiry relative to the pathology of progressive muscular atrophy, the question arises, Is the affection a disease primarily of the muscles or of the nervous system? From the fact that the wasting of a muscle so frequently resulted from its excessive use, it was, and still is, supposed by some pathologists that the lesion is essentially one of the muscles, and that the nerves and spinal cord are secondarily involved. But the conclusion does not logically follow the premise, and observation shows very conclusively that excessive use of a muscle exhausts, not the muscle primarily, but that part of the nervous centre from which the nerves come which supply that muscle. The consequence of this exhaustion is atrophy and disappearance of certain cells in intimate relation with the roots of the nerves supplying the atrophied muscles. By this theory is to be explained the occurrence of progressive muscular atrophy in the legs of the ballet-dancer, and the thumb of the faro-dealer, previously mentioned.

The destructive metamorphosis of the nerve-centre proceeds at a greater rate than its nutrition, owing to the extreme demand made upon it by the muscles put in excessive action, and hence their atrophy and disappearance.

A central disease being thus set up, it extends by contiguity and involves those nerve-cells which are nearest. These are the cells which supply the corresponding nerves of the opposite side, and therefore it is that the disease, like several others, tends to advance symmetrically, affecting homonymous muscles.

Of course it frequently happens that progressive muscular atrophy may originate from disease of the nerve-cells without the muscle having been put to inordinate use. In such cases the process is one of excessively chronic inflammation.

One other very important question remains to be considered, and that relates to the physiological character of the cells which have degenerated and have disappeared.

The spinal cord is admitted to be connected with two distinct faculties—motion and sensation. It is probable, therefore, that there are at least two kinds of nerve-cells in the gray substance of the cord, which, though alike in anatomical characteristics, differ essentially in their functions. One set is motor and one sensory. In those cases of spinal paralysis involving motion, and in which there is atrophy of nerve-cells, the motor cells are diseased; in those in which sensation is affected, and in which atrophy of nerve-cells is discovered, the sensory cells are the ones implicated.

Now, progressive muscular atrophy, pure and uncomplicated, is unattended by derangements of sensation, and unaccompanied by paralysis, except such loss of power as is directly due to the diminution of the volume of the affected muscles. The presumption is, therefore, that neither the motor nor the sensory cells have disappeared or become atrophied, and yet, on post-mortem examination, we find that nerve-cells of *some* kind have been diseased. The presumption is, and it is reasonable, that these are cells which are specially connected with the nutrition of muscles—trophic cells—and that progressive muscular atrophy is a symptom indicating the existence of disease of the trophic cells. The very existence of these cells is a matter of inference, but in my opinion the argument in favor of the affirmative is very much strengthened by the facts furnished by the morbid anatomy of progressive muscular atrophy. Dr. Handfield Jones[1] has recently written forcibly against the existence of any special trophic nerves, and, by extension of reasoning, trophic nerve-cells. But he was unaware of the more recent researches of Duchenne and Joffroy,[2] upon which, in accordance with these observers, I have based my views of

[1] Are there Special Trophic Nerves? St. George's Hospital Reports, vol. iii., 1868, p. 89.

[2] De l'Atrophie aiguë et chronique des Cellules nerveuses, etc. Archives de Physiologie, No. 4, 1870, p. 499.

the pathology of progressive muscular atrophy, and to which I have already alluded.

Treatment.—The treatment of progressive muscular atrophy is nothing without the use of the primary galvanic current. This should be applied every alternate day to the spinal cord, and to the sympathetic nerve. In the first instance the current should be employed in the manner recommended under the head of spinal anæmia; in the latter the positive pole should be applied to the cilio-spinal centre, and the negative to the sympathetic nerve in the neck. The current should be strong enough to give a decided sensation—that from fifteen to twenty Smee's cells will usually be sufficient—and it should be applied for about ten minutes to the cord, and five to the sympathetic every alternate day.

At the same time both the primary and induced currents are efficacious in improving the nutrition of the atrophied muscles, by applications made either directly, or to the nerves which supply them.

For internal treatment, the means recommended for posterior spinal sclerosis are the best. Iodide of potassium should of course be given when there is suspicion of syphilitic taint.

Hydrotherapeutics may be of service, and a few cases have been reported of benefit therefrom.

The treatment recommended should be continued for several weeks before any decided opinion can be given relative to its efficacy, and for many months before a cure can be expected.

CHAPTER II.

ATROPHY AND DISAPPEARANCE OF MOTOR NERVE-CELLS.

Although it is probable that there are several diseases which consist of atrophy and disappearance of motor nerve-cells, only one has been so far studied sufficiently as to warrant our associating it with the lesion in question, and that is—

GLOSSO-LABIO-LARYNGEAL PARALYSIS.

The first explicit account of this very remarkable disease is that of Duchenne,[1] who, in consideration of the tendency of the morbid process to advance unchecked, and of the parts affected, designated it "progressive muscular paralysis of the tongue, the veil of the palate, and the lips." The consequences of this condition, as pointed out by Duchenne, are difficulties of articulation and of deglutition, and at a late period of the disease frequent attacks of strangulation, during one of which the patient may die; or death may result either from inanition or syncope.

But, although Duchenne was the first to give a systematic description of the affection, it was observed by Trousseau in 1841, just twenty years before the publication of Duchenne's account, who recognized it as an affection he had not previously seen, and who wrote a memorandum of the existing phenomena.[2] Trousseau named the disease glosso-laryngeal paralysis, in his lecture on the subject, and

[1] De l'Électrisation localisée, etc., deuxième édition, Paris, 1861, p. 621.

[2] Lectures on Clinical Medicine, Bazire's translation, p. 117.

this was afterward amplified by Duchenne into glosso-labio-laryngeal paralysis. Many cases have been subsequently reported, and descriptions of the affection given, but no one has added any thing to the graphic symptomatology of Duchenne.

Eight cases of the disease have come under my observation during the past six years. I am enabled, therefore, to describe it from my own observation of its phenomena.

Symptoms.—It rarely happens that patients seek medical advice for the initial symptoms of the disease under notice. We are therefore, in general, obliged to rely on their accounts of the order and progress of the symptoms. In one instance only—and this patient is still under treatment—have I had the opportunity of observing a case from a very early point in the course of the disease.

The first evidence of disease, which in the majority of instances attracts the attention of the patient, is a slight difficulty of articulation, due to a want of rapidity and exactness in the movements of the tongue. This circumstance occurred in seven of my cases. In the other the symptom first noticed was a tendency in the lips to remain separate, and the consequent necessity of using some degree of mental action to keep them closed. In a short time the restraint in the motions of the tongue becomes more distinctly marked, and it is especially characterized by an inability to raise the extremity to the roof of the mouth, or to press it against the upper teeth. The words, therefore, which he experiences most difficulty in pronouncing distinctly are those which begin with lingual or dental consonants. The gutturals he can articulate without trouble; and the labials, except when the affection begins in the lips, do not yet give him inconvenience.

The next symptom to make its appearance is difficulty of swallowing. The food is not promptly grasped by the constrictor muscles of the pharnyx, and the tongue does not press it strongly against them. At times it enters the

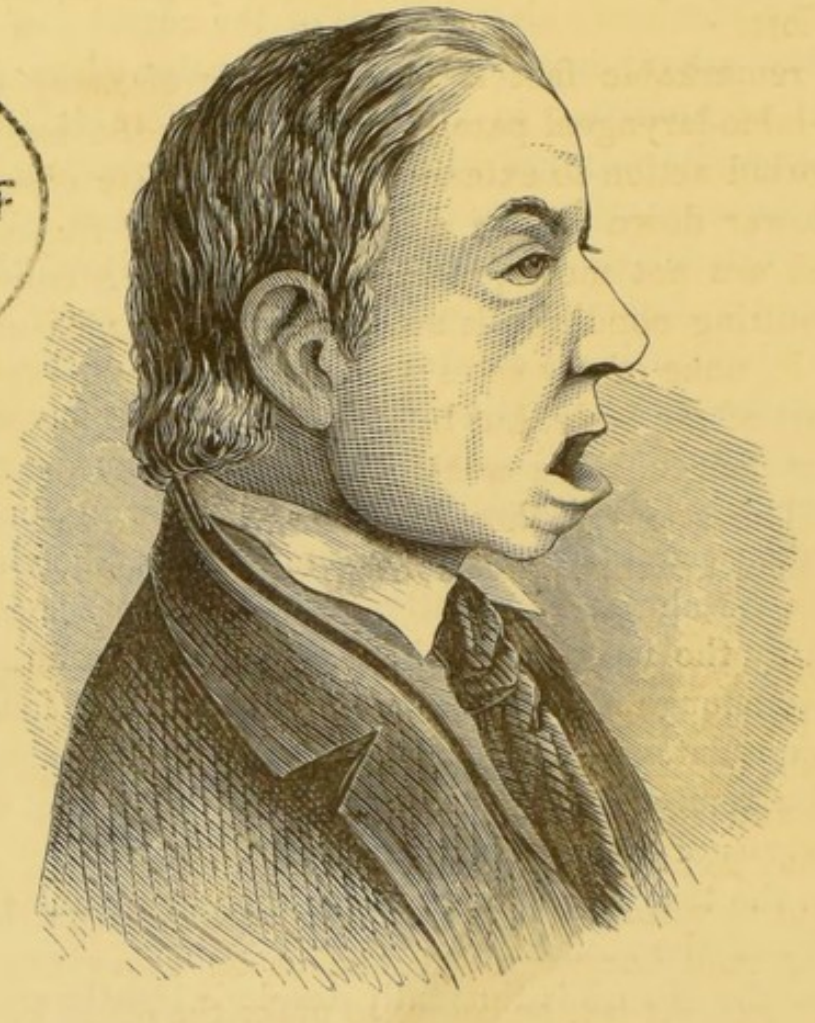
pharynx, and, not being carried onward by the muscles of deglutition, may slip into the larynx and occasion suffocation. Liquids are especially difficult to swallow, and are often ejected through the nostrils.

As the result of this paralysis of the muscles of deglutition, the saliva, instead of being swallowed as fast as secreted, accumulates in the mouth. Here it becomes stringy from its mixture with the buccal mucus, and when the patient opens his lips it runs out in streams. After a time the orbicularis oris becomes so far paralyzed that the lips cannot be kept closed without continual exertion, and then the viscid saliva is constantly flowing out of the mouth. In one of the cases mentioned as being under my charge, there was from the first some flow of saliva from the mouth, not apparently from any difficulty of swallowing, but from the existing paralysis of the orbicularis oris allowing the mouth to be almost constantly open. The other muscles supplied by the facial nerve in the lower part of the face, singularly enough, do not become involved. The food, it is true, accumulates between the gums and the cheeks, and has to be removed with the finger, but this is not due to any paralysis of the buccinator muscles, but to the want of power in the tongue to move the alimentary bolus around the cavity of the mouth.

When the disease is thus fully developed by the paralysis of the tongue, the veil of the palate, and the lips, the patient presents a pitiable spectacle. He is unable to talk; his teeth are exposed, from the impossibility of closing his mouth; the saliva either runs in streams over the lower lip, or he goes about with a handkerchief in his hand which he uses to absorb the perpetual flow; every attempt at deglutition causes him the utmost distress, and puts him in danger of his life from strangulation. When he opens his mouth the glutinous saliva is seen hanging in viscid strings from the roof, and his tongue, which he cannot move, lies torpid like an inert mass of muscles as it is.

The facial expression is well seen in the accompanying woodcut (Fig. 32), made from a very accurate sketch of one

Fig. 32.



LEEDS SCHOOL OF MEDICINE

of my patients suffering from the disease in question, and who entered my consulting-room with his handkerchief to his mouth to absorb the streams of saliva which were flowing.

The condition of the patient becomes still more painful from the implication of the respiratory muscles. The walls of the chest become paralyzed, and he is unable not only to breathe deeply, but to cough so as to keep the bronchial tubes clear of accumulations of mucus. So feeble is the respiratory power, that with all the effort he can make he cannot blow out a candle.

And, besides the impossibility of articulation, the larynx becomes paralyzed at a later period of the disease, and phonation becomes impossible. The patient is then doomed to perpetual silence, even the power of whispering being lost.

A remarkable fact is characteristic of many cases of glosso-labio-laryngeal paralysis, and that is the tendency of the morbid action to extend so as to implicate other nerve-cells lower down in the spinal cord. But the cells thus affected are not motor, but trophic, and as a consequence the resulting condition is not paralysis but muscular atrophy. In none of my cases was there muscular atrophy in any part of the body, but in one, to be presently referred to more at length, there was incipient paralysis of the right arm. The case was, therefore, similar to the one reported by MM. Duchenne and Joffroy in their memoir already cited.

Gradually, as the disease advances, the physical powers of the patient yield. He becomes unable to walk, not from paralysis, but from general debility, due to insufficient nutrition and imperfect respiration. His appetite remains good, but he is afraid to take any more food than is barely sufficient to sustain life, for experience has taught him that suffering and danger are attendant on every attempt at deglutition. At last he ceases to make the effort, and is fed with liquid food through a stomach-tube. The saliva during sleep runs down his throat, and fits of suffocation are the result. Too weak to walk, he remains in bed, his head turned to one side so as to allow free egress for the saliva, and he dies either from asphyxia, from the cessation of the action of the heart through the continued extension of the lesion to the cells supplying the pneumogastric nerve, or from some intercurrent affection.

Generally the mind remains clear to the last, but in a very interesting instance of the disease sent to me by my friend Dr. Fleming, of Pittsburg, this was not the case,

manifest dementia making its appearance toward the close. The emotions are, however, almost invariably easily excited.

The first case of this disease coming under my observation was one sent to me, over three years ago, by my friend Dr. Edward Bradley, of this city. The patient was a watchmaker, and very intelligent. Though unable to speak a word, I obtained a good deal of information from him relative to his disease by asking him questions, the answers to which he wrote. The accompanying *fac simile* of one of his written communications to me (Fig. 33) will, I doubt not, prove of interest. It was made partially in answer to questions, and partially at his own suggestion. The date (March, 1847) was given in answer to my question when the disease appeared, and the year mentioned is a mistake for 1867. As he states, there was a little trouble with his right arm. This was of the nature of paralysis, there being no muscular atrophy anywhere. The patient died about six months after I saw him, the disease lasting a little over a year.

The last case—the eighth—is a patient in the New York State Hospital for Diseases of the Nervous System. In him the affection began in the orbicularis oris, and has gradually involved, though as yet slightly, the tongue and muscles of deglutition. The left side was first involved, and then, a few weeks afterward, the paralysis extended to the right. There is nystagmus of both eyes. The mind is perfectly clear. He formed the subject of a recent clinical lecture on glosso-labio-laryngeal paralysis, which I delivered during the session of 1870–'71, at the Bellevue Hospital Medical College. The case is further remarkable as occurring in an exceptionably young person, the patient being but thirty-two years of age. Duchenne[1] states that he has never observed it in persons under forty. I subjoin a rep-

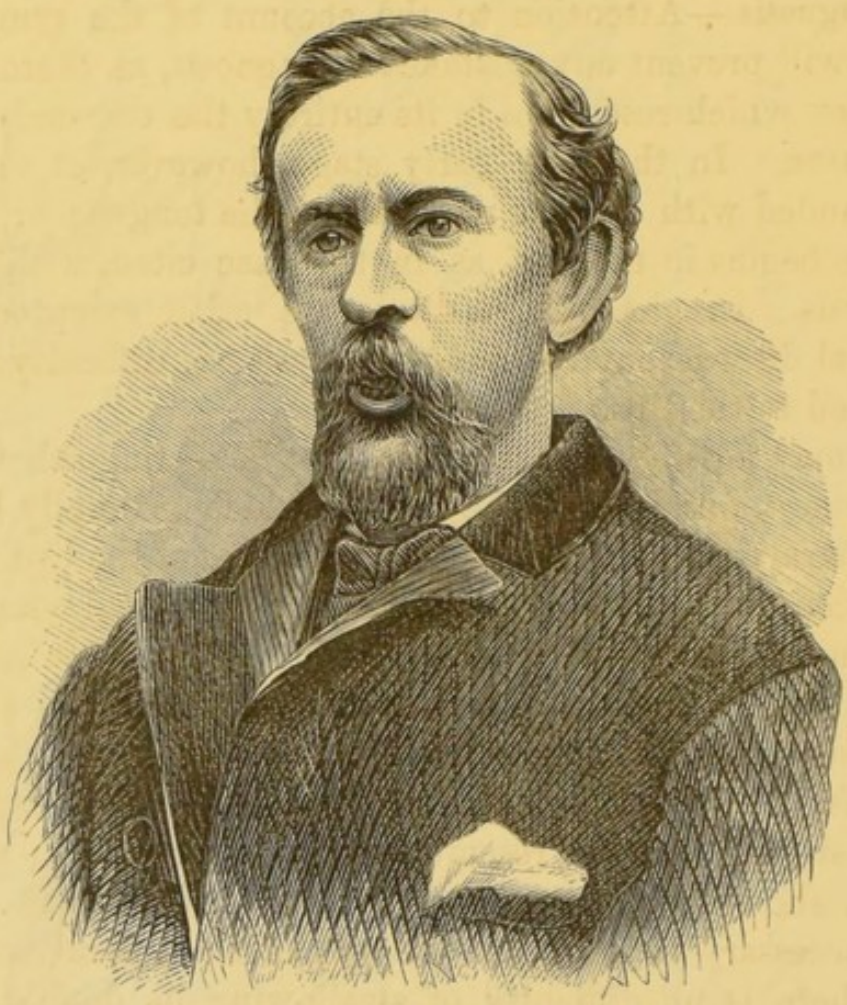
[1] De l'Électrisation localisée, Paris, 1861, p. 648.

Fig. 33.

March 1847 – Gradual tongue first
Cant swallow well.. No trouble with eyes
or ears, Lately a little in my right arm
breathing all right. taste unaffected,
smell well. Food lodges between the
gums & cheek – Sensibility re Effect –
Disturbed sleep from Suffocations – food
goes down wrong way – When I am
strongly affected the right side of
my mouth draws up –

resentation of this patient (Fig. 34), taken from a photograph. The paralysis of the orbicularis oris is evident, although it is partly concealed by the mustache. At the time it was taken the patient could swallow, but was conscious of a difficulty in beginning the act of deglutition.

Fig. 34.



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Causes.—The etiology of glosso-labio-laryngeal paralysis is very obscure. Duchenne attributes one of his cases to mental anxiety; two cases appeared to be due to syphilis and rheumatism. In no other instance could he assign a cause.

Of my own cases, one was apparently due to business troubles resulting from petroleum speculations; and in one, that of a gentleman from Kansas City, Missouri, who came here to consult Prof. Sayre and myself, excessive application

to business appeared to be the cause. In one other case, that of a gentleman of this city, the disease was evidently associated with syphilis. In none of the others could I assign any cause. All of my patients were between the ages of forty and sixty, except the one whose portrait has just been given.

Diagnosis.—Attention to the account of the symptoms given will prevent any mistake in diagnosis, as there is no affection which resembles in its entirety the one under consideration. In the very early stage, however, it may be confounded with simple paralysis of the tongue; or, if the disease begins in the lips, as in the case cited, with facial paralysis. In glossoplegia there are other symptoms of cerebral disorder, and in facial paralysis the difficulty is not confined to the lips.

It may possibly, in some cases, not be distinguished from the general paralysis of the insane, which generally begins with paralysis of the tongue and weakness of the lips. The facts that this disease is manifested also by mental symptoms, and that the paralysis gradually involves the other muscles of the body, will suffice for making an exact diagnosis. In facial diplegia the expression of countenance is very much like that of a patient suffering from glosso-labio-laryngeal paralysis, but here the resemblance ends, and careful examination shows even here many points of difference. It is only necessary to state that the tongue is not paralyzed, and that there is no difficulty of swallowing in double facial paralysis.

In progressive muscular atrophy, attacking the tongue, the veil of the palate, and the lips, a mistake might also be made. But, as Duchenne remarks, progressive muscular atrophy rarely begins in that way, and, when it does, other muscles of the body, especially the thenar and hypothenar eminences, will soon become involved. Charcot[1] has, however, recently reported a case in which progressive muscular

[1] Archive de Phys., No. 2, 1870, p. 247.

atrophy was clearly combined with glosso-labio-laryngeal paralysis, and in which, on post-mortem examination, though the volume of the tongue was not diminished, the muscular fibre had undergone degradation. In such a case, of course, a complete diagnosis could only be made after death. In ordinary progressive muscular atrophy, the fact previously insisted upon, that the atrophy comes on before the paralysis, is applicable here.

From diphtheritic paralysis, attacking the muscles of the pharynx, glosso-labio-laryngeal paralysis is readily distinguished by inquiries relative to the history of the case, and by the fact that the tongue is not involved in the first-named disorder.

Prognosis.—There is no instance on record of a cure. All my patients affected with the disease are dead but one, and with him the affection is slowly advancing. Ameliorations may certainly be produced, but probably no cure. The average duration of the disease is about two years.

Morbid Anatomy.—Previous to the very recent researches which have given us a clear insight into the morbid anatomy of glosso-labio-laryngeal paralysis, the lesions, detected by several observers, were atrophy of the roots of the hypoglossal, facial, spinal accessory, and pneumogastric nerves. But late investigations have shown that the lesions of the nerve-roots are secondary to others more central in their situation.

It has already been shown, under the head of progressive muscular atrophy, that the morbid process in that disease consists of atrophy and disappearance of nerve-cells forming the nuclei of origin of certain nerves. Very minute examinations, made in the cases of persons dying of the disease under notice, show very clearly that it also consists of atrophy and disappearance of nerve-cells.

Thus, in the case just cited from Charcot, the post-mortem examination revealed the fact of atrophy of the nerve-roots supplying the paralyzed muscles, and microscopical investi-

gation showed that the nerve-cells in relation with the filaments of origin of the hypoglossal, the spinal accessory, the pneumogastric, and the facial were altered, and had many of them disappeared. In the case which Duchenne has made the basis of some original views on the subject of atrophy of nerve-cells, and which has already been cited several times, it was found that the cells constituting the nuclei of origin of the hypoglossal, the facial, the spinal accessory, and the pneumogastric, had become atrophied, and had disappeared to a remarkable extent.

It may, therefore, be considered as satisfactorily determined, that the essential lesion in glosso-labio-laryngeal paralysis is found in the medulla oblongata and upper part of the spinal cord, and that it consists of atrophy and disappearance of certain nerve-cells constituting the nuclei of origin of the hypoglossal, the facial, the spinal accessory, and the pneumogastric nerves.

Pathology.—What is the nature of the nerve-cells which have been diseased? In progressive muscular atrophy, we saw that there was ample reason for supposing them to be cells that especially presided over the function of nutrition—trophic cells; for that disease is one in which the lesion, so far as the muscles are concerned, consists of deficient nutrition. Glosso-labio-laryngeal paralysis is not, however, a disease in which the muscles are defectively nourished, but one the essential feature of which is paralysis. It is reasonable, therefore, to suppose, with Duchenne, that the nerve-cells which have become diseased are motor cells. As regards the relation of the symptoms observed to the known distribution and functions of the nerves concerned, there is no difficulty. The affection of the hypoglossal causes the paralysis of the tongue, and the consequent impossibility of articulation, and of moving the food in the mouth. The implication of the facial accounts for the paralysis of the lips and the muscles of the veil of the palate, and the resultant impossibility of sounding certain letters, and of swallowing.

The extension to the spinal accessory explains the paralysis of the larynx, the loss of phonation, and the feebleness of respiration; and death, when it takes place as it sometimes does from the sudden stoppage of the heart's action, is due to the implication of the pneumogastric, to which cause other paralyses of the muscles of animal life are to be ascribed.

Treatment.—From what was said relative to the prognosis, it will have been perceived that there is not much to expect from treatment. I have, however, occasionally produced good results which have, for a time, at least, rendered the condition of the patient more tolerable. Thus, the first patient who came under my care was much relieved by faradization of the paralyzed muscles. He improved very much in his ability to swallow, and in power over his tongue and lips. These ameliorations were not permanent. In the case of the gentleman from Pittsburg, as well as in all the other cases but one, similar treatment, together with the use of the primary galvanic current and phosphorus, was without the least effect. In this latter case, which is now under treatment, some benefit has apparently resulted. The course of the disease is certainly less rapid than before treatment was begun, but it is nevertheless, in my opinion, slowly advancing.

CHAPTER III.

ATROPHY AND DISAPPEARANCE OF MOTOR AND TROPHIC NERVE-CELLS.

ORGANIC INFANTILE PARALYSIS.

Under the name of organic infantile paralysis, I have described at length[1] a form of paralysis occurring in young children, previously described by Rilliet and Barthez as the *Paralysie essentielle de l'enfance*, and by Duchenne[2] as *Paralysie atrophique graisseuse de l'enfance.* Previous to the writings of these authors, the affection in question was not distinctly recognized as a separate disease, but was confounded with a much less serious disorder, probably belonging to the class already considered under the head of anæmia of the anterior columns of the spinal cord. The tendency in the present affection to muscular atrophy, and the permanent character of the paralysis, are phenomena which sufficiently distinguish it from the temporary paralysis referred to.

Symptoms.—Organic infantile paralysis is generally preceded by febrile excitement and pain in the back. This pain marks the seat of the disease in the spinal cord to which the paralysis of the muscles is due. These symptoms last for a few days, or they may be so slight as in very young children not to attract attention.

[1] Journal of Psychological Medicine, No. 1, 1867, p. 49. Also, my translation of Meyer's Electricity in its Relations to Practical Medicine. New York, 1870, p. 228, note.

[2] Traité, clinique et pratique, des maladies de l'enfance. Paris, 1853, t. ii., p. 335.

Sometimes the paralysis is readily observed from the first, both by the extent and intensity; at others, it is not perceived till some one notices that the child does not use one hand or kick with one leg. The age of the patient of course exercises considerable influence on the question of ascertaining the existence of the paralysis at an early period.

The temperature of the affected limbs is always much lower than that of the corresponding sound ones. The difference is sometimes as much as eight or ten degrees, though generally it is not more than five. If, under appropriate treatment, amendment takes place, the first indication is shown by the return of the temperature toward the natural standard. It thus becomes important to have some means by which a very slight increase of heat may be noticed. A delicate thermometer graduated to tenths of a degree will generally suffice, but much more exact indications may be obtained by Becquerel's disks, which are placed in communication with a galvanometer. These disks consist of a very thin plate of copper, about the size of a half dime, soldered to a thin rod of bismuth. The latter is contained in a small tube of hard rubber furnished with a handle. The disks are two in number: one is placed on the sound limb, the other on the corresponding part of the paralyzed limb. Both are in connection, by delicate silk-covered wire, with the poles of a galvanometer. If the temperature of both limbs be the same, the needle of the galvanometer remains quiet. If either be warmer than the other, the needle is deflected to the north or the south, according as one or the other limb has the higher temperature. By this apparatus, very much less than the hundredth of a degree of temperature can be determined with absolute certainty.

Sensibility is not materially, if at all, lessened, though the reflex excitability is diminished, and often entirely abolished, from the very first.

But the most obvious and important change is that which takes place in the paralyzed muscles, and which consists of

atrophy and degeneration of the proper tissue. The process is very similar to that which constitutes the essential feature of progressive muscular atrophy, although it is far more rapid in its progress.

With this atrophy, the electric contractility of the muscles disappears, although it has begun to be lost at an earlier period, and hence the strongest induced currents fail to cause the slightest contraction, and in some cases even powerful primary currents are equally inefficacious. Owing to the disturbance in the normal equilibrium of the muscles consequent on the paralysis, distortions of various kinds are produced, and hence we have the most important causes of club-feet.

Causes.—Little is known of the etiology of organic infantile paralysis. In two cases under my observation, occurring in brothers, it was apparently induced by the nurse allowing the infants to lie on the damp ground for an hour or more; in several other cases, it came on while the children were suffering from teething, and in others it has followed diseases of various kinds, such as whooping-cough, measles, scarlet fever, etc. In the great majority of the cases that have come under my observation, no cause could be reasonably assigned.

Diagnosis.—The symptoms of organic infantile paralysis are so characteristic, that there is no danger of its being mistaken for any other affection.

Prognosis.—The prognosis depends very much upon the fact as to whether the disease has advanced so far as to have resulted in the abolition of the electric contractility of the affected muscles. If this is lost to the induced current, the cure will be difficult, and the treatment protracted; if the primary current is also powerless, a cure is impossible. I believe I was the first to use the primary current in the treatment of infantile paralysis, and to insist on its great value as a curative agent, and as an element in the prog-

nosis.[1] If the muscles can be made to contract with either the induced or primary currents, the cure is merely a matter of time and patience.

Morbid Anatomy.—The morbid anatomy of organic infantile paralysis is to be studied in the spinal cord, the nerves, and the muscles. As regards the latter, there has been a tolerable accord among observers, but there has been no approach to uniformity relative to the state of the spinal cord and nerves. The general opinion has, perhaps, been that there is no appreciable alteration. In one case in which I had the opportunity of making a post-mortem examination, and of inspecting the condition of the cord, I found a cicatrix partially filled with a very small clot. The paralysis in this instance was situated in the left lower extremity, and had begun four years previously. The lesion existed in the lower part of the dorsal region in the left anterior column. No microscopical examination was made; but, since the remarkable series of observations on the minute anatomy of the spinal cord, made by Dr. Lockhart Clarke, a new impetus has been given to studies of its morbid anatomy, and hence the results obtained in researches into the morbid anatomy of progressive muscular atrophy, glosso-labio-laryngeal paralysis, locomotor ataxia, and other spinal affections. In organic infantile paralysis some recent observations have thrown light on its nature. MM. Charcot and Joffroy have had the opportunity of making a minute investigation in a case of infantile paralysis existing in a patient in the Salpêtrière—a woman who died at the age of forty.

The disease began when she was seven years old. At first all the limbs were paralyzed, but by the end of a year the upper extremities had in a measure regained their power. The lower extremities remained nearly altogether without the power of motion.

On post-mortem examination, the spinal cord was found to be affected from the cervical to the lumbar enlargement.

[1] New York Medical Journal, December, 1865.

The alterations were chiefly in the gray matter, and especially in the anterior cornua. These were atrophied and distorted, and the cells had disappeared to a very great extent. The posterior cornua, though involved, were affected to a much less extent.

The anterior roots of the nerves coming from the diseased portions of the cord were atrophied.

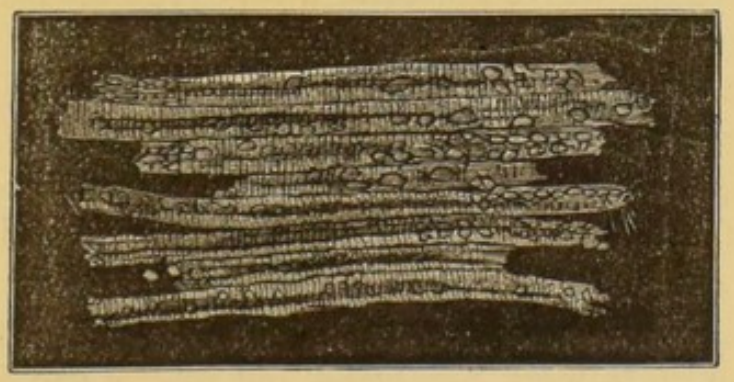
The paralyzed muscles had undergone fatty transformation, and the fibrillæ had, to a great extent, disappeared.

From these examinations we perceive that the lesion in organic infantile paralysis consists essentially of atrophy and disappearance of nerve-cells.

My own observations in the direction of the morbid anatomy of the disease under notice have been mainly limited to the condition of the muscles during life.

The nature of the morbid process is well shown in the accompanying woodcuts, made from my own drawings of the microscopical appearances of portions of diseased muscles removed by Duchenne's trocar. Fig. 35 represents a portion of the upper part of the tibialis anticus muscle of a

Fig. 35.

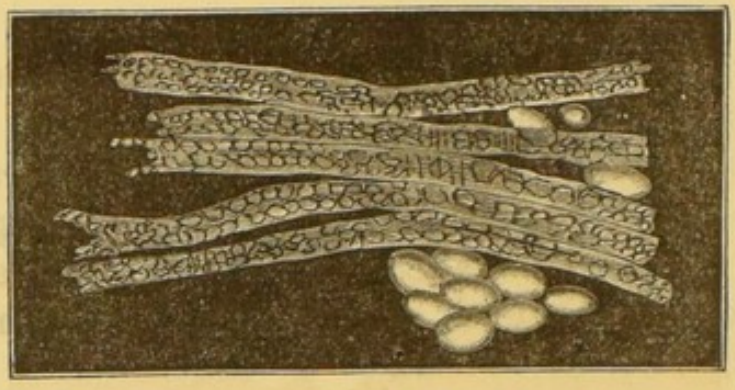


boy who had suffered from organic infantile paralysis for over two years. Oil-globules are seen along the course of the fibrillæ. These latter are irregular and torn, and the transverse striæ are becoming dim.

In Fig. 36 a still more advanced stage is shown. This cut represents a portion of the same muscle taken from the

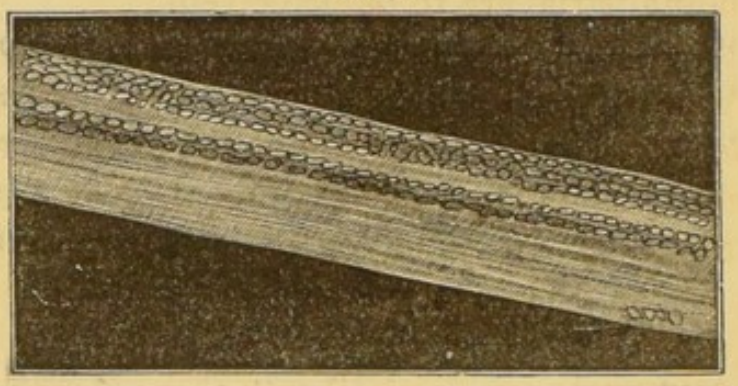
lower part. The transverse striæ have nearly disappeared, oil-globules are seen in large numbers, and fat-corpuscles are also abundant.

Fig. 36.



In Fig. 37 the progress of the disease is well shown.

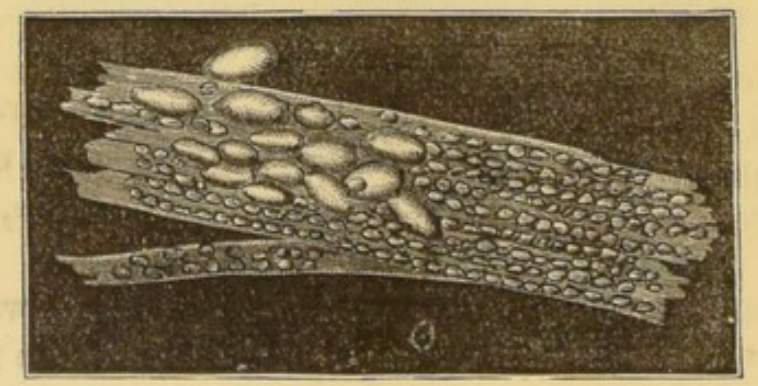
Fig. 37.



The upper margin of the specimen is a mass of fat-globules, and throughout the whole the transverse striæ are absent.

In Fig. 38 is shown a portion taken from the same muscle one month after the preceding specimens were removed.

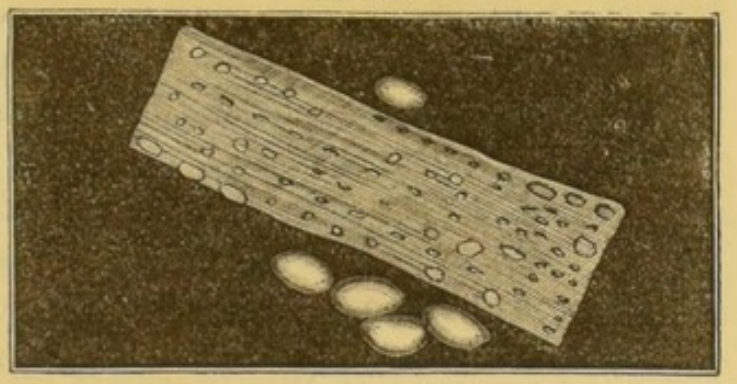
Fig. 38.



The transverse striæ are entirely gone, and the muscle is a mass of oil-globules and fat-vesicles.

Fig. 39 represents a piece of the same muscle six weeks later. It is now nothing more than a mass of connective

Fig. 39.



tissue, the fat being almost entirely absorbed; no transverse or longitudinal striæ are to be perceived.

But there is not, as Duchenne affirms, this degeneration in every case of organic infantile paralysis. In two cases, which had lasted over four years, I found the structure of the muscle unchanged. There were atrophy, loss of electric contractility, and reduction of temperature, but every specimen of the affected muscles that I examined showed no change from the normal character. In every other respect the symptoms were similar to those observed in ordinary cases of the disease. Improvement was very slow, but finally every muscle except the rectus femoris in one, and the tibialis anticus in the other, recovered, and the children were enabled to walk. The affection in both cases was confined to the left lower extremity.

I am hence led to the conclusion that fatty degeneration of muscles, though the ordinary result of organic infantile paralysis, is not an invariable consequence.[1]

Pathology.—It must be borne in mind that the disease under consideration is a paralysis primarily, and not an

[1] Journal of Psychological Medicine, No. 1, 1867, p. 57. Since the observations then published, other observers have arrived at the same conclusion.

atrophy. There can be no doubt, therefore, that Duchenne is wrong in considering it, as he does, an acute form of progressive muscular atrophy. In the first stage, the motor cells only are probably involved; in the latter, when the atrophy begins, the trophic cells are the starting-point. The disease is therefore, in my opinion, one which consists of atrophy, and disappearance of both motor and trophic cells, and hence we have, as its manifestations, paralysis and atrophy, each independent of the other.

Moreover, there is no tendency in organic infantile paralysis to extend beyond the limits of the muscles first paralyzed; on the contrary, there is a strong disposition toward repair of the spinal lesion, and the restoration of motility before the supervention of atrophy. These two features serve to increase the distinction between organic infantile paralysis and progressive muscular atrophy.

Treatment.—The treatment of the disease consists in the use of general and local means. Of these, the latter are of much the greater importance, especially after the first or febrile stage has subsided, and the disease is chiefly manifested in the paralyzed or atrophic condition of the muscles. During the acute stage, there is nothing of so much efficacy as rest in bed. I know of no medicines which are capable of producing any specific action on the spinal cord at this time, and, even after the spinal affection has become more chronic, the means mainly to be relied upon are those which are applicable to the local trouble of the muscles.

Strychnia is useful because it is capable of acting as a general stimulant to the nervous system, and is, moreover, a tonic to the muscles. I generally prescribe it in union with iron and phosphoric acid, according to the following formula: ℞. Strychniæ sul. gr. j., ferri pyrophosph. ʒ ss., acidi phosphorici ℥ ss., syrupus zingiberis ℥ iiiss. M. ft. mist. Dose, a teaspoonful or less, according to the age of the child.

The immediately local means of treatment are those which are calculated to promote the nutrition of the muscles,

and restore or augment their contractile power. The first end is effected by causing a greater amount of blood to flow through the diseased parts, the second is best accomplished by the persistent use of electricity, and active and passive exercise.

Under the first head are embraced heat, friction, and kneading.

Heat is best applied by means of hot water. A temperature of from 110° to 120° Fahr. may be used, and the limb should be thoroughly immersed, and allowed to remain so for half an hour; salt may be added to the water, with the view of augmenting the stimulant effect.

Frictions with a dry towel, a flesh-brush, or the hand, are also exceedingly useful; they should be practised several times in the course of the day, to the extent of reddening the skin.

Kneading the muscles affords a means of exercising them, and of increasing the amount of blood in the vessels. They should be pinched firmly between the fingers of both hands to the extent of producing some little pain; every paralyzed muscle should be gone over in this way daily.

Under the second head, electricity comes first. If the induced current will produce contractions in the affected muscles, it should be employed; but if, as often happens, it should fail to do so, the primary current must be brought into service. In the communication[1] already cited, I called attention to this valuable agent in the treatment of organic infantile paralysis. If a contraction can be induced by it, recovery is merely a matter of time.

During the period from December, 1865, to December, 1870, I have treated ninety-eight cases of organic infantile paralysis. Of these, the disease was so far advanced in eleven, as to render it very evident, after thorough examination, that success was out of the question. In the remaining eighty-seven, no contractions could be caused in the affected mus-

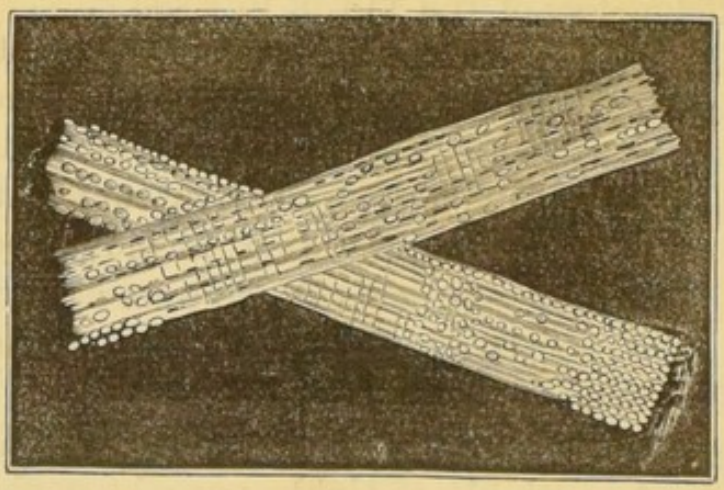
[1] New York Medical Journal, December, 1865.

cles by the strongest induced currents in thirty-nine; while in all of these the primary current produced decided contractions. Of the eighty-seven cases, fourteen were entirely cured; twenty-eight were greatly improved; thirty slightly improved, and the remainder—fifteen—discontinued treatment before sufficient time had elapsed to ascertain the effect.

At the best, however, the treatment must be of long duration, and even when the muscles are entirely restored they must be reëducated to the performance of their functions. Few parents, comparatively, have the patience to wait and to devote the necessary time to doing their part of the work; unless there is a reasonable assurance in regard to these points, it is better not to undertake the case. It is not, except in recent cases, a matter of days, or of weeks, but of months, and sometimes of years.

But, even when fatty degeneration is going on, the disease may be arrested by the proper use of the direct current. Fig. 40 shows the appearance of a portion of muscle as exam-

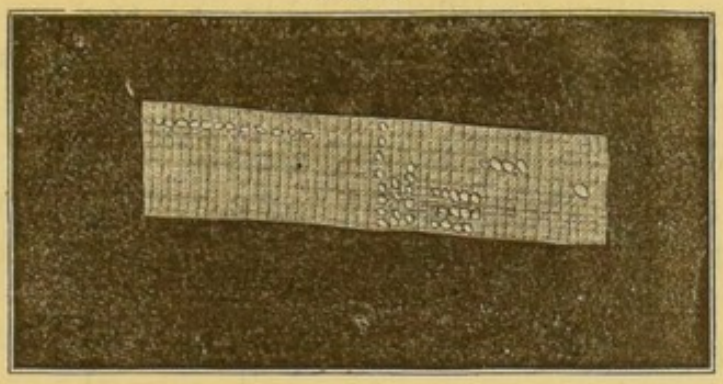
Fig. 40.



ined by the microscope, October 21, 1866. This specimen was removed from the belly of the gastrocnemius muscle before any treatment whatever had been employed, and after the disease had existed, with gradually-advancing atrophy, for about four and a half months.

Fig. 41 represents a piece of the same muscle from the same part, on December 3d, six weeks after the treatment was begun. In the first, oil-globules are seen to have displaced the muscular tissue to a great extent; the trans-

FIG. 41.



verse striæ have disappeared entirely from some parts, and are faintly seen even where they are present. In the second, the quantity of fat is perceived to be very much lessened, and the striæ are much more numerous and distinct. This case, which was one of paralysis of the left leg and foot, entirely recovered.

After the power of the will is to some extent restored over the muscles, the induced current may be used with more advantage than the direct.

Along with the electricity, passive motions of the joints should be made, and the child should be encouraged to direct the will to the affected muscles as often and as powerfully as possible.

HYPERTROPHY OF MUSCULAR CONNECTIVE TISSUE.

The first to call attention to this affection was Duchenne,[1] who described it under the name of *paraplégie hypertrophique de l'enfance de cause cerebrale.* He has since designated it *paralysie pseudo-hypertrophique, ou myo-sclérosique.*[2] Jaccoud[3] calls it *sclérose musculaire progressive*

[1] De l'Électrisation localisée, etc., Paris, 1861, p. 353.
[2] Archives Générales, etc., 1868. [3] Op. cit., p. 365.

(progressive muscular sclerosis). Dr. Foster[1] terms it paralysis with apparent muscular hypertrophy.

Regarding the affection as consisting essentially in disease of the motor and trophic nerve-cells, and as being manifested by hypertrophy of the muscular connective tissue at the expense of the muscular fibres, I have provisionally placed it in the present chapter. My personal acquaintance with the disease is limited to one case, and I am of the opinion that it is exceedingly rare in this country—one other case only having been reported, by Drs. William Ingalls and S. G. Webber, of Boston,[2] the latter of whom, in connection with the history of the case, has written a very excellent memoir on the disease.

Symptoms.—The first symptom observed is weakness in the lower extremities, which causes an inability to stand steadily, or to walk without stumbling or falling. The legs are separated widely in standing or walking, and thus a peculiar character is given to the gait, which somewhat resembles that of a duck.

Very soon an enlargement of the calf of one of the legs is perceived, the other before long is affected, and then the muscles of the thighs and gluteal region become involved.

As the child stands or walks, a remarkable incurvation of the spine in the lumbo-sacral region is perceived, so that, if, as Duchenne remarks, a plumb-line be allowed to fall from the most posterior spinous process of a vertebra, it passes far behind the sacrum. He considers this phenomenon to be due to weakness of the erector muscles of the spine. The muscles of the trunk may become involved, as may also those of the upper extremities—the deltoids being the first affected in the majority of cases, and the progress being much slower than in the lower extremities.

With the advance of the hypertrophy the paralysis

[1] Lancet, May 8, 1869.

[2] A case of Progressive Muscular Sclerosis, with a Paper on the same. Boston Medical and Surgical Journal, November 17, 1870.

becomes more strongly marked, and finally the child is confined to the recumbent posture. Distortions from disturbance of muscular equilibrium may take place, and the attempt at flexion or extension becomes painful.

Electric contractility is always lessened to the induced current, but, according to some observers, is increased to the primary current. In the case under my care, both currents failed to cause the normal amount of contraction in the affected muscles. The cutaneous sensibility is not affected.

The course of the disease is slow, its average duration being about five or six years. As it advances, there are symptoms indicating loss of mental power, and cerebral disturbance is sometimes also indicated by ocular troubles and pain in the head.

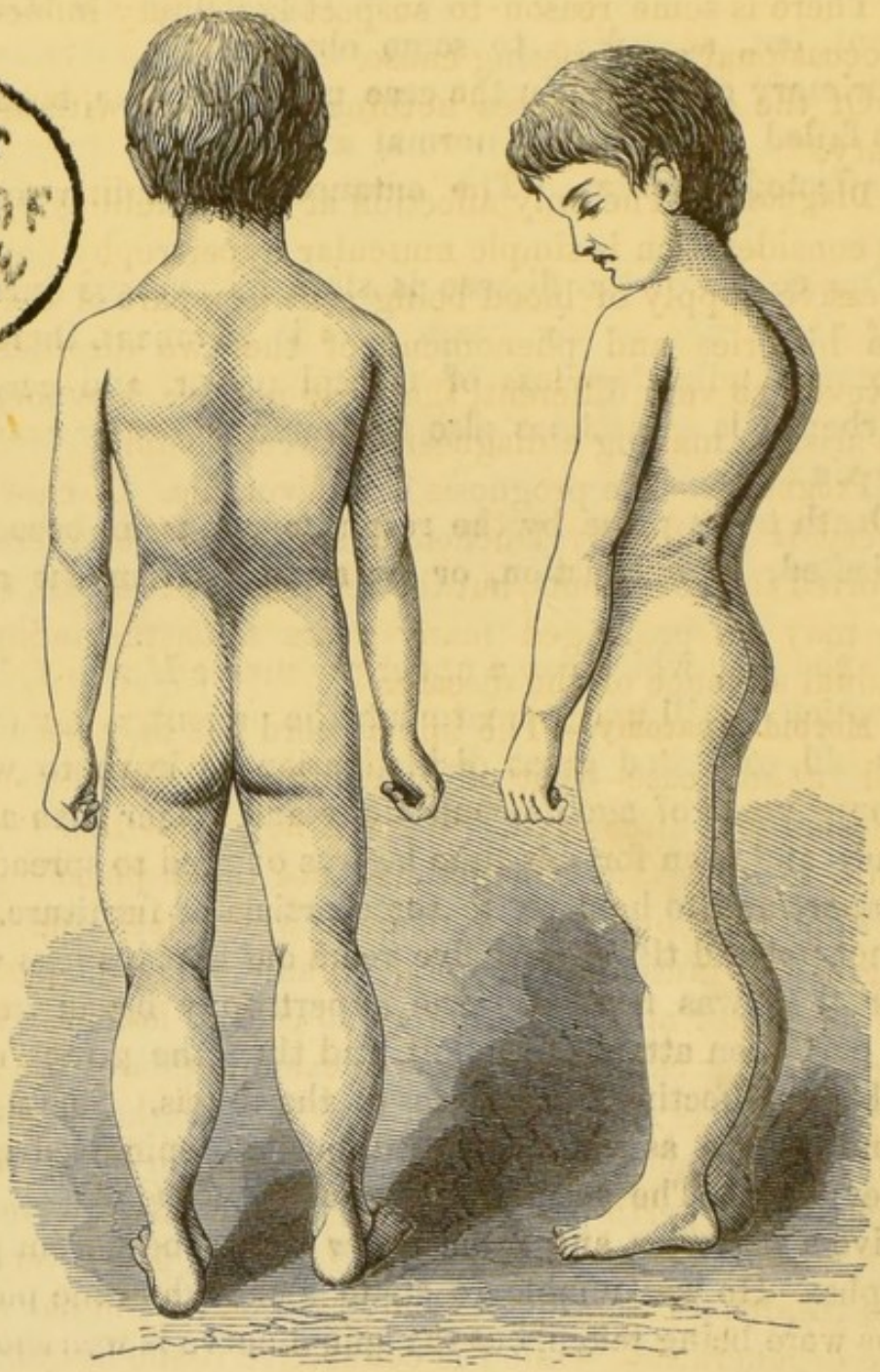
Death takes place by the respiratory muscles becoming implicated, by exhaustion, or by some intercurrent affection.

In the case which came under my notice March 7, 1871, and which is still under treatment, the patient, a boy seven years old, exhibited great disinclination to learn to walk. At three years of age he could not stand longer than a few seconds, and even for this time he was obliged to spread the legs apart and to hold on to some article of furniture. It was not noticed till he was five years old that his legs were larger than was natural. The hypertrophy began in the right calf, then attacked the left, and then the glutei muscles, before affecting the muscles of the thighs. The upper extremities are as yet unaffected, but the spinal curve is very evident. The accompanying woodcuts (Figs. 42 and 43) give a posterior and profile view of this boy, from photographs. He was unable to stand alone while the photographs were being taken, but the spinal curve is well shown, and the positions are those he spontaneously assumed.

Causes.—The disease is almost entirely confined to infancy, and boys are more liable than girls. From a table

containing analysis of forty-one cases given by Dr. Webber in his paper already cited, it appears that in one case the patient was twenty-six when the disease began, in one a few

FIG. 42. FIG. 43.



LEEDS SCHOOL OF MEDICINE

years under forty, and in one about twenty-eight. It is possible that those cases occurring in persons of adult age were instances of the simple hypertrophy of an extremity,

such as the case reported by Mr. Maunder,[1] and similar ones by other authors.

All the cases collected by Dr. Webber, except five, occurred in males.

There is some reason to suspect hereditary influence as an occasional predisposing cause.

Of the exciting causes nothing is known with any certainty.

Diagnosis.—The only affection at all resembling that under consideration is simple muscular hypertrophy due to an excessive supply of blood being sent to a part of the body. The histories and phenomena of the two disorders are, however, so very different, that I do not see how any error can arise in making a diagnosis between them.

Prognosis.—The prognosis is unfavorable. A case of recovery is related by Duchenne, and other observers have reported improvements, but the tendency is to death, though life may be prolonged many years notwithstanding the gradual advance of the disease.

Morbid Anatomy.—The spinal cord has only been examined in one case—that of Eulenburg, by Cohnheim—and no lesion was discovered. We are not, from this negative result, to infer that changes had not taken place. About the same time observers were everywhere declaring that in progressive muscular atrophy, organic infantile paralysis, and locomotor ataxia, there were no central lesions. I have no doubt that careful microscopic examination of the spinal cord, after the manner of Dr. Lockhart Clarke, will result in the detection of atrophy and degeneration of nerve-cells in cases of hypertrophy of muscular connective tissue.

Examination of the muscles, however, gives very uniform results. The proper tissue is atrophied and has undergone degeneration, while the connective tissue has not only taken its place, but has undergone extensive proliferation.

In the case under my care I have made repeated exami-

[1] Medical Times and Gazette, March 27, 1869

nations, removing the muscle with the trocar of Duchenne. The accompanying woodcut (Fig. 44) represents the histo-

FIG. 44.

logical character of a portion of muscle taken from the left gastrocnemius. The transverse striæ are seen to have entirely disappeared, the fibrillæ are in a state of disintegration, and the connective tissue is present in large amount.

Pathology.—The main point of difference between hypertrophy of the muscular connective tissue and organic infantile paralysis is that, in the former there is muscular atrophy with connective-tissue hypertrophy, while the latter is atrophy without this complication. It is highly probable that the lesion in the former is analogous to that in the latter disease. They are certainly not identical, but the phenomena of hypertrophy of the muscular connective tissue indicate the lesion to be situated in the motor and trophic cells.

Treatment.—The induced current has been useful in Duchenne's hands, and, as stated, he has reported one cure. He combines with it shampooing or kneading, and hydrotherapeutics. The primary current to the spinal cord and sympathetic nerve has been used by Benedikt[1] in five cases,

[1] Electrotherapie, Wien, 1868, p. 186.

and in three of them the induced current was applied to the hypertrophied muscles. Three cases were improved.

In my case the primary current is being used to the spinal cord and sympathetic nerve, and the induced to the affected muscles. These latter are also well kneaded every day. At the end of three weeks I am unable to detect any improvement.

CHAPTER IV.

FUNCTIONAL DERANGEMENTS OF MOTOR NERVE-CELLS.

PARALYSIS AGITANS.

Under the term paralysis agitans, several affections have been included which are very different in character. I have already considered two of them—multiple cerebral sclerosis and cerebro-spinal sclerosis; a third I propose to treat of under the name of paralysis agitans. Though the objections to its use are many, it possesses the advantages of being already known, and of expressing two of the main features of the disease to which it is applied.

The affection which Parkinson[1] described, and to which he applied the name "shaking palsy," has since been very carefully studied by many writers, and the fact has been clearly made out that it is not a single disease.

Charcot, in numerous memoirs and lectures to which reference has already been made, has very definitely shown that the affection which he designates *sclérose en plaques disséminées*—considered in this treatise under the name of cerebro-spinal sclerosis—must be regarded as a distinct morbid condition; and in the first section of this work I have made the same claim for multiple cerebral sclerosis. The term paralysis agitans I apply to a very different affection from either, but one which I am confident will be recognized as presenting well-defined characteristics. Ordenstein[2] has included it with multiple cerebral sclerosis,

[1] Essay on the Shaking Palsy, London, 1817.

[2] Sur la Paralysie agitante et la Sclérose en plaques généralisée, Paris, 1868.

and denies it any fixed seat; but Jaccoud[1] locates it in the pons Varolii, without, however, in my opinion, having any good reason for so doing. Of all writers Dr. Handfield Jones[2] appears to have the clearest ideas of the affection now under notice. Thus he says:

"It appears to me a question whether two distinct affections are not often comprehended under this name. For on the one hand it appears pretty certain that there is one form which is met with in old persons, is quite incurable, and is associated with, if not dependent on, organic wasting changes in the nervous centres; while another form occurs in younger persons, is more curable, and therefore is presumably not dependent on organic change." It is this latter disease which I propose to consider at present. The other embraces cases of multiple cerebral sclerosis and cerebro-spinal sclerosis.

Symptoms.—The primary manifestation is tremor, and this, like the same symptom in the severer forms of disease already considered, in which it forms an essential feature, may begin in a very restricted or more extensive region of the body. It is present whether voluntary movements are performed or not with the affected limbs, but is increased by mental excitement of any kind, by physical exertion, or by any cause capable of depressing the powers of the system.

It is not generally the case that the tremor shows any tendency to advance much beyond its original limits, however small or extensive these may be. When it does exhibit such a disposition, contiguous muscles are first attacked, and then the corresponding ones on the opposite side of the body.

From the very first there is muscular weakness, not to any very great extent, but still sufficiently evident to careful examinations with the dynamometer. As the tremor increases in violence or extent, the paralysis becomes more obvious.

[1] Op. cit., p. 424.

[2] Studies on Functional Nervous Disorders, London, 1870, p. 382.

Sensibility is rarely affected, there is no bending of the body forward, no festination, and no head-symptoms. The tremor always ceases during sleep, except in very extreme and long-continued cases, and there may be intermissions of longer or shorter duration while the patient is awake.

Causes.—Paralysis agitans may result from emotional disturbance, from continuous or severe muscular exertion, from some exhausting disease, such as dysentery, typhoid or typhus fever, or rheumatism, or from blows, falls, or other injuries. In many cases the cause cannot be ascertained.

Of twenty-one cases of which I have records, six were apparently due to mental causes, four to excessive physical exertion, four to diseases of various kinds, two to injuries, and in five no cause could be discovered.

Two cases of mercurial trembling, the symptoms of which affection are very similar to those of non-toxic paralysis agitans, are not included among the foregoing.

Diagnosis.—From multiple cerebral sclerosis, paralysis agitans is distinguished by the facts that there are no head-symptoms, no festination, and no derangements of sensibility. It is more apt to occur in persons under the age of fifty, and may be met with in quite young persons. The reverse of both these circumstances is true of multiple cerebral sclerosis.

From cerebro-spinal sclerosis, it is diagnosticated mainly by the absence of any head-symptoms, by the fact that the tremor usually comes on before the paralysis, and is independent of voluntary movements.

The character of the muscular action, and the history of the case, will prevent its being confounded with chorea.

Prognosis.—Paralysis agitans rarely terminates fatally, and when it does it is because the tremor has become so general that death results from exhaustion. It, however, often happens that all measures fail to relieve the agitation. Of the twenty-one cases occurring in my own experience, six were cured, four partially so, and in the rest no permanent effect was produced by any means I employed.

Morbid Anatomy and Pathology.—Nothing is known of the morbid anatomy. In a few cases, patients have died either from the disease or from some intercurrent affection, and post-mortem examinations have been made with negative results. Petræus, quoted by Dr. Handfield Jones, relates two severe cases, one of which proved fatal. At the autopsy nothing was found but fatty degeneration of the heart and pneumonic consolidation of the right lung. He remarks on the tremor not being constant in many cases, ceasing for some days and then returning with fresh force, or changing its seat from one part to another.

In my opinion, the disease under consideration is due to an irregular and diminished evolution of nerve-force from the motor nerve-cells in relation with the nerves supplying the muscles in which the agitation exists. The pathology of tremor, not the result of structural lesions, is a subject which is beginning to be studied, but which is not yet clearly understood. We know that, when we have strongly exerted an arm, for instance, the muscles are tremulous for some time afterward, and that the agitation is rendered very evident when we attempt to write or do any other act requiring delicate muscular adaptation. A period of rest must take place before steadiness is regained. Now, in such a case the agitation is not probably due to any cause inherent in the muscle, but is the result of exhaustion in the nerve-cells and the disengagement of insufficient force in an intermittent manner. I suppose paralysis agitans to be due to some such action in the motor nerve-cells in the gray matter of the spinal cord.

Treatment.—I have used electricity, both of the galvanic and faradaic kinds, in all the cases of paralysis agitans that have been under my charge, and in conjunction have employed many internal medicines, such as arsenic, iron, manganese, zinc, copper, phosphorus, strychnia, and sedatives of various kinds, including opium, bromide of potassium, conium, stramonium, Indian hemp, and many others. I am very decidedly of the opinion that the best treatment

consists in the use of the constant primary current to the spinal cord, sympathetic nerve, and the affected muscles, while at the same time strychnia and phosphorus, according to the formula given on page 58, are administered internally. By these means two of my six successful cases were entirely cured within two months. One of these was sent to me by my friend Dr. F. N. Otis. The affection was confined to the right arm, and was probably due to inordinate gymnastic exercise; the other was a gentleman from St. Louis, in whom the disease was also confined to the right arm, and had apparently resulted from writing excessively. Both had lasted several months.

The four other cases were, two of them, consequent on other diseases, and two were without known cause. Three were women; the tremor in two was in both arms, and in two, in one leg in each. The duration of the treatment was from three to seven months. A full and nutritious diet, and the avoidance of all mental excitement or strong physical exertion, are important features in the treatment.

In the mercurial form of the disease the iodide of potassium in large doses—twenty grains three times a day—must be administered in conjunction with the other remedies.

WRITER'S SPASM.

The disorder, which I think is best named writer's spasm, has been variously designated, according to the prominence which each author on the subject has given to some one symptom. Thus it has been called scrivener's palsy, writer's cramp, chorea scriptorum, mogigraphia, writer's dyskinesia, etc.

By the majority, if not by all writers who have given it a fixed seat, it is regarded as being a disease of the peripheric nerves. A consideration of its mode of origin and symptoms must, I think, tend to show that it is an affection of the motor nerve-cells, similar in several respects to paralysis agitans.

Although in my nomenclature of the disease I have termed it writer's spasm, I have done so simply for the sake of convenience, and because the affection is more frequently met with among writers than other professional people. It does, however, occur among those who are required by any employment to perform delicate, complex, or uniform actions with the fingers for many hours each day. It is thus not uncommon among violinists, pianists, watch-makers, jewellers, seamstresses, etc. The account I shall give of the affection, though specially applicable to writers, will be relevant to those of other professions who may suffer from this singular disease.

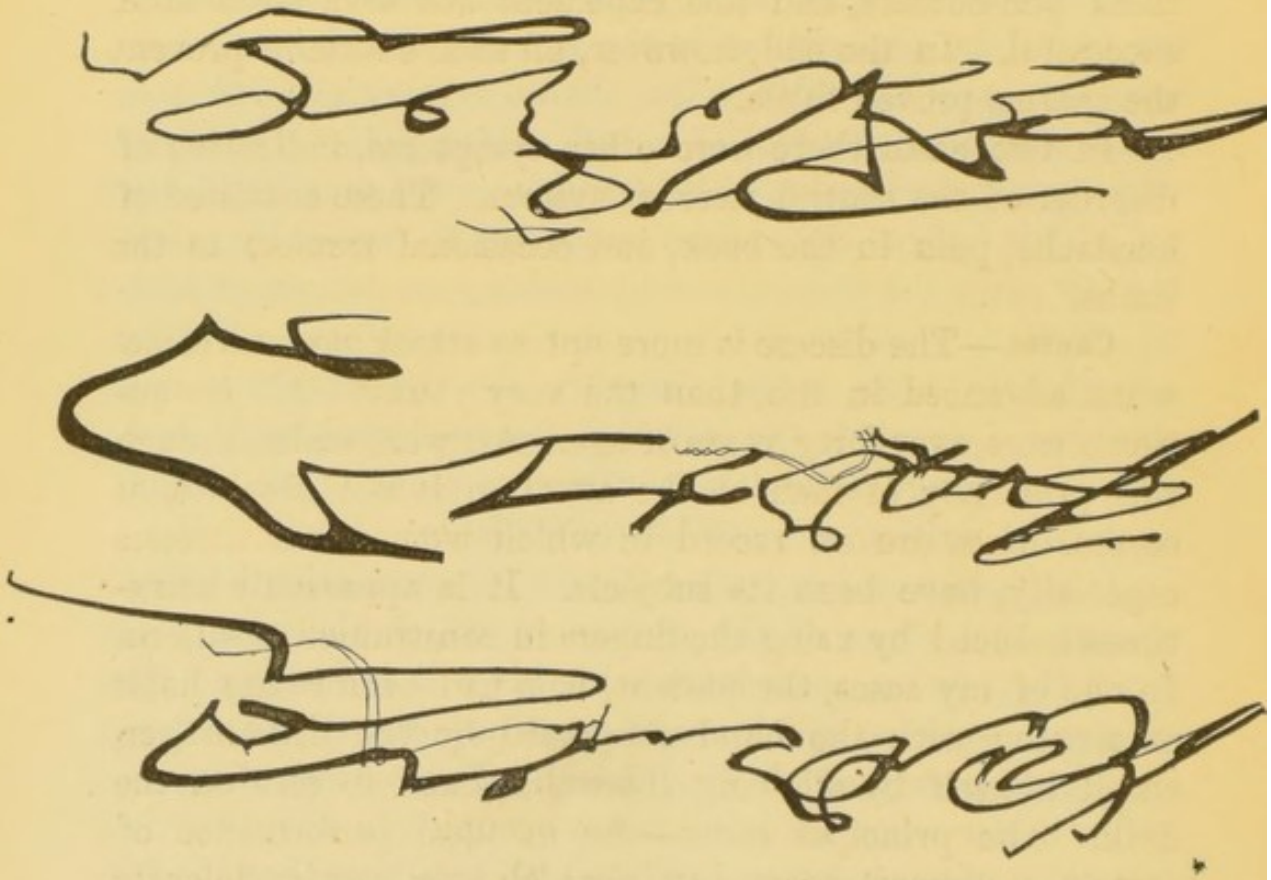
Symptoms.—The first symptom usually observed is a feeling of fatigue experienced in the muscles of the hand, forearm, arm, and shoulder. The thumb is especially affected, and is also often the seat of a dull, aching pain. Pains not very severe nor fixed are also common in the muscles higher up. This fatigue the patient endeavors to correct by grasping the pen more firmly, and by making an increased mental effort to regulate the muscular contractions. But he only thereby adds to the difficulty, for the weariness and pain are increased, the muscles become weakened, and, moreover, irregular and incoördinate actions ensue, which render the writing more or less unreadable.

If he perseveres day after day in his occupation he soon reaches that stage of the disease in which the ability to direct the pen in accordance with his will is lost, and the automatic actions, which are of great importance in writing, are likewise very much diminished. For a time, then, he writes better when his mind is not occupied in directing the formation of every letter, but in which he allows the muscles as it were to take care of themselves. Constantly, however, he feels the necessity of mental action, and this action invariably increases the difficulty, until at last, the moment the attempt is made to write, the pen, actuated by the muscles of the fingers, executes such disorderly movements as to bear

no analogy with the words attempted to be written. A distinct paroxysm is thus induced, which lasts as long as the patient persists in the attempt to write. When he discontinues, the spasm ceases, and he can perform any other act with the fingers without there being the slightest convulsive movements. In some cases there is pain in the fingers, the muscles between the metacarpal bones, and in those of the forearm. The spasm is much worse if the patient be excited or particularly anxious to do his best.

In the accompanying woodcut (Fig. 45) are represented three attempts of a patient to write the name "James Ely."

Fig. 45.



At first some resemblance to the letter J is made, but in the second trial it is less distinct, and in the third is lost altogether.

I have witnessed eleven cases of this disease during the past six years—all of them in writers except one, an interest-

ing case occurring in an engraver. This patient was seized with the spasm in the fingers of the right hand whenever he grasped his burin. He could write for hours perfectly well.

In three of the other cases the individuals had acquired the power to write with the left hand, but the spasm soon appeared in it on any attempt at writing.

All of my patients had resorted to various expedients to obviate the spasms, under the idea that they were produced by metallic pens carrying off the electricity from the arm; several had, for a time, made use of quills, or hard rubber pens, and for a time relief had been obtained, but the paroxysms soon became as bad as ever. Others had used very thick pen-holders, and this expedient was also, for a time, successful. In the end, however, all such efforts to prevent the spasms proved futile.

In two cases there were other symptoms, indicative of disorder of the central nervous system. These consisted of headache, pain in the back, and occasional tremors in the limbs.

Causes.—The disease is more apt to attack persons somewhat advanced in life, than the very young. All my patients were over forty years of age. All were males, though this proclivity of men to the affection is not absolute, as several cases are on record in which women, seamstresses especially, have been its subjects. It is apparently sometimes induced by using the fingers in constrained positions. In one of my cases, the patient, who had been in the habit of writing with the hand supported by the little finger, cured himself by allowing the whole hand to rest on the desk. The principal cause—the habitual performance of certain restricted movements—has already been sufficiently considered.

Diagnosis.—Attention paid to the characteristic symptoms of writer's spasm will prevent its being mistaken for lead-paralysis, progressive muscular atrophy, or any other disease.

Prognosis.—In the early stage, writer's spasm admits of cure. When it has existed a long time, and when the patient cannot rest, a cure is almost impossible.

Of the eleven cases that have been under my care, seven were incurable, having lasted several years, and resisting all means of cure, even long rest. Of the other four, one was in process of cure by his own expedient of changing the attitude of the hand in writing, when he came under my observation. The remaining three cases were successfully treated by means to be presently described.

Morbid Anatomy and Pathology.—As regards the morbid anatomy, there are no data, and the lesion is probably not one which can be detected by our present means of observation. The affection is, however, doubtless due to disorder in the normal action of the motor-cells, and this disorder is the result of over-exertion of a particular set of muscles in a particular way. Examples of cerebral exhaustion by the predominance of one idea, or a series of ideas for a long time, are often witnessed. Writer's spasm is, I conceive, the result of a similar action on spinal motor cells.

Treatment.—The most indispensable means of cure is rest, and, unless this can be secured, it is useless for the physician to undertake the treatment. In some cases it has succeeded without any assistance. The abstinence from writing should be absolute during at least six months.

The constant galvanic current has proved the most effectual agent in my hands; I apply it to the sympathetic nerve, the spinal cord in its upper part, and to all the muscles and nerves of the upper extremity. A half an hour three times a week, with a current of considerable intensity (forty cells), will be sufficient. Faradization, in my experience, is more productive of harm than benefit.

With the galvanism I have administered the combination of phosphide of zinc, and extract of nux-vomica, recommended on page 58 of this treatise.

When a cure cannot be effected, well-devised prothetic

apparatus will enable the patient to write. Division of tendons or muscles is not admissible.

LEAD PARALYSIS.

The frequency with which lead paralysis is met with, and the fact that the loss of power in certain muscles is generally unaccompanied by symptoms referable to the system at large, must be my excuse for including it within the limits of the present treatise.

Symptoms.—Before the occurrence of paralysis, the patient has probably suffered from attacks of lead-colic, though this is not invariably the case. The immediately precursory symptoms connected with the loss of power are slight numbness and tremors in the muscles of the upper extremities. Occasionally, the muscles of the trunk and lower extremities become involved in the trembling.

Ere long the patient observes that he has difficulty in extending the fingers or wrist, and that there is a general loss of strength in one or both hands. These symptoms go on increasing in severity, and eventually he loses the power to raise the hand or fingers. In extreme cases, the ability to extend the forearm, or to raise the arm from the side, is lost through the paralysis of the triceps and deltoid. Occasionally, the extensors of the lower extremity are involved in the paralysis.

The predominance of the loss of power in the extensors has led to the idea that they alone are affected. The dropping of the hand, the flexion of the forearm on the arm, the hanging of the arm against the side of the body, and, when the lower extremity is affected, the inability to raise the toes so as to avoid striking them against the ground in walking, all give countenance to this supposition. But careful observation shows that the difference is merely one of degree, and that there is a very considerable loss of power in the flexor muscles. Indeed, of many cases of the disease that I have observed in hospital and private practice, I have never

seen one in which the flexors were not implicated with the extensors.

Owing to the disuse of the muscles, atrophy takes place, and this is frequently exceedingly well marked, and, from the disturbance of the normal equilibrium between the several groups of muscles, contractions and distortions ensue. The circulation in the affected limbs becomes languid and weak, and painful swellings result in consequence.

It is generally supposed that the right arm is more generally affected than the left; such, however, does not appear to be the case. Thus, Tanquerel des Planches,[1] of seventy-nine cases in which the upper extremities were the seat of the paralysis, found both affected in fifty-one, the left twenty-three times, and the right twenty-four. Of thirty-two cases of lead-paralysis occurring in my own practice, the upper extremities were affected in all; in twenty-seven both limbs were the seat; and, of the remaining five, three were in the left, and two in the right. The left upper extremity was therefore affected thirty times, and the right twenty-nine.

In some cases, the muscles of respiration become very seriously paralyzed through the influence of lead, and death then soon takes place. In two of my cases there was aphonia, and in several the voice was materially weakened.

The electric sensibility and contractility are always greatly reduced in all cases of lead paralysis. In the majority of cases, no faradaic current, which it is safe to employ, will produce contractions, and strong primary currents are necessary. The cutaneous sensibility is rarely impaired.

The saturnine cachexia is almost always present, and the blue line on the gums can readily be distinguished.

Causes.—The fact that paralysis follows the introduction of lead into the system admits of no doubt. This introduction may take place through the stomach, the air-passages, or the skin. The two latter are the more common channels for contamination.

[1] Traité des Maladies de Plomb. Paris, 1839, t. ii., p. 39.

It is, of course, more frequently encountered among those who work in lead, such as lead founders and smelters, the makers of white and red lead, painters, plumbers, printers, etc.; although it may occur among those who are only temporarily or accidentally exposed to the toxic influence. Thus, it may be caused by drinking water which has passed through lead pipes, or been kept in lead vessels, by using tobacco which has been wrapped in lead foil, two cases of which have happened in my experience,[1] and which is so common a cause that, in France, Belgium, and Prussia, strong laws have been passed against packing tobacco in lead; by the use of hair-dyes containing lead, of which I have seen three cases; the use of powders and enamels for the face, two cases of which I have observed, one in consultation with my friend Prof. Lewis A. Sayre; and by several other less common causes. The majority of cases, however, occur in painters, probably for the reason that workers in white and red lead, though more exposed, are aware of their danger, and take effectual measures to prevent absorption.

Diagnosis.—The history of the case, including a knowledge of the occupation of the patient, will generally prevent any error in diagnosis. The presence of the peculiar cachexia, the existence of the blue line around the gums, and the predominance of the paralysis in the extensors, especially those of the wrist, will tend still further to render the diagnosis accurate.

Prognosis.—The prospect of recovery depends altogether on the ability to produce contractions in the paralyzed muscles by electricity. If the induced current will effect them, the cure will be rapid, if the interrupted primary current is required, a longer time must elapse before success is attained; but, if the muscles will not react to either the induced or primary currents, a favorable result is not to be expected.

[1] See my translation of Meyer's Electricity in its Relations to Practical Medicine, New York, 1870, p. 181, for reference to other cases.

The extent of the atrophy is also an important element in the prognosis.

Morbid Anatomy and Pathology.—So far as the central nervous system is concerned, no spinal lesions have been found unless in those cases complicated with cerebral symptoms, and when inflammation and softening have existed. The difficulty is one which probably affects the motor nerve-cells of the spinal cord, and this in a way to be undiscoverable—as much so as the effects of opium, alcohol, hydrocyanic acid, strychnia, and other substances—by our present means of investigation.

The muscles have been examined by Andral,[1] Gendrin,[2] and Tanquerel des Planches,[3] and analogous results obtained. The fibres have been found to be pale and yellowish, to be friable, atrophied, and desiccated. I have repeatedly removed small portions with Duchenne's trocar, and have always found the transverse striæ disappearing, and fatty degeneration making its appearance.

The hypothesis, that the affection is, primarily, one of the muscles, is not supported by facts. Such a thing as muscular paralysis independent of nervous derangement somewhere is unknown in the whole range of pathology. And those cases of apparent loss of muscular irritability, resulting from certain poisons, adduced by Longet, Bernard, Mitchell, myself, and others, were simply instances in which the loss of nervous irritability took place from the periphery to the centre. The present state of our neurological knowledge is altogether against the idea of muscular irritability independent of the nerves. When a muscle is no longer capable of contracting, it is because the nerves are dead.

Facts, too, are against the notion that the lead acts by contact with the muscles, and the circumstance of the pa-

[1] Clinique Médicale, t. ii., p. 227.

[2] Maladies de l'encéphale, par Abercrombie, traduction, séconde édition, p. 576.

[3] Op. cit., pp. 77, 144, 149.

ralysis occurring so generally in the hands of painters, for instance, is adduced in proof. But we have seen that the left hand is just as frequently affected as the right, while it is certainly less in contact with the lead. Moreover, those cases of paralysis in the extensors of the hand, which have resulted from hair-dyes and other cosmetics, are altogether against the hypothesis in question.

Treatment.—The first thing to do in the treatment of a case of lead-paralysis is to remove the lead from the system. This is done by the administration of the iodide of potassium, through the agency of which the mineral is converted into a soluble compound, the iodide of lead, which is excreted from the system, mainly by the kidneys. Some authors advise caution in the use of the iodide of potassium, on the ground that the resulting compound is very poisonous, and may produce highly-deleterious effects. In a great many cases of lead-paralysis and other consequences of lead-poisoning in which I have given the iodide, I have never seen the least untoward result, and I always use it in large doses from the beginning. In many cases the lead can be readily detected in the urine. If there is great debility, or if the cachexia be marked, iron, quinine, and strychnia, may be employed with advantage.

But with all these measures the paralysis remains, and would continue indefinitely, without the use of measures directed specially against it: chief among these is electricity. The induced current, if it will cause the muscles to contract, is to be preferred. Each paralyzed muscle must be acted on for two or three minutes every alternate day, so that for both upper extremities the duration of a *séance* would vary from a half to three quarters of an hour. In ordinary cases two months will suffice to effect a cure.

But it often happens that the electric contractility of the paralyzed muscles is so completely abolished that the induced current is without effect. In such cases the primary interrupted current must be used, and continued till, as will

eventually be the case, the induced current causes contractions. I have never seen a case in which the primary current would not produce contractions. One of the worst examples of the affection in question I ever saw is the patient who formed the subject of a recent clinical lecture to the class at the Bellevue Hospital Medical College.[1] His improvement under the circumstances has been rapid, and he is now (March 31st) able to earn his living again. Induced currents of great power failed to produce contractions, and but for the use of the primary current he would have been incurable.

If the primary current fails to act on the muscles, success is out of the question.

In addition to electricity, frictions, kneading the muscles, and passive exercise, are useful. Contractions may be overcome by suitable prothetic apparatus. In a case under the care of Prof. Sayre, and which I had the opportunity of seeing, the patient, a young lady, was able to play the piano —though paralyzed in both hands—by means of an admirable appliance devised by Dr. E. D. Hudson, of this city.

[1] Journal of Psychological Medicine, January, 1871, p. 43.

SECTION V.

DISEASES OF PERIPHERAL NERVES.

I do not propose to include under this head all the diseases to which the peripheral nerves are liable, but to take one or more as types of others which are different merely from their situation. Thus, any nerve of the body may be paralyzed from injury or disease, or from some contiguous affection capable of interfering with the due performance of its functions. It would be useless to give such paralyses separate consideration, as their general features and the treatment proper can be sufficiently pointed out under the head of a typical representative.

Besides, many affections, which are often regarded as located in the peripheral nervous system, are really central in situation. Among them are various cases of paralysis, spasm, hyperæsthesia, and anæsthesia, which have already been considered as symptoms of centric diseases.

I shall divide the affections of the peripheral nerves into four groups: paralysis, spasm, anæsthesia, and hyperæsthesia.

CHAPTER I.

PERIPHERAL PARALYSIS.

FACIAL PARALYSIS.

Paralysis of the facial nerve has already been considered as a symptom of several central lesions, but it may exist as an affection of altogether peripheral origin. As such, it is often known as Bell's paralysis, on account of its real nature having been first clearly pointed out by Sir Charles Bell. The nerve in question, the facial or portio-dura of the seventh pair, was, at one time, regarded as one of sensation, and, in accordance with this view, was often divided for neuralgia. The experiments of Bell and Magendie established the fact of its being entirely a nerve of motion.

Symptoms.—The facial nerve is distributed to nearly every muscle of the face. Its paralysis therefore causes such decided change of expression as to be readily recognizable. The most marked phenomenon, and one which is of importance in the diagnosis, is the inability to close the eye of the affected side. This is due to the fact that the orbicularis palpebrarum has lost its contractile power, while the levator palpebræ superioris, not supplied by the facial, but by the third nerve, is not paralyzed, and keeps the upper lid elevated. In consequence of this condition, the eye is constantly exposed to the action of the atmosphere, and to contact with extraneous substances. The patient cannot wink, and thus the tears, not being distributed over the surface of the eyeball or carried off by the nasal duct—the tensor tarsi

also being paralyzed—run over the lower lid, and scald the cheek. From this inability to wink, dust and other small particles of matter are not removed, and hence considerable irritation is produced. Exposure to strong light or to wind adds to the inconvenience. Comparative comfort may be obtained by the patient frequently closing the eye with the finger, or by keeping the lids together with a piece of adhesive plaster.

The next most prominent group of symptoms is due to the loss of power in one lateral half of the orbicularis oris. As a consequence, the patient cannot purse up the mouth on that side, as in the act of whistling or spitting. From this loss of tonicity the saliva is not retained on the affected side of the mouth, but runs out over the lip, to the great annoyance of the patient.

The muscles of mastication, the masseter, temporal, and external and internal pterygoid, are supplied by the third branch of the fifth pair of nerves, and hence the ability to chew is not impaired. The buccinator, the function of which, in conjunction with the tongue, is to press the alimentary bolus against the jaws, and thus keep it submitted to their action, is supplied by the facial, and hence its office is not performed. The food consequently accumulates between the jaws and the cheek, and it must be continually removed by the finger.

The muscles which expand the face, as in the action of laughing or smiling, are supplied by the facial, and their paralysis destroys the normal equilibrium, and hence the face is drawn toward the sound side. This loss of antagonism is most evident when the patient opens his mouth, and particularly when he laughs or smiles, for the paralyzed muscles, the zygomatici, and the risorius, are incapable of responding to the emotion, while those on the sound side contract vigorously.

The paralysis of the occipito-frontalis and of the corrugator supercilii prevents the raising of the eyebrows, or frown-

ing, and obliterates all wrinkles from the brow. As Romberg remarks, there is no better cosmetic for elderly ladies than facial paralysis ("Für alte frauen kein wirksameres cosmeticum existirt").

Among other symptoms, it is noticed that the ala nasi is depressed, and does not expand as air is drawn in through the nostril, and that the articulation, especially of words containing labials, is very indistinct.

The expression of one side of the face is therefore destroyed; it is a complete blank, incapable of responding to any emotion, and unable to execute those motions which in the normal condition are performed by its muscles.

Such are the obvious and superficial symptoms of an ordinary attack of unilateral facial paralysis. For the full understanding of other important phenomena, a few words in relation to the anatomy and physiology of the nerve are necessary.

The facial nerve takes its origin from the posterior border of the pons Varolii and the lateral tract of the medulla oblongata. Some of its fibres of origin may be traced to the floor of the fourth ventricle, and even to the lateral columns of the spinal cord. A knowledge of its course and connections enables us to determine with a good deal of accuracy the seat of the lesion by which it is paralyzed, and thus we have an important element in making a prognosis.

From its point of apparent origin the facial passes forward and outward, resting on the crus cerebelli, and leaves the cranial cavity by entering the internal auditory meatus with the auditory nerve. It next enters the aqueductus Fallopii, and, passing through its whole length, makes its exit from the skull by the stylo-mastoid foramen; while in the aqueductus Fallopii it gives off three branches, the two superficial petrosal nerves, and the chorda tympani. The great superficial petrosal passes to Meckel's ganglion, and through this supplies the levator palati and the azygos uvulæ muscles; the small superficial petrosal—regarded by

some as a branch of the glosso-pharyngeal, though communicating with the facial—runs to the otic ganglion which supplies the tensor-palati and tensor-tympani muscles, and also, according to Bernard, presides over the secretion of the parotid gland, through the auriculo-temporal nerve; the chorda tympani goes to join the gustatory branch of the fifth, and is in part distributed with this to the tongue, but another portion of its fibres enters the submaxillary ganglion which presides over the function of the submaxillary gland.

With this brief *résumé* of the anatomical and physiological points of the facial nerve, we are prepared to study other symptoms to which I have not as yet alluded; for, in the account given, I have simply considered the phenomena of facial paralysis when the lesion is situated on the distal side of the stylo-mastoid foramen. But the nerve may be affected farther back, and, though in such a case we have the symptoms already described, there are others which vary according to the seat of the disease.

Thus, if the morbid process is in action above the origin of the chorda tympani, but below that of the petrosal nerves, the patient will experience a diminution but not a complete abolition of the sense of taste upon the corresponding side of the tongue. This fact led to the supposition that the chorda tympani was a sensitive nerve, but the experiments of Bernard and others have clearly shown that it is an efferent nerve, conveying influence from the brain, not to it. One of its actions is to increase the flow of submaxillary saliva. In addition, it supplies the lingualis muscle, and probably erects the papillæ of the tongue, and modifies the circulation of this organ. When, therefore, a lesion of the facial exists above the origin of the chorda tympani, the sense of taste on that side is lessened because the dryness of the mouth prevents the ready solution of the sapid substance. The difficulty is augmented through the non-erection of the papillæ, and perhaps, also, by the change which has ensued in the circulation. This diminution of the sense of taste

therefore shows that the lesion is seated on the central side of the origin of the chorda tympani nerve.

Again, if the lesion be situated behind the gangliform enlargement, from which the petrosal nerves arise, but anterior to the meatus internus, we have, of course, all the symptoms mentioned, and in addition those due to paralysis of the petrosal. One of them is the depression of the palatine arch on the affected side; it hangs lower than the opposite one, and its edge is nearly straight instead of curved. This condition results from paralysis of the levator-palati muscle, which, as we have seen, is supplied by the great petrosal through Meckel's ganglion. One of the two little muscles of the uvula being powerless, the other draws the uvula into a bow shape, with the concavity toward the sound side. The uvula and the velum are also pulled *en masse* toward the sound side by the action of the tensor palati, the other being paralyzed through the implication of the small petrosal nerve. The connection of the small petrosal through the otic ganglion with the parotid gland causes a diminution of the secretion of this gland when the lesion of the facial is in the situation described.

Acuteness of hearing on the paralyzed side is sometimes observed. This is accounted for by Landouzy,[1] on the ground of the paralysis of the tensor-tympani muscle, which, as we have seen, is supplied by the otic ganglion, but Brown-Séquard attributes it to hyperæsthesia of the acoustic nerve from vaso-motor spasm.

This last category of symptoms, therefore, indicates the seat of the lesion to be at or behind the gangliform enlargement.

When the lesion is within the cranium, we have all the symptoms mentioned, but they are complicated with others indicative of derangements of other nerves, or of cerebral

[1] De l'Altération de l'ouïe dans la Paralysie faciale, Gazette Médicale, Paris, 1851.

disease. These have already been considered under other heads.

In the foregoing account of facial paralysis, the unilateral form, which is by far the most common, has alone been considered, but both nerves may be paralyzed, producing what is called double facial paralysis, or facial diplegia. The condition has been well described, among others, by Wachsmuth,[1] and by Pierreson,[2] the latter of whom has collected twenty-eight cases as the basis of his memoir. Both sides may be paralyzed simultaneously, in which instance the disease is probably central, or one may follow the other. In either case, the face presents a complete want of expression, and the symptoms previously mentioned are duplicated in full. Two excellent representations of the affection are given in the report of a case by Mr. Wright.[3] Only one case has come under my observation. It was of long standing and incurable. I lost sight of the patient before I could have his photograph taken.

Causes.—Cold is a prominent cause of facial paralysis. It is most apt to cause that form of the disease in which the lesion is external to the temporal bone. Exposure to intense cold, especially when the wind was blowing, has caused several cases in my experience. The patient has gone to bed feeling pretty well, and has awakened with one side of the face paralyzed.

Rheumatic inflammation, occurring in the course of the nerve, may also induce facial paralysis, as may likewise tumors of the parotid gland, or other cause capable of making pressure on the nerve. I have seen several cases which had resulted from sleeping with the closed hand under the face; and it may occur in new-born children, as the result of

[1] Ueber progressive Bulbar-Paralyse und die Diplegia facialis. Dorpat, 1864.

[2] De la Diplégie faciale. Archives Gén. de Médecine, Août, 1867, p. 139.

[3] Notes of a Case of Double Facial Palsy, British Medical Journal, 1869, p. 184.

pressure by the forceps. Wounds and injuries of other kinds may, of course, produce it.

Within the temporal bone, facial paralysis may result from tumors from periostitis, from caries of the petrous portion of the temporal bone, from disease of the middle ear, from hæmorrhage into the aqueductus Fallopii, and from fractures of the temporal bone.

Within the cranium it may be caused by disease of the pons Varolii, or of the medulla oblongata, by atrophy of the nerve, by tumors, and as the consequence of injury.

Diagnosis.—Facial paralysis is distinguished from glosso-labio-laryngeal paralysis, by the facts that in the latter the symptoms affect only the lower part of the face, and that they are accompanied by paralysis of the tongue and of the muscles of deglutition. From the facial paralysis of hemiplegia it is diagnosticated by the marked circumstance that, in the latter disorder, the patient can close the eye, while in the former it remains wide open. There are no other affections with which facial paralysis can be confounded, if the slightest attention be given to its symptoms.

Prognosis.—The prognosis varies according to the seat and the cause of the lesion, and the duration of the paralysis. If this latter is due to cerebral or intra-cranial difficulty, or to disease existing within the aqueductus Fallopii, the prospect of cure is remote. But, if the lesion exists outside of the skull, and is capable of removal, or if the paralysis be the result of exposure to cold, or subjection to pressure, and if the electric contractility of the muscles be not destroyed, the case, under suitable treatment, will probably terminate favorably. By electric contractility, I do not mean the ability to respond to the excitation of the induced current, for this is lost at an early period in the majority of cases, but to contract upon the application of as strong a primary current as can with safety be applied to the face.

In deep-seated lesions, if a clinical history of syphilis

can be made out, the prognosis becomes more favorable.

If the affection has lasted a long time, and if contractions of the paralyzed muscles from atrophy have taken place, the probability of recovery is very slight, even if there is some glimmering of electro-contractility.

Morbid Anatomy and Pathology.—When facial paralysis results from cold, it may be from consequent neuritis, or from inflammation excited in contiguous parts. In the latter case lymph is effused and pressure is exerted upon the nerve. Most of the other causes act by the pressure they make on the nerve, and, though, as in the case of sleeping with the fist under the face, the action may not be long continued, the consequence is very lasting. The effects of pressure upon the nerve are experienced when we sit too long in one position, so as to compress the sciatic nerve, or when persons go to sleep with one arm thrown over the back of the chair on which they are sitting. The axillary plexus is compressed, and paralysis, more or less complete, of the muscles supplied by it, is the result. Several such cases have come under my observation, and the resulting paralysis is generally most difficult to remove.

Treatment.—The indications are: to remove the cause if possible; to put the nerve under the best possible conditions for regaining its lost power; and to preserve the organic integrity and irritability of the muscles till this can take place. When there is reason to suspect the existence of a syphilitic taint, and the growth of exostoses of syphilitic character in the aqueductus Fallopii, the iodide of potassium with the bichloride of mercury should be given, according to the formula on page 322. In two cases I succeeded in effecting a cure by this treatment, conjoined with electricity, when the latter by itself had produced no improvement.

For the restoration of the nerve function, we can do little beyond securing healthy nutrition of the general system, by the use of proper hygiene and tonics. Among the

latter, strychnia is especially useful. I have never found blisters or liniments to be of the slightest service.

The third indication is to be met by passive exercise, such as can be produced by pinching and kneading the muscles, and, above all, by the persistent use of electricity. Without this latter agent facial paralysis cannot be cured.

If the induced current will cause the muscles to contract, it should be employed. One pole is placed over the nerve at its exit, from the stylo-mastoid foramen, and the muscles of the paralyzed side are separately excited by the other. A *séance* should last about fifteen minutes, and should be repeated every alternate day, or every day in bad cases.

If the induced current will not cause contractions, the primary interrupted current should be used for the purpose. Care should be taken not to employ a current of too great a degree of intensity, as serious consequences have resulted to the vision by neglect of this precaution. As a rule, fifteen Smee's cells will be sufficient; but, if the current be passed through a column of water before it reaches the face, a larger number of cells may be used with safety. Means must be taken to interrupt the current, as contractions are only produced when the circuit is closed and opened. When the primary current has been employed for a few weeks, it will generally be found that the induced will cause the muscles to contract, in which case it should be substituted.

The first muscle to recover power is usually the orbicularis palpebrarum, but several weeks, and sometimes months, are requisite to bring about a complete cure.

Other peripheral paralyses, such as those occurring in the muscles of the eye and its appendages, the muscles of the larynx, the muscles supplied by the motor branch of the fifth, the deltoid, etc., are to be treated upon the same general principles as are applicable to facial paralysis, the causes and pathology being very similar.

CHAPTER II.

PERIPHERAL SPASM.

There are two affections which may be taken as the types of peripheral spasm in general: these are spasm of the facial muscles—the mimic or histrionic spasm of Romberg, the convulsive tic of the French—and torticollis, or the spasm in the muscles of the neck supplied by the spinal accessory nerve.

FACIAL SPASM.

The spasms in the disease under notice may be either clonic or tonic, the former being by far the more common. In the clonic form, the muscles of the face, or a portion of them, generally on one side, are suddenly and violently contracted, and as suddenly relaxed. Sometimes, the angle of the mouth is drawn back again, the upper lip, and the alæ of the nose are elevated; and again, the spasm affects the orbicularis palpebrarum. In a case now under my charge, occurring in a gentleman from Rahway, New Jersey, both orbicularis muscles are affected with clonic and tonic spasms, the eyes sometimes being closed for several minutes at a time.

The spasms come on in paroxysms which are of variable duration. I have seen them last continuously for over an hour. Generally, they continue from a few seconds to one or two minutes, and are repeated at intervals of about the same time. They may generally be excited by emotional disturbance of any kind; by muscular exertion, by a current

of wind, or other cause capable of exciting reflex actions. In the case above referred to, they are always induced by walking. They can be made to cease by pressure upon the facial nerve at various points, and they are generally arrested by powerful mental occupation and by sleep.

In the tonic form of the affection the spasm persists, and causes more or less distortion of the face. It interferes with articulation, mastication, and especially with emotional expression.

The tendency is for either form to become habitual, and hence to be difficult of cure.

Causes.—Cold is a common cause, as are also wounds and injuries, and carious teeth. I have seen two cases recently, from this last-named influence.

The **Diagnosis** calls for no special consideration, and the **Prognosis** depends very much upon the duration. Generally, it is unfavorable.

There are no facts bearing on the **Morbid Anatomy**, and the **Pathology** is to be explained by the principle of reflex excitation which, in this case, probably takes place through the intermediation of the fifth pair. The analogy with chorea is very great.

Treatment.—Of thirteen cases that have been under my charge, five were cured. The means which I have found most useful are, daily hypodermic injections of a mixture in water of five drops of Fowler's solution, and the one-fiftieth of a grain of atropia, and the daily use of the constant primary current to the facial nerve and the convulsed muscles.

In several cases I have obtained good results from permanent pressure over the facial nerve. The gentleman previously referred to has had, at my suggestion, a steel spring constructed which terminates in two pads, and which he wears over the head in such a way as to compress the facial nerves at their exit from the stylo-mastoid foramen. While he wears it he has no spasms, but he is unable to endure the pressure longer than a couple of hours.

Division of the affected muscles has been practised with very moderate success.

TORTICOLLIS.

In this disease the spasms—which, as in the corresponding affection of the face, may be either clonic or tonic—occupy the sterno-cleido-mastoid, the trapezius, the rhomboids, and the levator anguli scapulæ, separately or collectively. The movements of the head in the clonic form depend upon the seat of the spasms, the action being in the direction of the tractile force of the affected muscles. Sometimes the contractions are very rapid, and again they are slow and regular; as in facial spasm, they are aggravated by emotional excitement or physical exertion. They cease during engrossing mental occupation, and during sleep. Occasionally both sides are affected.

The reverse of facial spasm, the tonic form, is much the more common, and it is to it that the term torticollis is usually applied by surgical writers. The sterno-cleido-mastoid is generally its exclusive seat. The contraction is often accompanied by pain.

Causes.—The etiology is not essentially different from that of facial spasm.

Diagnosis.—There is no difficulty about the diagnosis of the clonic variety. The tonic form is, however, liable to be confounded with a similar affection so far as appearances and consequences go, which is a veritable myositis, but which is not an affection of the nervous system. The transitory character of the latter affection and the severe pains are sufficient diagnostic marks.

Prognosis.—The prospect of recovery from the clonic form is very remote. Of seven cases that I have had under my charge, two only were cured.

Of the **Morbid Anatomy**, or of the **Pathology**, nothing is known.

Treatment.—I have made use of every remedy, in the

clonic form, which could in my opinion be of service. Iron, belladonna, arsenic, morphia, chloral, chloroform, ether, bromide of potassium, strychnia, zinc, and many other medicines, have all failed. In one case I administered morphia hypodermically in gradually-increasing doses, till at last two grains were given twice a day, but without any permanent effect. I have divided the muscles in four cases without benefit. In one of them I cut both sterno-cleido-mastoids, the left trapezius at its insertion into the occipital bone, the left levator-anguli scapulæ, and finally, with the concurrence of my friend Prof. Markoe, the left complexus. But as soon as one muscle was cut another became affected, and, after the division of the complexus, the expectation of obtaining a cure by myotomy was given up. The patient, a lady, from the South, is still affected, though she appears to be getting better gradually.

Electricity in any form has never cured a case in my hands, though I have employed it steadily, for weeks at a time, both as the primary and induced currents.

In the two successful cases, many means were tried without success. In one, that of a young man from Newark, in addition to other means, I divided the right sterno-cleido-mastoid muscle twice, and it was afterward cut by my friend Prof. Sayre. All the operations were unsuccessful, although, as in the other cases, an apparatus was worn to prevent the too rapid union of the muscle. This patient was finally cured with large doses of the bromide of potassium.

In the other case, that of a lady of this city, every means used failed, till I tried the oxide of zinc; she began with doses of two grains three times a day, which were gradually increased. When she reached fifteen grains at a dose, the spasms ceased and did not return.

For the tonic variety, myotomy is the proper remedy, and it is generally successful if a suitable apparatus be subsequently worn.

CHAPTER III.

PERIPHERAL ANÆSTHESIA.

Almost any part of the body may be deprived of sensation from causes acting on the peripheral nerves. One of the most familiar examples of this fact is the anæsthesia produced in the foot and leg by pressure on the sciatic nerve in the act of sitting too long in one position; another is the loss of sensibility produced in the hand and arm by pressure on the ulnar nerve as it passes over the elbow.

Anæsthesia originating from cerebral, spinal, and cerebro-spinal causes, has already been considered, and the present remarks will be strictly limited to the anæsthesia of peripheral origin.

ANÆSTHESIA OF CUTANEOUS NERVES.

Symptoms.—The symptoms of anæsthesia from peripheral causes do not vary materially from those which result from central lesions. They consist of the various sensations of numbness, such as tingling, "pins and needles," a feeling as if ants are crawling over the skin, water trickling over it, and, in complete cases, of absolute abolition of sensibility. The conducting power of the nerve may be impaired in so much as only to cause a retardation of the velocity of excitations, and thus an impression made on the terminal extremities of a nerve is not felt for a much longer time than would normally be the case. Peripheral anæsthesia may be accompanied with disorders of nutrition from irregularity of blood-supply. One form of the affection, of which I have seen several examples, and which probably owes its

complication to vaso-motor spasm, is characterized by unnatural whiteness and shrinking of the skin, usually in the hands. If an incision be made, little or no blood escapes. In a young lady from Savannah, who was under my charge a short time since, this condition existed to an extreme degree, but disappeared with the removal of the anæsthesia. Anæsthesia of peripheral origin in the cutaneous nerves is often accompanied by more or less loss of power.

Causes.—Peripheral cutaneous anæsthesia may be produced by a variety of causes. Among the chief are wounds and injuries of various kinds, whereby the nerve is divided or its conducting power impaired; pressure such as that caused by tumors, tight clothing, or accidental influences; rheumatism; exposure to intense cold; the action of certain drugs, such as aconite locally applied; and diseases of the nerves.

Diagnosis.—The important point in the diagnosis of peripheral anæsthesia is the discrimination between it and the anæsthesia, due to central causes. The elements of the diagnosis have been dwelt upon at some length by Romberg, and perhaps needlessly so, for there can scarcely be a case in which any difficulty in forming a correct opinion can arise except in those cases of anæsthesia in which the fifth pair is involved, and they will presently be more especially considered. As regards the cutaneous nerves, the existence of a peripheral cause, and the non-existence of evidences of cerebral or spinal derangement, will be sufficient indications of the nature of the affection. It could scarcely happen that anæsthesia, the result of central lesions, could exist without other marked symptoms being present, not connected with cases of peripheral origin.

Prognosis.—This depends very much upon the cause, and the ability to remove it. In cases of simple division of a nerve, union may be effected after a time, and the functions restored, but if any considerable portion of the nerve has been destroyed, the case is hopeless. Even when the cause is removed, as may be accomplished for instance in

cases due to pressure, a long period often elapses before complete restoration takes place.

The **Morbid Anatomy** and **Pathology** call for no special remarks after what has already been said.

Treatment.—The most important therapeutic measure consists in the removal of the cause. Unless this can be effected, it is useless to attempt other treatment. If this can be accomplished, electricity is the most efficient agent to be employed toward restoring the irritability to the nerves. Sometimes the primary current is to be preferred, at others the induced. In the latter case the wire brush should be used as one of the electrodes, and the anæsthetic parts be stroked with it at each *séance*.

ANÆSTHESIA OF THE FIFTH PAIR.

Symptoms.—These vary according to the seat of the lesion. If the ophthalmic branch alone be implicated, the anæsthesia is situated in the forehead, the upper eyelid, the conjunctiva, and the lining membrane of the nostril. Irritating substances, therefore, coming in contact with the eye or the pituitary membrane, are not felt.

If the difficulty is limited to the superior maxillary branch, the skin of the upper part of the face and the teeth of the upper jaw are insensible. When the inferior maxillary branch is affected, the temporal region, the skin covering the upper and lower jaw, the under lip, the chin, the lining membrane of the mouth, the anterior third of the tongue, and the teeth of the lower jaw, lose their sensibility; mastication becomes difficult, and the saliva flows from the mouth. In either of these cases the seat of the lesion must be anterior to the Gasserian ganglion. When all the branches of the fifth are involved, and, as a consequence, anæsthesia exists throughout the whole of one side of the face, it is very certain that the ganglion is affected, or that the main trunk of the nerve is itself the seat of the disease. Anæsthesia of the fifth nerve due to lesion of the Gasserian ganglion,

or of the main trunk, is very generally accompanied by disorders of nutrition and derangement of the senses of sight, smell, and taste. Fungoid growths on the gums and defective circulation in the face are common in such cases; but ulceration of the cornea and congestion of the conjunctiva do not occur unless the lesion is situated in the Gasserian ganglion, or anterior to it in the ophthalmic branch.

The **Causes** of peripheral anæsthesia of the fifth pair are analogous to those which produce the corresponding affection in the cutaneous nerves; but the **Diagnosis** requires a few special remarks, and these may be stated in the form of Romberg's propositions:

"*a.* The more the anæsthesia is confined to single filaments of the trigeminus, the more peripheral the seat of the cause will be found to be.

"*b.* If the loss of sensation affects a portion of the facial surface, together with the corresponding facial cavity, the disease may be assumed to involve the sensory fibres of the fifth pair before they separate to be distributed to their respective destinations; in other words, a main division must be affected before or after its passage through the cranium.

"*c.* When the entire sensory tract of the fifth nerve has lost its power, and there are at the same time derangements of the nutritive functions in the affected parts, the Gasserian ganglion, or the nerve in its immediate vicinity, is the seat of the disease.

"*d.* If the anæsthesia of the fifth nerve is complicated with disturbed functions of adjoining cerebral nerves it may be assumed that the cause is seated at the base of the brain."

The **Prognosis**, the **Morbid Anatomy**, the **Pathology**, and the **Treatment**, call for no remarks additional to those made when peripheral cutaneous anæsthesia was under consideration, except that, as regards the treatment, if the primary current is employed, care should be taken that the tension be not too high, a point to which reference has already frequently been made.

CHAPTER IV.

PERIPHERAL HYPERÆSTHESIA (NEURALGIA).

Under this head I propose to consider the principal painful affections embraced under the term neuralgia. No designation in medical nomenclature has been more abused than this. Any pains, the origin of which cannot readily be ascertained, and many which are well known to depend upon central lesions, are called neuralgic. I propose, in the present remarks, to include under it those affections only which, so far as can be ascertained, are not due to disease either of the brain or spinal cord, but the seat of which is in the nerves themselves. Following the classification of Valleix, I shall consider—

a. Neuralgia of the fifth pair.
b. Cervico-occipital neuralgia.
c. Cervico-brachial neuralgia.
d. Dorso-intercostal neuralgia.
e. Lumbo-abdominal neuralgia.
f. Crural neuralgia.
g. Sciatic neuralgia.

NEURALGIA OF THE FIFTH PAIR OF NERVES.

Symptoms.—Either division of the fifth pair of nerves may be the seat of the disease, or all may be simultaneously affected.

1. *Ophthalmic Division.*—This branch of the fifth is distributed to the side of the nose, the eyelids, the lachrymal gland, the globe of the eye, the conjunctiva, the forehead,

and the scalp. The long root of the ciliary ganglion communicates with the nerve, and anastomoses take place with the superior maxillary branch.

Valleix has shown that there are particular spots in which neuralgic pains are always more severe than in others, and that these are the points where the nerve either passes through a foramen in a bone, or penetrates a fascia. In the ophthalmic nerve several of these points are to be found. The most prominent is in the nerve as it passes out of the supra-orbital foramen to ramify on the forehead and scalp; another is seated in the upper eyelid; another in the long nasal branch as it passes to the skin through the line of union of the nasal bone with the cartilage; another is located in the eyeball, and another at the inner angle of the orbit. Besides these which are peculiar to the ophthalmic branch, there is another situated near the parietal eminence, and which corresponds to the inosculation of various branches.

The most common form of neuralgia affecting the ophthalmic division of the fifth nerve is hemicrania. The occurrence of the paroxysms is marked by a tendency to periodicity. The pain is exceedingly sharp and lancinating, and occupies the frontal, temporal, or parietal regions, being especially intense at the point corresponding to the supra-orbital foramen, or at that situated near the parietal eminence. It frequently happens that this latter spot is the only part affected. The paroxysm usually comes on in the morning, and rarely lasts longer than twenty-four hours; frequently it disappears at nightfall. The pain is greatly aggravated by mental or physical exertion, by loud noises or bright lights. It is often complicated with nausea and vomiting, in which case it constitutes what is known as sick-headache. In other cases the pain is mainly confined to the eyeball and the accessory parts. There is then lachrymation, from the fact that the lachrymal gland is supplied from the ophthalmic division, and there may be visual

troubles from the relation which the nerve bears to the ciliary ganglion.

This form may also be distinctly periodical in its occurrence, and it rarely lasts longer at one time than twenty-four hours.

2. *Superior Maxillary Division.*—The distribution of this branch is to the teeth of the upper jaw, the lower eyelid, the side of the nose, the upper lip, to the lining membrane of the nose and mouth, and to the temple and cheek. It inosculates freely with the ophthalmic division, and is in intimate relations with the spheno-palatine ganglion.

The painful points of Valleix for this nerve are, in the infra-orbital nerve as it emerges from the infra-orbital foramen to be distributed to the lower eyelid, the side of the nose, and the upper lip; over the most prominent part of the malar bone, where the nerve is very superficial; an uncertain point on the gums of the upper jaw; a similar point on the upper lip, and another on the palate. Neuralgia of this division occurs in paroxysms, and may, like that of the ophthalmic, be periodical in its attacks.

3. *Inferior Maxillary Division.*—This nerve is distributed to the cheek, the tongue, the lower jaw and teeth, and to the sub-maxillary gland. It is also in connection with the otic and sub-maxillary ganglia.

Its painful points are a spot on the auriculo-temporal branch, just in front of the ear; another on the inferior dental nerve, where it emerges from the inferior dental canal, through the mental foramen.

It is generally the case that facial neuralgia is limited to one side, but both are sometimes affected. It may also be confined to very restricted boundaries, the extreme terminal branches alone being involved.

Causes.—According to my experience, facial neuralgia is rarely met with in young persons, but is more common during adult life. It is certainly more apt to attack females than males, and is often transmitted by hereditary influence.

The most common exciting cause is, in this country, malaria, and this is especially the case with the affection in the ophthalmic division, as manifested in hemicrania and supra-orbital neuralgia. This latter is often popularly known as "brow-ague."

Among other causes are to be mentioned mental excitement, anxiety, intense intellectual exertion, exposure to cold and damp, the loss of blood, as in the case of women after child-birth, or from menorrhagia, prolonged lactation, and the changes due to the cessation of the menses.

Another very common cause is syphilis, and there is reason to think that the gouty diathesis may also excite it.

But, as Anstie[1] remarks, it is after the powers of life begin to decline that the most formidable varieties of facial neuralgia are encountered. Those forms which are attended with muscular spasm, constituting the "tic douloureux" of the French, and another still more violent which Trousseau has designated "tic épileptiform," are almost peculiar to advanced life. The pain in these affections is atrocious, and is excited by the least muscular action in the face, by a touch, however light, or even by a breath of air. They are often accompanied by an hereditary tendency to insanity, and they eventually wear away the life of the miserable sufferer.

Facial neuralgia may also result from tumors compressing the nerves, from thickening of the bones, or of the periosteum, causing narrowing of the foramina through which they pass, and from interstitial organic changes taking place in the nerve-trunks.

The **Diagnosis** requires no special remarks, and the **Prognosis** depends upon the cause, and the ability to remove it. In general terms it may be stated that the malarial and syphilitic forms are usually readily cured, while others are seldom thoroughly relieved. The intense varieties, coming on for the first time late in life, are absolutely

[1] Article Neuralgia, in Reynolds's System of Medicine, vol. ii., p. 726.

incurable, and are very seldom capable of even being mitigated.

CERVICO-OCCIPITAL NEURALGIA.

In this affection the pain is situated in the sensory branches of the first four cervical nerves, though the great occipital which arises from the second cervical is mainly the one affected. These nerves are distributed to the occipital and posterior parietal regions, as well as to the neck and lower part of the cheek. The painful points are those at which the nerves become most superficial.

The pain in cervico-occipital neuralgia, though severe, is not in general so intense as that of the facial variety. There is a tendency in the affection to extend so as to involve the inferior maxillary nerve, and, when the disease has lasted some time, a paroxysm rarely occurs without this nerve being implicated. After the acute stage of a paroxysm has passed off, there remains a dull, heavy pain, which continues several days, and which is increased by the pressure of the clothing, by mental exertion, or by moving the head.

The **Causes** are similar in general character to those of facial neuralgia, though cold is probably a still more powerful factor in the etiology.

The **Diagnosis** and **Prognosis** call for no special remarks.

CERVICO-BRACHIAL NEURALGIA.

In this form the brachial plexus, the nerves which go to form it—the five lower cervical and first dorsal—and those which arise from it, are the seat of the affection. The pain may therefore be felt in the subclavicular region, along the whole length of the upper extremity, or in the situation of the mammary gland. The exact seat varies of course with the particular nerve affected. It is often accompanied by various sensations of numbness, and interferes more or less with the movements of the limb. The principal painful points are the axillary in the arm-pit, and corresponding to

the brachial plexus, the scapular near the inferior angle of the scapula, the acromial in the angle between this process and the clavicle, the median cephalic in the bend of the elbow, the ulnar corresponding to the most superficial portion of the ulnar nerve at the back of the elbow-joint, and the radial at the point where the radial nerve becomes superficial at the lower part of the forearm.

Among the **Causes** of cervico-brachial neuralgia, excessive muscular exertion and injuries are preëminent. It is not so frequently the result of malaria as the corresponding affection of the facial nerve.

There is nothing special to be said relative to the **Diagnosis** and **Prognosis.**

DORSO-INTERCOSTAL NEURALGIA.

In this affection the dorsal and intercostal nerves are the seat of the pain. In the first case the disease is often regarded as rheumatic or muscular, and has received the popular name of lumbago; in the latter it is often known as pleurodynia. Whether in the dorsal or intercostal form, the pain does not often occur in well-marked paroxysms, but is more or less continuous in character, and is much increased by muscular exertion. In the dorsal form, the mere act of straightening the back causes great suffering, and in the intercostal respiration is exceedingly painful.

The painful points are very numerous, and in general correspond to the situations where the nerves become most superficial.

The association of intercostal neuralgia with herpes zoster of unilateral form is an interesting fact, and one which has led to the recognition of other skin-diseases as being essentially nervous affections.

The **Causes** of dorso-intercostal neuralgia are cold, exhaustion, and, in women, the depression of vital power, due to profuse menstruation or prolonged lactation. Anæ-

mia, both in males and females, is also a common cause, however produced.

The **Diagnosis** of the dorsal form is not a matter of difficulty; the intercostal has, however, often been mistaken for pleurisy. The **Prognosis** is more favorable than in the other neuralgias described.

Lumbo-abdominal and *crural neuralgias* are not very common. The latter is seldom a primary affection.

SCIATIC NEURALGIA.

This form is characterized by the occurrence of pain in the course of the sciatic nerve and its branches, mainly in those distributed to the skin. It may be restricted to the gluteal region and upper part of the thigh, or may extend to the sole of the foot or toes. The principal painful points are those which correspond to the sacral foramina, where the large and small sciatic nerves emerge from the pelvis; a series corresponding to the emergence of cutaneous branches through the fascia, a fibular point at the head of the fibula, an external malleolar, and an internal malleolar.

Sciatica generally begins as a dull, heavy ache, which gradually becomes more and more intense, and which, like all the other forms of neuralgia, is aggravated by muscular exertion. It is subject to exacerbations of violence, during which the least agitation of the body still further increases the intensity of the suffering. Sometimes the pain darts through the nerves like electric shocks, while at others it retains its original situation. It is often accompanied by muscular contractions. Anæsthesia is generally present in the parts which are or have been the seats of the pain, and can readily be detected with the æsthesiometer.

The affection generally lasts two or three months, and is liable to recur.

Causes.—The etiology of sciatica is not materially diffreent from that of other neuralgias, except so far as it is

modified by local circumstances. Among these latter, are enlargement of the prostate gland, by which pressure is exerted on the nerve, various tumors of the abdominal organs, the pressure of the fœtal head in child-birth, accumulations of fæces in the large intestine, etc. It is also occasionally induced by the pressure on the nerve which results from sitting long on a hard chair. Several cases of this kind have come under my observation.

The **Diagnosis** is not a matter of any difficulty, though I have many times seen cases mistaken for diseases of the spinal cord, and *vice versa*. The **Prognosis** depends greatly on the ability to remove the cause.

Morbid Anatomy and Pathology.—The remarks which might be made under this head have already been expressed to some extent in the foregoing pages, and there is not much more that could be said without entering the domain of pure speculation. I may, however, state my opinion that neuralgia, not directly the result of some physical cause interfering with the integrity of the nerve in which it is situated, is almost invariably induced by a depressed state of the system. Its existence in such cases is evidence, therefore, of deficient physical stamina, and of the fact that the nervous system is not duly nourished. The remote factor may be malaria, syphilis, rheumatism, gout, or some other cause capable of lowering the vitality of the organism, and, as a consequence, that of the nerves. It is of course of the utmost importance, with reference to the treatment, to ascertain whether there is, or is not, any such constitutional taint, but, whatever the result of our inquiries in this direction, that system of therapeutics is best which, in addition to physical measures, embraces restorative means.

Treatment.—The measures which it is proper to employ in neuralgia may be divided into two categories, the constitutional and the local.

Among the constitutional remedies must be included

those which are for the correction of any taint which may be present. If there is reason to suspect the existence of syphilis, iodide of potassium is an indispensable remedy, and should be given in large doses. If malaria can be ascertained to have exerted an influence, quinine must be administered; and, indeed, it is safe to act upon the theory that this has been the cause, unless some other can be clearly made out. It must be recollected that malaria may give rise to neuralgia, especially in the facial nerve, without there having been any other manifestation of its toxic effect; and that the affection is often cured by large doses of quinine, when the patient has not apparently been subjected to the malarious influence. Should there be no relief after three or four ten-grain doses of quinine, it should still not be decided that the disease is not of malarious origin, but arsenic should be administered. I have seen many cases of supra-orbital neuralgia, undoubtedly the result of miasmatic poisoning, effectually cured by arsenic, when quinine had failed. From my own experience, I am very well convinced that it acts much more efficaciously when administered by hypodermic injection than by the stomach. Four drops of Fowler's solution, diluted with an equal quantity of water, should be given twice a day, and the quantity should be gradually increased to eight or ten drops at a dose.

If a gouty diathesis is present, colchicum should be used; and, if rheumatism be clearly made out, the blood should at once be rendered alkaline by liquor potassæ.

Whether any specific difficulty be discovered or not, general tonics are always indicated; among them cod-liver oil occupies the front rank, and iron is not far behind it in value; strychnia is also very generally useful. Among constitutional remedies, ergot has proved of very decided benefit in my hands, and this especially in sciatica. It should be given in large doses, a drachm or more of the tincture three times a day.

A full and nutritious diet is of great value in the treat-

ment of neuralgia, as are likewise sunlight, and pure and fresh air.

In addition to these purely constitutional measures, there are others which, though administered to act upon the system at large, are given for the purpose of arresting a paroxysm, or deadening sensibility, so as to prevent the pain being felt. The medicines embraced in this category are included among the stimulants, narcotics, and anæsthetics. Opium and its various preparations are preëminent as palliatives of the neuralgic paroxysm, and morphia stands first among them. It is most efficaciously administered hypodermically, in doses varying from one-eighth grain to half a grain, or even more in extreme cases. Great care should be exercised in its use, and the smaller quantity mentioned should not be exceeded except by regular gradations. It is immaterial in what part of the body the injection is made, so far as its influence over the pain is concerned.

Among other medicines of this class are belladonna, or its alkaloid atropia, Indian hemp, aconite, bromide of potassium, hydrate of chloral, hyoscyamus, conium, and some others of minor importance.

Of very great value are chloroform and ether, administered by inhalation, and the various forms of alcoholic liquors.

The chief local means of treatment in neuralgia are counter-irritation—preferably in the form of repeated blisters, which should be applied over the course of the painful nerves, and which are especially valuable in sciatica—and the local application of tincture of aconite, and of veratria in the form of an ointment, or an alcoholic or ethereal solution.

But, above all local means, not only for relieving the pain of any particular paroxysm, but also for effecting a permanent cure, electricity stands first. I have employed it in every possible form, and am satisfied that the primary galvanic current is the preferable agent. Indeed, I very much doubt if the induced current, unless in a few cases, when

the wire-brush has been employed, has ever, in my experience, accomplished any very decided benefit. In the employment of the primary current, the positive pole should be applied over the seat of the pain, and not more than fifteen or twenty Smee's cells should be used. The application should be continuous for at least half an hour, and should be repeated every day for several weeks, and in extreme cases longer. I have cured a number of severe cases of nearly every kind of neuralgia by the aid of electricity when other means had entirely failed. I rarely, however, employ it without at the same time insisting on such constitutional treatment as the case seems to require.

I have several times used acupuncture with success, and have likewise employed electro-puncture with decided benefit in ten cases of sciatica. In either operation the needles should be introduced at the most painful parts, and, when galvanism is also used, the current should be passed continuously through the needles. In a notable case of sciatica which I saw in consultation with my friend Dr. John Gallaher, of this city, a severe attack of sciatica was at once cut short by electro-puncture. Two or three cells will afford a current of sufficient tension.

As to surgical operations on the affected nerves, either of section or excision of a portion of their continuity, the success which has hitherto followed them has not been such as, in my opinion, to warrant their repetition.

LEEDS SCHOOL OF MEDICINE

INDEX.

Abscess, chronic cerebral, 254.
Active cerebral congestion, 33.
  symptoms, 34.
  apoplectic form of, 37.
  epileptic form of, 39.
  maniacal form of,
  first stage of, 34.
  second stage of, 37.
  third stage of, 41.
Acute cerebral meningitis, 219.
Acute myelitis, 456.
  symptoms, 456.
  causes, 459.
  diagnosis, 459.
  prognosis, 460.
  morbid anatomy, 460.
  pathology, 460.
  treatment, 460.
Acute spinal meningitis, 444.
Affective insanity, 336.
Anæmia, cerebral, 61.
Anæmia of antero-lateral columns of cord, 430.
  symptoms, 432.
  causes, 432.
  diagnosis, 433.
  prognosis, 433.
  morbid anatomy, 434.
  pathology, 434.
  treatment, 437.
Anæmia, spinal, 396.
  of posterior columns of spinal cord, 397.
Anæsthesia of cutaneous nerves, 735.
  symptoms, 735.
  causes, 736.
  diagnosis, 736.
  prognosis, 736.
Anæsthesia of the fifth pair, 737.
  symptoms, 737.
  causes, 738.
  diagnosis, 738.
  prognosis, 738.
  morbid anatomy, 738.
  pathology, 738.
  treatment, 738.
Anæsthesia, peripheral, 735.
Antero-lateral columns of cord, anæmia of, 430.
  sclerosis of, 471.
Aphasia, 166.
Athetosis, 654.
  cases of, 654.
  description of, 654.
Atrophy and disappearance of motor nerve-cells, 677.
  of motor and trophic nerve-cells, 689.
  of trophic nerve-cells, 663.

Brain, tumors of, 301.

Catalepsy, 590.
  symptoms, 590.
  causes, 594.
  diagnosis, 594.
  prognosis, 595.
  morbid anatomy, 595.
  pathology, 595.
  treatment, 596.
Cerebral abscess, chronic, 254.
Cerebral anæmia, 61.
  symptoms, 61.
  causes, 64.
  diagnosis, 66.
  prognosis, 67.
  morbid anatomy, 67.
  pathology, 67.
  treatment, 69.
Cerebral arteries, obliteration of, 119.
Cerebral congestion, 33.
  causes, 43.
  diagnosis, 46.
  prognosis, 49.
  morbid anatomy, 50.
  pathology, 52.
  treatment, 54.
Cerebral hæmorrhage, 74.
  symptoms, 74.
  causes, 85.
  diagnosis, 90.
  prognosis, 95.
  morbid anatomy, 97.
  pathology, 99.
  treatment, 106.

Cerebral meningitis, acute, 219.
symptoms, 219.
causes, 224.
diagnosis, 225.
prognosis, 225.
morbid anatomy, 226.
pathology, 227.
treatment, 228.
Cerebral meningitis, chronic, 231.
tubercular, 234.
Cerebral rheumatism, 223.
Cerebral sclerosis, diffused, 260.
multiple, 278.
Cerebral softening, 137.
symptoms, 137.
causes, 145.
diagnosis, 147.
prognosis, 148.
morbid anatomy, 149.
pathology, 151.
treatment, 157.
Cerebritis, 246.
Cerebro-spinal diseases, 545.
Cerebro-spinal sclerosis, multiple, 637.
Cervico-brachial neuralgia, 743.
causes, 744.
diagnosis, 744.
prognosis, 744.
Cervico-occipital neuralgia, 743.
causes, 743.
diagnosis, 743.
prognosis, 743.
Chronic cerebral abscess, 254.
Chronic cerebral meningitis, 231.
symptoms, 231.
causes, 232.
diagnosis, 232.
prognosis, 232.
morbid anatomy, 232.
pathology, 232.
treatment, 232.
Chronic spinal meningitis, 445.
Chorea, 600.
symptoms, 601.
causes, 609.
diagnosis, 610.
prognosis, 611.
morbid anatomy, 612.
pathology, 612.
treatment, 615.
Classification of insanity, 335.
Congestion, cerebral, 33.
spinal, 385.
Cretinism, 338.
Crural neuralgia, 745.
Cutaneous nerves, anæsthesia of, 735.

Definition of insanity, 332.
Degeneration, secondary, of spinal cord, 523.
Delirium, 343.
Delusion, 341.
Dementia, 336, 338, 373, 374.
Dementia, organic, 337.
senile, 337.
Diffused cerebral sclerosis, 260.
symptoms, 260.
causes, 272.
diagnosis, 273.
prognosis, 273.
morbid anatomy, 274.
pathology, 275.
treatment, 275.
Diseases of nerve-cells, 663.
of peripheral nerves, 721.
of the spinal cord, 385.
Dura mater, hæmatoma of, 116.

Eccentricity, 331.
Ecstasy, 597.
symptoms, 597.
causes, 598.
diagnosis, 598.
prognosis, 598.
morbid anatomy, 598.
pathology, 598.
treatment, 598.
Embolism, 127.
symptoms, 127.
causes, 131.
diagnosis, 132.
prognosis, 134.
morbid anatomy, 134.
pathology, 134.
treatment, 136.
Emotions, 328.
Emotional insanity, 338, 351.
Encephalitis, 246.
Epilepsy, 560.
symptoms, 560.
paroxysm, varieties of, 563.
causes, 572.
diagnosis, 575.
prognosis, 576.
morbid anatomy, 577.
pathology, 578.
treatment, 582.
Epileptic insanity, 337.

Facial neuralgia, 739.
Facial paralysis, 722.
symptoms, 722.
causes, 727.
diagnosis, 728.
prognosis, 728.
morbid anatomy, 729.
pathology, 729.
treatment, 729.
Facial spasm, 731.
symptoms, 731.
causes, 732.
diagnosis, 732.
prognosis, 732.
morbid anatomy, 732.
pathology, 732.
treatment, 732.

Fifth pair, anæsthesia of, 737.
hyperæsthesia of, 739.
neuralgia of, 739.
Functional derangements of motor nerve-cells, 706.

General paralysis, 338, 366.
General principles of insanity, 324.
Gliomata, 320.
Glosso-labio-laryngeal paralysis, 677.
symptoms, 678.
causes, 684.
diagnosis, 685.
prognosis, 686.
morbid anatomy, 686.
pathology, 687.
treatment, 688.

Hæmorrhage, meningeal, 115.
Hæmorrhage, spinal meningeal, 440.
Hæmorhage, spinal, 440.
symptoms, 440.
causes, 441.
diagnosis, 441.
prognosis, 441.
morbid anatomy, 442.
pathology, 442.
treatment, 442.
Hæmatoma of the dura mater, 116.
causes, 117.
prognosis, 117.
diagnosis, 117.
morbid anatomy, 118.
pathology, 118.
treatment, 118.
Hallucination, 340.
Hydrophobia, 545.
symptoms, 545.
causes, 551.
diagnosis, 553.
prognosis, 554.
morbid anatomy, 556.
pathology, 556.
treatment, 557.
Hyperæsthesia peripheral, 739.
Hypertrophy of muscular connective tissue, 699.
symptoms, 700.
causes, 701.
diagnosis, 703.
prognosis, 703.
morbid anatomy, 703.
pathology, 704.
treatment, 705.
Hysteria, 619.
symptoms, 619.
causes, 629.
diagnosis, 631.
morbid anatomy, 632.
pathology, 632.
treatment, 634.

Idiocy, 336, 338, 373.
Illusion, 339.
Imbecility, 336.
Incoherence, 342.
Infantile paralysis, 689.
Inhibitory paralysis, 431.
Insanity, 324.
classification of, 335.
causes, 376.
diagnosis, 377.
prognosis, 377.
morbid anatomy, 378.
pathology, 378.
treatment, 380.
definition of, 332.
affective, 336.
emotional, 338, 351.
epileptic, 337.
intellectual, 338, 345.
perceptional, 338, 343.
paralytic, 337.
simple, 337.
volitional, 338, 357.
Intellect, 328.

Lead paralysis, 715.
symptoms, 715.
causes, 716.
diagnosis, 717.
prognosis, 717.
morbid anatomy, 718.
pathology, 718.
treatment, 719.
Locomotor ataxia, 484.
Lumbo-abdominal neuralgia, 745.

Mania, 336, 338, 358.
Melancholia, 336.
Meningeal hæmorrhage, 114.
causes, 115.
prognosis, 116.
diagnosis, 116.
morbid anatomy, 116.
pathology, 116.
treatment, 116.
Meningitis, cerebral, chronic, 231.
cerebral, acute, 219.
cerebral, tubercular, 234.
spinal, 444.
senile, 223.
Monomania, 336.
Motor nerve-cells, atrophy and disappearance of, 677.
Motor nerve-cells, functional derangements of, 706.
Motor and trophic nerve-cells, atrophy and disappearance of, 689.
Multiple cerebral sclerosis, 278.
symptoms, 269.
causes, 288.
diagnosis, 289.
prognosis, 290.
morbid anatomy, 290.
pathology, 291.

Multiple cerebro-spinal sclerosis, 637.
symptoms, 637.
causes, 651.
diagnosis, 651.
prognosis, 652.
morbid anatomy, 652.
pathology, 652.
treatment, 652.
Muscular connective tissue, hypertrophy of, 699.
Myelitis, acute, 456.

Nerve-cells, diseases of, 663.
Nerve-cells, motor, functional derangements of, 706.
Nerves, cutaneous, anæsthesia of, 735.
Neuralgia, 739.
varieties of, 739.
causes, 344.
diagnosis, 745.
prognosis, 745.
morbid anatomy, 746.
pathology, 746.
treatment, 746.
Neuralgia of fifth pair of nerves, 739.
symptoms, 739.
causes, 741.
diagnosis, 742.
prognosis, 742.
Neuralgia, cervico-brachial, 743.
cervico-occipital, 743,
crural, 745.
dorso-intercostal, 744.
lumbo-abdominal, 745.
sciatic, 745.

Obliteration of cerebral arteries, 119.
Organic dementia, 337.
Organic infantile paralysis, 689.
symptoms, 689.
causes, 691.
diagnosis, 691.
prognosis, 691.
morbid anatomy, 692.
pathology, 695.
treatment, 696.

Paralysis agitans, 706.
symptoms, 707.
causes, 708.
diagnosis, 708.
prognosis, 708.
morbid anatomy, 709.
pathology, 709.
treatment, 710.
Paralysis, general, 338, 366.
infantile, 689.
inhibitory, 431.
reflex, 431.
facial, 722.
lead, 715.
peripheral, 722.
Paralytic insanity, 337
Partial cerebral anæmia, 119.
Passive cerebral congestion, 41.
symptoms, 41.
first stage of, 41.
apoplectic form of, 42.
second stage of, 42.
epileptic form of, 42.
maniacal form of, 42.
Pathetic insanity, 336.
Peripheral anæsthesia, 735.
hyperæsthesia, 739.
nerves, diseases of, 721.
paralysis, 722.
spasm, 731.
Perception, 327.
Perceptional insanity, 338, 343.
Posterior columns of spinal cord, anæmia of, 397.
Posterior columns of spinal cord, sclerosis of, 484.
Progressive muscular atrophy, 663.
symptoms, 664.
causes, 668.
diagnosis, 670.
prognosis, 670.
morbid anatomy, 670.
pathology, 674.
treatment, 676.

Reflex paralysis, 431.

Sciatic neuralgia, 745.
causes, 745.
diagnosis, 746.
prognosis, 746.
Sclerosis, multiple, cerebral, 278.
Sclerosis, cerebro-spinal, 637.
Sclerosis, diffused cerebral, 260.
Sclerosis of antero-lateral columns of spinal cord, 471.
symptoms, 471.
causes, 476.
diagnosis, 477.
prognosis, 477.
morbid anatomy, 477.
pathology, 477.
treatment, 482.
Sclerosis of posterior columns of spinal cord, 484.
symptoms, 484.
causes, 496.
diagnosis, 496.
prognosis, 498.
morbid anatomy, 498.
pathology, 499.
treatment, 513.
Secondary degeneration of spinal cord, 523.
symptoms, 524.
causes, 526.
diagnosis, 526.
prognosis, 526.
morbid anatomy, 527.
pathology, 527.
treatment, 527.

Senile dementia, 337.
Senile meningitis, 223.
Simple insanity, 337.
Softening, cerebral, 137.
spinal, 463.
Spasm, peripheral, 731.
facial, 731.
writer's, 710.
Spinal cord, anæmia of posterior columns of, 397.
sclerosis of antero-lateral columns of, 471.
sclerosis of posterior columns of, 484.
secondary degeneration of, 523.
tumors of, 517.
Spinal anæmia, 396.
Spinal congestion, 385.
symptoms, 385.
causes, 388.
diagnosis, 389.
prognosis, 390.
morbid anatomy, 391.
pathology, 391.
treatment, 392.
Spinal hæmorrhage, 440.
Spinal meningeal hæmorrhage, 440.
Spinal irritation, 397.
symptoms, 408.
morbid anatomy, 416.
pathology, 416.
diagnosis, 419.
treatment, 422.
Spinal meningitis, 444.
symptoms, 444.
causes, 446.
diagnosis, 447.
morbid anatomy, 448.
pathology, 448.
treatment, 448.
Spinal softening, 463.
symptoms, 463.
causes, 467.
diagnosis, 467.
prognosis, 468.
morbid anatomy, 468.
pathology, 468.
treatment, 469.
Suppurative encephalitis, 246.
symptoms, 246.
causes, 251.
diagnosis, 251.
prognosis, 253.
morbid anatomy, 253.
pathology, 253.
treatment, 258.

Tetanus, 529.
symptoms, 529.
causes, 533.
diagnosis, 534.
prognosis, 536.
morbid anatomy, 537.
pathology, 537.
treatment, 539.
Thrombosis, 119.
symptoms, 119.
causes, 121.
prognosis, 122.
diagnosis, 122.
morbid anatomy, 123.
pathology, 123.
treatment, 126.
Trophic nerve-cells, atrophy and disappearance of, 663.
Tubercular cerebral meningitis, 234.
symptoms, 234.
causes, 240.
diagnosis, 241.
prognosis, 242.
morbid anatomy, 242.
pathology, 242.
treatment, 244.
Torticollis, 733.
symptoms, 733.
causes, 733.
diagnosis, 733.
prognosis, 733.
morbid anatomy, 733.
pathology, 733.
treatment, 733.
Tumors of the brain, 301.
symptoms, 301.
causes, 311.
vascular, 311, 315.
parasitic, 312, 317.
diathetic, 312, 319.
tuberculous, 312, 319.
syphilitic, 312, 320.
accidental, 312, 320.
diagnosis, 313.
prognosis, 315.
morbid anatomy, 315.
fibro-plastic, 320.
osseous, 321.
lipomatous, 321.
enchondromatous, 321.
mucous, 321.
treatment, 322.
Tumors of spinal cord, 517.
symptoms, 517.
causes, 520.
diagnosis, 520.
prognosis, 521.
morbid anatomy, 621.
pathology, 521.
treatment, 521.

Volitional insanity, 338, 357.

Will, 328.
Writer's spasm, 710.
symptoms, 711.
causes, 713.
diagnosis, 713.
prognosis, 714.
morbid anatomy, 714.
pathology, 714.
treatment, 714.

Page numbering jumps from XXV to p34 after the intro.