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L. E. LA FÉTRA, M.D., Associate Editor

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By

B. K. RACHFORD, M. D.

PROFESSOR *of* DISEASES *of* CHILDREN, MEDICAL COLLEGE *of* OHIO, UNIVERSITY *of* CINCINNATI. PEDIATRIST *to the* CINCINNATI, GOOD SAMARITAN *and* JEWISH HOSPITALS. MEMBER *of* AMERICAN PEDIATRIC SOCIETY, ASSOCIATION *of* AMERICAN PHYSICIANS, *etc.*

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PREFACE

In 1893-94 I published a series of papers in the *Archives of Pediatrics* entitled "Some Physiological Factors of the Neuroses of Childhood."

In these papers I made an effort to study the physiological peculiarities of the immature nervous systems of infants and children, and to note the all-important bearing which these peculiarities had in producing and in giving individuality to the neuroses of childhood.

It is a well-known fact that infants and children are especially predisposed to serious and complicated nervous disorders, and that this class of diseases has been very little understood by the general practitioner, and has, in fact, not been a matter of special study by neurologists.

For these reasons I decided to revise the papers previously published in the *Archives of Pediatrics* and make of them the nucleus of a book on the Neurotic Disorders of Childhood.

Part I. of this book contains these revised papers, with the addition of chapters on "Gastro-Intestinal Toxæmia," "Auto-Intoxications" and "Chronic Systemic Bacterial Toxæmias."

Part II. of this book deals with the individual neuroses. Here I have attempted a careful study of the many neurotic disorders of childhood, and have endeavored to

so present the etiology, symptomatology, and treatment of these diseases that the student of medicine and the general practitioner will not only be able to better comprehend these common and little understood diseases, but will also be able to apply successful lines of treatment.

B. K. RACHFORD, M.D.

CINCINNATI, OHIO, *September*, 1905.

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PART I

Neurotic Disorders of Childhood

CHAPTER I

NORMAL FUNCTIONS OF NERVE CELLS

THE term "neuroses of childhood" is used to cover all local and general nervous disorders which do not depend on known local pathological lesions of the nervous system. This definition of the term neuroses does not imply that these diseases have an entirely unknown pathology, but that they cannot be morphologically classified. In these diseases we know more of the symptoms than we do of the lesions, more of the effect than we do of the cause, more of the disordered functions of nerve cells than we do of the widely varying pathological conditions which produce these disordered functions, and these are the reasons why these diseases are incorrectly called functional nervous diseases.

The first requisite to the study of perversions in the functions of nerve cells should be a knowledge of the normal functions of nerve cells. For this reason the following preliminary physiological outline is introduced.

Nerve cells have three important functions, viz., to generate, to discharge, and to inhibit energy.

The highest function of the nerve cell is to generate energy. By this it is meant that the cell transforms and appropriates existing energy. The amount of existing energy is constant; the cell does not and cannot

originate energy, but in the chemical metabolism necessary to the life of the cell force is developed, which is transformed into that form of nerve energy which is the special function of the individual cell (Professor J. Gad—personal communication), and this nerve energy is stored up to be discharged in the exercise of the cell's peculiar function. From this it would follow that the generation of nerve force would be directly dependent on the healthful chemical metabolism of the nerve cell; but it does not follow that the amount of energy thus developed would always be commensurable with the physical waste or the chemical metabolism going on in the cell. This disproportion between cell activity and the amount of force developed is especially noticeable in *the immature nerve cells of the child.* A most marked example of the slight amount of energy developed by the cell activity of immature cells may be noted in the cortical cells of the brain of the infant and the brain of the unintelligent adult. In such brains the cortical cells concerned in the development of mental energy have going on within them an active chemical metabolism with the development of very little mental energy, and this failure of chemical metabolism to develop commensurate mental energy is due to the incomplete functional development of these cells. The same amount of force may be developed, but the cell has not the power of converting this force into mental energy. Of all the cells in the body, the cell that develops mental energy is the slowest in reaching the degree of functional perfection for which it is destined, and it does so only after a judicious training, in the exercise of its peculiar func-

tion, throughout a long period of about twenty-three years.

The functional development of the motor cell is much more rapid, and during this development the disproportion between the amount of cell activity and force produced is not so great as in the mental cell, but nevertheless it may be stated as a fact true for all nerve cells, that the amount of energy which a cell is capable of generating will depend on the degree of functional development which the cell has attained. But these facts, concerning the difference in the amount of cell energy developed by different cells under the same conditions, do not in any way modify the force of the statement made above, that nerve energy is directly dependent on the chemical metabolism of the nerve cell. It will therefore be permissible for us to say that, *other conditions being the same, the amount of energy developed by a nerve cell will directly depend on the amount of healthful chemical metabolism going on within it.* This point in the physiology of the development of cell energy is very important, since upon it rests the conclusion that insufficient nourishment will diminish the capacity of the nerve cell for the generation of energy. The maximum amount of energy will, therefore, be found stored in the well-nourished cell and the minimum amount of energy in the starved cell. We shall see later that this statement, which has important clinical bearing, can be strongly supported by experimental evidence.

Discharge of nervous energy is a function of the nerve cell only second in importance to the generation of energy. The more or less constant discharge of force is

an automatic function of the nerve cell, and this unconscious discharge of nerve energy is the regulating function that controls the whole body mechanism. As an example of this automatic discharge of nerve force, one may cite the influence of the central nervous system over involuntary muscular tissue, whereby the "muscular tone" of involuntary muscles is maintained. The vasomotor center in the medulla oblongata has such an influence on the muscular coats of blood vessels as to keep them in a state of normal contraction best adapted for the purposes which they serve. This vascular tone remains much the same at all times, except when the functions of the center are perverted by some change in the metabolism of the cells, or by influences acting on the center either directly or in a reflex manner. But possibly of even greater importance to us, in this study, is the tonic influence of the spinal motor cells on the sphincter muscles of the anus and the bladder, which are dependent on the spinal cord for their normal muscular tone. The "muscular tone" of these sphincter muscles is easily disturbed by reflex stimulation, producing on the one hand spasmodic stricture and on the other incontinence. The muscular tone of the skeletal muscles is likewise said to be maintained by an automatic discharge of nerve force, and a perversion of this function may in the same manner produce complete relaxation, or irregular spasmodic contractions of these muscles. These examples on the part of the muscles are sufficient to illustrate how nerve cells, by the automatic discharge of nerve force, regulate the whole body mechanism. It would be of no value for us to discuss whether this more or

less constant discharge of nerve force is purely an automatic function of the cell, or whether it is due to unconscious afferent impulses producing a reflex discharge of force. It is sufficient for us to know that these phenomena exist, and it is a matter of words whether we speak of them as automatic or as reflex.

Nerve force may also be discharged *voluntarily*. This power of willing the discharge of nerve impulses resides in the cortical cells of the cerebrum. The influence of the will over the discharge of force, by the spinal motor cells, is a physiological fact of great clinical importance in the study of the neuroses of childhood.

Thirdly, and lastly, and most important of all, so far as our present study is concerned, *nerve force may be discharged reflexly*. This reflex discharge of force occurs when nerve cells are acted on by outside stimuli. If the stimulus be mild, the reflex discharge of energy from the normal motor cells of the cord occurs only through the paths of least resistance, viz., the efferent nerves in the same plane, and on the same side, as the nerve fiber that carried the afferent stimulus; but if the stimulus be more severe the reflex discharge of force will also occur in the same plane, but on both sides of the cord. We shall see later how these simple laws of reflex action have little control over the reflex discharge of nerve force under certain pathological conditions.

Inhibition of nervous energy is the third important function of the nerve cell. Certain cells throughout the central nervous system have the power of inhibiting energy discharged by other cells, and it is also possible that some cells of high functional development may have

the power of inhibiting their own energy. But however this may be, it is a well-established fact that inhibition does exist, and that this power of inhibiting nervous energy may be either voluntary or involuntary. Voluntary inhibition of mental and motor force is a function peculiar to the cells of the cerebral cortex, but involuntary inhibition of nerve force is a function of cells everywhere distributed throughout the central nervous system; but the higher centers are always the predominating centers when the nervous system is intact. The spinal cord contains cells, or collections of cells (centers), which are capable of being excited reflexly, so as to give motor expression to sensory stimulants, and inhibition can best be understood by studying the inhibitory influence of the higher centers on spinal reflex acts. The spinal reflex centers can act quite independently of higher centers. Gad demonstrated that after section of the spinal cord at any point the centers below the section are still active and capable of translating sensory impressions into motor acts. But this absolute autonomy of the spinal reflex centers does not exist when the spinal cord is in normal communication with the brain; then the reflex centers in the cord are more or less under control of other centers higher up in the cord (the medulla oblongata and the brain). These centers may influence the lower spinal centers, not only in causing them to discharge force, as we have above noted, but also in inhibiting their reflex acts, which are discharged from any cause whatsoever. Some of the inhibitory influences coming from the brain are voluntary, and probably originate in the cells of the cerebral cortex; for

example, we can by voluntary inhibition control the urinary bladder reflexes and prevent urination even when the micturition center, in the lumbar cord, is strongly stimulated; and again there are spinal reflexes over which voluntary inhibition has no control, as, for example, erection, ejaculation, movement of the iris.

Of even more importance to us in the study of the neuroses of childhood are the involuntary inhibitory centers which are distributed throughout the central nervous system. They are found in the brain, the medulla oblongata, and the spinal cord, and without voluntary effort or apparent reflex stimulation these centers seem to exert a constant inhibitory influence on the lower spinal centers. Setchenow's inhibitory center, in the medulla oblongata, is an example of similar centers which we have reason to believe exist in the large ganglia at the base of the brain. The inhibitory influence of this center on spinal reflex acts has been quite satisfactorily demonstrated. It is also easy to demonstrate, in a brainless frog, that stimulation of the sciatic nerve will inhibit spinal reflex acts. It is clear, therefore, that spinal inhibition may be brought about by other impulses than those that come from predominating centers in the brain and medulla oblongata; that is to say, by impulses which are not in themselves of a specific inhibitory nature, but originate in the peripheral stimulation of sensory nerves. But it is not necessary for me to narrate experiments bearing on the subject of inhibiton of nerve force, for such experiments are so satisfactorily detailed in the physiologies that I need here only say that experimental physiology teaches us to believe that there are cells, every-

where distributed throughout the central nervous system, which have the power of inhibiting nervous energy. It matters little to us in the present study whether this inhibition is always the special function of certain cells or whether it may also be the function of the nucleus of the cell that discharges the energy; but it is important for us to know that inhibition exists both for mental and motor acts, and we will see later why a clear understanding of the influences that control and disturb inhibition is of the utmost importance to us in the study of the neuroses of childhood. If kept in mind, the above outline of the normal functions of the mature nerve cell will materially assist in the study of the functional peculiarities that are manifested by the immature cells of the rapidly developing nervous system of the child.

CHAPTER II

PHYSIOLOGICAL PECULIARITIES OF THE NERVOUS SYSTEM DURING INFANCY AND CHILDHOOD

We have some knowledge of a few of the physiological peculiarities of the immature nervous system of the child that have a most important etiological import in the study of the neuroses of childhood, and it is my purpose here to outline such of these peculiarities as I believe to have a bearing on neurotic disease.

In the young infant the dura mater is very closely adherent to the skull, and the blood vessels of the pia mater are so abundant and so fragile that hemorrhage into the subarachnoid space may result from causes which produce high blood pressure.

At birth the brain is morphologically and functionally the most immature of all the great organs of the body. From birth up to seven years of age it develops enormously in weight, in structure, and in function. At this time the brain has attained 90 per cent. of its maximum weight (Boyd), and after this slowly increases in weight up to the age of eighteen; but increase of function does not keep pace with increase of weight,—the brain of a child of eight is almost as large as the brain of an adult,—but, as Clouston aptly says, "the difference between what the brain of a child of eight and the brain of a man of twenty-five can do and can resist is quite indescribable. The organ at these two periods might be-

long to two different species of animals, so far as its essential qualities go."

The chief structural deficiency at this time is in the cortex, and from this time on the increase in cortical matter is relatively greater. While the rapid increase in weight of the brain does not continue after the seventh year, the rapid increase in the brain's functional development goes on, and still continues, long after the brain at eighteen has reached its maximum weight. Clouston says: "The unique fact about the nerve cell is the extreme slowness with which it develops function after its full bulk has been attained." "In this it differs from any and every other tissue." "We may say that after most of the nerve cells of the brain have attained their proper shape and full size, it takes them the enormous time of eighteen or nineteen years to attain such functional perfection as they are to arrive at." It is an important fact that should always be kept in mind that the entire nervous system, of the normal infant and child, is constantly undergoing structural and functional development, and that the structural development, so far as we are able to judge by our instruments of precision, is much more rapid than is the development of function. It is also a fact that even with normal children this development of structure and function does not always go on with the same rapidity, nor does it always follow a regular order in its development. It is quite within the limits of health that certain functions may be rapidly developed and that other functions may be unusually retarded in development. The innumerable conditions of heredity and environment have their influence on the

nervous system of the child in developing and retarding both structure and function, and this interference with the order of development is not an abnormal condition if, within a reasonable time, the delayed functions reach a normal state of development. But it is not my purpose to enter deeply into this phase of my subject. I only wish to call attention to the following important facts:

1. At birth the nervous system is structurally, but more especially functionally, immature.

2. Throughout infancy and the earlier years of childhood the brain normally undergoes rapid structural development.

3. Throughout the entire period of infancy and childhood the brain normally undergoes rapid functional development.

4. Innumerable conditions of heredity and environment have much to do with the rapidity and the order of development of the functions of the nervous system of the normal child, as well as with the failure and retardation of their development in the abnormal child.

5. The metabolism in the normal immature nerve cell of the child must be rapid enough not only to supply the functional waste, but also to supply the material for the growth and development of cells.

6. This structural instability of the functionally weak and immature nerve cell of the child makes it much more irritable and excitable than the stable mature nerve cell of the adult.

With these general considerations of some of the functional peculiarities of the nervous system during childhood, let us pass to the consideration of certain special

functions of the nervous system which are not the same in childhood as in adult life.

The feeble inhibition of nerve energy is from a clinical standpoint the most important physiological peculiarity of the nervous system in infancy and childhood. The inhibitory function of the nerve cell is the last to be developed. The cell first acquires the function of generating energy, then the function of discharging energy, and lastly the function of inhibiting and coördinating energy. These functions of the cells are developed in the order in which they are needed. Until energy is generated there can be no occasion for a discharging function, and until energy is discharged there can be no occasion for an inhibiting function. *Feeble inhibition* is therefore one of the physiological characteristics of the immature nervous system of infancy and childhood, and it plays a most important rôle as a predisposing factor to the neuroses of childhood. Inhibition is very feeble in young animals, and becomes stronger as the animal grows older. The inhibitory function of cells is, therefore, in this regard like the generating function—it gradually becomes stronger as the cells get older, up to the time when they reach their complete functional development. But it must be remembered that the inhibitory function of a cell is always developed later than that function of the cell which generates the force to be inhibited. In the normal order of things the function of inhibition should closely follow the development of the function which generates the force to be inhibited.

The inhibitory mechanisms which control the discharge of nerve force that regulates such vital processes as the

action of the heart and the lungs are fairly well developed at birth, while those that regulate reflex phenomena are slowly developed during infancy and early childhood, and voluntary inhibition of motor and *mental* force does not not find its complete development till childhood has passed. The late development of the function of inhibition is a fact of prime importance from a clinical standpoint, because this is the last function of the cell to develop, and is the one that is most likely to be still further retarded in development by unfavorable conditions of heredity and environment. *It is therefore the abnormally feeble inhibition which occurs in the abnormal child that is such a potent factor in the production of neurotic disease in infancy and childhood.*

It is my belief that this functional immaturity of the inhibitory centers is most important in explaining the manner in which childhood acts as a predisposing cause of such neuroses as convulsions, epilepsy, hysteria, and incontinence of urine.

It is evident that inhibition is most feeble at birth, and gradually becomes stronger as the child grows older. This is especially true of voluntary inhibition. At birth voluntary inhibition, if it exists at all, must be very feeble, and it gradually grows stronger as the higher functions of the brain are more and more developed. We have a good example of voluntary inhibition in the influence of the will over urination. One wills to urinate, and the impulse passes down the cord to the lumbar center that presides over urination, and it is there translated into the reflex act of micturition; or, on the other hand, one wills not to urinate, and the impulse travels

down the cord to the lumbar center, and the act of urination is inhibited.

But the functional immaturity of the *involuntary* inhibitory centers is of even more importance to us as clinicians than the voluntary; for these centers having most to do with coördinating and regulating spinal movements, the lack of inhibition on the part of these centers would make it possible for an overflow of spinal reflex movements to occur passing up and down the cord, and in this way predispose to such convulsive disorders as eclampsia, chorea, and epilepsy. As previously noted, the reflex discharge of energy from the spinal motor cells occurs normally through the paths of least resistance, that is to say, in the same plane and on the same side, or in the same plane and on the opposite side, of the cord from where the nerve fiber entered that carried the afferent stimulus. But if the resistance to the spreading of the reflexes up and down the cord be reduced, or if the exciting stimulus be increased, then we may have an overflow of energy up and down the cord, exciting general spinal reflex movements. As above stated, these spinal reflex movements are inhibited, and an "overflow" of energy prevented by the action of involuntary inhibitory centers higher up in the cord (the medulla oblongata and the brain). The normally feeble inhibition of infancy predisposes to an "overflow" of spinal reflexes, or, in other words, to convulsive disorders of all muscles operated through spinal motor nerves. It is also easy to understand how unfavorable circumstances of environment and heredity, having their greatest retarding influence on the development of the inhibitory

function of the immature nerve cells of the infant and child, would still further predispose to overflow of spinal reflexes, and in this way to convulsive disorders. By this *overflow* of energy we may have a large number of spinal reflex movements as the result of a single exciting stimulus. Extensive convulsive movements of almost the entire body may in this way be caused by some simple discharging stimulus. It is one of the functions of the reflex inhibiting mechanisms to prevent this overflow, so that an impulse sent to one portion of the cord may not overflow and spread to other portions of the cord; but this mechanism being inefficient, incoördinated and spasmodic muscular movements occur. This overflow of nerve force is not peculiar to spinal cells exhibiting motor energy, but it also occurs in the cortical cells exhibiting mental energy (insanity). An inhibition against this overflow is quite as important in the brain cortex as in the spinal cord.

It is of clinical importance that we should here note that both the reflex centers and the conducting fibers by which reflex movements overflow, spreading up or down the cord, are in the sensory tracts of the cord, for this gives us a partial explanation of how certain drugs, such as cimicifuga, the bromides, antipyrin, and gelsemium, by depressing the sensory tracts of the cord, can control reflex spinal movements.

INCOMPLETE DEVELOPMENT OF PYRAMIDAL TRACTS

It is a fact of very great physiological and pathological importance that the fibers of the pyramidal tracts are the latest to become myelinated. At birth they have

no myeline sheaths, and, until their myeline sheaths are developed, it is believed that impulses cannot be carried from the convulsive centers at the base of the brain to the spinal cord cells. It has been noted that electrical excitation of the cerebral motor cortex, in dogs, at birth is not followed by movements (of extremities, etc.) presided over by spinal motor cells. This phenomenon was for a time explained on the supposition that the cerebral cortex is non-excitable in very young animals. This non-excitability of the motor cortex was thought to be due to a lack of development of these motor areas. While this hypothesis may serve as a partial explanation of the failure of spinal movements to respond to stimulation of the motor cortex, it is now known that on or about the tenth day of the life of the dog, when the pyramidal tracts have acquired their myeline sheaths, an excitation of the motor cortex will produce motion in muscles over which the spinal motor cells preside. The absence or partial development of the myeline sheaths of the fibers of the pyramidal tracts in very young animals may interfere, wholly or partially, with the lines of communication between the cerebral motor centers and the spinal motor cells. Day by day, as these myeline sheaths are developed, the cerebral and spinal motor cells are brought into closer communication, and very early in the life of the animal (ten days in the dog, and perhaps three or four months in the human infant), communication may be said to be fairly well established; prior to this time the communications are imperfect.

The above physiological facts may be offered in explanation of the comparative immunity which young infants

enjoy from convulsive disorders during the first few months of their lives. At this time it is probable that the motor areas of the cortex are not sufficiently well developed to respond readily to stimulation. It is also probable that the reflex centers of the cord are not fully developed at this early period; but most important of all is the fact that in certain young animals, and probably also in the human infant, the incomplete functional development of the pyramidal tracts makes the communication between the convulsive centers at the base of the brain and the spinal motor centers much more difficult than it is a few months later, when the myeline sheaths of the fibers of the pyramidal tracts are fully developed.

Convulsive Centers.—The true convulsive centers are located at the base of the brain, and probably all general convulsions are produced by a discharge of nerve force from these centers through the pyramidal tracts into the motor cells of the spinal cord.

The cortical motor centers are not true convulsive centers, but they are in such close touch with the convulsive centers at the base that any violent irritation of these cortical centers may produce general convulsions by causing a discharge of force from the basal convulsive centers. General convulsions of cortical origin may begin with convulsive movements in a single member, such as an arm or leg, and these become general through the action of the convulsive centers. The localized convulsive movements which precede the general convulsion may not only help to determine that the convulsion is cortical in its origin, but it also determines the portion of the cortex from which the irritation proceeds,—the

arm, leg, or face center, as the case may be. The motor fibers which pass directly from the motor areas of the cortex to the arm, leg, and face centers in the cord carry the impulses which produce the localized convulsive movements in these parts. At the same time, the same cortical irritation (the impulse possibly slightly delayed in transmission by the necessary relay of force) excites the basal convulsive centers to discharge their force into the cord, and a general convulsion follows very quickly the local convulsive movements. Localized convulsive movements followed by general convulsions always mean severe cortical irritation from some local organic condition. Localized convulsive movements not followed by general convulsions also mean localized organic disease, if not of the nervous system itself, then in such a location as to impinge upon or irritate certain of the peripheral nerves and ganglia. General convulsions, however, not preceded or marked by localized convulsive movements, are in the great majority of instances toxic in their origin. Where strong predisposition exists, either from hereditary influences or constitutional disease, general convulsions may be touched off by reflex causes.

Another reason for the infrequency of the reflex neuroses (including certain convulsive disorders) in the young infant is that the peripheral endings of the sensory nerves are not so perfectly developed in the early days of life as they are some months later.

In the light of the above physiological facts the feeble inhibition of early infancy is not so potent a factor in producing disease as it is a little later on, when inhibi-

tion is found not to have kept pace with the development of other functions of nerve tissue. The reflex centers of the cord and motor areas of the brain early in life take on the abnormal excitability of young nerve centers and are put in close communication by the functional development of the pyramidal tracts; but the inhibitory function of higher nerve centers over spinal cells and centers is very slow in reaching full development. In this way feeble inhibition, after the first few months of life, comes to play an important rôle as a predisposing factor to the neuroses of childhood.

The non-excitability of reflex centers in the spinal cord of the young infant has yet another important bearing, since it is in great part responsible for the *lack of tone* of the sphincter muscles of the infant. I have previously noted that the muscular tone of the sphincters was maintained by an automatic function of the central nervous system so closely analogous to reflex action that it seems a difference of name rather than of function. Now these reflex or automatic functions of the cord are so immature, in the newly born, that there is a lack of tone of all sphincter muscles—that is to say, an absence of the normal amount of contraction which afterwards fits them for the purposes they are to serve, and which depends in great part upon the action of normal reflex centers in the cord. This lack of sensitiveness of the reflex centers of the cord in the infant is, in my opinion, a most important factor in producing the incontinence which is characteristic of infantile sphincters. The incontinence of infantile sphincters passes away with the functional development of the centers whose function

it is to maintain in them the normal amount of muscular tone that fits them for the purposes they are to serve. Abnormal conditions of heredity and environment may much delay the functional development of these centers, and for this reason a complete or partial incontinence may continue long after the period when it should normally disappear. During this period, when involuntary inhibition is so feeble, voluntary inhibition is of great service in preventing, as it usually does, the diurnal incontinence. But at night, when the will is asleep, a minimum reflex will overcome the feeble involuntary inhibition and cause a relaxation of the sphincters. Besides this, any abnormal conditions of heredity or environment which increase the irritability of these reflex centers will also make it possible for slight reflex causes to disturb the "muscular tone" of sphincters, and cause either spasmodic stricture or incontinence. The pathological conditions, therefore, which produce feeble inhibition and excitable nerve centers are sufficient explanation for the not infrequent condition of incontinence of sphincters during childhood, and it is not necessary to invoke a cause which does not as a rule exist, viz., insufficient muscular development.

CHAPTER III

SOME PHYSIOLOGICAL FACTORS OF THE HIGH FEVERS AND THE VARIABLE TEMPERATURES OF CHILDHOOD.

It is a well-known fact that children are more prone to fever than adults, and it is also well known that the temperature is more variable in the fevers of infancy and childhood than it is in the fevers of adults. Why this is so is a question which we now wish to study from a physiologic standpoint. But first let us clearly understand what we mean by the terms high temperature and fever.

By high temperature is meant an increase of the body heat, whether it be due to increased heat production or diminished heat dissipation. When high temperature is due to increased heat production it is a symptom of fever, but when it is due to diminished heat dissipation it is not a symptom of fever.

By fever is meant an abnormal increase of those tissue changes by which the normal heat of the body is produced, that is to say, an abnormal increase of the chemic changes which result in disorganizing tissues and breaking them up into carbonic acid, water, urea, and other products of retrograde metamorphosis.

The fever process is characterized by a chain of symptoms with which every clinician is familiar; the most characteristic of these symptoms is increase of body temperature. But it must be remembered that the height

of the body temperature does not always mark the severity of the fever process, and that even a severe and wasting fever may exist with a subnormal temperature. One may note at least two reasons why the temperature is not an index of the severity of the fever process: First, increased heat production is but one of the results that is ordinarily but not necessarily produced by the same causes that produce fever; second, even should heat production keep pace with the severity of the fever process, heat dissipation may be so rapid or so variable that the body heat at any given time would not be an index of the fever process. With this understanding, the terms fever and temperature will be used as above defined, and we may proceed to study the influence of the nervous system on these processes.

Increased tissue metabolism, which is the one great cause of increased heat production, is under the direct control of the nervous system, and the centers which control this metabolism, and indirectly the production of body heat, are called heat centers.[1] Certain of these heat centers have the function of discharging force which will increase tissue metabolism and thereby increase the body heat; they are for this reason called *thermogenic* centers.

Other so-called heat centers have the power of inhibiting or controlling the discharge of force from the thermogenic centers, and they are, for this reason, called *thermo-inhibitory* centers. These thermo-inhibitory centers have no direct influence over the processes whereby the body heat is produced. Yet they are of the greatest

[1] Metabolism centers might be a better name for these centers.

clinical importance because of their control over the thermogenic centers.

The thermogenic and thermo-inhibitory centers have their functions so nicely balanced in the normal adult nervous mechanism that with the aid of the heat-dissipating centers they are able to maintain the body at almost an uniform temperature under the most adverse circumstances, and this temperature equilibrium can be disturbed only by some maladjustment of this nervous mechanism, which would produce either increase or decrease of the body temperature.

Where are These Heat Centers Located?—Ott, Richet, Sachs, Aronson, Wood, Reichert, Girard, Baginski, and White agree that the dominating thermogenic or heat-producing centers are situated at the base of the brain, in or near the corpus striatum. Eulenberg, Landois, Wood, Ott, Reichert, and White agree that important thermo-inhibitory centers are located in the cerebral cortex, and they are known as the cruciate and Sylvian centers.

As a prelude to the use of these physiologic data in the explanation of some important clinical phenomena associated with the diseases of infancy and childhood, let us first inquire, what would one expect, in the light of these facts, would be the influence on the body temperature of disease or injury of the parts of the brain containing these centers?

1. *Destruction* of that portion of the cerebral cortex containing the cruciate or Sylvian inhibitory heat centers should cause a rise of temperature, because the inhibitory influence of these centers on the basal thermogenic cen-

ters would be wholly or partially withdrawn. Experimental physiology confirms this deduction. This is probably the explanation of the fever that follows cerebral hemorrhage into the cortex, and a partial explanation of the fever of insolation.

2. *Irritation* of these cortical inhibitory centers should cause a subnormal temperature by strengthening the inhibitory control which they exercise over the thermogenic centers; this is also evidenced by physiologic experiments. We have here an explanation of the subnormal temperature which may result from cortical meningitis and from hemorrhage, foreign bodies, or depressed bone, all of which may first act by irritating these cortical centers (subnormal temperature), and later by destroying them (increase of body temperature).

3. *Destruction* of the basal thermogenic centers should cause a decrease of the body heat. But clinically there is little opportunity to observe the effect of destructive lesions of this portion of the brain, since any lesion sufficiently severe to destroy the basal heat centers would cause immediate death by the involvement of adjacent centers controlling vital processes. In shock we possibly have an example of subnormal temperature from partial paralysis of these centers, and in the compression stage of basilar meningitis we may have a subnormal temperature due to enfeeblement of these centers.

4. *Irritation* of the basal thermogenic centers should cause an increase of body heat; this fact, which is proven by physiologic experiment, is the explanation of the increased temperature that accompanies the specific fevers.

When are the Heat Centers Developed?—The answer to this question is in great part the answer to the question, Why are infants and children more prone to high temperatures than adults? The heat-dissipating centers situated in the medulla oblongata are well developed at birth, but these centers, because of their special clinical importance in infancy and childhood, will be given separate consideration later on. Here it is my purpose to note and especially emphasize the time of functional development of the heat-producing and the heat-inhibiting centers.

Before birth the thermogenic centers are in a state of immature functional development. In the human infant born prematurely they are so imperfect that artificial heat is necessary for a time to keep the body heat up to the normal. In this respect the immature human fœtus resembles cold-blooded animals, who are more or less dependent on their surroundings for their body heat. But as the fœtus matures the thermogenic mechanism reaches a state of fair development, so much so that one may say that the thermogenic centers are functionally competent at birth; this of course must be so, since the formation of body heat is a vital process, and is, as we have seen, probably controlled by the same mechanism that controls the all-important processes of tissue metabolism. While the thermogenic heat centers have a fair degree of development at birth, they are yet immature and unstable, and are therefore, like all the nerve centers in the unfinished brain of the child, more easily excited to abnormal action than are the mature heat centers of the adult brain. All the nerve cells of the rapidly growing

brain of the infant and child are in a state of more or less structural instability, since the metabolism going on within them must not only be rapid enough to supply waste, but also to furnish material for the growth and development of new cells. This structural and functional instability of the cells makes them more irritable and excitable than the nerve cells in the finished brain of the adult. For this reason one would expect to find the thermogenic heat centers of the child more excitable than those of the adult, and such in fact is the case. This is one important reason why the temperature of the infant is so variable and unstable under slight disturbing influences, and why like causes produce higher temperatures in the infant and child than in the adult.

But important as this normal excitability of the immature thermogenic centers of the child may be, yet of far greater importance from a clinical standpoint is the greatly increased irritability from unfavorable conditions of heredity, nutrition, and environment. The thermogenic heat centers of the nervous, anæmic, delicate child are in a state of abnormal excitability, so that a slight excitation will produce an abnormal discharge of force, resulting in fever and high temperature.

But after all, probably the most important cause of the instability of the body temperature in infancy and childhood is to be found in the feeble control exercised by the cortical thermo-inhibitory centers. The thermo-inhibitory centers, like other cortical inhibitory centers previously spoken of, have very imperfect functional development at birth, so that at this time they do not exert a very strong controlling influence over the basic thermo-

genic centers, and are not able to inhibit these centers from discharging increased energy under slightly increased excitation; for this reason slight causes may produce an elevation of temperature in the infant. Hale White says, in speaking of the thermo-inhibitory centers: "In the human adult they are fairly competent and active, as is proved by our pretty constant temperature." "In the lower animals and in children they are probably not so completely evolved, for I have found that the normal temperatures of rabbits vary several degrees, and rapid fluctuations of temperature are common in children even when slightly ill."

Ott, in a personal communication, says: "It seems to me that children are more prone to high temperatures because of a loss of control of the cortical centers."

It is, on the whole, a justifiable conclusion from all the evidence in our possession that the high and variable temperatures of infancy and childhood are in part due to the normal immaturity and instability of the cortical thermo-inhibitory centers. But, as I have previously noted, the feeble inhibition in the normal child is not of so much clinical importance as the abnormally feeble inhibition of the abnormal child; this is as true of the heat-regulating mechanism as it is of all other nervous mechanisms. The inhibitory part of the heat mechanism in its feeble and unstable state is the portion of this mechanism which suffers most from disease, and in its development is still further retarded by unfavorable conditions of heredity and environment. McAlister says: "The inhibitory is the first portion of the heat-regulating mechanism to fail under injury or disease." All of this

is quite in accord with the general observation previously made, that the amount of energy developed by a nerve cell will depend entirely on the amount of healthful chemical metabolism going on within it. The maximum amount of energy being stored up in the well-nourished cell and the minimum amount of energy in the starved cell, one can readily understand how a malnutrition of the nerve elements resulting either from heredity, impoverished blood, or bad hygiene can still further weaken the physiologically incompetent cortical thermo-inhibitory centers of the child, so as to make it more prone to variable and to high temperatures from slight causes than the normal child is, since in this condition the energy from the thermogenic centers would be discharged under much less restraint from the inhibitory centers than it is in the normal child. It may not be out of place here to state that the best explanation we have for the high and rapidly varying temperatures that not infrequently occur in hysterical women, is that they are due to the instability of the cortical thermo-inhibitory centers which have given way under the combined influence of environment, bad heredity, bad hygiene, and impoverished blood.

From what has been said the following summary may be made of the reasons why children are more prone than adults to high and variable temperatures:

1. In normal children the thermogenic centers are more unstable, and therefore more easily excited than in the adult.

2. In normal children the thermo-inhibitory centers are weaker, more excitable, and therefore more incapable

of exercising proper control over the thermogenic centers than they are in adults.

3. In malnourished, anæmic children the thermogenic centers are far more excitable than in normal children; such children are therefore more prone to high and variable temperatures.

4. In malnourished, anæmic children the thermoinhibitory centers are even weaker than in the normal child, and therefore still more incapable of restraining the discharge of force from the thermogenic centers; this is a most important reason for the variable and high temperatures of such children.

HEAT-DISSIPATING MECHANISM

The heat-dissipating mechanism is the mechanism by which we keep ourselves cool. This may be done in three ways:

1. By radiation and conduction of heat from the surface of the body.

2. By constant evaporation of water from the surface of the body.

3. By evaporation of water from the air passages.

Dissipation of heat by radiation from the surface of the body is by far the most important means of heat dissipation. In this process the vasomotor nervous mechanism is all-important. When unusual heat loss is demanded the vasomotor nerves dilate the blood vessels of the skin, and in this way expose more blood to the lower temperature of the air.

Loss of heat by evaporation is dependent on the

activity of the sweat glands, which are controlled by sudoriparous nerves and sweat centers. When unusual heat loss is demanded these centers respond by increasing the activity of the sweat glands, which cover the surface of the body with fluid, and the temperature is lowered by its evaporation. Both the dominating vasomotor and sweat centers are located in the medulla oblongata, and have reached good functional development at birth. But in the infant and young child they respond more readily and energetically to the demands for heat reduction than they do in the adult.

It must also be kept in mind that heat loss from both radiation and evaporation is greater in the infant than in the adult, because its area of surface is greater in proportion to its body weight; the infant has, in fact, a threefold greater radiation. These are the reasons why the high temperatures of infancy and childhood are so readily reduced by the heat-dissipating mechanisms. The increased activity of the heat-dissipating mechanisms acting on a proportionately larger surface compensates for the increased activity of the thermogenic centers. In the play of function between the heat-generating centers and the heat-dissipating centers we have an explanation of the rapid variations of temperature so characteristic of the fevers of infancy and childhood.

Evaporation of water from the air passages is a means of heat dissipation which we have yet to consider.

In certain animals, the dog, for instance, which do not sweat, the evaporation of water from the air passages is the chief means of reducing the body temperature. Richet calls the rapid respirations of the panting dog

Polypnœa. By these rapid respirations, amounting to as many as four hundred in a minute, the heat of the body is rapidly given off. Richet located the polypnœic center in the medulla oblongata. Ott later located it in the tuber cinerium. Richet proved that the polypnœic center was not affected by the amount of carbonic acid or oxygen in the blood, and that it was solely for the purpose of heat dissipation.

In answer to the question, How is the polypnœic center excited to activity? we have the experiments of Sihler, demonstrating that increased respiration of an animal exposed to heat is due to two causes, warmed blood and stimulation of the skin by the heat, and that skin stimulation is the more important factor. Gad and Mertschinsky also demonstrated that an increased temperature of the blood stimulates the respiratory centers and causes an increased number of respirations, and Ott produced polypnœa by electrical stimulation of the tuber cinerium.

Does the polypnœic center exist and is it functionally active in infancy and childhood? The answer to this question has important clinical bearings. Ott says: "In infants we see a polypnœa during fever; the respiration rises in frequency with the rise in temperature." Every physician must have seen many cases of rapid respiration in children that could not be accounted for by pulmonary disease. It not infrequently happens that a child with fever will have sixty, eighty, and one hundred respirations per minute, without presenting any sign or symptom of lung trouble. Polypnœa is, to my mind, the explanation of this phenomenon. Very rapid breathing

is a common symptom of summer complaint, and in many cases means nothing more than nature's attempts at heat dissipation. The importance of recognizing polypnœa as a symptom of fever in infancy and childhood is great. If we do not do this, we may often be led, by the rapid breathing, away from the real cause of the disease. Fortunately for us as clinicians, there is a marked difference between the character of the polypnœic breathing and the rapid respirations due to lung or heart disease. In polypnœa, the breathing is regular, easy, and rapid, but is not as it is in lung and heart disease, irregular, labored, and accompanied by cyanosis.

CHAPTER IV

GASTRO-INTESTINAL TOXÆMIA[1]

Gastro-intestinal toxæmia as differentiated from auto-intoxication is a systemic intoxication produced by poisons formed in and absorbed from the gastro-intestinal canal. Autotoxins, excreted into and afterward absorbed from the intestinal canal, cannot, therefore, be classed as intestinal toxins.

Our knowledge of the toxins which contribute to intestinal intoxication is not as yet upon a very satisfactory chemical basis. We know, however, that the intestinal contents, even under normal conditions, are toxic, and we know, also, that under certain pathological condition the toxins produced by the bacterial fermentation of foods in the intestinal canal very greatly increase this toxicity, with the result that a profound systemic intoxication is produced. Our present knowledge, however, is not as yet sufficient to make it profitable for us to attempt to differentiate, clinically, between the symptom groups produced by normal and abnormal intestinal toxins.

INTESTINAL TOXINS

The poisonous albumoses, which are the intermediate bodies formed in the digestion of albuminous food stuffs, may be mentioned as contributing to the toxicity of the

[1] Read before the sixteenth annual meeting of the American Pediatric Society, Detroit, Mich., May 30, 1904.

intestinal contents, but these bodies, under normal conditions, are robbed of their toxicity in their passage through intestinal epithelium and liver cells, during the process of absorption. The body is, therefore, carefully guarded against intoxication from this source. It may be, however, that when these protective mechanisms fail, through disease or functional disturbances of these filtering organs, these bodies may act as toxins.

Bacterial fermentation is the great source of intestinal toxins. It is possible that the poisons produced by bacteria in the intestinal canal may have their origin from three different sources:

1. The components of dead bacteria may furnish a certain amount of proteins; some of these bodies we know to be poisonous, as, for example, tuberculin, which is a protein of the tubercule bacillus. The bacillus of glanders and other bacilli also contain poisonous proteins, and it is not impossible that poisons of this type may at times be a factor in producing intestinal toxæmia. The rôle, however, which these bodies play in intestinal toxæmia has not as yet been determined, and is probably not very great.

2. Living bacteria in the intestinal canal may and do excrete ferments or toxins capable of producing the most profound nervous symptoms. These specific toxins of bacteria are believed to be very potent factors in producing intestinal toxæmia. It is evident that the character and virulence of these toxins will depend largely upon the micro-organisms producing the fermentation, since certain micro-organisms are capable of eliminating much more virulent toxins than others. Whether all of

these cell toxins belong to the ferment class is a question as yet undecided. Pathologists, at the present time, incline to the view that the specific toxins are closely allied to, if not identical with, the ferments.

3. Substances produced by bacteria from the culture media are possibly the most important source of intestinal intoxication. Among the poisons of this class the ptomains probably hold the most important place. These basic compounds, resembling the alkaloids in chemical structure, are capable of producing the most severe systemic intoxication. The virulence of the ptomain formed depends not only upon the micro-organism, but also upon the character of the food material in which it is growing. Certain food materials, such as cheese, milk, meat, and other albuminous foods, when acted upon by certain bacteria, may become very poisonous owing to the development of poisonous ptomains.

While ptomains are the most poisonous, and, so far as the etiology of intestinal toxæmia is concerned, the most potent of the transition products produced during the process of putrefaction, yet there are a large number of other bodies produced in this way which may also be factors in producing intestinal toxæmia. Among these bodies may be mentioned indol, skatol, phenol, cresol, leucin, tyrosin, ammonia, sulphuretted hydrogen, volatile fatty acids, oxalic acid, uric acid, and the xanthin bodies. Of these bodies, however, it must be said that indol is probably the only one whose etiological relationship to nervous symptoms has been satisfactorily demonstrated. Herter and others have recently shown that indol, one of the most constant and readily absorbed products of

the bacterial fermentation of albuminous food stuffs in the intestinal canal, is toxic, and capable of producing headache and increased nervous excitability. It is not improbable, therefore, that this body may at times, under the conditions named, contribute to the production of these symptoms.

Indirect Etiological Factors.—Constipation is the most important predisposing factor of both acute and chronic intestinal toxæmia. It acts by retarding the passage of food materials along the intestinal canal; this gives time for hyper-fermentation, and for the production of toxins by bacterial action on the intestinal contents.

Too much food, indigestible food, and food that has already undergone bacterial fermentation may also be important factors in producing, or in prolonging, either an acute or chronic intestinal toxæmia.

Lack of fresh air and exercise are also important predisposing factors.

The digestive idiosyncrasies of the individual are predisposing factors which manifest themselves not infrequently. These idiosyncrasies are sometimes made manifest by the taking of such foods as milk, eggs, oatmeal, shell-fish, strawberries, acids, and wines. One of the most common of the nervous syndromes developed in this way is urticaria. This symptom group is not uncommonly associated with an acid fermentation in the intestinal canal, and may, in certain instances, be classed as an acid intoxication of intestinal origin.

In studying the etiology of intestinal toxæmia it is necessary to bear in mind the important rôle which the liver plays in protecting the body against intestinal

toxins. These bodies can reach the general circulation, and thereby produce systemic intoxication only by passing through the liver; and in leaving the intestinal canal these bodies pass through the portal vein, and thence through the liver, to be worked over by the liver cells, and filtered through by way of the hepatic vein, and lymphatics to the general circulation. In passing through the liver, however, these intestinal poisons are under normal conditions rendered harmless. This so-called filtering function of the liver stands guard between the poisons absorbed from the intestinal canal and the general circulation.

One may suppose that so long as the toxins in the intestinal canal are not greatly increased in quantity, and so long as the filtering function of the liver is intact, the intestinal toxins cannot produce systemic intoxication, and one may also suppose that systemic intoxication may be produced by failure on the part of the liver to perform its function of filtering the poisonous blood in the portal vein, or from a great excess of intestinal toxins, which may so overwhelm the liver that these poisons find other channels of entrance into the general circulation.

It is well to keep these facts in mind, since in some instances the liver itself may be primarily at fault in chronic intestinal toxæmia. This organ, therefore, must be kept under supervision in the treatment of all chronic neuroses which are supposed to be either wholly or partly caused by intoxications from the intestinal canal.

Chronic intestinal toxæmia may also be an indirect cause of nervous symptoms, by the profound changes it

sometimes produces in the blood, contributing, as it does, to the production of chlorosis and other forms of chronic anæmia. These anæmic conditions may in turn cause malnutrition of nerve centers, and in that way produce chronic nervous irritability, and the long chain of nervous symptoms which are not uncommonly associated with profound anæmias.

Chronic appendicitis, especially in the adult, is not an uncommon cause of intestinal toxæmia. This condition may exist for a long time without being marked by distinct attacks of acute appendicitis. In these cases the symptoms of intestinal toxæmia, such as headache and general nervous irritability, may be associated with a mucous colitis, tenderness in the region of the appendix, an occasional colic, and ofttimes a sensation of weight and discomfort in that region on active exercise.

ACUTE INTESTINAL TOXÆMIA

Acute intestinal toxæmia is more common in the infant and young child than it is in the adult. This greater susceptibility to the acute forms of intestinal toxæmia is probably due to a number of causes, the most important of which is the great irritability and the immaturity of the nervous system of the child. This instability of the nervous system of the child makes it possible for slight disturbing factors to produce maximum results. A small quantity of poison absorbed from the intestinal canal may, through its action on the susceptible nervous system, produce high fever, convulsions, and other pronounced nervous symptoms, while the same quantity of

poison might be easily resisted by the stable nervous system of the adult.

It is also true that severe albuminous fermentations capable of producing virulent toxins are more common in the child than they are in the adult. This may be due to the fact that the hydrochloric acid function of the stomach is not so well developed in the young child as in the adult, and therefore not capable of exercising the same control in preventing intestinal fermentations. It may also be possible that the filtering function of the liver, which is at all ages one of nature's safeguards against intestinal toxæmia, is not so well carried out in the child as it is in the adult. But whatever may be the explanation, acute intestinal toxæmia is much more common in the young child than in the adult.

While our knowledge of the poisons which produce acute intestinal toxæmia is very unsatisfactory, yet there is no doubt that such a toxæmia exists, and that it is one of the most important factors in producing nervous symptoms in the young child, and a somewhat less important factor in producing nervous symptoms in the older child and the adult. Acute intestinal toxæmia occurs so commonly in the infant and young child that we are justified in suspecting this cause, where no other apparent cause presents, as a factor in producing sudden rises of temperature and acute convulsive disorders.

The nervous symptoms which result from acute intestinal toxæmia may vary in severity from a slight fever, with exaggerated reflexes, to a high fever and convulsive disorder so severe as to produce death. Every physician recognizes the importance of acute intestinal toxæmia as

an etiological factor in the production of dangerous nervous symptoms in the infant and child, and everyone has seen these convulsive disorders, followed by high fever and unconsciousness, quickly relieved by cathartic medication, and cured by intestinal antiseptics and diet.

CHRONIC INTESTINAL TOXÆMIA

Familiar as we are with acute intestinal intoxications, we are slow to recognize the importance of chronic intestinal intoxications which are produced no doubt by the same intestinal toxins, absorbed in smaller quantities and over a longer period of time.

As the child gets older and the nervous system develops and acquires greater powers of resistance against these toxins, then the acute intoxications become less, and the chronic intoxications more important, so that in the older child these severe forms of acute intestinal toxæmia are infrequent as compared with chronic intestinal intoxications.

It is the chronic form of intestinal intoxication to which I wish to call special attention, because its great importance as an etiological factor in producing nervous symptoms in children is commonly overlooked.

Chronic intestinal toxæmia may be associated with diarrhœa, but is not uncommonly associated with constipation. It must be remembered that constipation may exist even when the child has a movement from the bowels every day, or even two or three movements. These movements may be fragmentary and therefore incomplete, or they may be hard and dry, showing that

they have remained in the intestinal canal twenty-four or more hours longer than they should.

In order to prevent hyper-fermentation, and increased absorption of intestinal toxins, the food materials must not be retarded in their passage through the intestine, and when ejected should be moist and have the appearance and form of the normal intestinal evacuation.

So important, do I believe, is the rôle played by chronic intestinal toxæmia in the production of neurotic diseases in the child that I invariably begin the treatment of nervous diseases, whatever the symptom group may be, by a careful investigation of the intestinal canal, and throughout the treatment of these cases I give careful attention to any abnormalities of digestion.

The child should be fed upon food carefully adjusted to its digestive capacity, and there should be no retardation of food stuffs in their passage through the intestinal canal.

Chronic intestinal toxæmia is probably a factor in the production of a large group of nervous symptoms, and may, as I believe, aggravate the symptoms of certain neuroses which are produced by etiological factors entirely foreign to the intestinal canal. Among the symptoms which may be produced or exaggerated by intestinal toxæmia may be mentioned malnutrition, anæmia, headache, general malaise, fever, heightened reflexes, convulsive disorders, restlessness at night, night-terror, general nervous irritability, hysterical and neurasthenic symptoms, incontinence of urine, brachicardia, hyperesthesia, paresthesia, nervous anxiety, psychoses, and, in rare cases, a syndrome resembling meningitis.

The following case, while somewhat unusual in the character of the nervous symptoms which it presents, may be taken as a good example of neurotic disease produced by intestinal toxæmia.

SEVERE NERVOUS SYMPTOMS PRODUCED BY INTESTINAL TOXÆMIA

Boy, age five years, has never been strong, has had stomach and intestinal trouble very frequently during his life. At the present time he is thin, anæmic, and has the appearance of being malnourished. During the last year he has been very nervous, and this nervousness has recently very greatly increased, so that at the present time he is irritable, cries on slight provocation, is very restless at night, and has certain peculiar nervous attacks, which led his mother to seek medical advice. These attacks come on suddenly with dizziness; the boy falls to the ground, and his mother thinks he does not lose consciousness, and is sure he has no convulsive movements. Some minutes elapse before the boy is able to regain his feet. These attacks are followed immediately by severe headache and more or less nausea. Following these attacks, the boy is put to bed, and soon falls into a profound sleep, which may last some hours. From this sleep he awakes almost or quite as well as before the attack.

He has had in all seven attacks during the last year, and three of them have occurred within the last two months. These attacks, which seem to occupy the borderland between migraine and epilepsy, have not only increased in frequency, but also in severity.

The family history throws no light on the etiology of these attacks. The father is phlegmatic, the mother is somewhat nervous, but there is no history of neurotic disease in the family.

A careful examination failed to show any reflex factors which might be etiologically related to this neurosis.

The mother says that for a long time the boy has not been normal in his intestinal canal. Diarrhœa has alternated with constipation. There has been much flatulency. The boy has a fitful appetite, and craves food he should not eat. He has been fed almost anything, because "he ate so little" the mother thought "it would not hurt him."

This history strongly suggested the probability that the nervous symptoms might be due to intestinal toxæmia.

An examination of the urine, which was high colored, and had a specific gravity of 1023, showed no albumin, no sugar, but a marked excess of indican.

Treatment.—A dose of castor-oil, followed by a diet carefully regulated to suit the patient's digestive capacity; active life in the open air; a diatase and iron preparation after meals. Under this treatment the boy's general health gradually improved. He gained slowly in strength and weight. His general nervous irritability, which was greatly improved from the beginning of the treatment, gradually disappeared. He never had any of his peculiar attacks after the treatment was begun. Three months later he was discharged, well.

Intestinal toxæmia, acute and chronic, is not an uncommon complication of other diseases, such as typhoid

fever, malaria, tuberculosis, and chronic diseases of the gastro-intestinal canal.

As a complication this condition is probably met with more commonly in the convalescence from typhoid fever than in any other disease. The physician's unjustifiable fear of cathartics in this disease is ofttimes prolonged into the period of convalescence, and as a result fœcal accumulations occur, which result in an intestinal toxæmia which prolongs the period of convalescence many weeks.

The urine furnishes valuable evidence of the existence of intestinal toxæmia, and ofttimes our attention is called to this condition by the presence of an excess of indican, and the ethereal sulphates in the urine. The presence of these substances in the urine is sufficient reason to suspect a hyper-fermentation of albuminous food stuffs in the intestinal canal, and also usually means the retardation of these food stuffs in their passage through the canal, but the absence of indican does not rule out intestinal toxæmia, since we may have severe forms of intestinal toxæmia in which indol is not an etiological factor.

The etiological importance of the vegetable organisms in producing intestinal toxæmia must not cause us to altogether overlook the possible rôle which animal parasites may play in these pathological processes. In the present state of our knowledge, however, it is not possible for us to make any positive statements as to the exact rôle which intestinal worms play in producing gastro-intestinal toxæmia. Lynch in Grancher and Comby's "Maladies de l'Enfance" says that intestinal worms may be responsible for a large group of toxic symptoms, such

as urticaria, ringing in the ears, syncope, vertigo, palpitation of the heart, insomnia, mental anxiety, hypochondria, general nervous irritability, fever, delirium, and convulsions.

While this group of severe toxic symptoms may be associated with intestinal worms, it does not necessarily follow that they are produced by the absorption of poisons which have been excreted by them. Lynch himself points out the possibility of these symptoms being produced in part, at least, by the secondary fermentations which the presence of these parasites excite in the intestinal canal, and notes, also, the possibility of severe symptoms being produced, reflexly and mechanically, by their action.

Leukart observed that the ascaris lumbricoides excreted a poison capable of producing toxic symptoms. Huber, von Linstow, Chanson, and Raillet state that this same worm contains an irritating substance capable of producing a toxic effect on the human organism, and a number of other French authors state that this worm excretes an irritating and convulsive substance. Cao, on the other hand, after carefully investigating the subject, concludes that the evidence at the present time does not justify us in attributing toxic symptoms to the action of poisons excreted by intestinal worms.

While there is considerable difference of opinion as to the etiological relationship which exists between animal parasites and intestinal toxins, it seems to be rather generally conceded that these parasites may either directly or indirectly produce intestinal intoxication. In this connection the following case is of interest.

SEVERE GENERAL CONVULSIONS PRODUCED BY NEMATODES IN THE INTESTINAL CANAL

I was called in consultation to see a girl, twelve years of age, who had been having convulsions for several hours. She was a strong, healthy German, with no tendency to neurotic disease. After a few days of slight indisposition, on the part of the intestinal canal, she suddenly had a severe convulsion. On my arrival I found that the physician in attendance had been working with her for some hours, and that during this time she had had a number of severe convulsive seizures. In the intervals between the convulsions she was unconscious and had considerable muscular rigidity.

After controlling the convulsions with chloroform, the urine, drawn with a catheter, was found to be normal. Sulphate of magnesia was given by the mouth, and by rectal injections. Some hours later a large ball of round worms (ascaris lumbricoides) was passed with a large amount of fœcal matter, and very soon thereafter the patient was restored to consciousness, and the next day was well.

There can be no doubt that this was a case of intestinal toxæmia. The child's age, previous good health, and stable nervous system speak against the reflex origin of these convulsions; and I may further state that while it is impossible for one to exclude absolutely other causes of intestinal intoxication, the discharges from the intestinal canal were not of such a character as to indicate that the poisons were produced by the fermentation of the intestinal contents.

CHAPTER V

AUTO-INTOXICATIONS

Auto-Intoxication, which, strictly speaking, is due to the presence of autogenetic toxins in the blood, is one of the most important, and one of the least understood, of all the causes of neurotic disease both in adults and in children.

The close relationship of auto-toxins to certain nervous disorders is accepted by almost all physicians as axiomatic, notwithstanding the fact that we have very little accurate knowledge of these poisons. Such nervous syndromes as occur in diabetes, uræmia, and gout and certain forms of neurasthenia, hypochondriasis, and hysteria are among the nervous disorders which are at the present time believed to be etiologically related to auto-intoxication.

The poisons of this class may have their origin in any of the three following ways:

First.—They may be formed by the various cells of the body to serve some physiological purpose, and may become toxic only when accumulated in abnormal quantities in the blood and tissues.

Second.—They may be substances which are abnormally formed through the perverted functional activity of the cells. Incomplete products of cell metamorphosis belong to this class of poisons.

Third.—They may be produced by retrogade tissue

metamorphosis incident to the death and disintegration of cells. Poisons of this class become pathological factors when they are formed in excess, or when there is defective elimination, or failure in the physiological processes which convert them into harmless bodies.

It is evident that the body may be protected up to a certain point against these poisons by the increased activity of such excretory organs as the intestinal canal, the kidneys, the sweat glands, the liver, and the lungs, and again it is evident that the functional incapacity of these organs may be potent factors in precipitating an attack of auto-intoxication. One may therefore understand how in the pathology of auto-intoxications defective elimination and neutralization of poisons may be almost as important as increased production.

The early experiments of Bouchard and others to discover by an examination of normal urine the auto-toxins responsible for nervous symptoms were the first to place this subject on a scientific basis. These researches gave a wonderful impetus to the systematic study of auto-toxins, and led to the recognition of these bodies as distinct factors in the production of disease.

THYROID-INTOXICATION

The thyroid gland is one of the organs which has its greatest functional activity during the early years of the life of the animal. This gland furnishes a secretion which is absolutely necessary to the normal body chemistry by which normal growth and development is carried on. This function of the thyroid is so nicely adjusted

to the needs of the organism over whose chemistry it exercises such a marvelous influence that in the vast majority of instances it furnishes a secretion both in quantity and quality nicely adjusted to the purposes it is to serve.

In a few instances, however, this gland is congenitally absent, and in others its functional capacity is diminished or destroyed, producing cretanism and myxœdema. The absence of the thyroid secretions in these conditions, by perverting the normal chemistry of the body, no doubt leads indirectly to auto-intoxications.

On the other hand, from an increased functional capacity of the thyroid gland we may have an excess of its secretions poured into the body-media, producing a well-known group of nervous symptoms. This symptom group may be produced experimentally in man by feeding excessive quantities of thyroid, or it may be observed in exophthalmic goiter, a disease the symptoms of which are now recognized by pathologists as being in part at least produced by thyroid-intoxication. Among these nervous symptoms may be mentioned headache, general nervous irritability, and rapid heart action. Every clinician has observed this symptom group to develop from thyroid feeding.

In the infant and child we know practically nothing of thyroid-intoxications, yet it is my belief that such intoxications exist, and that they are not an infrequent cause of general nervous irritability and rapid heart action in the child. We know that thyroid secretions increase the excitability and stimulate the growth and functional development of the nervous system. It seems

very probable, therefore, that, since childhood is the period of life when great thyroid activity is an important factor in producing the rapid growth and functional development of the nervous system, it may also be a factor in increasing the irritability of the nervous system in the young child. It may, and undoubtedly does, happen that the amount of thyroid secretion varies with the individual child, and that when this secretion is excessive it may be a factor in producing the too rapid growth and development of the nervous system which not uncommonly occurs in children, and which, when it does occur, is recognized by the physicians as a factor in producing neurotic disease. It is not improbable, therefore, that rapid body growth and rapid functional development of the nervous system, associated with nervous irritability, mental precocity, tachicardia, headache, and other nervous symptoms, may be produced by an excessive activity of the thyroid gland. This hypothesis may explain one of the most common and one of the most obscure syndromes of childhood; viz., the rapid, irregular heart and general nervous irritability that accompanies the rapid growth of children.

It is a well-known fact that thyroid feeding will increase the output in the urine of purin bodies, and will aggravate the arthritic and certain other symptoms in the gouty patient. This may lead to the inquiry whether or not the symptoms of auto-intoxication produced by excessive quantities of the thyroid secretion is not in part due to an increase in the products of retrograde tissue metamorphosis which occur as a result of the stimulating effects of thyroid secretion on the chemical pro-

cesses incident to the rapid growth and functional development of cells. The exact *modus operandi* of thyroid-intoxication is yet to be determined.

It may also be worthy of inquiry whether or not the increased activity of the thyroid gland which occurs at the menstrual period, especially in girls and young women, may not be a factor in producing the headache, rapid heart action, and general nervous excitability which occur so commonly at these periods.

BILIARY TOXÆMIA

Biliary toxæmia is a form of auto-intoxication resulting from the absorption of bile. Bouchard has shown that the biliary salts and the biliary coloring matters are poisons, the latter being much the more poisonous. These substances when injected into the veins of rabbits produce convulsions and death. From his experiments on rabbits Bouchard estimates that man forms in eight hours enough biliary poison to kill himself. These experiments, however, are not fully corroborated by clinical experience, since the absorption of considerable quantities of bile may go on over a long period of time, producing well-marked jaundice, without producing very severe symptoms of intoxication. The symptoms commonly produced by biliary toxæmia are languor, depression of spirits, headache, slow heart action, and itching of the skin, and this symptom group may continue with variable intensity for a long period of time without producing more acute or dangerous symptoms. In the catarrhal jaundice of children we have more or less fever,

nervous irritability, and headache—symptoms which in part, at least, may be due to the biliary toxæmia. Children suffering from more severe forms of jaundice may be drowsy, irritable, and may even have convulsions, followed by coma and death.

It is impossible to say what part biliary toxæmia plays in producing these severe cerebral symptoms, which sometimes develop very suddenly in icteric patients. It has been suggested that they may be due to an acid intoxication, because of the resemblance of this symptom group to diabetic coma. When the common bile duct is ligated in rabbits it is noted that the alkalinity of the blood gradually diminishes day by day until the death of the animal. But the acid intoxication in these experiments is not sufficient to justify the belief that acid intoxications are the sole cause of the severe nervous symptoms above noted. Other factors not yet discovered probably play a rôle in producing these severe toxic symptoms.

Since the above was written a paper (not yet published) was read before the Association of American Physicians by Meltzer and Salant on "The Toxicity of Bile." They found that bile contained two elements: first, a depressing or coma-producing substance; and, second, an exciting or tetanizing substance. These active principles of bile are antagonistic and normally exist in proper proportions to neutralize each other. This theory of Meltzer is offered in explanation of the fact that the blood may contain considerable quantities of bile without the existence of marked toxæmia. In such conditions the exciting and the depressing substances in the bile are so nicely balanced as to neutralize each other and

thus produce no toxic symptoms. On the other hand, such an individual may be suddenly and violently poisoned by one or the other of the toxic principals if for any reason this equilibrium be destroyed.

ACID INTOXICATIONS

Perhaps the most tangible results of recent studies in auto-intoxications relate to acid intoxications. By acid intoxications is meant an increase of normal or abnormal acids in the body media. This increase in acids may result from their increased absorption from the stomach and intestinal canal, from their increased formation by the cellular elements of the body, and from the diminished combustion of acids.

The inorganic acids,—hydrochloric acid, sulphuric acid, and phosphoric acid,—and the organic acids,—the volatile fatty acids, sarcolactic acid, β-oxybutyric acid, diacetic acid, oxalic acid, uric acid, and carbonic acid,—may appear in excess in the body media and contribute to acid intoxications.

It may be suggested that these acids act in producing pathological conditions in any one of four ways.

First.—The acids themselves may be directly toxic.

Second.—By union with the calcium, potassium, sodium, and magnesium of the blood and tissues they may, by the removal of these bases, produce symptoms directly due to the diminished quantity of one or more of these alkalies in the blood and tissues. These alkalies in definite quantities in the body media are known to be absolutely necessary to normal physiological processes,

and any diminution of these quantities is fraught with serious results. The alkaline content of the body media may also be reduced by a minimum intake of alkaline food.

Third.—The excess of acids in the body may be combined with more or less poisonous bases, such as ammonium, which increase their toxicity, and the resulting intoxication may be partly due to the presence of large quantities of ammonium and other poisonous bases which are carried in this way through the circulating media on their way to excretion.

I called attention, in a paper on "The Comparative Toxicity of Ammonium Compounds," [1] to the fact that in acid intoxications the degree of toxicity may in part depend upon the base with which the acid is united, and since this base is commonly ammonium, and since ammonium salts of the various acids are much more toxic than the acid themselves or the sodium or potassium salts of these acids, it is not impossible that the symptoms of an acid intoxication may be partly due to the ammonium ion rather than to the acid ion. This seems the more probable as under normal conditions there is a very small quantity of ammonium circulating in the blood; but in acid intoxications the ammonium, which under normal conditions is combined with CO_2, and is converted by the liver into urea, is diverted from this, its normal channel, to form ammonium salts of the acids, and in this form is carried through the blood and tissues in enormous quantities to be excreted by the kidneys.

[1] "The Comparative Toxicity of Ammonium Compounds," by B. K. Rachford and W. H. Crane.—*Transactions of Association of American Physicians.* 1902.

Fourth.—Organic or mineral acid in the blood may, as C. A. Herter suggests, displace the diffusible carbon dioxide, and appropriate the ammonium and sodium with which it is united. In this way an accumulation of carbon dioxide may occur in the fluids and cells of the body which will interfere with oxidations essential to life, and occasion coma and death.

DIACETIC AND β-OXYBUTYRIC ACID INTOXICATIONS

The protective mechanism which guards the body against alkaline loss is so effective that the alkalinity of the blood remains almost constant under ordinary pathological conditions, even those which are characterized by mild acid intoxications. In certain severe acid intoxications, however, such as occur in the last stages of diabetes mellitus, it appears that the alkalies of the blood may be drawn upon to assist in the neutralization and elimination of the enormous quantities of β-oxybutyric and diacetic acids which are present in the body in this condition. In this severe acid intoxication, after all the available ammonia is used up by these acids, the alkaline bases of the blood and tissues are seized upon and are thus carried in combination with acids to the kidneys to be excreted. The intensity of the acid intoxications in the later stages of diabetes mellitus is shown by the fact that eight or ten grammes of ammonium (not to mention other alkaline bases) may be excreted in combination with these acids in twenty-four hours.

The symptoms which characterize severe acid intoxications are a peculiar dyspnœa, increased pulse rate,

lowering of the body temperature, decrease in the alkalescence of the blood, and increased excretion of ammonia. Later somnolence, coma, and death.

Many other milder forms of β-oxybutyric acid intoxications have been described by Von Jacksch and others. Von Jacksch found acetone, diacetic acid, and β-oxybutyric acid in excess in diseases accompanied by high fever, such as typhoid fever, scarlet fever, and pulmonary tuberculosis. Acetone and diacetic acid are also found in considerable quantities in the later stages of carcinoma. And they also commonly occur in severe malnutritions, gastro-intestinal diseases, migraine, recurrent vomiting, and toxic epilepsy. What rôle acid intoxications play in producing systemic toxæmias in the above-named diseases has been determined.

Of β-oxybutyric acid Von Noorden says: "Owing to the fact that this acid is so closely related chemically to acetone and diacetic acid, one is justified in suspecting its presence in the urine whenever these two bodies are excreted in considerable quantities. As a matter of fact, one always succeeds in finding the acid under these circumstances." He also believes that "all acid intoxications produced by the presence in the tissues of acetone, diacetic, and β-oxybutyric acids are due largely, if not wholly, to an insufficient intake of carbohydrate food, or to some fault in the carbohydrate metabolism. Feeding carbohydrates and cutting down the quantities of fats and albumens will always diminish and sometimes entirely overcome this form of acid intoxication."

The origin of the acetone bodies is not at all clear, but in the present state of our knowledge it may be assumed

that they are synthetic products derived from the breaking down of the carbohydrate portion of proteid molecules, and from the disintegration of fat molecules, and that the disintegration of fat and proteid molecules which results in the formation of acetone, diacetic, and β-oxybutyric acids is influenced by the absence or scarcity of carbohydrate molecules.

Clinical and laboratory experiments have apparently demonstrated that an excess of acetone bodies in the blood is due to some defect in the oxidative processes not altogether dependent upon a deficiency in the respiratory intake of oxygen. Von Noorden believes that the absence of carbohydrates from the food influences unfavorably these oxidative processes, and leads to the formation of the acetone bodies. He is led to this opinion by the fact that this form of acid intoxication cannot occur when carbohydrates are taken and assimilated in proper quantities.

It is my belief that the metabolic processes, the disturbance of which is responsible for diacetic and β-oxybutyric acid intoxications, are largely carried on by the liver. The functional incapacity of the liver which occurs as a secondary condition in so many nutritional disorders may so disturb carbohydrate metabolism and interfere with oxidation processes that this form of acid intoxication may be produced. This theory is strongly supported by the occurrence of secondary acid intoxications in such diseases as migraine and recurrent vomiting, as well as by the liver findings in cases which have had terminal acid intoxications. Von Noorden says: "Magnus-Levy, however, discovered that β-oxybutyric

acid was a product of the autolysis of the liver, and his observation may, therefore, possibly be considered an argument in favor of the view that the liver has something to do with the formation of acetone bodies."

It is a well-established fact that carbohydrate starvation will produce an excess of the acetone bodies, and it occurs to me that this carbohydrate starvation may be due either to a deficient intake of carbohydrate food or to a disturbance of the liver functions which interferes with the intake and output of carbohydrates by the liver.

This functional incapacity of the liver which may thus be a factor in producing diacetic and β-oxybutyric acid intoxications also throws into the circulation large quantities of ammonia which, under normal conditions, would be manufactured into urea. This ammonia combines with and saturates these acids, thus attempting to protect the body against the threatened acid intoxications.

CARBONIC ACID INTOXICATION

Carbonic acid poisoning is an auto-intoxication which may occur in certain heart and lung diseases and severe anæmias and may complicate other severe acid intoxications.

CO_2, which is one of the products of retrograde tissue metomorphosis, is formed in the tissues and conveyed by the blood plasma and corpuscles, to be excreted largely by the lungs. Poisoning from CO_2 may occur in three ways:

First.—By a failure on the part of the blood to carry the CO_2 from the tissue to the lungs for excretion. This

may occur in heart diseases, anæmias, or in any disease of the circulatory system which enfeebles the capillary or general systemic circulation. This failure of the blood to remove CO_2 from the tissues results in its accumulation in such quantities in the venous blood that an auto-intoxication results.

Second.—In diacetic and β-oxybutyric acid intoxications, as C. A. Herter suggests, the bases which ordinarily combine with CO_2 are in great part exhausted by other acids, thus allowing the CO_2 to circulate as such in the blood, and in that way poisoning and irritating the higher nerve centers.

Third.—Diseases of the lungs, by interfering with the elimination of CO_2, may also result in its accumulation in the blood and tissues.

Among the symptoms ordinarily ascribed to CO_2 poisoning are the following: Dyspnœa, mental dullness, stupor, unconsciousness, coma, and death. It should also be noted that CO_2 in the form of a salt may under certain pathological conditions contribute to the toxicity of the body media. For example, the ammonia of the tissues unites with CO_2 to form a carbonate of ammonium, and this salt is in turn converted into urea. A failure of the urea-forming function of the liver may therefore throw into the general circulation a considerable quantity of carbonate of ammonium, which is passed through the blood to be excreted by the kidneys. Since carbonate of ammonium possesses a considerable degree of toxicity, it may be possible that it contributes to the systemic intoxications which occur as a result of the functional inactivity of the liver.

OXALIC ACID INTOXICATION

The medical profession is by no means agreed that oxalic acid is ever a factor in producing auto-intoxication. It is agreed, however, that oxalic acid is poisonous, and that the ammonium salt of this acid, in which form it is ordinarily excreted, is a very poisonous salt. Ammonium oxalate is in fact four times as poisonous as the ordinary ammonium salts, such as chloride. It is the oxalate ion rather than the ammonium ion which gives toxicity to this salt, and in experimental oxalic acid poisoning it is probable that this salt plays a very important rôle.

For a long time the medical profession associated a certain rather definite symptom group with the appearance of an excess of oxalates in the urine. Patients of this type were found to be irritable, to suffer from headache, digestive disturbances, and insomnia, and were as a rule melancholic. But from the fact that an excess of oxalic acid might occur in the urine without producing any of the above-named symptoms, it was thought that in those cases in which nervous symptoms coincided with an excess of oxalates in the urine, the symptom group was produced by other toxins, probably intestinal in origin, which were formed at the same time.

It seems to me, however, rather illogical, in the light of the uncertain chemical knowledge which we have of the conditions underlying the formation of oxalic acid in the intestinal canal and in the body tissues, and of the form in which it traverses the body media, to assert that oxalic acid, an excess of which in the urine is not uncommonly

associated with the constitutional symptoms above named, has no causative relation whatever to these symptoms. The relationship which oxalic acid bears to this symptom group must be determined by further investigations.

URIC ACID INTOXICATIONS

In addition to the above-named syndromes associated with acid intoxications, there are many other symptom groups which are believed to be etiologically related to acid intoxications: such, for example, as urticaria, recurrent vomiting, migraine, certain types of epilepsy, and other of the nervous syndromes associated with the uric acid diathesis.

Perhaps no opinion not capable of absolute demonstration is more firmly fixed in the medical mind than that the nervous symptoms associated with gout, and the so-called uric acid diathesis, are due to auto-toxins which are closely related in their formation and chemic properties to uric acid and its compounds. A discussion of auto-intoxication, therefore, must include the possible rôle which the purin bodies may play in the production of these symptoms. These bodies are uric acid, adenin, hypoxanthin, xanthin, guanin, epiguanin, paraxanthin, heteroxanthin, episarkin, and carnin, and the trend of thought at the present time is that all of these are formed by the disintegration of exogenous and endogenous nucleins. The five first-named are known to be derived from this source.

A most remarkable change in uric acid theories followed Horbaczewski's discovery that uric acid could be

formed by heating spleen pulp in the presence of fresh blood or peroxide of hydrogen. In these experiments the uric acid was formed from the disintegration of the splenic leucocytes—the fresh blood and peroxide of hydrogen acting as oxidizing agents. He observed that oxidizing agents were necessary to the formation of uric acid from nucleins, and that, when nucleins were broken up by heat in the absence of oxidizing agents, the xanthin bases were formed.

Previous to these observations Kossel and Salomon had produced adenin and hypoxanthin from the degeneration of nucleins, and a number of observers had noted the excessive excretion of the purin bodies in leucocythemia and other diseases attended by leucocytosis. The relationship that exists between leucocytosis and an increased excretion of the purin bodies is not definitely understood. Leucocytosis does not, as Küknau has shown, always mean an increased formation of purin bodies—the death and disintegration of the nucleins of these cells must precede the formation of the purin bodies. They have their origin, therefore, in the death and not in the physiological life of these cells. Excessive cell destruction followed by excessive nuclein disintegration always results in excessive formation of the purin bodies. Leucocytosis accompanied by an excessive destruction of nuclein will, as a rule, produce an excess of uric acid; but this, however, is not always the case. In the leucocytosis of the severe anæmias it is a notable fact that the xanthin bases are increased and uric acid is decreased. The explanation for the excretion of xanthins and the diminished excretion of uric acid in severe anæmic con-

ditions associated with leucocytosis, is, as Küknau has suggested, to be found in the experiments of Horbaczewski above recorded. The nuclein catabolism in anæmic conditions takes place under conditions of deficient oxidation, and as a result xanthin bodies are formed instead of uric acid. It seems important, therefore, in the present state of our knowledge, to lay stress on the following facts:

First.—Uric acid is formed when nuclein is broken down in the presence of oxygen.

Second.—Xanthin bases are formed when nuclein is broken down in the absence of oxygen.

Third.—Xanthin bases are not oxidized into uric acid, and are not, therefore, intermediate bodies in its formation.

Fourth.—Both uric acid and the xanthin bases may be oxidized into urea. The liver plays a part in this process.

When the statement is made that uric acid and the xanthin bases owe their presence in the human body largely to the catabolism of nucleins, it must be remembered that not only the leucocytes, but all the cellular elements of the body, may contribute, through their death and disintegration, to the formation of these bodies. It is evident that as a result of normal cellular destruction and nuclein disintegration a certain amount of the alloxuric bodies must be daily formed as a normal physiological retrograde process; and it must also be remembered, as Umber has demonstrated, that the alloxuric bodies formed in the body are derived in part from the nucleo proteids of the food. Burian's and Shur's recent investigations indicate that about forty to sixty per cent.

of the total purin content of the urine is derived from this source. It may be roughly estimated, therefore, that under normal conditions about one-half of the purin content of the blood is exogenous and the other half endogenous. It is evident that this proportion between endogenous and exogenous purins may vary greatly in different individuals, and also from time to time in the same individual.

Under certain pathological conditions accompanied by an increased cell destruction and a nuclein disintegration the endogenous purins may be greatly increased.

The increase of the nuclein-content in the food may also greatly increase the exogenous purin-content of the blood, and the present tendency is to attach more pathological importance to the exogenous than to the endogenous purins. The excessive intake of nucleo proteids in the food is therefore looked upon as probably the most important factor in producing an excess of purins in the blood.

In studying the variation of the purin-content of the blood, the functional capacity of the liver must also be considered. This organ under normal physiological conditions not only converts a large proportion of the endogenous purins into urea, but it also stands guard between the purin-content of the intestinal canal and the general circulation.

These bodies derived from the nucleo proteids of the food are filtered through the liver, and for the most part converted into urea before they reach the general circulation. The liver may in this manner for a long time protect the body against an excessive intake of exoge-

nous, or intestinal purins. Auto-intoxications from these purins may in this manner be prevented just so long as the filtering and urea-forming function of the liver can hold out under the increased strain of this overwork. But in this crisis the liver often fails, and by reason of its temporary functional incompetency the antecedents of urea, namely, the ammonium compounds and both the exogenous and endogenous purins, are thrown into the circulation, and, the excretory organs not being able to excrete them rapidly enough, an acute auto-intoxication results. Under these conditions the urine shows a decrease of urea and an increase of ammonia and the purin bodies.

It will be observed that in the above argument the temporary functional incompetency of the liver plays a most important rôle in precipitating attacks of acute systemic intoxication. In the chronic forms of auto-intoxications, also, it is probable that the liver, either from overwork or from hereditary causes, is in a state of more or less chronic incompetency, and that at all times it permits a certain excess of exogenous purins to filter through. Acute attacks in these chronic cases being also caused by the occasional complete incompetency of the liver, and after a few hours or days of rest the liver again resumes its function, complete or incomplete as the case may be, and the acute attack is over.

It is believed that the purin-content of the blood is under normal conditions in organic combination, and that the dissociation is brought about by the kidney, and the purin bodies excreted as such. We do not know under what conditions this dissociation may occur in the blood,

or under what conditions the urates are deposited in the tissues, especially about the small joints. Many pathologists at the present time believe that these deposits are secondary to necrotic changes in the part which have been produced by auto-toxins; that is to say, the uratic deposits in the tissues are secondary, and not primary, pathological changes.

THEORY OF ACTION OF THE PURIN BODIES

Uric acid and its compounds were for a long time considered to be the all-important *materies morbi* of the nervous syndromes grouped under the general term lithæmia. This view has, however, been so modified in recent years that at the present time it is believed that uric acid as compared with other purin bodies plays an unimportant rôle in the production of these symptoms.

The hypothesis that the xanthin bodies play an important part in producing the nervous symptoms associated with the uric acid diathesis is, as we shall see, supported by considerable evidence. This hypothesis naturally presented itself when it was demonstrated that uric acid and its compounds were not sufficiently toxic to account for the nervous symptoms of the uric acid diathesis, and, working upon this hypothesis, experimenters have demonstrated that a number of the xanthin bodies are sufficiently toxic to place them under suspicion as being, at least partly, responsible for the toxic symptoms with which they are associated.

Gaucher demonstrated that hypoxanthin and xanthin when repeatedly injected into the bodies of animals would

produce degenerative changes in the excreting cells of the parenchyma of the kidney.

Crofton, who recently confirmed these findings, produced in guinea pigs by the daily injection, for six or eight weeks, of five per cent. solutions of xanthin and hypoxanthin, "a granular degeneration of the epithelial cells lining the tubuli contorti, and a proliferation of the endotheleum of the intertubular capillaries was found. The picture corresponds with the nephrite epitheliale of Gaucher."

Kolish also produced parenchymatous degeneration of the kidneys of rabbits and guinea pigs by injecting small quantities of hypoxanthin for periods of one or two months.

Hager says that a necrosis of the joint tissues is brought about in gout by the irritating action of certain of the purin bodies, particularly adenin, which he says is the most harmful of these bodies, and produces necrosis of tissue cells; and Kolish also believes that the xanthin bodies, by producing disease of the kidneys, prepare the way for the deposit of uric acid.

Minkowski found that 0.5 adenin administered daily to dogs produced malaise, vomiting, and, after five or six days, death. Before death the urine of these animals contained albumen, casts, and epithelial cells, and after death the kidneys showed inflammatory changes and uric acid deposits, and he further observed that these deposits occurred quite independently of the amount of uric acid in the urine, or of the concentration or alkalinity of the urine.

Mandel found that the injection of four milligrams of

xanthin produced an elevation of temperature in a monkey. He also demonstrated that in aseptic fevers there is a "distinct relation between the rise of temperature and the appearance of certain incomplete products of cell oxidation, as shown by the excretion of the purin bodies." He concludes that the purin bodies are important factors in the production of febrile temperatures.

These experiments are very suggestive when one considers the close relationship that exists between lithæmia, arterio-sclerosis, and kidney disease in later life. I have been much impressed, by careful clinical observations extending over a number of years, with the fact that lithæmia is one of the most important etiological factors in the production of arterio-sclerosis. One can well imagine that this condition of the arteries might result from their long-continued irritation by reason of the presence of an excess of the purin bodies in the blood. Hypoxanthin, xanthin, and adenin, therefore, which have been demonstrated to have an irritating effect upon the kidneys, may not only be factors in the acute auto-intoxications of lithæmia, but may also have something to do with the slow arterial changes which take place in these patients, and in that way explain why the sick headaches and bilious attacks of former years are in later life accompanied by transient albuminurias, and why these patients often succumb in later life to cerebral hemorrhage, or uremia.

In addition to the irritating action of xanthin on the kidney, it should also be stated that according to Filehne it has a toxic action on the nervous system, producing

in the frog a decided muscular rigor and paralysis of the spinal cord.

Paraxanthin is the most poisonous of the alloxuric bodies. Its physiological action has been studied by Salomon, who observed that it produced dyspnœa and a rigor-mortis-like contraction of muscles, followed, in the mouse and guinea pig, by convulsions and death. In the mouse these symptoms were preceded by reflex excitability.

Heteroxanthin is also poisonous, and produces, according to Krüger and Salomon, the same group of symptoms as paraxanthin. Heteroxanthin possesses, however, only one-third of the toxicity of paraxanthin.

It is plain from the foregoing *résumé* that the xanthin bodies are capable of producing disease if found in excess in the body media. It is important to know, therefore, what pathological conditions are associated with an excess of the xanthin bodies.

Some years ago the author advanced the theory that the xanthin bodies are very important factors in producing the auto-intoxications grouped under the term lithæmia, and asserted the belief that these bodies are etiologically related to migraine, migrainous epilepsy, and recurrent lithæmic vomiting. He found an excess of the xanthin bodies in the urines of patients suffering from these conditions, and also found that the "final fluids" containing these bodies, eliminated from such urines, were poisonous to mice and guinea pigs. But he failed to find an excess of these bodies in the urines of these same patients in the intervals between the attacks.

Crofton found the xanthin bodies in excess in a large

number of cases belonging to the "uratic diathesis," including gout.

Kolish found in gout that uric acid was diminished and the xanthin bodies increased in quantity.

A large number of observers agree that the blood in gout contains a considerable excess of uric acid, and that there is in the condition a deficient elimination of the purin bodies.

The xanthin bases are spoken of as leucomains, and the auto-intoxication which is supposed to be produced by them has been called leucomain poisoning. It is my belief that these leucomains are factors in producing the forms of auto-intoxications which are described under the term lithæmia. No one, however, in the present state of our knowledge can say that these bodies are the only important factors of this phase of auto-intoxication. Future investigations will add much to our knowledge of the poisons engaged in this process, and will also no doubt disprove many things which to-day are thought to be of value in the study of the pathology of lithæmia.

EXCRETION OF PURIN BODIES

The purin bodies are excreted by the kidneys, the skin, and the intestinal canal. This is a matter of much clinical importance, since one of the most important questions which presents itself to the physician in the treatment of lithæmia is, How can the elimination of poisons be increased?

The kidneys play the most important rôle in the excretion of these bodies. Uric acid and the xanthin

bodies are removed by the kidney cells from the blood into the urine, and their presence in excess in the urine means that immediately before their excretion they were in solution in excess in the blood. The kidney eliminates but does not manufacture or destroy these bodies. Severe diseases of the kidney may, therefore, cause their abnormal retention in the blood and other body media, and in this manner contribute to the nervous symptoms of acute and chronic Bright's disease. However this may be, it is certain that these bodies are excreted largely by the kidneys, and that we take advantage of this fact by stimulating these organs to increased work in the treatment of lithæmia.

Purin bodies are also excreted by the skin. The skin is much more active in this function during the hot summer months than during the winter. And this may be one explanation for the increased liability to lithæmic attacks during the winter months. The undoubted value of many of the thermo-alkaline springs in the treatment of lithæmic conditions depends partly upon the fact that the hot bath promotes the cutaneous elimination of the purin bodies. In the depurative treatment of lithæmic attacks the skin is often stimulated to excessive action to relieve the acute intoxication.

The gastro-intestinal canal is probably the most important channel through which the purin bodies may be eliminated when there is defective excretion through the kidneys; this fact is quite empirical, and is based on the accumulated testimony of the medical profession for many years. The value of laxative medication in these cases, however, is probably not entirely due to the fact

that in this way the absorption of exogenous or intestinal purins may be largely prevented, but it is also probably due to the fact that the intestinal canal, by proper cathartic medication, may be stimulated to the more rapid excretion of endogenous purins and other poisons circulating in the body media.

CHAPTER VI

CHRONIC SYSTEMIC BACTERIAL TOXÆMIAS

Bacterial toxins, formed in the blood and tissues of the body, play an important rôle in the etiology of the neuroses of childhood. We know from both laboratory and clinical observations that bacterial products can, by their direct action on nerve elements, produce most profound nervous symptoms.

Bacterial products are by far the most important of the exciting causes of fever and high temperature in children. The variations in temperature accompanying the acute infections are largely due to the action of bacterial products on the heat centers. Bodies capable of producing fever and variations in temperature may be formed by bacterial action, either within the blood and tissues of the animal or in wounds and cavities such as the intestinal canal. But wherever these bacterial products may be formed, they are capable of producing fever and variable temperatures by their direct action on nerve centers.

Centanni investigated seventeen species of bacteria and found in cultures of all of these, substances which when injected into animals caused fever with the following symptoms: high temperature, prostration, emaciation, and finally death. The toxins produced by the tetanus bacillus were shown, by Brieger, to be the cause of the

profound nervous symptoms of that disease. From pure cultures of this bacillus he isolated bacterial products capable of producing tonic and clonic muscular spasms.

Since these early investigations, poisonous bacterial products, which when injected into animals produced marked nervous symptoms, have been isolated from cultures of a large number of bacteria, including those of diphtheria, cholera, tuberculosis, typhoid fever, and septicæmia, so that clinicians have now very generally come to believe that the nervous symptoms of the acute microbic diseases are in great part due to the action of bacterial toxins on the nervous system.

The purpose of this chapter, however, is more especially to call attention to the relationship of certain nervous diseases to those blood intoxications which result from such chronic microbic diseases as tuberculosis, malaria, rheumatism, and syphilis. Not that the toxins formed in the body during the progress of acute microbic diseases are not all-important factors in the production of nervous symptoms, but that these poisons are not quite so intimately associated with the acute and chronic neuroses of childhood as are the manifold blood changes which the above-named chronic diseases produce.

Tuberculosis, of all the chronic microbic diseases, stands in closest etiological relationship to the neuroses of childhood.

The relationship of tuberculosis to certain nervous diseases, more especially idiocy and insanity, has been noted by many medical writers. Dr. Langdon Downs says: "I have made an analysis of the last one hundred

of my post-mortem records, at the Earlswood Asylum for Idiots, and I find no fewer than 62 per cent. were subjects of tubercular deposits."

Dr. Ireland says: "Perhaps two-thirds, or even more, of all idiots are of the scrofulous constitution, and fully two-thirds of them die of phthisis. The scrofulous diathesis, therefore, seems to favor, or at least to accompany, the production of idiocy."

Dr. Clouston says of tuberculosis and insanity: "It is very common to find these two diseases in different members of the same family, and there is every reason to suppose from the facts that an heredity towards phthisis may determine insanity, and vice versa. The percentage of death from tuberculosis is four times higher among the 'insane than among the general population of the same ages."

While many other writers might be cited who have called attention to the close clinical relationship which exists between tuberculosis and certain neuroses, yet I believe that the importance of this relationship to all the neuroses of childhood has not been fully recognized by writers upon this subject.

The following figures, taken from the records of my children's clinic, prove that tuberculosis is a very common etiological factor of the neuroses of childhood.

Of 407 cases of tuberculosis under fourteen years of age, 139 cases had, as a complication, one of the neuroses; that is to say, 34 per cent. of all cases of tuberculosis occurring in dispensary practice have some well-marked nervous affection.

Of the 139 neurotic cases, 30 had chorea, 23 had in-

continence of urine, and 80 had such other neuroses as persistent headache, epilepsy, night terrors, laryngismus stridulus, and hysteria.

It does not follow, of course, from the above statistics that tuberculosis was the sole factor in all of these cases. Some five or six of these children had in addition to their tuberculosis some rather vague evidences of chronic malaria, and a few of the cases of chorea, here included, gave slight evidence of rheumatism. Yet the tuberculosis was the predominating disease in every case, and I think the inference is just that it was the most important factor in bringing about the blood state which produced neurotic disease in these children.

If, instead of noting the cases of tuberculosis complicated with neurotic disease, we inquire into the percentage of cases of neurotic disease showing evidence of tuberculosis in dispensary practice, we find the figures not less convincing. Of 300 cases of neurotic disease, I found that between 35 and 40 per cent. presented more or less marked evidences of tuberculosis.

While I am quite assured that chronic tuberculosis in childhood is one of the most important of the etiological factors of the neuroses of childhood, I am not prepared to say that the toxins of tuberculosis are directly responsible for the nervous symptoms. It may be that the blood changes incident to the chronic anæmia of tuberculosis may, even apart from the specific action of the toxins upon the nervous centers, be etiologically related to these neuroses. The malnutrition of nerve elements must necessarily follow such profound blood changes.

If tuberculosis is so closely related to nervous diseases

in children, then this fact is of great importance, since it is, especially among the poor, the most common disease of childhood. The records of my children's clinic show 407 cases in 4400, that is to say, 10 per cent. of all cases treated were tuberculous. Of 10,000 cases treated in Steener's clinic 12 per cent. were tuberculous. And even this large percentage, according to Carmichael, is very much increased when children are crowded together under bad hygienic conditions and insufficiently fed. He concludes as follows: "On closer examination of 400 or 500 children in the House of Industry, it was found that more than one-half of these unhappy children had the characteristic signs of scrofula in their necks."

The prevalence of tuberculosis among the poor makes it a much more important etiological factor in producing nervous diseases among the children of this class than it is among the children of the rich. The reasons why tuberculosis is more prevalent among the poor than the rich are largely questions of heredity, bad hygienic conditions, and improper food.

Christopher lays much stress on improper food and bad hygiene as important factors in the production of neuroses, but there can be but little doubt that these factors exert their worst influences among tuberculous children, and in this way act as contributing factors to the development of neurotic disease.

Lymph node tuberculosis is the most common form of tuberculosis in childhood, and this is the form of the disease which produces the most profound blood changes. Children suffering from well-marked lymph node tuberculosis are profoundly anæmic; and this

profound anæmia must result in malnutrition of nerve elements.

It is evident, therefore, that the relationship of tuberculosis to the neuroses of childhood may be more or less complex. In part it may be due to the action of tuberculous toxins on the nerve elements. The profound blood changes accompanying this disease may, apart from the toxins, be strong contributing factors; and bad hygiene and improper food, which have contributed to the development and progress of the tuberculosis, may also be more or less indirectly related to the nervous symptoms which so commonly accompany this disease.

Rheumatism is recognized as having a close etiological relationship to certain of the neuroses of childhood, such as chorea, hysteria, and incontinence of urine. Here again the relationship between the primary disease and the nervous symptoms may be more or less complex. There can be little doubt that the toxin of acute rheumatism may, by its direct action on the nervous system, produce chorea, since chorea is not infrequently the first manifestation of the rheumatic poison; joint symptoms, heart symptoms, and other rheumatic manifestations developing later. It is also a well-known fact that rheumatism in the child may be a more or less chronic disease, producing the profound blood changes which are characterized by the term chronic anæmia. Goodhart believes that children of rheumatic parentage are often habitually anæmic. Cheadle says that "the presence of the rheumatic poison appears to be inimical to the red corpuscles. It either produces their disintegration or interferes with their production."

Trousseau affirms that there is perhaps no acute disease which produces anæmia so rapidly as rheumatism. Certain it is that rheumatism is one of the diseases of childhood which produces most profound blood changes, and in this way brings about a malnutrition of nerve elements which may act as a factor in the production of neurotic disease in children.

Malaria is another of the chronic microbic diseases which holds close etiological relationship to neurotic disease. Headaches, neuralgias, hysteria, night terrors, and other nervous symptoms are frequently either directly or indirectly produced by the malarial poison.

Certain periodic neuroses, such as headache and neuralgia, may undoubtedly be produced by the direct action of the malarial poison on the nerve elements.

It is also probable that certain other neuroses, such as hysteria, incontinence of urine, general nervous irritability, and neurasthenia, are more or less indirectly related to malaria through the profound blood changes which occur in this disease.

Forchheimer says: "The prime and principal lesion of malaria is that of the blood." . . . "Malarial cachexia is the usual concomitant of chronic malaria in children, and children having the cachexia are emaciated and extremely anæmic." The relationship which this cachexia bears to neurotic disease in children is a well-established clinical fact, and it probably depends not alone upon the direct action of the malarial poison upon the nervous system, but also on the profound blood changes which produce malnutrition of nerve elements, in that way causing and predisposing to neurotic disease.

Hereditary syphilis is another chronic disease of childhood which is also closely related to neurotic disease. The blood changes which occur in this condition are very profound, and these changes, no doubt, are responsible for the etiological importance of inherited syphilis to the neuroses of childhood.

I wish here to call attention to the fact that the four diseases, tuberculosis, rheumatism, malaria and syphilis, which in this chapter are noted as being closely related to the neuroses of childhood, are the four important chronic diseases which have latent stages and which produce morbid changes, especially in the blood-forming organs of children, the lymph glands, spleen, tonsils, and bone marrow.

There are many other microbic diseases, such as scarlet fever, diphtheria, measles, and in fact all of the other zymotic diseases, which are capable, through the action of their specific poison, of producing marked nervous symptoms, but they are not so closely related to the chronic forms of nervous disease in children, since the blood changes which they produce are usually acute, and the diseases themselves have no tendency to chronicity.

The importance, however, of these acute zymotic diseases, as factors in producing nervous diseases in children, must not be overlooked.

CHAPTER VII

CHRONIC ANÆMIA

The nervous symptoms resulting from a venous condition of the blood are almost the same as the symptoms produced by an arterial anæmia of the same centers. The reasons for this are plain, since following the ligation of arteries we have not only an arterial anæmia of the nerve centers, but also a compensatory venous congestion, so that in both artificial venous congestion and arterial anæmia we have the nerve centers bathed in venous blood.

It is thought by Landois and Sterling that "the stimulation of the nerve centers which results from the ligation of arteries is due to the sudden interruption of the normal exchanges of gases between blood and tissues."

It must be remembered, however, that a venous condition of the blood which is associated with arterial anæmia means not only a decrease of O, and increase of CO_2, but it also means more urea, more purin bodies, and more of all the effete products of retrograde tissue metamorphosis. That is to say, the nerve tissues are not only deprived in part of all the substances which are necessary for their nutrition and healthful action, but they are also exposed to the irritating and poisonous influence of the effete products previously noted. It seems, therefore, a safer explanation of the symptoms which result from experimental arterial anæmia or ven-

ous congestion of nerve centers to say that they are caused not only by an interruption in the normal exchange of all substances necessary to the nutrition and healthful action of nerve tissues, but also by the presence in the blood of CO_2, and other effete and poisonous products.

In this connection we may note the following physiological facts concerning the influence of the above-named blood conditions on important nerve centers.

A venous condition of the blood in the medulla oblongata will stimulate the vasomotor centers and cause constriction of the small arteries; this has been thought to be due to the direct stimulation of the centers by CO_2 (Landois and Sterling). The same result may also be produced by an arterial anæmia of these centers due to ligation of arteries.

In the medulla oblongata there is a center whose stimulation causes general spasms. This center may be excited either by a venous congestion or an arterial anæmia of the medulla oblongata.

The respiratory center may also be excited by either a venous condition of the blood or by an arterial anæmia.

Lauder Brunton cites the following experiment to show the relation existing between convulsive movements and a venous condition of the blood supplying nervous centers: "In fowls killed by cobra poison the convulsions came on at the moment the comb became livid, and when artificial respiration is begun, the convulsions disappear as the comb again regains its normal color." Brunton believes this to be an instance of asphyxial convulsions, due to irritation of the higher

brain centers, thus diminishing their coördinating or inhibiting action on the lower centers of the cord. He also says that "drugs which stimulate the circulation and increase the nutrition of the higher nerve centers in this way strengthen their coördinating power and tend to prevent spasm; alcohol and ether act in this way."

That this weakening of the inhibitory power of the brain and medulla oblongata may result from arterial anæmia as well as from venous congestion is shown by the following experiments. If the arteries going to the brain be ligatured so as to paralyze the medulla oblongata, then, on ligaturing the abdominal aorta, spasms of the lower limbs occur, owing to the anæmic stimulation of the motor ganglia of the spinal cord (Sigm. Meyer). That the anæmic condition of the cord produced by ligaturing the abdominal aorta is incapable of producing spasms when the medulla oblongata is in normal condition, is a striking example of the inhibitory influence of the oblongata centers on the motor centers of the cord.

V. Aducco made a series of valuable experiments on dogs. He produced anæmia of the nerve centers by cutting off a portion of the blood supply from the spinal motor centers. He compared the excitability of these centers before and after the artificial anæmia thus produced, and in this way he determined "the effect that partial anæmia exercised on the motor centers of the cord."

Aducco concludes his paper as follows: "The researches I have just described have led me to draw the following conclusions: In anæmia, that is to say when the flow of blood is diminished, the active materials of

the nerve centers are found in a state of great excitability. In this condition excitants from the exterior act much more energetically than in the normal condition, and this state of excitability increases, very probably, during the entire duration of the anæmia. It seems to me that one should, within certain limits, admit that there is an inverse relation between nutrition and the excitability of the nerve elements. This latter augments during the time that the nutrition diminishes."

In these conclusions, Aducco wrongly interprets artificial arterial anæmia to mean a *simple innutrition,* and concludes that the excitability of the nerve centers is due to this innutrition rather than to the numerous blood changes which we have previously noted as accompanying arterial anæmia.

I have repeated Aducco's experiments, and quite agree with him that the excitability of the nerve centers increases with the duration of the arterial anæmia; but I have also shown by a series of experiments, made upon rabbits and dogs, that the complete closure of the veins, returning the blood from the spinal motor centers, will produce the same symptoms that are produced by the ligature of the arteries supplying the same spinal centers.

In these experiments I studied the increase in the electrical excitability in the muscles of the hind legs as well as the increase in the reflex excitability of these parts, and always obtained practically the same results from ligation of arteries as from ligation of veins supplying these same nerve centers.

From the observations above cited in this chapter the following inferences may be made:

1. Both arterial anæmia and venous congestion can produce an excitable condition of the nerve centers, and may therefore be factors in the production of nervous symptoms.

2. The nervous symptoms resulting from arterial anæmia are very similar to those resulting from venous congestion, and this is because in both conditions there is a venous condition of the blood supplying the nerve centers.

3. Arterial anæmia and venous congestion produce nervous symptoms by producing a *malnutrition* rather than a simple *innutrition* of the nerve centers.

4. Arterial anæmia and venous congestion weaken the inhibitory centers, and this results in the discharge of force from reflex centers on comparatively slight excitation.

5. Arterial anæmia and venous congestion make more excitable both the reflex centers in the cord and the more important reflex centers in the medulla oblongata.

It is my belief that the above experiments offer at least a partial explanation of the long chain of nervous symptoms that are commonly associated with the complex blood condition known as chronic anæmia. These chronic anæmias produce a chronic malnutrition of nerve centers, and thus take rank among the most potent etiological factors of the neuroses of childhood.

Chronic anæmia is a term used to express an inconstant and very complex blood condition. The chronic anæmias of infancy and childhood are due to a great variety of causes, the most important of which are tuberculosis, rheumatism, malaria, syphilis, rachitis,

scurvy, intestinal disease, and improper food and bad hygiene.

The blood in chronic anæmia is weak in proteids and hemoglobin, and must necessarily therefore produce a proteid and oxygen starvation of nerve cells. Chronic anæmia may also mean a diminished quantity of fat and of inorganic salts and an increase of the poisonous and irritating products produced by retrograde tissue metamorphosis and bacterial action.

In the chapters on auto-intoxication, intestinal toxæmia, and bacterial toxins we have discussed the etiological relationship of bacterial poisons and auto and intestinal toxins to nervous symptoms.

It is the purpose of this chapter to study certain of the other phases of the blood condition known as chronic anæmia in their etiological relationship to the neuroses of childhood.

In chronic anæmias we may have the conditions which Christopher has described as "partial starvations" of nerve elements, and these conditions may be important factors in producing irritability of nerve cells. Such qualitatively starved cells are yet sufficiently well nourished to store up a large amount of nerve energy to be fitfully discharged.

These partial starvations may consist in a diminished amount of fat, albumin, hemoglobin, oxygen, and the inorganic salts. It is my belief that the character of the nervous symptoms may vary with the character of this partial starvation.

Fat starvation is a form of malnutrition which can best be studied in the chronic anæmia produced by rachi-

tis. The works of Cheadle and others clearly demonstrate that fat starvation may be one of the important causes of rachitis, and the feeding of some easily digested fat is now accepted as a most important means in the cure of this disease. It must not be understood that the blood condition in rachitis is described by saying there is a diminution in the amount of fat, since there are probably many other blood changes, including a diminished amount of calcium, phosphorus, and proteid, which are contributing factors to the blood impoverishment of this disease.

But while the blood improverishment of rachitis is very complex, yet by far the most important factors of this condition are the diminished quantity of fat and calcium. The deficiency of fat is a constant condition, and one that we know is etiologically related to rachitis. The inference, therefore, is probable that fat starvation is a form of malnutrition which may predispose to certain well-defined neuroses, such as laryngysmus stridulus and other local and general convulsive neuroses.

Calcium starvation may also play a part in the etiology of the nervous symptoms associated with rachitis. Just the rôle, however, which it plays in this condition has not yet been determined; certain it is, however, that experimental physiology teaches us that calcium starvation, whatever the conditions may be that bring it about, is capable of producing profound nervous disturbances. This subject has been studied to advantage by W. H. Howell, who demonstrated that the normal irritability of nerve and muscle tissue is in great part dependent upon the proper supply of calcium to these tissues. If

the heart be deprived of calcium salts, by feeding it with blood deprived of its calcium salts, it stops beating very soon, and this action is so rapid that it could only result from nervous influence. The most plausible explanation of this fact is that the nerve ganglia of the heart, in the absence of the calcium, fail to discharge the nerve force which stimulates the heart muscle to contraction. If, on the other hand, the heart be fed with a calcium solution in distilled water, it will continue to beat for a long time. In this instance the calcium keeps up the irritability of the cardiac ganglia, so that they continue to discharge nerve force into the cardiac muscle, and the heart's action continues. In this explanation, which I have taken the liberty to make from Howell's experiments, I have attributed to calcium an important influence over the discharge of nerve force from automatic centers. The presence of calcium in normal quantities causes these centers to discharge their nerve force into the cardiac muscle, as they normally do; and the absence of calcium inhibits the discharge of nerve force from these automatic centers, and as a result the heart stops.

If a certain amount of calcium is necessary to the normal irritability of nerve centers, and if the absence of calcium inhibits the discharge of force from nerve centers, then it is reasonable to infer that a diminished amount of calcium would have an influence on the irritability of nerve centers which would find expression in clinical manifestations. That an insufficient quantity of calcium in the blood may produce nervous symptoms, is, I think, proven by Howell's experiments. He says: "When a frog is irritated with oxylate solutions (that

is to say, calcium free solutions) the muscles are affected quickly and in a peculiar manner." . . . "Twitching movements of the toes begin in a few minutes, and soon extend to muscles of the leg and trunk. In some cases these movements were violent; strong convulsive contractions of muscles and limbs followed each other rapidly, and were often so violent as to throw the animal out of the position in which it was lying. The convulsions resembled those caused by strychnia; the violent tetanic contractions had the appearance of being caused by stimulation of the cord." This extremely excitable condition of the reflex nervous mechanism was followed after a time by the complete loss of irritability of this mechanism.

These observations of Howell's seem to me to show that between the stage of the normal irritability of this reflex mechanism, when the calcium salts are supplied to it in normal quantity, and the complete paralysis or loss of irritability of this mechanism, due to the more or less complete absence of calcium salts, which have gradually been washed away by the calcium free circulating fluid, there is a stage of extreme irritability and reflex excitability of this reflex nervous apparatus which corresponds to the period when this nervous mechanism is supplied with a diminished amount of calcium salts; that is to say, there is a partial calcium starvation of the nerve elements. This explanation of Howell's experiments is supported by his further experiments. In animals in which the irritability of the reflex nervous apparatus had been destroyed by calcium starvation, as in the above experiments, it was found that if calcium

solution was added to the circulating fluid of the muscle, the primary effect was to again produce a twitching movement of these muscles, "lasting for a short while," to be followed by a more or less distinct return of the muscle to its normal irritability.

From these and other experiments along the same line I conclude that calcium starvation of the nerve elements may be a factor in the production of the convulsive neuroses of childhood. By way of parenthesis it may here be stated that rachitis has by some pathologists been classed among the acid intoxications, and the deficiency of calcium and other inorganic salts is thought to be due to this cause (see chapter on Acid Intoxications).

However unsatisfactory our knowledge may be of the exact blood changes in rachitis, the fact remains that these blood changes, whatever they may be, are among the most important etiological factors in producing certain neuroses in infants and young children. Among the nervous symptoms associated with rachitis may be mentioned restlessness at night, muscular spasm, laryngismus stridulus, tetany, and general convulsions.

Scurvy is due to some error in diet. The exact nature of the partial starvation which results in scurvy is not known. The cooking or sterilization of food, however, has something to do with producing this food deficiency, which results in the general cachexia and profound anæmia which characterize well-marked scurvy. Tremor, sleeplessness, pseudo-paralysis, pain, muscular tenderness, and general nervous irritability are among the nervous symptoms which accompany scurvy, and some of these, notably the sleeplessness and general irrita-

bility, continue long after the active symptoms of scurvy have disappeared.

Chronic gastro-enteritis is one of the important etiological factors in producing nervous symptoms in infants and young children. Among the nervous symptoms closely associated with this condition may be mentioned general nervous irritability, disturbed sleep, and convulsive disorders, and, in older children, hysteria, neurasthenia, incontinence of urine, and chorea.

These symptom groups are, no doubt, partly due to a proteid, hemoglobin and oxygen starvation. These factors, however, are probably secondary in etiological importance to the poisoning by intestinal toxins, which occurs in these diseases. This phase of the subject has been discussed in the chapter on intestinal toxins.

Oxygen starvation, which results from impure air and bad hygienic surroundings, is a very important factor in producing anæmia, general malnutrition, and nervous symptoms in children. This factor is especially potent for evil during the first two years of life.

Impure air and bad hygienic surroundings, by predisposing to and aggravating all of the chronic diseases of infancy and childhood, act as powerful indirect factors in producing neurotic disease. The nervous symptoms of rachitis, scurvy, tuberculosis, intestinal diseases and lithæmia are greatly aggravated by oxygen starvation, and their cure is promoted by pure air and good hygienic conditions. The direct influence, however, of these factors in producing anæmia and nervous symptoms in otherwise healthy children must not be overlooked.

CHAPTER VIII

REFLEX IRRITATION

Reflex irritation is one of the most important etiological factors of the neuroses of childhood. Many able pediatrists in recent years have waged an active crusade against this proposition, which previously was thought to be one of the axioms of medical knowledge. While these men have not been able to convince the medical world that reflex irritation is an unimportant factor of neurotic disease, they have very much modified the view, which so long obtained, that reflex irritation was the all-important factor in producing these diseases. In the proposition as stated at the beginning of this chapter, I have taken position between these extreme views, and it will be the purpose of this chapter to show that the influence of reflex irritation in producing nervous diseases in childhood has been as much underrated in recent years as it was exaggerated by earlier writers, who taught that almost every nervous disease was caused by some reflex factor. It is a matter of common clinical observation that such neuroses as hysteria, incontinence of urine, night-terrors, chorea, convulsions, fever, and headache are at times etiologically related to some form of reflex irritation, and this relationship is not infrequently absolutely demonstrated when removal of the reflex irritation cures the neurosis.

The common sites of reflex irritations which are recognized factors of nervous diseases in children are the genito-urinary organs, the gastro-intestinal tract, the eye, the ear, and the nose. The importance of this subject does not end with recognizing that reflex irritation from all of the above-named sites are common factors of neurotic disease, but it is of equal importance that we should recognize that, as a rule, reflex irritation acts conjointly with other factors in producing the neuroses of childhood. It is a well-known fact that reflex irritation, of apparently a severe type, may exist without producing nervous symptoms. In such instances, the center, which is the most important part of the reflex arc, is normally stable, and not easily excited to discharge its stored-up nerve energy. It is most important, therefore, that we should recognize the fact that the reflex irritation which excites neurotic disease is made potent by reason of its connection with an abnormally irritable reflex center. In previous chapters we have studied the influence of heredity, sex, age, environment, and various blood conditions in producing an increased irritability of nerve centers; and it is chiefly with the aid of these factors of neurotic disease that reflex irritation can produce such a wide range of nervous symptoms. The study of this subject embraces, therefore, not only how each of these factors may act in producing nervous symptoms in children, but it must also inquire in individual cases into the interdependence and relationship of these factors in producing these symptoms.

The fact that reflex irritation is commonly associated with other factors does not in the least diminish its

importance as a factor of neurotic disease, since the removal of the reflex excitant very commonly cures the neurosis, even though the other factors remain, and since our best efforts at removal of other factors of neurotic disease, as a rule, are futile for good, as long as the reflex excitant remains to constantly excite the nerve centers. The explanation of these clinical facts is that reflex irritation does not act simply as an excitant in discharging nerve force from irritable centers, but it also acts by keeping up the irritability of these centers, and, if long continued, by producing changes in the nerve centers, recognizable under the microscope, which make these centers more irritable and more susceptible to reflex excitation.

If this be true, then, reflex irritation at once assumes an important position among the factors of neurotic disease in children; such a position, as in recent years, has not been accorded to it, and it is the special purpose of this chapter to bespeak for reflex irritation the high position which it merits among the factors of neurotic disease in children; a position only a little less important than that which it formerly occupied, and from which it has been unjustly removed.

The microscope has gradually revealed to us the fact that all cellular activity is accompanied by definite chemical and morphological changes in the cell itself. The tired cell differs from the rested cell, not only in morphological changes, which can readily be noted in nucleus and cell protoplasm, but also in the reaction of cell protoplasm and nucleus to coloring matters.

The changes which result from the functional activity

of cells may be called fatigue changes, and it is evident that the longer the cell is worked, the more marked will be these changes. It is also a physiological fact that fatigue changes in the tired cell will disappear after a period of rest, and the cell will again be found morphologically and chemically a rested cell, but it requires a longer period of time for a cell to return to its rested condition than it does for the same cell to tire under ordinary work.

The fatigue changes resulting from the functional activity of glandular epithelium are, as a rule, very pronounced. These changes, while not the same in all gland cells, may be noted in the shrunken condition of both nucleus and cell protoplasm and in the changed reactions to coloring matters of both nucleus and cell protoplasm. Fatigue changes in the tired muscle cell are also shown in the shrunken and vacuolated condition of its protoplasm. And both the tired muscle cell and the tired gland cell are only restored to their rested condition by a period of prolonged rest—the period of rest required being considerably longer than the period of activity.

The nerve cell, like the gland and muscle cell, shows marked morphological and chemical fatigue changes. C. F. Hodge, in a very clever piece of work, has shown that definite changes occur in the nerve cells of the brain and spinal ganglia of certain birds and bees as a result of their normal daily activity. He compared the nerve cells of sparrows and swallows shot in the early morning with the nerve cells of sparrows and swallows shot in the evening after a day of hard flight. Experiments of

this kind on birds and bees invariably showed fatigue changes in the nerve cells tired from the day's work. Hodge also found definite changes to occur in the spinal ganglion cells of the frog, the cat, and the dog under electrical stimulation, and these changes were very similar to the changes which he had observed to result from the normal daily activity of nerve cells.

These fatigue changes in the nerve cells, whether resulting from normal daily activity or electrical excitation, are as follows:

Nucleus was "much smaller, and had a jagged, irregular outline. It took a darker stain, and lost its reticular appearance."

Cell protoplasm "did not take stain so readily, and was much shrunken. In spinal ganglia it was vacuolated."

Hodge also observed that the nerve cell recovered much more slowly than it tired, and that the recovery of the nerve cell might be represented by a curve quite similar to the curves obtained by Mosso and Lombard for the muscle cell in its recovery from fatigue. He concludes that "individual nerve cells after electrical excitation recover if allowed to rest for a sufficient time, but the process of recovery is slow. From five hours' stimulation recovery is scarcely complete after twenty-four hours' rest."

The changes above noted in nerve cells, as resulting from electrical stimulation and normal fatigue, have a plain bearing on the study of the changes which occur in the spinal ganglia from reflex irritation, since reflex irritation can do nothing more than greatly exaggerate the functional activity of these cells, and must, therefore,

result in changes within the cells similar to those above described.

Satovski, in a careful research on "Changes in Nerve Cells Due to Peripheral Irritation," has made an important advance in our knowledge of this subject. He irritated a peripheral nerve by ligature, and thereby caused a peripheral, but not a central, degeneration of the nerve. In this way he produced a chronic reflex irritation of that portion of the cord to which this nerve belonged, and on microscopical examination of the cord at this point he found on the injured side, using the uninjured side for a control, many cells exhibiting great vacuolation and shrinking of the protoplasm from the capsule. The nuclei of these cells were oval instead of round, they stained easily, and were sometimes so much shrunken that they were zigzag in outline and left a space between the protoplasm and the nucleus of the cell.

Ternowski, in a research on "Changes in the Spinal Cord from Stretching the Sciatic Nerve," found changes very similar to those previously noted by Satovski.

From the observations quoted, it is plainly evident that chronic reflex irritation can produce very marked changes in the nerve cells of the spinal ganglia, and that the longer and more violent this irritation is, the more pronounced will these changes be. It is also plain that a considerable length of time must be required to restore to their normal condition cells which have been subjected to reflex irritation for months and years. It has even been noted that nerve cells, under electrical stimulation, can be so exhausted that the nuclei will entirely disappear, and the cells be unable to recover their normal

condition even after the removal of the stimulus which produced the change. Here we have an explanation of the ofttimes slow recovery of an irritable spinal cord, after the removal of the reflex cause which brought about the irritability. In the application of these facts to clinical medicine, we must remember that the spinal cord has but two functions, viz.: conduction and reflex action. We must also remember that a reflex irritation of an afferent nerve carrying impulses to any of the cells of the cord does not confine its morbid influence to those cells, but by reason of the physiological law of "overflow of reflexes" the impulse spreads up and down the cord, producing changes in adjacent cells; and if the reflex irritation be severe and long continued, the impulses may spread throughout the cord, involving all its cells and producing a general spinal irritability, in this way helping to produce in the individual a great variety of reflex nervous symptoms.

In the above observations we have not only a physiological but also a morphological explanation of how and why chronic reflex excitation may be an important factor in producing general spinal irritability, and we have also a sufficient explanation of the fact that the removal of the reflex cause, which has been acting for years in producing spinal irritability, may not at once be followed by the cure of the spinal irritability, but that it may even require years of comparative rest for the irritable spinal centers to become stable (normal), even after the removal of the reflex cause which produced the irritability of these centers. These observations also justify the belief, arrived at by clinical observations, that reflex

irritations, acute and chronic, are among the most important causes of neurotic disease in children.

In the adult such reflex factors as are produced by eye-strain, and diseases of the male and female genito-urinary organs, may be important factors in producing nervous symptoms. Yet reflex irritation is much more important in producing functional nervous diseases in the child than it is in the adult, for the following reasons:

1. Reflex disturbances, such as intestinal irritation, adherent prepuce, and uncorrected eye-strain, are much more frequent in the child than in the adult.

2. The nervous system of the child is more irritable and unstable by reason of its incomplete functional development.

3. The inhibitory control of higher nerve centers over spinal reflex movements is feebly developed in the child.

4. Blood changes such as we have described in previous chapters are much more common allies of reflex factors in producing nervous diseases in children than they are in adults.

5. The functional development of the male and female genital organs which marks the approach of puberty is a source of marked reflex disturbances which greatly predispose to neurotic diseases.

CHAPTER IX

EXCESSIVE NERVE ACTIVITY

Excessive nerve activity (the term including brain work and nerve excitement) is recognized as one of the most powerful etiological factors in producing neurasthenia, hysteria, and other neuroses in the adult, but notwithstanding the attention which these factors have received at the hands of neurologists as factors in producing neurotic disease in the adult, I fear that pediatrists have rather underrated them in their etiological relationship to the neurosis of childhood. At any rate, I feel sure that too little has been done to educate those who have the rearing and tutelage of the young to the importance of this subject. For only in this way can children be protected against the baneful influences which excessive brain work and nerve excitement produce.

There can be no doubt that neurotic disease is, especially in our large cities, greatly increased by subjecting the immature nervous systems of young children to the almost constant excitement, strain, and mental activity with which our social order has surrounded them. An all-important question, therefore, to pediatrists who should be especially interested in making of the child the strongest possible man, is: How can these influences which are playing such havoc with the nervous systems of children be guarded against? How can they be

counteracted? How can parents, guardians, nurses, and teachers be made to comprehend the importance of this subject?

If these questions are to be answered, if the campaign against the evil of constantly subjecting children to the nervous strain resulting from the artificial conditions which obtain in all cities, is to be in any degree successful, then the whole subject must be placed upon a more exact physiological basis than it has ever been before, so that those who have charge of the young may be told not only that nervous strain is an important cause of neurotic disease, but that they may also be told why this is so. And in this series of papers on the etiology of the neuroses of childhood I have attempted to outline some of the physiological facts by which this goal is to be approached.

The teachers and guardians of the young must be told that the nervous system of the child differs very materially from the nervous system of the adult; they must be told that the child, especially in his nervous organization, is not a *little man;* that his nervous system is structurally and functionally immature; that it is excitable, unstable, and under feeble inhibitory control; that the sources of reflex irritation in the child are many, and that the nerve centers discharge their force more fitfully and more readily than in the adult; that the period corresponding with the onset and establishment of the reproductive function in girls is a time when they are especially predisposed to nervous disease. And they must also be told that these and other physiological peculiarities of the nervous system of childhood are made

much more potent for evil when they are associated with the various "blood conditions" which, in previous chapters, I have shown to be etiologically related to the neuroses of childhood.

In order to approach this subject in a physiological way, I shall call attention to a very extensive research by Dr. Wm. Townsend Porter, which has, I believe, great practical importance in the study of the influences of school life in producing the neuroses of childhood.

Dr. Porter demonstrated that children who are advanced in their studies are, on the average, heavier, taller, and of larger girth of chest than less advanced children of the same age. Thus, boys aged eleven were found in Grades I, II, III, IV, V, and VI of the St. Louis public schools. The average weight of the six classes was respectively 64, 66, 68, 71, 72, and 74 pounds. The ability to succeed in school life is, in the average, a measure of mental power, and if successful scholars are, as a rule, better developed physically than the less successful, it follows that mental ability is, in the average, greater in large children than in small children of the same age.

Dr. Porter makes a practical deduction from the law thus established. The entrance to any grade in a graded school system is guarded by examination, and the children found in that grade are such as have passed the entrance examination, and have in this way shown their capacity to do the mental labor exacted in this grade. The greater number of these children are of the same age. The work of this grade is, then, normal for this age, and the average height, weight, and girth of chest

of this age form the physical development most often found in children able to do the work of the grade. No child younger than the average age of any grade should be permitted to enter it until a physical examination has shown that his strength shall probably be sufficient. In determining this, the relation of weight and girth of chest to height is of special importance. Abnormal height is undoubtedly a disadvantage, yet such children may be strong provided their physical development is in proportion to their height. If the contrary is the case, the child will be much less able to resist the strain of school life.

Dr. Porter points out the importance of frequent weighing of growing children. Persistent loss of weight in an adult is a matter of grave concern. The failure of a child to make the normal gain in weight is no less grave, and should lead to an inquiry into his school tasks, for the effects of prolonged overwork are very serious in children, and often irremediable.

It is my belief that if there were a rule, such as Dr. Porter suggests, guarding every grade in our public school system by a physical as well as a mental examination, it would prevent the development of a considerable portion of the neurotic disease which is now so prevalent among school children. With children of good physical development working in the public schools within the limitations of their proper grades there is almost no danger that a moderate amount of school work will in any way assist the development of neurotic disease, provided always that the hygienic conditions of the school, especially the light and ventilation, are good.

But the strain of ordinary school work is a very different matter with children of poor physical development, many of whom are, unfortunately, precocious. A large number of those children, by reason of bad heredity, are neurotic, poorly nourished, and anæmic, and many of them have tuberculous, rheumatic, or syphilitic inheritances, while others, from accidental causes, such as bad hygiene, improper food, etc., are below the normal in physical development. The nervous systems of such children are in a condition of malnutrition, and are therefore not capable of doing the ordinary work of their grades in the public schools, and if they are permitted to do this work, or if, as is often the case, these children are encouraged to push on into higher grades than the one to which their years and strength should assign them, disastrous consequences will surely follow, and their nervous systems may be injured beyond repair.

These children under the mental strain of school work may develop chorea, hysteria, and other neuroses. The important duty, therefore, of every physician is to advise against much school work in children of feeble physical development, and to explain to parents and teachers why such children as these should first have their physical defects looked after, and should then be placed in a grade lower than that to which their age and intelligence would assign them.

It is my belief that a normal dwarf, with no bad hereditary influences behind him, may without injury to himself keep pace in mental development with fellows of his own age; the dwarfish body is not of itself an indication that school work might be injurious if there is

every other evidence of perfect physical development. Dwarfishness of body in school children of good physique does not mean dwarfishness of mind. But dwarfishness among children, as indicated by weight and chest development, is, as a rule, the result of disease and bad heredity, and this is the reason why children who are under weight and have poor chest development are, as a rule, incapable, without injury to their nervous systems, of doing the same amount of school work as their fellows of the same age. It is my belief, therefore, that the physical basis of precocity and dullness in children depends upon the facts that bad heredity and disease are the chief causes of abnormal dwarfishness or poor physical development in the young. It is also my belief that children of this class are, as a rule, anæmic and poorly nourished, and that their nervous systems are therefore in a condition of malnutrition, and not capable of doing an amount of work in keeping with the age of the child.

The reasons, then, are clear why we should not allow a child of poor physical development to be pushed to rapid brain development. If we do, their nervous systems will surely suffer from the strain, and whatever predisposition they may have to neurotic disease will be greatly increased. In dealing with individual cases, it will be of the utmost importance to the physician to know the child's heredity; if the child has a bad family history, it should be the imperative duty of the physician to protect it against mental overwork. We cannot, of course, change the child's ancestry, but we can speak out against the crime of pushing children with hereditary physical defects to rapid brain development, and in this way

developing an hereditary nervous weakness into actual disease. School work may therefore be classed as a cause of neurotic disease in children of poor physical development, and it acts chiefly in calling out hereditary defects of the nervous system. In speaking of school work as a cause of neurotic disease in children, it must be understood that this term embraces not only brain work, but also the mental excitement which attends examinations, and the eye strain which results from imperfect vision and bad light, the latter being one of the most common causes of reflex nervous disease in children, and one of the physical defects which should be promptly removed.

It must be remembered that what is here said of the physical basis of precocity and dullness is a matter of proof and not of opinion, and that it applies to children only, and has nothing whatever to do with the question of whether, in adult life, a healthy body adds strength and capacity to the nervous system.

In this demonstration of the injury which results to the nervous system of the delicate child from the nervous strain of school life we have a most important warning against the pernicious habit of encouraging mental precocity in early childhood. It is a matter of almost daily experience to see a poorly nourished tuberculous child brought forward for the purpose of demonstrating its "wonderful" precocity. The proud mother and overzealous nurse commence the process of mental cramming even before infancy has passed into childhood. From this time on children are daily being taught, apparently with the idea of destroying their childhood and making of

them little men and women. And this unphysiological process is not infrequently a factor in the production of the nervous disorders of late childhood, puberty, and adult life. Mothers must be told that *early precocity is an abnormal condition in the human infant,* which, if encouraged, may result in actual disease and permanent mental impairment. They must be told that *vegetation* is the ideal life of infancy and early childhood. Look to the physical and retard the intellectual development of the young child. It must not be taught, it must not be trained. It must have plenty of exercise, fresh air, proper food, and, if possible, a large portion of the year should be spent in the country, away from the clamor and excitement of city life. In the country also the child can have a certain amount of solitude, the importance of which can scarcely be overestimated in giving independence of thought and character to the future man.

It is my belief that the nurse and governess in the modern home are doing much to destroy the development of the individuality in children. The modern child has someone to do his thinking, someone to minister to his every want, and is almost constantly being trained. He has no time to himself, and a very small portion of his day is spent in play with his intellectual equals. If there is one crying evil common to all of our large cities it is the scarcity of playground for children, and the attention of humanitarians should be called to this fact. If our generous citizens would pause long enough in the building of hospitals, libraries, and places of learning to realize that there is a field almost totally neglected

by the humanitarian, and one of as much importance to the welfare of our communities as the building of hospitals, libraries, and institutions of learning, then, possibly, a portion of the vast sums of money annually spent in this way would be spent in providing playgrounds for children. These playgrounds should not be covered with beautiful grass plots guarded by policemen, but they should be playgrounds in the best sense of these words; places where ball, tennis, and all kinds of healthful sport could be enjoyed. And I believe the day is not distant when the physiological importance of the physical, as opposed to the mental, development of children will be so generally recognized that many philanthropists will prefer to hand their names to posterity associated with "playgrounds" rather than with fountains, art museums, music halls, and other worthy enterprises.

PART II

CHAPTER X

FEVER

In the chapter on the "Physiological Factors of the High and Variable Temperatures of Childhood" I have discussed the physiological peculiarities of the heat-regulating mechanisms of the young nervous system; they are, briefly, as follows:

PHYSIOLOGICAL CAUSES OF FEVER IN CHILDHOOD

1. The thermogenic or heat-producing centers situated at the base of the brain are more easily excited to increased heat production in the immature brain of the young child than they are in the mature brain of the adult.

2. The thermo-inhibitory or heat-controlling centers are weaker, and therefore less capable of exercising proper control over the thermogenic centers in the young child than they are in the adult.

3. The heat-dissipating mechanism is much more efficient in the young child than it is in the adult.

The marked excitability of the thermogenic centers, and the feeble control which the inhibitory centers exercise over them, make it possible for comparatively trivial exciting causes to produce high fever in the young child, but, on the other hand, the quick response

of the very efficient heat-dissipating mechanism quickly lowers the body temperature. This rapid play of function between the heat-generating and heat-dissipating mechanisms gives the great variability of body temperature which characterizes the fevers of childhood.

PREDISPOSING CAUSES OF FEVER

1. A neurotic inheritance may increase the irritability of the thermogenic centers and diminish the control of the thermo-inhibitory centers, and in that way make the individual child more prone to high and variable temperatures than his fellows of the same age. A neurotic family history may, therefore, assist the physician in explaining why certain children are especially predisposed to high body temperatures from slight exciting causes.

2. Chronic malnutrition, by increasing the irritability of the thermogenic centers and by retarding the development of the thermo-inhibitory centers, becomes a very powerful predisposing cause of fever in young children. Chronic malnutrition implies that the nervous system, as well as other parts of the body, is malnourished. This malnutrition, as has been previously pointed out in Chapters II and III, increases the instability of the nervous mechanism which controls the body temperature.

Improper food, bad hygiene, and unfavorable climatic conditions, with the rickets, scurvy, chronic anæmia, and intestinal diseases they produce, are important causes of malnutrition in the young child, and are therefore potent predisposing causes of fever at this period of life.

EXCITING CAUSES OF FEVER

The exciting causes of fever in infancy and childhood may be classified as follows:

1. Bacterial products.
2. Auto-toxins.
3. Heat stroke.
4. Muscular action (convulsive).
5. Mechanical and reflex irritation.

1. *Bacterial products* are by far the most important of the exciting causes in children. The variations in temperature accompanying the acute infections, including all forms of external and internal bacterial toxæmias, are due to the action of bacterial products on the heat centers. Bacterial products capable of producing fever may be formed by bacterial action, either within the blood and tissues of the animal or outside of the blood and tissues of the animal, in wounds, or in cavities, such as the intestinal canal, which communicate with the external air. But wherever these bacterial products may be formed, the soluble ones are absorbed and produce fever by their direct action on the thermogenic centers. As a rule the soluble bacterial products which produce fever also produce a continuous increase of body temperature, and the increase of temperature is a valuable indication of the severity of the fever process, but this is a rule which has many exceptions, as is shown by the normal and subnormal temperatures that occasionally attend pneumonia, typhoid fever, influenza, scarlatina, and other acute infections. The subnormal temperatures which may occur in these fevers may be explained

by the increased action of the heat-dissipating mechanism, or it may be explained by a variability in the potency of the bacterial products, or by a failure on the part of the thermogenic centers to continuously respond to the bacterial poisons.

Centanni investigated seventeen pathogenic species of bacteria, and found in cultures of all of these germs a substance, not a peptone, which when injected into animals caused fever, with the following symptoms: high temperature, prostration, emaciation, and finally death. Omitting further discussion, I will say that the evidence justifies the conclusion that bacterial products excite fever by acting directly upon the thermogenic centers, and that the variations in body temperatures which characterize the fevers of childhood are due to a disturbance in the play of functions which these poisons produce between the heat-producing and the heat-dissipating centers.

Why do bacterial products produce fever so much more readily in children than they do in adults? This question has in part been answered by our previous study of the peculiarities of the nervous mechanism in childhood which controls the body temperature.

(*a*) The thermogenic centers being more unstable and irritable in the child, are more readily excited by bacterial products. Fever and increased temperatures are therefore more easily produced.

(*b*) The thermo-inhibitory centers being immature and feeble in the child, exercise but a weak restraining influence over the discharge of force from the thermogenic centers, which are being excited by bacterial

products; for this reason fever and increased temperatures are more easily produced by bacterial products in the child than in the adult.

(*c*) Still another possible reason why microbic poisons produce fever and increase of temperature more readily in the child than in the adult was suggested to me by Professor Charles Richet, in a personal communication. His explanation depends on the potency of the fever poison more than upon the peculiarities of the nervous mechanism. Richet asks, Is it not possible that the microbic fever-producing toxins may be stronger or more toxic when they are produced in young organisms that are not protected by previous attacks of acute infections? That is to say, in infants and young children who have not had previous microbic infection and who are not, therefore, protected against these diseases, pathogenic microbes may develop more potent fever-producing toxins than they can later in life.

2. *Auto-toxins* probably play a very unimportant rôle as exciting causes of fever in infancy and childhood. Some recent experiments, however, by Mandel, indicate that the purin bodies, when they occur in excess in the body tissues, may produce fever. He found that the injection of four milligrams of xanthin produced an elevation of temperature in a monkey. He also demonstrated that in aseptic fevers there is a distinct relation between the rise of temperature and the appearance of certain products of incomplete cell oxidation, as shown by the excretion of the purin bodies. He concludes that the purin bodies may be important factors in the production of febrile temperatures.

Our knowledge, however, of auto-toxins as fever producers is not sufficient to justify the further discussion of this subject. We do not commonly associate high temperatures among the symptoms of the auto-intoxications with which we are clinically familiar, and, moreover, auto-toxins are perhaps less important disease producers in infancy and childhood than they are in adult life.

3. *Heat stroke* is an important cause of fever and high temperature in infancy and childhood. Probably the best explanation of the fever of heat stroke is that the feeble inhibitory centers of the child are still further weakened by the heat, so that practically no restraint is exercised over the thermogenic centers. Intestinal fermentation is one of the constant complications of heat strokes in the young child. This secondary condition, by the formation of intestinal toxins, acts as an important factor in keeping up the fever in these cases. Cases of heat stroke in infancy and childhood are, for these reasons, ordinarily classed as cholera infantum, or enteritis, and this classification greatly obscures the direct etiological importance of heat as a fever producer in these cases. Forchheimer has for many years taught that many of the cases of so-called cholera infantum were cases of heat stroke, and that in such cases the intestinal fermentation is a complication rather than the original cause of the disease.

The remarkable influence which outside or artificial heat produces on the body temperature of certain infants is noted by Holt, who says: "Some very puzzling and alarming temperatures are seen in infants as the result

of the application of artificial heat. In one of my patients, an infant two days old, a temperature of 107° F. was caused by the close proximity of two large hot-water bags placed in the baby's basket. The younger and feebler the child the more readily are such temperatures produced."

It is evident that if such high temperatures can be produced in delicate children by the temporary application of artificial heat, the long-continued application of excessive heat that occurs during the mid-summer months must be a most potent factor in the production of fever and high temperature, especially in delicate children whose surroundings are unhygienic.

4. *Convulsive muscular action* is not an infrequent cause of increased temperatures in infants and young children. The manifestation of muscular energy is always accompanied by the evolution of heat, and excessive muscular action, such as occurs in general convulsions, is always accompanied by increased production of heat. This is a partial explanation of the increase of temperature that occurs in general spasms, but a portion of the increase of body heat that occurs in this condition may be attributed to the increased friction of the muscles, tendons, and articular surfaces, which transforms kinetic energy into heat. It should be remembered, therefore, that excessive muscular action may be a factor in producing increased body heat, and that this source of heat production is quite distinct from that which results from the normal metabolism going on in muscles and other organs at rest, and from the abnormal metabolism going on in these organs during fever. I do not wish to convey

the idea that increased muscular action is the most common or the most important cause of the increased body temperature which occurs during muscular spasm, but only to impress the fact that violent muscular action is a factor in producing the increased body heat rather than that the increased body heat is a factor in producing the spasm.

When the spasm is purely reflex in origin, the excessive muscular action may be the most important cause of the increased body heat; but when the spasm results from microbic poisons, then, no doubt, the increase of temperature is chiefly due to the action of these poisons on the heat centers. For these reasons one would expect to find the temperature during reflex spasm not so high as it is in spasm due to microbic infection.

5. *Direct mechanical and reflex irritations* may produce fever in the young child. This is more especially true of the nervous, malnourished infant.

In Chapter III I have noted the fact that foreign bodies, growths, and exudations could act directly on the heat centers, to disturb the body temperature, so that here there only remains the consideration of the reflex causes of variations in the body temperatures of infants and young children.

Ott says: "After the use of large doses of atropin I have seen the temperature rise greatly upon sciatic irritation. It was also found that this increase of temperature was accompanied by an increased production and augmented dissipation of heat." In these experiments we have proof that not only high temperatures, but also fever, may be produced reflexly. It is my belief

that variations in the body temperature in infancy and childhood are not infrequently of reflex origin, and that the intestinal canal and the genitalia are the sites where reflex irritation is most likely to produce these symptoms. Increased temperature may occur in the malnourished infant and young child from the cutting of a tooth, from worms, undigested food, and other foreign bodies in the intestinal canal. The irritating products of an intestinal fermentation may also produce increase of temperature, unaided by the soluble bacterial poisons previously spoken of. It is a matter of every-day experience with clinicians that the removal of such simple causes as are here narrated will cause the temperature of the sick child to fall to normal, and all the other symptoms of fever to disappear. It will be well, therefore, in these days when chemistry and bacteriology are dominating medical pathology, for us to remember that a purely reflex fever can and does sometimes occur during infancy and early childhood.

From the preceding outline of the possible causes of fever it is evident that bacterial products are by far the most important of these causes, and the much more common occurrence of fever in infancy and childhood is due not alone to the physiological peculiarities of the nervous system of the child, and the prevalence of the predisposing blood factors at this time of life, but it is also largely due to the fact that bacterial infections are much more common in the child than they are in the adult.

The fevers of bacterial origin in *infancy* are largely due to acute and chronic gastro-intestinal toxæmias, and

the fevers of *childhood* are due largely to acute and chronic systemic bacterial toxæmias. These facts should be noted for the purpose of directing attention to the most common causes of fever in the infant and child, but should not obscure the fact that fevers due to other causes may occur at any time during the life of the child.

TREATMENT

It is always well to begin the treatment of any fever with a cathartic, and this is especially true if the patient be an infant or young child. At this period of life we not uncommonly observe the fever and its accompanying symptoms disappear when the intestinal canal has been swept clear of offending matter, but even in those cases where the fever is due to causes entirely apart from the intestinal canal, the cathartic has a very favorable influence in unloading the intestine and preparing it for the special diet which is necessary in all of these cases.

The importance of cathartic medication in the beginning of the treatment of all fevers is important not alone for the purpose of overcoming an intestinal fermentation, or a constipation which may predispose to the development of intestinal toxins, but it is also important for the purpose of removing all reflex intestinal factors which may complicate and aggravate the fever by their influence on the excitable nervous systems of infants and young children.

The intestinal canal of the child should receive attention not only in the beginning of the fever, but it should be carefully guarded by a proper diet, and, if necessary, laxative medication through the course of the disease.

Diet.—A fluid diet free from milk should be prescribed until the cause of the fever is determined. In fact, during the first few hours of the treatment it is better to keep food out of the stomach, and then small quantities of broth, meat juice, cereal-waters, and whisky may commence the dietetic treatment of the fever. On the second day the diet of the patient will depend largely upon the cause of the fever and the age of the patient. If the fever be due to an intestinal toxæmia, or to a systemic infection, such as la grippe or typhoid, with an accompanying intestinal infection, then the diet must be carefully chosen with reference to the control of the intestinal intoxication. In such cases milk, as a rule, is to be excluded from the diet for a number of days, or until the intestinal infection is under control. But if on the second day one finds that the intestinal intoxication is not a factor in producing the fever, then milk becomes an important article of diet, and from this time on the diet should be selected with reference to the age and digestive capacity of the patient, remembering always that both digestion and absorption are somewhat impaired during the fever process, and the patient must, therefore, be carefully protected from taking food in quantity and quality beyond his digestive capacity.

Jacobi says: "In ordinary fevers the food must be liquid and rather cool; in vomiting, cold; in respiratory diseases, warm; in collapse, hot. The best feeding time is the remission; in intermittent fevers nothing must be given during the attack except water, or acidulated water, now and then with an alcoholic stimulant; in septic fevers nothing during a chill, except either cold

or hot water, according to the wishes of the patient, with alcoholic stimulant. Common ephemeral catarrhal fevers may do without food (except water) for a reasonable time. Sleep must not be disturbed, except in conditions of sepsis and depressed brain action. In both there is no sound sleep, but sopor, which should be interrupted. In sepsis (diphtheria and other) this rousing from sopor is an absolute necessity. Unless they are roused frequently to be fed sufficiently and stimulated freely the patients will die. Besides, in most of the cases the temperatures are not high, and there is no contra-indication to feeding on that account. Chronic inflammatory fevers bear and require feeding as generous as it must be careful."

Antipyretics.—The ice-bag, not too closely applied to the head, is, in my opinion, one of the most important of the measures we have for the control of high temperatures in the infant and child. No harm can come from its use if the patient is under the observation of a competent nurse. In very young and frail children it is necessary that the temperature should be taken frequently, so that when it approaches the normal the ice-bag may be removed. In older children the application of the ice-bag does not require such careful watching. The ice-bag is not only a very satisfactory antipyretic measure, but it acts very kindly in the control of the nervous symptoms which accompany fever.

The bath, properly applied, is the most important of all agencies for reducing body temperatures in the fevers of infancy and childhood. It must be remembered, however, that the cold bath cannot be used so indiscrimi-

nately and with such uniform success in children as it can in the adult. Infants and young children, especially if they be frail and nervous, do not stand the cold bath well. Their young nervous systems are so profoundly shocked by this measure that more harm than good comes from it. The character of the bath, therefore, will depend on the age and strength of the child. In young and delicate children a warm or tepid bath, or a sponge bath with alcohol and warm water, will quickly reduce the body temperature. In older and sturdier children colder baths may be given, but it is rarely necessary to use a bath below 80° F. for the control of high temperatures in children. If the baths, as described above, be combined with the intelligent use of the ice-bag, high fevers may be readily controlled.

Phenacetin and antipyrin, in proper doses, are, as a rule, perfectly safe antipyretics in all of the ephemeral fevers of childhood, and their good offices are marked not only in the reduction of the body temperature, but also in the control of the nervous symptoms, the child commonly falling asleep under their influence.

It must be remembered that the efficiency of mild antipyretic measures in reducing high temperatures in infancy and childhood is due to the fact that the heat-dissipating mechanism in the young child is very efficient and responds readily to slight exciting causes. In Chapter III the remarkable efficiency of this mechanism has been studied from a physiological standpoint.

In the treatment of fever, older children should be kept in bed, and at all ages the patient should be kept as quiet as possible, avoiding all excitement.

CHAPTER XI

ECLAMPSIA IN INFANTS AND CHILDREN

A convulsion is a sudden discharge of motor nerve force, resulting in violent and rapid muscular contractions of one or more parts of the body. It is not a distinct disease, but a symptom group which may be produced by a great variety of causes.

ETIOLOGY

Predisposing Causes.—Age.—Infants during the first few months of life have comparative immunity from eclampsia, but from the fourth month to the third year they are especially predisposed to all kinds of convulsive disorders. In the third year of life convulsions become less frequent, and from this time on in the normally developed child they are but slightly more common than they are in the adult. In Chapter II I have detailed the physiological peculiarities of the nervous system which explain the varying predisposition of the infant and young child to convulsive disorders. These physiological facts are, briefly, as follows:

The stimulation of cortical motor centers, and of the convulsive centers at the base of the brain, cannot so readily produce convulsive disorders in the very young infant, because the discharge of nerve force from these centers is not readily communicated to the spinal reflex

centers, since at this early period of life the fibers of the pyramidal tracts have not fully developed their myeline sheaths, and are not therefore capable of readily transmitting impulses from the convulsive centers to the spinal cells. The development of these myeline sheaths, however, gradually goes on, so that the pyramidal tracts have their functions sufficiently developed to place the spinal cells and the cerebral convulsive centers in close touch by the time the child is three or four months of age.

The frequency of convulsions from this time on to the end of the second year of life is due to the fact that all of the nerve centers of the infant, including the cortical motor centers and the convulsive centers at the base of the brain, are much more irritable, and therefore much more easily excited than they are later in life. At this period a mild exciting cause acting upon these centers may cause them to send a severe convulsive discharge through the now developed pyramidal tracts into the spinal cells. And owing to the fact that feeble inhibitory centers are exercising but a mild restraining influence over spinal reflex movements, we have as a result of the discharge of this force into the spinal cells an "overflow" of reflex movements spreading up and down the cord, producing more or less general convulsions.

Later in the life of the child convulsive disorders are less common because the whole nervous system, including the convulsive centers, is now less irritable and more stable, and the convulsive centers at the base of the brain and the spinal cord reflexes are under better inhibitory control.

The above physiological facts are a sufficient explana-

tion for the varying frequency of convulsive disorders at different periods in the life of the infant and child. It should, however, be noted that the comparative immunity which young infants have from convulsive disorders is in part due to the fact that at this period of life the acute systemic bacterial toxæmias, and the gastro-intestinal toxæmias, which are such potent factors in producing eclampsia in infants and children, are, especially in young breast-fed infants, comparatively infrequent. This partial immunity from the acute infectious diseases protects them against some of the most potent factors of the convulsive neuroses.

Heredity.—That the tendency to convulsive disorders may be a direct inheritance is indicated by the fact that now and then whole families of children will be found who have convulsions upon the slightest provocation. It is not improbable that the hereditary tendencies in such families is due to the direct inheritance of an abnormally feeble inhibitory control of convulsive centers. On the other hand there may be a general neurotic inheritance of unstable and irritable nerve centers from neurotic ancestors which may predispose the child not only to convulsive disorders, but to all kinds of functional nervous diseases.

Rachitis plays such an important rôle in the etiology of infantile convulsions that it is sometimes classed as a direct rather than an exciting cause. It matters little, however, whether rachitis be classed as a direct or predisposing factor so long as one remembers the close etiological relationship which exists between this condition and infantile convulsions. So close, indeed, is this

connection that convulsions during infancy should always lead to a careful search for other signs of rachitis.

It has been asserted that this close relationship is due to the fact that the cranial bones in rachitis are in a state of hyperæmia, and, on the other hand, it has been apparently demonstrated that there is an acute anæmia of the brain during convulsions. My own belief, however, is that rachitis is a predisposing rather than an exciting factor, but that it is such a powerful predisposing factor that in advanced cases of this disease the infant's nervous system is in such a state of extreme irritability, and the basal convulsive centers and the cord centers are under such feeble inhibitory control, that even a slight irritation, toxic or reflex, which under normal conditions would be harmless, is sufficient to produce general convulsions. In examples of this kind, rachitis, while acting as a predisposing factor, is really the all-important factor in producing the eclampsia.

Rachitis predisposes to neurotic disease in general, and to convulsive disorders in particular, because it is the most common and the most profound form of malnutrition which occurs in infancy, and because these evil influences are brought to bear on the unstable and immature nervous system while important functions, such as inhibition, are being developed. In rachitis, as noted in Chapter VII, the nerve and other tissues are suffering from an albumin, fat, calcium, and oxygen starvation, and this profound starvation of nerve elements occurs, as a rule, during the first and second year of life, when the nervous system is most immature and most in need of good food and normal conditions for

structural and functional development. Little wonder, then, that rachitis exaggerates all the physiological weaknesses of the infantile nervous system, and still further predisposes the child of this age to convulsive disorders, by increasing enormously the excitability of the motor centers in the brain and cord, and still further weakening the inhibitory control which the higher centers should exercise over the convulsive centers at the base of the brain and the motor centers in the spinal cord.

The above hypothesis, I believe, explains the relationship which exists between infantile convulsions and rachitis, and also determines the advisability of classifying rachitis as a predisposing rather than as a direct exciting factor. The degree of predisposition may vary with the extent and severity of the rachitic process, from a condition of the nervous system but slightly removed from the normal to one in which the nervous system has become so unstable that even a slight excitant will produce a severe convulsion.

I have discussed the relationship of rachitis to convulsive disorders, not only because this disease is itself such an important factor of convulsions in infancy, but also because it may be taken as a type of other forms of malnutrition, to explain how profound nutritional changes may be related to convulsive disorders and other neuroses of infancy and childhood.

Among the other conditions which produce profound malnutrition in the infant, and which act as strong predisposing factors to convulsions, and neurotic disease in general, may be mentioned hereditary syphilis, lymph node tuberculosis, chronic malaria, scurvy, chronic

gastro-enteritis, and all other diseases producing chronic anæmia. As contributing factors to the chronic malnutritions which are present in all these diseases may be mentioned improper food, bad air, unhygienic surroundings, and absence of sunlight.

Chronic reflex irritation as a predisposing factor of convulsive disorders and other neuroses of infancy and childhood is a subject which deserves special consideration. In Chapter VIII I have detailed at length the profound influences which chronic reflex irritation may have upon spinal-cord cells. These cells, under the constant irritating influence of nagging reflexes, show marked pathological changes, and are put in a condition of extreme chronic nervous irritability. This condition, which may be spoken of as an irritable cord, is not an uncommon one in infants, and predisposes them to all forms of nervous disorders which have their manifestations through the discharge of force from the spinal motor cells.

Among the reflex causes which may bring about this chronic irritability of the nervous system may be mentioned adherent prepuce, diseases of the rectum, bladder, and naso-pharynx, and, in older children, eye-strain.

Exciting Causes.—Acute bacterial toxæmia is by far the most common exciting cause of infantile convulsions. Soluble products of bacteria, capable of producing convulsions by their action on the convulsive centers at the base of the brain, may be formed within the blood and tissues, as in the acute infectious diseases so common in childhood; or they may be formed within the intestinal canal,

as in the acute gastro-intestinal infections so common in infancy. Since intoxications from bacterial poisons are by far the most common causes of eclampsia in infancy and childhood, and since the intestinal form of this bacterial intoxication is very much more common in the infant, and the systemic form rather more common in childhood, it follows that convulsions in infancy should suggest to the clinician acute intestinal toxæmia, and convulsions in childhood should suggest the onset of some acute systemic bacterial infection. These suggestions, however, do not imply that gastro-intestinal toxæmia may not in rare instances produce convulsions even in older children,[1] and that acute systemic bacterial infection may not occur, and be ushered in by convulsions, even during the first year of life. It simply calls attention to the very important clinical fact that acute intestinal toxæmia is such an important factor in producing convulsions during the first and second year of life that in the early treatment of these cases the physician is justified in suspecting this cause where no other palpable cause presents, and it further calls attention to the fact that systemic bacterial infection is such an important factor in producing convulsions in childhood that the physician is also justified in suspecting, and is especially called upon to search for, other signs of acute systemic bacterial infection.

Pneumonia, scarlet-fever, measles, and polio-myelitis, may be ushered in by convulsions, or the convulsions may occur later in these diseases. In severe types of malaria the convulsion may take the place of the chill.

[1] See the chapter on "Gastro-intestinal Toxæmia."

Pertussis, occurring in the first and second year of life, is not an infrequent cause of convulsions. Holt says that several factors may be present in producing these convulsions: "Asphyxia, due to a severe paroxysm, cerebral congestion, or hemorrhage resulting from such a paroxysm, or simply the peculiar susceptibility of the patient, brought about by the disease itself."

Uræmia is a factor always to be looked for in the convulsions of childhood, especially if the child has, or recently had influenza, scarlet fever, diphtheria, or other of the acute infections.

Hemorrhage, if severe, may be a cause of convulsions by producing an acute anæmia of the nervous system.

Insulation is a not uncommon cause of convulsions in infants. It probably acts by still further weakening the feeble inhibition of the infant, and by the high fever which it produces.

Asphyxia, produced by any cause, may produce convulsions, especially in the young and feeble infant.

Reflex Factors.—I have already noted the influence of chronic reflex irritation as a predisposing factor to convulsive disorders. This, I believe, is the most important rôle which these factors play in the production of neurotic disease. It should be noted, however, that severe reflex irritation, such as may be produced by undigested food, worms, and other irritants in the intestinal canal of susceptible infants whose nervous systems have been rendered extremely irritable and unstable by chronic malnutrition, may produce convulsive disorders. In such instances as this, however, the reflex factors would be powerless to produce neurotic

disease, if the nervous system had not been prepared for these explosions by powerful predisposing factors.

The cutting of teeth in highly nervous and malnourished children suffering from rachitis or other forms of malnutrition may also produce severe nervous symptoms, and possibly at times may be the slight exciting factor which touches off the highly irritable convulsive centers. It must always be kept in mind, however, that when reflex excitations are capable of producing such profound nervous symptoms as convulsions, there are other powerful and contributing factors which must be discovered.

Epilepsy, which is a symptom group characterized by recurring convulsions, must always be suspected when convulsions are repeated from time to time without apparent cause. It must also, however, be kept in mind that infants having repeated convulsions from any of the causes previously named have a predisposition to convulsions which may disappear before they reach the age of six years. The retardation of development of inhibitory centers may explain these cases. Recurring convulsions, therefore, do not always mean that the child is to develop into an epileptic, even though the convulsions be repeated from time to time from slight exciting causes up to the fifth or sixth year.

Convulsions may also be produced by organic lesions pressing on or irritating the cerebral motor centers. Among such causes may be mentioned meningeal hemorrhage, meningitis, tumor, abscess, hydrocephalus, embolism, thrombosis, enlargement of the thymus, and injury to the skull or brain. The rôle, however, which

these organic lesions play in producing general convulsions in infants and children is slight as compared with the other non-organic factors previously detailed. Moreover, these organic lesions should not be discussed under the present heading, except for differential diagnosis.

SYMPTOMATOLOGY

Eclampsia is a syndrome, and not a disease. Healthy children, however, do not suffer from convulsions. This symptom group always means some serious acute or chronic disease, the nature of which must be determined by other symptoms and the general history of the case. Notwithstanding this fact, the importance of this syndrome demands that it should have separate consideration.

There are ofttimes premonitory signs which indicate that eclampsia is threatened, such as sudden twitchings of the muscles of the arms, legs, or face. These manifestations occur especially while the child is asleep. In many instances the physician will be called, not because the little patient has fever or intestinal disturbances, but because the mother has become alarmed at the occasional sudden jerkings or spasmodic contractions of the legs, arms, or face of her sleeping child. The child may continue to toss restlessly in its sleep for a time, and then without awakening pass suddenly into a general convulsion. These premonitory symptoms are more likely to occur during sleep, because in this condition voluntary inhibition is no longer active, and the excited motor centers for this reason the more readily respond

to very slight reflexes, such as sudden noises and movements of the bed-clothes.

In a large number of instances, however, possibly in the majority, the convulsive storm occurs without warning out of a clear sky. A sudden pallor of the face is followed by a convulsive stiffening of the muscles, the eyes roll up and become fixed, spasmodic contraction of muscles occur; these clonic contractions may almost immediately become tonic, producing rigidity of the entire body; the face is distorted, the head drawn to one side, the hands are clinched upon the thumbs. Very shortly clonic convulsive jerkings of the head and extremities supervene, and these severe spasmodic movements continue for three or four minutes, leaving the child relaxed, exhausted, and in a condition of more or less profound sleep, from which it may awake bright and conscious or without awakening may pass into a second convulsion. The sleep which follows the convulsion is ofttimes so profound as to resemble stupor or coma, and when the convulsions recur at short intervals the unconsciousness which always attends them is continued through the interval, producing a profound coma. The number of convulsions in any case will depend on the character and severity of the disease of which they are a symptom, and upon the physician's ability to remove or control the immediate exciting cause of the convulsive seizure. In the ordinary eclampsia of infancy, however, the patient on awakening from the sleep which follows the convulsion is, as a rule, bright and conscious, and gives little evidence of the severe nervous storm through which it has just passed.

During the convulsion incontinence of urine and fæces is the rule; there may be spasm of the respiratory muscles, the breathing may be shallow, irregular, and spasmodic, a choking sound may be produced in the larynx; more or less cyanosis may occur, and in severe cases life may be threatened by asphyxia.

It must not be understood, however, that the entire symptom group above presented will occur in every case of eclampsia. The severity of the convulsive seizure may vary from a momentary unconsciousness, with slight twitching of some portion of the body, to a general convulsion so severe as to take the life of the child, and comprehending in its symptomatology the entire syndrome above presented. Unconsciousness and clonic or tonic muscular contractions (be they ever so slight) are the only necessary symptoms of eclampsia.

Convulsions may be general, involving the whole body, or partial, involving only a member, or they may begin in a member and then become more or less general.

One attack of eclampsia does not predispose to another unless some organic injury to the nervous centers results from the convulsive seizure. The same predisposing causes, however, which made possible the first convulsion may account for subsequent attacks from slight exciting causes.

PROGNOSIS

Age is a very important factor in prognosis. Eclampsia is a very serious condition when it occurs during the early days of the life of the child. It is very much less serious when it occurs between the end of the third month and the second year of life. It is again more

serious in the third year of life, and becomes more so as the child grows older.

In the newly born the prognosis is bad, because only serious brain lesions, such as cerebral hemorrhage or congenital defects, are likely to produce general convulsions at this age. For these reasons convulsive disorders which have their origin during the first week of life have a very serious prognosis, since spastic palsies or epilepsy commonly supervene.

After the first week of life, when the infant has recovered from the accidents of birth and has become accustomed to its new surroundings, convulsions are rather uncommon until after the third month of life. If, however, convulsions should begin between the second week and the fourth month of life, while they are not so ominous in their import as those occurring during the first week, yet they are more serious than those that occur during infancy *after* the third month, because the nervous system has not yet sufficiently developed to predispose the infant to convulsive disorders, and it therefore requires some severe form of irritation (organic or toxic) to produce convulsive disorders.

Between the fourth month and the end of the second year, as has been previously noted, is the time when convulsions most readily occur and when the prognosis is at the best. At this period of life, however, convulsions may be fatal, or may herald some serious disease; yet in the great majority of instances they are from comparatively trivial and quickly removable causes, and for these reasons the prognosis is especially good.

After the second year, as the growth and functional

development of the nervous system, in the normal child, gradually removes the predisposition to convulsive disorders, eclampsia requires for its production more potent etiological factors, has a graver prognosis, and becomes less and less frequent, so that at the age of five or six years it suggests some severe constitutional intoxication, such as uræmia, or one of the acute infectious diseases, such as meningitis, scarlet fever, or pneumonia.

Apart from the age of the child there are a number of indications which assist us in making an early prognosis.

1. A severe initial convulsion, deep supervening coma, and a tendency to tonic contractions in the intervals between the convulsions suggest a most unfavorable prognosis, not only so far as the ultimate recovery of the child is concerned, but also as to its immediately perilous condition.

2. Partial convulsions preceding general convulsions, and possibly continuing in the interval, suggest serious brain lesions of the cortex, and speak against the ultimate complete recovery of the patient; partial convulsions, however, may occur from non-organic causes.

3. Severe general convulsions recurring at intervals without apparent exciting causes, especially if the patient has inherited a strongly neurotic temperament, should make one apprehensive that the child's predisposition to convulsive disorders is so great that epilepsy may develop.

4. Convulsions occurring after traumatic injury to the head are not infrequently followed by true epilepsy or serious structural diseases of the brain.

5. Convulsions characterized by profound cyanosis and spasmodic breathing may produce such dangerous complications as asphyxia and cortical cerebral hemorrhage.

6. Prolongation of convulsive seizures adds to the seriousness of the prognosis not only in the greater immediate danger to life, but also in the fact that it indicates a more serious exciting cause which may threaten the future welfare of the child.

7. To make an early definite prognosis is unwise because of its uncertainty. Even if one has an accurate knowledge of prognostic indications, a careful study of an infant that has but just recovered from its first convulsive seizure will not furnish the data for a safe prognosis. At this time the prognosis should be provisional, awaiting further developments to determine the causes which have produced, or the results which may follow, the convulsive seizure, since, after all, the character of the disease which produces the convulsion is the most important factor in prognosis. Pertussis and advanced rachitis, however, are among the grave etiological factors which may be determined or excluded at once.

DIAGNOSIS

The diagnosis in eclampsia comprehends not alone the fact that the child has convulsions, but also the character and cause of the convulsions.

The diagnosis of convulsions is easily made, and is rarely if ever mistaken for any other syndrome, and the differential diagnosis as to the characteristics of individual convulsions has been sufficiently dwelt upon under

Prognosis. There yet remains, however, something to be said on the importance of determining the cause of the convulsion, since upon this phase of the diagnosis depends all rational treatment.

It may aid us in making the differential diagnosis of the disease or diseases responsible for the convulsive seizure to remember that all eclampsias in young children may be placed in one of the four following groups, named in order of their etiological importance:

1. Those produced by acute systemic intoxications from bacterial toxins, as in the acute infections, especially those from the intestinal tract, or from autotoxins, as in uræmia.

2. Those produced by chronic malnutrition (rachitis, etc.), or powerful hereditary factors (feeble inhibition, etc.), plus some slight exciting causes, such as fright, teething, or undigested food.

3. Those produced by organic diseases of the nervous system, such as cerebral hemorrhage and meningitis.

4. Those produced by an epilepsy, with no apparent anatomical basis.

In determining to which of these four groups the eclampsia belongs, one must make a comprehensive study of the individual case, including age, hereditary tendencies, previous condition, character of convulsions, surrounding circumstances, and especially all accessory symptoms pointing to the nature of the present illness.

Since systemic intoxications are by far the most important of the etiological factors, a careful study should be made of the child's present and past intestinal condition. Intestinal fermentation and disease of the

digestive tract are most important. Symptoms of the specific infectious diseases should be looked for and the urine must always be examined.

Failing to find indications pointing to acute systemic intoxication, the infant should be examined for rachitis and other forms of malnutrition, and its hereditary tendencies should be investigated on the suspicion that some slight exciting factor, aided by these powerful predisposing factors, may have been the cause of the convulsions.

Failing to discover a satisfactory explanation for the convulsive seizures, organic disease of the nervous system should be suspected. If the convulsions occur during the first few days of life, cerebral hemorrhage is a probable cause; but if the convulsions occur later, meningitis or other organic nervous diseases may be suspected.

Lastly, no other exciting cause being apparent, epilepsy may be suspected in those cases where the convulsions have been repeated from time to time without apparent cause.

TREATMENT

In the majority of instances convulsions are self-limited, and last such a short time that the physician is not called upon to resort to any very active measures to control the convulsive movements. In these milder cases his energies must be chiefly directed to preventing a recurrence of the convulsive attack.

It should be remembered, however, that in not a small percentage of the cases the convulsion itself is a source of danger, not only to life, but also to the subsequent well-being of the child, and that the longer the convulsion

lasts, the greater are these dangers. It is the all-important duty, therefore, of the physician to terminate the convulsion as soon as possible, regardless of its cause. This may be done by inhalations of chloroform. The convulsive movements quickly disappear when a few drops of chloroform are placed upon a handkerchief and held to the child's nose. The administration of chloroform may be repeated at any time for the purpose of cutting short the return of convulsive movements, and the chloroform treatment is to be continued until the convulsive movements cease or have been brought under the control of other remedies.

As soon as the convulsions have subsided under the first inhalations of chloroform the child is placed in a bath, the temperature of which will depend upon a number of conditions. If the child have high fever, begin with a lukewarm bath and cool down to 80° F. This not only reduces the body temperature, but exerts a soothing and tonic effect upon the nervous system. If, however, the patient be a very young or a very delicate infant, the bath is not to be cooled below 90° or 95° F. Some writers deprecate the efficiency of the bath in the treatment of convulsions; for my own part, I am, with the laity, a firm believer in the warm-bath treatment of convulsions in infants and children. It reduces fever, promotes the action of the skin and kidneys, and exercises a sedative and tonic influence upon the nervous system, and for these reasons it justly holds a high place in the routine treatment of eclampsia.

Immediately the child is taken from the bath, an ice-cap should be applied to the head. This application

of cold to the head helps to keep down the temperature and acts as a sedative to the nervous system. The bath and ice-cap are to be used in the subsequent treatment of the case if high fever and nervous symptoms demand their use.

A cathartic should be given as soon as possible in the treatment of infantile convulsions, regardless of their cause. The selection of the cathartic will depend upon the condition of the child's stomach; castor-oil is to be preferred if the stomach will retain it; if not, calomel is to be given in quarter of a grain doses every half-hour, until one or one and a half grains are given, according to the age of the child. The importance of the cathartic in the treatment of infantile convulsions does not depend wholly upon the fact that gastro-intestinal toxæmia is the most important etiological factor of convulsions, for, even in those cases which have their origin entirely apart from the intestinal tract, it is important in the treatment of the case that this canal be unloaded to prepare for the special dietetic treatment that is necessary in the subsequent management of almost all of these cases.

A high rectal enema of a pint or more of physiological salt solution should be given immediately or very soon after removing the child from the bath. The object of this is to unload and irrigate the large intestine, so as to remove any possible source of irritation and prepare it to receive medicines which it may not be possible to give by the mouth.

Chloral hydrate is the best and safest of all remedies used for the control of convulsive movements. It should

be given dissolved in starch water by high rectal enema half an hour after the lower bowel has been washed out. The dose for a child of six months is five grains, and for a child of two years, ten grains. If the chloral be given, however, by the mouth, these doses are to be cut in half. In the beginning of the treatment it is advisable to give the chloral by rectum rather than by mouth, even if the child can swallow, since it is most important that the cathartic which has been given should be retained. It is wise, therefore, not to risk upsetting the stomach either with food, stimulants, or medicines until the cathartic has acted. If the choral is retained by rectum for half an hour, and the convulsive movements are under control, the physician may safely leave the case for the time being in the hands of a competent nurse, with directions that the chloral injections be repeated in one or two hours, if necessary. After twelve or twenty-four hours the child is, as a rule, able to take by the mouth small doses of chloral combined with four or five grain doses of bromide of potash. It is not necessary, however, to continue this sedative treatment for any great length of time, as thirty-six hours usually suffices for the removal of all indications for sedative treatment.

Morphine is the most certain of all the remedies we possess for the control of convulsions. A remedy, however, which acts so powerfully must be used cautiously and in the proper dosage, and only when the other measures above outlined have failed to control the convulsive movements.

In not a few instances the chloral is not retained by

the rectum. In others the eclampsia may be so severe that the chloral fails to act. In such instances the child may have to be kept constantly under the influence of chloroform to control the convulsions. In these cases morphine is the safest remedy. It should be given hypodermically, in doses varying from a fiftieth of a grain, for a child six months of age, to a twentieth of a grain, for a child two years of age. These doses are perfectly safe, and they act specifically in the control of convulsive movements. The dose may be repeated in an hour, and thereafter as necessary. It is rarely necessary, however, to give more than one injection of morphine. After this, as a rule, the convulsions may be controlled by the other remedies above named. If in very severe eclampsia which requires morphine for the control of the convulsive symptoms, a prolonged period of coma or unconsciousness should follow the use of the morphine, it is advisable, especially in older children, to resort to venesection, followed by the injection into the vein, or subcutaneous tissues, of half a pint or more of sterile normal salt solution. This treatment, especially in uræmic poisoning, is frequently followed by a return of the child to consciousness.

Absolute quiet for the nervous system and rest for the stomach are necessary during the first few hours, in the treatment of eclampsia. Food and stimulants by the mouth should be avoided until the intestinal canal has been unloaded. If, during this time, the child's condition demand stimulation, a rectal or subcutaneous injection of normal salt solution is the most effective remedy we have.

During the early treatment of the case, following the control of the eclampsia, the child may be allowed water, barley water, or weak beef broth, provided it craves something to drink.

After the cathartic has acted, and the cause of the eclampsia has been ascertained, the case is to be treated with reference to the control of the disease which caused the convulsion. If the trouble be of intestinal origin, as is the rule, then a period of careful feeding must follow. If nutritional disorder is an underlying cause, then the treatment must be directed towards the cure of the special form of malnutrition which is present. If the eclampsia be due to organic disease of the nervous system, the subsequent history of the case must determine the treatment of the existing disease.

CHAPTER XII

LARYNGISMUS STRIDULUS.

Synonyms.—Cerebral croup, child-crowing, inward spasms.

Definition.—Laryngismus stridulus is a reflex neurosis rarely observed outside of foundling hospitals and similar institutions for the care of infants. It is characterized by spasm of respiratory muscles and especially of the adductor muscles of the larynx, which results in a sudden closure of the glottis, with a temporary shutting off of air from the lungs.

ETIOLOGY

Rachitis is by far the most important etiological factor in the production of this syndrome. All observers are agreed that there is a close relationship between rachitis and laryngismus stridulus.

Kassowitz found in three hundred and seventy cases well-marked evidences of rachitis in all but one, and in 87 per cent. of these cases he found a well-marked craniotabes. Other writers, among them Jacobi, have called attention to the relationship of craniotabes to laryngismus stridulus.

While the close association of rachitis, craniotabes, and laryngismus stridulus is recognized by all writers, it is

not at all certain that craniotabes or any other one manifestation of rachitis is directly responsible for the spasm of the glottis.

This symptom group is more especially associated with the acute advanced form of rachitis in which craniotabes is so common, and in which there is also a more marked irritability of the nervous centers than can be found in any other form of malnutrition occurring in infancy. This extreme excitability of the nervous centers, which makes it possible for slight reflex factors to bring on a spasm of the glottis, is the all-important etiological factor of laryngismus stridulus.

Lymph node tuberculosis producing a profound malnutrition and irritability of nerve centers may also be an etiological factor in the production of this syndrome. It may be associated with rachitis or with other forms of malnutrition in producing the irritability of the nervous centers, which is the underlying cause of this symptom group.

Enlarged lymph nodes pressing on the recurrent laryngeal nerve may also be an exciting cause of the paroxysm.

The malnutrition produced by hereditary syphilis in premature infants, as well as the malnutritions produced by long-continued gastro-enteritis, may also bring about an irritability of the nervous centers which predisposes the infant to laryngeal spasm.

Clinicians in studying the malnutritions associated with laryngismus stridulus may easily overlook a concealed tuberculosis or hereditary syphilis and report only the presence of certain rachitic symptoms, which may or

may not explain the full character of the malnutrition. I do not wish to convey the opinion that rachitis is not the most important factor in producing laryngismus stridulus, but I do wish to insist that there are other forms of chronic malnutrition which are not uncommonly associated with this disease.

Age.—Laryngismus stridulus occurs as a rule between the sixth and the eighteenth month, just at the period of life when rachitis, lymph node tuberculosis, hereditary syphilis, and gastro-intestinal diseases are most common, and when these diseases produce the most extreme irritability of the nervous system. This is also the period in the life of the child when, on account of the lack of inhibition, the convulsive neuroses are most common.

Season.—This symptom group occurs most commonly during the months of January, February, and March. These are the months during which infants are most commonly confined to hot, illy-ventilated rooms, and it is during these months that bad air, absence of sunlight, and bad hygiene contribute to the malnutritions above noted.

Reflex Factors.—Among the reflex causes of laryngismus stridulus may be mentioned stomach indigestion acting through the vagus, enlarged lymph nodes acting through the recurrent laryngeal nerve. It is also possible that mucus or foreign substances which may accidentally drop into the larynx may excite a paroxysm. The cutting of a tooth, fright, anger, enlarged tonsils, adenoids, and nasal irritation have also been mentioned as exciting causes.

SYMPTOMS

A nervous child suffering from some profound malnutrition may, with little or no warning, be seized in the early hours of the night with a spasm of the glottis, the adductor muscles playing the most important rôle in this spasm. The closure of the glottis completely shuts off inspiration. As the glottis is closing the child sometimes in its struggles gives vent to a strident noise produced by the rushing in of air before the stricture of the glottis is complete. With the shutting off of air the child struggles for breath, and its face soon becomes cyanotic, the head is thrown back, convulsive movements of the diaphragm occur, the body stiffens, and the child's life seems in imminent danger, when suddenly a loud crowing inspiration announces the fact that the spasm has relaxed and all immediate danger is over. It is the strident crowing sound that marks the close of the paroxysm which characterizes the symptom group and gives it its name.

Following this strident inspiration the child breathes rapidly, is greatly excited, cries and frets, and finally falls asleep, possibly to be awakened some hours later with a second attack.

The spasm of the glottis which produces these paroxysms lasts only twenty or thirty seconds; if it lasts longer the child is in great danger from asphyxia or general convulsions.

Second and third attacks almost always occur within a few hours after the first attack, and in severe cases the

child may have a dozen or more paroxysms in twenty-four hours.

Convulsive movements of the diaphragm and other muscles of respiration are, as a rule, a part of the attack.

Carpo-pedal spasm, which is one of the classical symptoms of tetany, is so commonly associated with laryngismus stridulus that some writers, among them Cheadle, believe the two conditions to be the same. In about one-half the cases of laryngismus stridulus the fingers and toes are spasmodically flexed, just as they are in tetany, but otherwise the two syndromes differ widely.

Eclampsia occurs in about one-third of the cases, the general convulsions supervening as the laryngeal spasm relaxes.

An attack of laryngismus stridulus may occur at any time during the day or night, but the first attack of the series most commonly occurs during the most profound sleep in the early hours of the night.

Holding the breath spells, which occur in older children, are closely allied to but not identical with laryngismus stridulus. In this condition the spasm of the larynx is usually brought on by a fit of anger.

Spasms of the larynx occur also in acute laryngitis, whooping cough, and other diseases, but the clinical pictures they produce are quite different from that of laryngismus stridulus.

PROGNOSIS

Prognosis is good so far as the paroxysm is concerned, and if the underlying malnutrition can be successfully treated, then the prognosis, so far as ultimate recovery,

is also good. Some of the more severe cases die from asphyxia or general convulsions.

TREATMENT

Treatment of the Attack.—The child should be taken up and cold water dashed into the face, or cold wet towels applied to the chest. If this does not relieve the paroxysm, chloroform may be given by inhalation. In the vast majority of instances, however, the physician never has an opportunity to personally supervise the treatment of the attack. After one attack has occurred, however, he may leave directions that subsequent attacks are to be treated with cold water and chloroform.

Prevention of the Attack.—For the first twenty-four hours the child is to be kept somewhat under the influence of chloral, one or two grains every two or three hours. After the first twenty-four hours the bromides may be used; strontium bromide in three- to five-grain doses every four hours is to be preferred.

Treatment of the Underlying Causative Condition.—This is the all-important part of the treatment, and should be followed up until complete recovery takes place. To accomplish this may require years.

The special treatment indicated will depend entirely upon the character of the underlying malnutrition, but the most important part of that treatment will be dietetic and hygienic. The infant must have a carefully selected diet suited to its age and digestive capacity. It must also live in the open air and sunlight as much as possible. Cod-liver oil and other tonics may enter into the treatment. If the child has any disease of the naso-pharynx

or throat, or any other source of reflex irritation, these conditions must be treated and removed.

The paroxysm of laryngismus stridulus may indirectly be instrumental in saving the life of the child, in that the profound malnutrition from which the infant is suffering is thus brought to the attention of the physician, who recognizes the importance of the underlying causes which have produced this alarming local spasm.

CHAPTER XIII

TETANY—IN CHILDHOOD

Tetany is a neurosis characterized by tonic contractures of muscles. These contractures may be intermittent, but as a rule they are persistent and subject to exacerbations at irregular intervals. The favorite site for these contractures is in the extremities; the muscles of the trunk, neck, and face may also be affected.

ETIOLOGY AND PATHOLOGY

Tetany occurs with far greater frequency in Europe than in this country. The consensus of opinion is that tetany is comparatively a rare disease in America. The epidemic form of the disease has not been noted here. Koplik says that the disease is not a rare one in New York. He has observed in his clinic a number of cases yearly. They appear in groups in the early spring months.

Holt seldom sees more than four or five cases a year in a large hospital service in New York. Morse says the disease is a very unusual one in Boston, as shown by the statistics of the Medical Out-Patient Department of the Infants' Hospital, where 7114 cases of disease were treated during the years 1896 and 1897, and among them was one case of tetany. In my own experience

in the Cincinnati Hospital the disease is rare. In the average not more than one case occurs in this institution in a year.

Griffith found that 72 cases (some of these doubtful) had been reported in American medical literature up to 1894, and Morse, from 1894 to 1897, inclusive, found 13 additional cases; while Griffith himself reports 5 cases, and Morse 6. The reported cases do not give a very accurate estimate of the number of cases that actually occur, yet these figures justify the conclusion that tetany is a rather rare disease in America, and that in this country it occurs perhaps much more frequently in New York than elsewhere.

Age.—Tetany may occur at any age, but it is much more frequent in infancy About 50 per cent. of the reported cases are under two years of age. Of the 95 cases collected and reported by Griffith and Morse, 38 were under one year of age, 8 were in the second year of life, 15 were between two years of age and puberty, and 34 above puberty. From this it appears that in America tetany is vastly more common in the first year of life than it is at any other period, two-fifths of all the reported cases being under one year of age. During the second year the disease is much less common than during the first year, yet it is still much more frequently observed during this period than in any subsequent year of the life of the individual. After the fourth year cases occur more frequently about the period of puberty than at any other time.

The study of tetany in childhood is therefore largely a study of this disease as it occurs during the first year

of life, and the reasons for its more common occurrence during this period are found in the facts that gastro-intestinal diseases, rachitis, and other forms of severe malnutrition are most common at this time.

The changes in the nervous system which produce the syndrome of tetany are not known, and for this reason it is classed as a neurosis. We do know, however, that whatever may be the character of these changes, they are temporary, since the great majority of cases of tetany terminate in complete recovery. Among the lesions of the nervous system which have been found in patients who have died during an attack of tetany are hydrocephalus, hyperæmia and œdema of the brain, and hyperæmia; and a slight degree of poliomyelitis of the cord, especially the cervical portion, in which the motor cells of the anterior horns may show changes not unlike those noted under "fatigue" changes in the chapter on Reflex Irritation. These changes are not characteristic, neither are they constant. Many observers have failed to find anything noteworthy in the nervous system, and such changes as those above recorded may in large part be due to the long-continued action of toxins on the nerve centers.

In the present state of our knowledge the action of toxins on the nerve centers is the most plausible theory of the production of the syndrome of tetany as it occurs in the young infant. It is probable, however, that the mechanism of the production of this syndrome is not always the same. Some of the cases of tetany occurring in the adult cannot be explained in this way. The epidemic form of this disease which has been described,

especially by French writers, lends support to its toxic origin.

Gastro-intestinal toxæmia is perhaps the most important etiological factor in producing infantile tetany. Gastro-intestinal disturbances are present in nearly every case occurring during the first two years of life, and even in the adult dilatation of the stomach and intestinal disease are spoken of as causal conditions. In about 75 per cent. of all cases of tetany, including the adult cases, gastro-intestinal disturbances are present.

Gastro-intestinal disease, occurring during the first two years of life, not only subjects the nervous system to constant irritation and poisoning by intestinal toxins, but it also produces a general profound malnutrition, which interferes with the development and increases the instability of the infantile nervous system.

Rachitis.—More or less marked evidences of rachitis are found in nearly all infants suffering from tetany. The rachitis, however, in these cases is perhaps a phase of the malnutrition which has been produced by the gastro-intestinal disease and the character of the feeding which this disease has led up to. The rachitis associated with tetany is not commonly of a very severe type, and in this particular it differs from laryngismus stridulus.

Rachitis, however, must be noted as one of the most important etiological factors of tetany, and it matters little whether or not we consider it a secondary malnutrition resulting from intestinal disorders. Other acute infections beside those of the intestinal canal may be responsible for the production of tetany. Cases have

been observed to follow measles, typhoid fever, rheumatism, and pertussis.

Season.—Most of the cases occur during the winter and spring, when the infant living in tenement houses and foundling asylums has been housed and subjected to the influences of bad air, absence of sunlight, and other unhygienic conditions which aggravate the more important causative factors above noted.

Sex.—In all the statistics there is a slight preponderance of males, the proportion being about as 5 : 4. It is a little difficult to understand why a male infant should be more susceptible to this disease than a female, since females, as a rule, are more susceptible to neurotic disease. The explanation, however, may probably be found in the fact that balanitis and adherent prepuce are important reflex factors in developing the disease in the male child. Later in life girls are more susceptible than boys. This is probably due to the reflex influence of menstruation.

Reflex Factors.—It is probable that reflex factors, pure and simple, play a very unimportant rôle in the etiology of tetany. It must be remembered, however, that when the nervous system of the young infant has been placed in a state of extreme irritability by malnutrition and the action of toxins, it requires a minimum reflex excitant to produce rather profound nervous symptoms. Under conditions such as these one can understand how foreign bodies, worms, and undigested food in the intestinal canal, or adherent prepuce and adenoid growths, may be factors sufficient to touch off a paroxysm of tetany.

The epidemic form of this disease, which apparently does not occur in this country, and the form of the disease which follows the removal of the thyroid gland, and the adult type of this disease, will not be discussed here.

SYMPTOMATOLOGY

The most characteristic symptoms of tetany are tonic muscular contractures, which occur in almost any part of the body; but the most common and characteristic locations for these contractures are in the forearms, hands, and feet, producing the characteristic carpo-pedal spasms.

The positions assumed by the hands and feet during the spasm are characteristic: the fingers are flexed at the metacarpo-phalangeal joints, the phalanges are extended, and the thumb is drawn across the palm of the hand. In some instances the phalanges, instead of being extended, are flexed over the thumb, as it crosses the palm of the hand. The wrist is sharply flexed on the arm, and the whole hand is drawn towards the ulnar side. In the more severe cases the forearms are flexed on the arms and pressed against the sides of the thorax.

In moving the elbow the resistance is not so great or so painful as in moving the wrist. In milder cases the shoulder and elbow are freely movable, while the contractures of the wrist and hand are very strong.

The pedal spasm usually accompanies the carpal spasm; the feet are extended, and the first phalanges of the toes are flexed, and the others extended. The foot is curved inward, and the tendo-Achilles is very tense.

The knee and hip-joints are usually free, but in some cases the thighs are adducted.

While these contractures are commonly confined to the forearm, hands, and feet, it is not uncommon in more severe cases, especially those under one year of age, to have contractures of muscles of the trunk and neck, producing opisthotonos and stiffening of the body. I have seen cases of this kind in which the infant's body remained rigid when lifted from the bed by placing one hand under the hips and the other under the occiput. In rare instances the muscles of the face and eyes are involved.

A paroxysm of tetany may continue for a few days, or it may last for weeks, and during this time the muscular contractures are, as a rule, continuous. There may, however, be periods during the paroxysms in which there is a marked remission or even a short intermission of the spasm. When the paroxysm has subsided the child under proper treatment, as a rule, progresses slowly to a satisfactory recovery, and this may occur without relapses. In other cases, however, second and third attacks recur at variable intervals, weeks or months intervening.

Pain, as a rule, accompanies the spasm, especially in the severe cases. It may be severe enough to cause the child to cry out. The pain is greatly increased by any attempt to move the contractured part. Stretching or pressing a contractured muscle will produce pain. There is no loss of consciousness in this disease, unless general convulsions supervene as a complication. While general convulsions are not so common in this disease as they

are in laryngismus stridulus, they may occur, producing one of the most dangerous complications. Œdema of the feet, ankles, and wrists may be present.

Temperature.—There may be an elevation of two or three degrees. This fever, however, does not belong to the tetany as much as it does to the intestinal fermentation, which is nearly always present. When the intestinal condition is properly cared for, tetany is, as a rule, an afebrile disease.

The increased irritability and excitability of peripheral nerves which occurs in tetany is responsible for some of its most characteristic symptoms. The increased nerve and muscle irritability is noted in the increased electrical excitability of both nerves and muscles, with changes in their qualitative reaction to galvanism. It is also shown in the facial phenomenon known as "Chvostek's symptom," where spasm of the facial muscles is produced by percussing over the facial nerve, and by Trousseau's symptom. This remarkable observer noted that in patients suffering from tetany the spasm could be greatly exaggerated by pressure upon the large nerve trunks and arteries of the extremities. All of these phenomena, due to the increased excitability of the peripheral nerves, may be observed not alone during the acute paroxysm, but may be observed in some cases for a considerable time after the muscular contractions have disappeared. In such cases the contractures may be developed in the manner described by Chvostek and Trousseau. So long, therefore, as Trousseau's or Chvostek's symptoms can be elicited, and so long as there is an increased electrical excitability of the muscles,

first noted by Erb, the patient is still to be considered as not thoroughly convalescent from the attack. It is only when all of these evidences of the irritability of peripheral nerves have disappeared that the patient is to be considered convalescent, but even then the danger of second and third attacks is not removed until the underlying intestinal disease and malnutrition have been cured.

DIFFERENTIAL DIAGNOSIS

Tetany is to be differentiated from tetanus by the location of the contractures, and by their intermittency, and especially by the absence of trismus, which is one of the earliest and most characteristic symptoms of tetanus. Trousseau's, Erb's and Chvostek's symptoms are not present in tetanus. The age and previous history will also assist in the differential diagnosis.

TREATMENT

In beginning the treatment of a case of tetany it is absolutely necessary to give close attention to the intestinal canal; calomel, followed by castor oil, will serve the purpose of removing all sources of irritation from the intestine, and prepare the patient for the very careful dietetic treatment that is to follow. The child must be carefully fed with a view not only of correcting the existing malnutrition, but also preventing further intestinal intoxication. The feeding of the child is therefore the all-important part of the treatment. For the control of the spasm, chloral and bromides may be used in

moderate quantities for a short time only. These remedies are to be dispensed with as soon as possible, and only resorted to when the spasms are severe and painful. Luke-warm baths at intervals during the day will not only help in the relief of the spasm, but will benefit the intestinal condition. The child should be given sunlight and fresh air; these are almost as necessary in the treatment of this condition as they are in tuberculosis. As the child improves, cod-liver oil and iron are of great value in overcoming the malnutrition produced by rachitis or other causes. A search should also be made for every possible cause of reflex irritation. The prepuce and rectum should be examined, and as the child convalesces the throat and nose should be inspected. The removal of such reflex factors may facilitate the child's ultimate recovery.

CHAPTER XIV.

ENURESIS

Incontinence of urine in children is a true neurosis, and is not, as a rule, due to muscular incompetency of the sphincter vesicæ. It is commonly associated with other nervous symptoms, with anæmia, and with reflex irritation. This condition, like the other neuroses of childhood, commonly rests upon a tripod of etiological factors, viz.: First, irritable and unstable nerve centers, due to age and heredity; second, bad blood and consequent malnutrition; third, reflex irritation.

In many cases these three factors coexist. We may, however, have incontinence of urine without malnutrition or malnutrition without incontinence. We may also have incontinence without apparent reflex irritation, and very strong reflex excitation, even on the part of the genital organs, without incontinence. It is not wise, therefore, to assume that phimosis, vesical irritation, or some other reflex factor is the sole cause of enuresis, or that general malnutrition, in any individual case, is the sole cause of this condition; nor can it be said that a neurotic inheritance is alone sufficient to produce enuresis, since the great majority of neurotic children do not suffer from this condition. A rational inquiry, therefore, into the etiology of a case of enuresis must inquire into the presence or absence of each of these factors and their relative importance in producing this syndrome. In order

to do this it is necessary to keep in mind the nervous mechanism of micturition.

The longitudinal and circular muscular fibers which by their contraction empty the bladder are enervated by sensory and motor nerves from the lumbar region of the cord, and the external sphincter, in the prostatic portion of the urethra, which by its contraction prevents the escape of urine from the bladder, is also enervated by sensory and motor nerves from the lumbar cord. Von Zeissl's researches on the innervation of the bladder give us a better understanding of this subject. He found that "the erector nerve" was not only the motor nerve of the muscular coat of the bladder, but that it carried inhibitory fibers to the sphincter vesicæ, and that "the hypogastric nerves" carry motor fibers to the sphincter vesicæ, and inhibitory fibers to the muscular coats of the bladder. These researches explain the manner in which reflex excitation may act in starting or checking the flow of urine. For example, a reflex carried to the proper center in the lumbar cord would, through the motor fibers of the erector nerve, contract the muscular coat of the bladder and, through the inhibitory fiber of the same nerve, relax the sphincter vesicæ, and in this manner allow the urine, which is being expelled by the contracting bladder, to pass without hindrance through the relaxed sphincter vesicæ. This is, indeed, a simple and beautiful mechanism that must be understood if we are to estimate the importance of various etiological factors in their play upon this mechanism. Another important fact that should also be mentioned in this connection is that the urination center

in the spinal cord is partly under the inhibitory control of higher centers, including the voluntary centers in the brain cortex. The act of urination is, for this reason, partly under control of the will. We will to urinate or not to urinate, and the message passes down to the centers in the lumbar cord where, by the mechanism just described, the reflex act is completed.

From this sketch of the nervous mechanism of urination it is evident that the etiological factors of incontinence of urine may also be divided, with reference to the manner of their action, into three classes: first, those that act upon the higher centers in the brain, diminishing their inhibitory control over the urination center in the lumbar cord; second, those that act directly on the centers in the lumbar cord, making them more irritable and unstable, and in that way increasing their reflex excitability; third, those that act by reflex irritation indirectly on the spinal centers, touching off the nervous impulses which produce urination.

With this general introduction we are better prepared to consider the coöperation of these factors in producing incontinence of urine.

GENERAL ETIOLOGY

Predisposing Causes.—Age is a most important and little understood predisposing factor of incontinence of urine. We are greatly indebted to Clouston for his careful study of the relationship of neuroses of childhood to the rapid metabolism and growth of brain tissue during this period. In early life the nerve centers are more

excitable by reason of their immaturity, and the great metabolism of nerve tissue incident to its rapid growth and development increases the sensitiveness of the nerve centers and exaggerates reflex phenomena. This, for the most part, is a cause more or less active in all children, and is largely responsible for the prevalence of the neuroses in early life. The importance of age as an etiological factor takes yet greater prominence when one remembers that there is, during childhood, a functional immaturity of the centers inhibiting reflex acts. In early infancy inhibition is so feebly developed that we have during the first year of life a normal incontinence of urine. During this time the urinary center in the spinal cord, being under little or no inhibitory restraint, is excited to action by such very slight reflex causes as a small quantity of urine in the bladder. As the child grows older the mechanism inhibiting reflex acts becomes better developed, and, as a result, in the second year of life the normal incontinence of urine gradually disappears. Delayed development, however, or other pathological factors may continue the incontinence of urine into the third year of life. When this occurs, the condition is considered pathological. Enuresis may continue until relieved by treatment, or until the inhibitory centers are better developed, and the nervous mechanism which controls urination is more stable; this, as a rule, occurs before the seventh year, but it may continue into adult life.

Heredity.—Nearly all children suffering from enuresis have a neurotic inheritance. A family history of enuresis, hysteria, neurasthenia, chorea, and other neuroses

is common. Two or three children in the same family may suffer from incontinence of urine. This inherited neurotic tendency is a very important etiological factor, and depends upon an hereditary feeble inhibition and general nervous irritability, which, under favoring conditions, may find expression in incontinence of urine.

Direct Causes.—Chronic malnutritions are most important factors of enuresis. They act by bringing about a malnourished condition of nerve centers, which not only increases the reflex irritability of the spinal centers, but also lessens the functional activity of the higher centers of the brain, in this way still further weakening the inhibitory control which these centers exercise on spinal centers. The causes of chronic malnutritions, therefore, may be classed among the direct causes of enuresis. The most important of these causes are tuberculosis, enteritis, rheumatism, malaria and syphilis. Improper food and bad hygiene are contributing factors in all of these conditions.

About one-half of the cases of incontinence of urine seen in dispensary practice in Cincinnati, Ohio, have a tubercular malnutrition. Intestinal, rheumatic, malarial, and syphilitic malnutritions are also common. There is a well-marked chronic anæmia in 80 per cent. of the cases of enuresis seen in dispensary practice, and the anæmia in these cases can, for the most part, be traced to one or more of the five diseases above named. Chronic malnutritions may, therefore, be classed as the most important of all the blood factors of incontinence of urine.

Auto-intoxication is an important factor in producing

enuresis. The form of auto-intoxication which is most commonly associated with enuresis is that which occurs in the uric acid or gouty diathesis. Fothergill says: "Lithuria is a very common occurrence in children of the uric acid diathesis. . . . Wetting the bed at night has close relations with uric acid, and in all cases of nocturnal incontinence the urine should be examined. In my experience, wetting the bed occurs mainly in two classes of children—in very bright, vivacious, neurotic little girls, and in comparatively dull and backward children of low nervous organization. In either case the uric acid present plays a part."

It is my own opinion that the enuresis which occurs so commonly in gouty or lithæmic children is due, not only to the action of auto-toxins on the nervous system, but also to the irritation of the bladder and genital organs which occurs in this condition as a result of the concentration and increased acidity of the urine.

Bacterial Intoxication.—Chronic intestinal intoxication, which is usually bacterial in origin, may produce profound malnutrition, and in that way furnish a basis for the development of enuresis. The acute infectious diseases are sometimes followed by incontinence of urine. The chronic bacterial intoxications which are also potent in producing this syndrome have been spoken of above under "Chronic Malnutritions."

Malformations of the genital tract may also be responsible for incontinence of urine; and organic diseases of the brain or spinal cord may have as one of their symptoms incontinence of urine. But these conditions have nothing to do with the neurosis under consideration.

All of the direct causes above noted act by bringing about an increased irritability of the general nervous system, and decreasing the inhibitory control which the higher centers exercise over the lower. Usually, more than one of these causes are found to be coöperative in producing enuresis, and they are usually associated with some form of reflex irritation.

Exciting Causes. — Some form of reflex irritation probably acts as an exciting cause in every case of incontinence of urine. But in perhaps one-half of these cases the causes of the reflex irritation are so unimportant that they cannot be found. In such cases the reflexes may be a distended bladder, or even a small quantity of urine in a slightly irritated bladder, or some other condition that varies so slightly from the normal that it could only excite to action nervous centers made hypersensitive by one of the direct causes above mentioned. In other words, the reflex irritation is so unimportant that it can scarcely be spoken of as a real factor in producing the enuresis.

In the other half the reflex factor is important; it can, as a rule, be located, and its removal in many cases is necessary to successful treatment. Even in those cases, however, in which the enuresis disappears on the removal of a reflex factor, it does not follow that the reflex was the sole or even the most important cause. Other factors, such as grave nutritional disturbances, may have coexisted with the reflex irritation and yet not be made manifest by the continuance of the enuresis after the reflex has been removed. If relapses are to be prevented and the patient, as well as the enuresis, is to be

treated, a careful search for other factors should be made, even if the enuresis disappears on the removal of the reflex irritation.

On the other hand, if little or no improvement immediately follows the removal of an apparently potent reflex factor, it does not follow that this factor was unimportant, since while it may not have played an important rôle in touching off the urination center in the lumbar cord, it may have produced a general spinal irritability, involving the lumbar as well as other centers, which remains long after the reflex irritation has been removed. The effects of chronic reflex irritation on the spinal cord do not disappear at once on the removal of the reflex factor which produced them. They do, however, slowly disappear when these factors are removed. If the reflex irritation is strong and long-continued, it produces the condition of general spinal irritability described in the chapter on "Reflex Irritation." The changes which take place in the spinal cord cells under long-continued reflex irritation bring about an irritability of the spinal centers which the removal of the reflex and time alone can cure.

The reflexes which are most closely associated with enuresis have their origin, as a rule, in genital, vesical, or rectal irritations, such as phimosis, preputial adhesions, contractions or granulations in the meatus, polypi in the rectum, fissure of the anus, acid and irritating urine, cystitis, and contracted and intolerant bladder, and vaginitis.

Reflex irritations having their origin in diseases of distant organs, such as the throat, nose, eye, and intestinal

canal, are not infrequently associated with enuresis. Diseased adenoids are very commonly associated with enuresis.

Habit.—It should be remembered that whatever may have been the important etiological factors in producing enuresis, the condition may continue even after these factors are apparently removed. The continuance of the enuresis under these conditions is due not alone to the spinal irritability which may persist, as we have noted above, but is also due to the habit which is formed in these cases of emptying the bladder when it contains but a small quantity of urine. This habit is apparently engrafted upon the nervous mechanism which controls urination.

SYMPTOMS

Enuresis in about 55 per cent. of the cases occurs only at night. About 40 per cent. are both noctural and diurnal, and about 5 per cent. are diurnal only. Incontinence of urine may occur once or several times during the night. In other cases milder in character it occurs at irregular intervals, days or weeks intervening. Nocturnal incontinence occurs most commonly soon after the child goes to bed. At this time sleep is most profound, and the brain fails to perceive the symptoms of vesical irritation from a full bladder, and the unconscious higher brain centers there fail to exercise inhibitory control over the urination centers in the spinal cord.

Ordinary enuresis being a pure neurosis, and not due to paralysis or lack of development of sphincter muscles, does not have as one of its symptoms the dribbling of the

urine. On the other hand, the contraction of the bladder empties this organ as thoroughly as under normal conditions, but it responds so quickly to reflex irritation that the patient is not able to control even for a short time the discharge of urine.

Cases of enuresis that have apparently yielded to successful treatment not uncommonly have relapses. Enuresis is commonly self-limited, and even those cases which have not yielded to treatment get well, as a rule, before the child is seven years of age, the growth and development of the nervous system effecting a cure. In a few cases, however, the disease may continue into adult life and in those cases in which the condition is dependent upon an incurable organic disease it may continue indefinitely.

The urine of patients suffering from enuresis may be concentrated and increased in acidity, or it may be of low specific gravity, alkaline in reaction, and greatly increased in quantity. Uric acid, urates, oxalates, and phosphates are commonly found in excess; occasionally mucus, pus, and albumin are found, indicating disease of the genito-urinary tract; sugar occurs in a few cases, apart from those of true diabetes.

PROGNOSIS

When the enuresis is a symptom of organic disease of the brain or cord or of some malformation of the genito-urinary organs, the prognosis will vary with the prognosis of the organic disease which produces it. In the ordinary enuresis of childhood, however, the prognosis as to ultimate recovery is absolutely good, and as to

cure in a limited time is also fairly good, since these cases, as a rule, yield to careful systematic treatment within a period of from two to six months.

TREATMENT

In the treatment of no other neurosis of childhood is it of so much importance to remove every possible cause of reflex irritation that can be discovered. It is an absolute waste of time to begin medical or other treatment until a most careful search for reflex factors has been made. Phimosis when present can, as a rule, be relieved by stretching the prepuce; circumcision is to be advised only in those cases which do not yield to this treatment. There is no more common error in surgical practice than that of sacrificing the prepuce for simple contractions of this organ. Under dilatation the foreskin can be separated from the glands, breaking up the preputial adhesions and removing the smegna; the parts are then to be anointed with vaseline, and this process is to be repeated daily for a period of eight or ten days. This treatment is simpler and far more efficacious than circumcision in the great majority of these cases. Adherent prepuce is the normal condition in the young child, and in my experience is to be found in almost every case that has not been previously treated. It is the retention of the smegma and the consequent low grade of ballanitis which this condition brings about that makes it pathological. At any rate, in every case of incontinence of urine this routine treatment pertaining to the hygiene of the genital organs should be followed.

The intestinal canal throughout should receive careful

attention; worms and undigested food are to be removed by proper medication; rectal irritation from polypi, fissure, pin worms, or other causes must be treated, and fermentations must be corrected and prevented by proper food and medication. A small meatus may demand nicking and stretching. Stone in the bladder, cystitis, vaginitis, or any other abnormal condition of the genito-urinary organs must receive appropriate treatment. Adenoids, which strangely enough are often associated with enuresis, must be removed. In short, all reflex irritations capable of producing an increased irritability of the nervous system, even though they come from distant organs like the eye, nose, or throat, must be removed before other treatment is instituted.

General hygiene is most important in the treatment of enuresis. The child should be removed from all excitement and nervous strain, should be taken out of school, and, if possible, sent into the country, where it can lead a quiet outdoor life. Wherever the child is treated it is imperative that his nervous system should be carefully protected. He should go to sleep early and at a regular hour and be fed upon a diet carefully selected to suit the individual case. If the child be tuberculous or suffering from any other form of grave malnutrition, the diet should consist largely of meat, eggs, milk, and bread, with such additions as the age and digestive capacity of the child may suggest. If, however, he has inherited a gouty diathesis, or has lithæmic symptoms other than the enuresis, or if at times his urine is very acid or concentrated, depositing urates on standing, then his diet is to be slightly different. Such a child is to be allowed

milk, cereals, cooked fruits, potatoes, and other well-cooked vegetables, but meats and eggs are to be partaken of sparingly. In all cases of incontinence of urine, beef juice, beef tea, alcohol, coffee, tea, sweets, and pastry, as well as all foods that may be beyond the child's digestive capacity, are to be prohibited.

The moral treatment in these cases is important. The child should neither be punished nor be threatened with punishment for the incontinence. If the child is old enough, he should be made to understand the importance of overcoming the habit by retaining his urine for as long a time as possible during the day, provided the case is not one of diurnal enuresis. If the child can be taught to thus accustom the bladder to hold considerable quantities of urine for some hours during the day, then the habit on the part of the bladder of discharging urine, when only partly filled, may not be carried over into the night.

When the incontinence occurs at night the child should take as little water as possible after four o'clock in the afternoon, and in the worse cases should be awakened about an hour and a half after going to bed, so that the bladder may be emptied and thus avoid the unconscious discharge later on.

The foot of the bed should be raised so that the child's shoulders will be lower than his hips. This may prevent the urine in a partially filled bladder from running down into its neck and starting the reflex which finds expression in incontinence.

Cold daily douches to the spine are indicated in well-selected cases due to long-continued chronic reflex irrita-

tion and not associated with profound nutritional changes. The cold douche acts as a tonic to the irritable cord in these cases, and not infrequently the enuresis rapidly disappears.

Treatment of the Malnutrition.—The malnutrition which occurs in perhaps 50 per cent. of all cases of enuresis must be successfully treated before one can hope to cure the incontinence. If the malnutrition be due to concealed tuberculosis, as it so commonly is, iron, cod-liver oil, fresh air, and good food are indicated. If it be due to chronic malaria, quinine and arsenic are indicated. If it be due to hereditary syphilis, anti-syphilitic treatment is indicated. If it be due to chronic digestive disturbances, carefully selected diet, pure air, outdoor life, and appropriate medication are indicated.

In the treatment of enuresis, after reflex factors have been searched for and removed, the next important step is the differential diagnosis of the type of malnutrition upon which this neurosis may rest. If one can successfully treat the malnutrition, the enuresis which in large part is dependent upon it will disappear.

MEDICAL TREATMENT

Belladonna is the one drug which all writers recommend in the treatment of enuresis, and is no doubt the most valuable. Belladonna, it should be remembered, is well borne by children, and to get the results the dose must be gradually increased until the enuresis is controlled, or until disagreeable physiological symptoms are produced. The dilatation of the pupils and the dryness

of the throat will indicate when a maximum dose has been reached.

For a child of six years, it is well to begin with a dose of three minims of the tincture, three times a day. After a day or two this is slowly increased a drop or two a day until physiological symptoms are produced, or until the child is taking 25 or 30 drops a day. Some authorities prefer atropin. Holt says: "A convenient method of administration is to use a solution of atropin, 1 grain to 2 ounces of water, of which one drop (1-1000 of a grain) may be given for each year of the child's age. For nocturnal incontinence this dose should at first be given at 4 and 10 P. M. After a few days at 4, 7, and 10 P. M. Usually this may be gradually increased until double the quantity is given. A child of five years would then be taking 10 drops (1-100 of a grain) at each of the hours mentioned. I have rarely found it advisable to go above these doses."

In cases that are benefited or controlled by the belladonna treatment, this drug should be continued in smaller doses for months. The belladonna being excreted by the urine acts as a local anodyne to the genito-urinary tract, and it is believed that the benefit which is derived from its use is largely, if not wholly, due to this local action. By allaying the irritability of sensory nerves it diminishes the potency of reflexes coming from these organs and increases the tolerance of the bladder. The curative influences of belladonna are therefore probably indirect, in that it controls the enuresis until the underlying conditions can be removed by the treatment previously outlined. The belladonna treatment also helps

to overcome the habit of frequent urination, which is such a potent factor in keeping up the incontinence when it has once been well established. The indications, therefore, for this treatment continue long after the enuresis has been controlled, and greater success will follow the use of this drug if it be given over a long period of time.

Alkalies are invaluable in the treatment of those cases dependent upon the lithæmic diathesis and having an excess of urates and acids in their urine. In such cases the belladonna is to be combined with benzoate of soda or bicarbonate of potash, and this prescription may be made more palatable by the addition of peppermint water and essence of pepsin. For a child of six years of age 5 grains of either of these alkalies may be given after meals. It is perhaps better to prescribe the alkali and belladonna in separate bottles, giving them at the same time, but allowing for an increase of the belladonna without increasing the alkali. In this type of case also the constipation which is usually present is to be overcome by phosphate of soda, or sulphate of soda put up in palatable solution. In older children effervescing carbonated waters may be used to cover the taste of these drugs. If the lithæmic condition is recognized and successfully treated, the enuresis will, as a rule, take care of itself.

In very nervous hysterical children not of the lithæmic type, the bromide of potash may be used, combined with the belladonna treatment, to assist in getting control of the enuresis. The bromide treatment, however, is not to be continued for any length of time.

Ergot is a drug highly spoken of by many writers, and must therefore be of value in the treatment of cer-

tain cases of enuresis. I must confess, however, that I have not been impressed with its efficacy. Aromatic tincture of rhus in 5 to 10 drop doses three times a day is at times a very efficacious remedy and should be tried when the above treatment fails.

Strychnine is another drug universally used and universally commended by the very best writers in the treatment of certain cases of enuresis. I, however, believe that this drug is of little or no value, and in many cases is absolutely contra-indicated. I believe that its use originated in the belief that the incontinence of urine was due to a weakness rather than to a lack of proper innervation of the sphincter muscles. The fact, however, that so many writers have used it apparently with good results would justify its further use in these cases.

Electricity is another therapeutic measure in which I have little faith. In those cases which have yielded to faradism, when locally applied to the bladder and rectum, the result, I believe, was due to suggestion rather than to electricity. It should be stated, however, that the electrical treatment of enuresis has been very highly extolled by some writers, and that galvanism to the spine is perhaps of real value in these cases.

Cathelin's method of epidural injections into the sacral canal between the periostium of the vertebræ and the dura mater, of 10 to 25 cc. of sterile decinormal salt solution given in the average once in seven days, has recently been used with some success in the treatment of these cases.

CHAPTER XV

MIGRAINE

Synonyms.—Megrim, sick headache, hemicrania.

Definition.—Migraine is an auto or intestinal intoxication which finds expression in recurrent self-limited attacks of severe paroxysmal headaches, usually unilateral, commonly accompanied by nausea, vomiting, vertigo, and visual phenomena, and followed by a profound sleep, from which the patient awakes free from pain.

ETIOLOGY

I. Predisposing Causes.—Age is an important predisposing factor; the majority of cases appear in late childhood or early adult life. Not a small percentage, however, begin between the fifth and the tenth year; in these cases occurring in early childhood the stomach symptoms are, as a rule, more pronounced and the hemicrania less severe. Migraine once established does not, as a rule, spontaneously disappear until the fifth decade of life. The disappearance of migraine at this period is probably due to the arterio-sclerotic changes which occur rather early in individuals of this type. The hard arteries of the migrainous patient of fifty protect him from the vasomotor disturbances which are an essential part of the migrainous attack. In women the cessation of menstruation at or about the fiftieth year removes

one of the most common of the exciting causes of migraine, and offers another explanation for its common disappearance at this time of life.

Sex.—Migraine is, among the poor and uneducated, much more common in women than in men, the proportion being as four or five to one. Among the rich and refined, however, there is but a slight preponderance of females, and this is perhaps due to the influence of the menstrual function in precipitating these attacks.

Season.—Migraine occurs more frequently during the winter than the summer months in the Middle and Northern States. This is probably a matter of diminished outdoor life, change of food, and decreased action of the skin.

Heredity is by far the most important of the predisposing factors. There is, as a rule, a history of direct migrainous inheritance, which may run back a number of generations; or there may be a general neurotic inheritance, other members of the family having suffered from functional nervous diseases. A gouty inheritance is also commonly observed, and this may carry with it the history of a family tendency to functional disturbances of the liver, which manifests itself in so-called attacks of biliousness.

Constipation, which is one of the most common etiological factors of migraine, is closely associated with the so-called bilious temperament. The hyper-fermentation of the intestinal contents which results from constipation may produce intestinal toxins which an incompetent liver is not able to destroy.

Occupation is a very important predisposing factor

and makes this disease very prevalent among the poor, because of their indoor life, lack of fresh air and sunlight. For this reason the disease is very common among factory girls and tenement house dwellers. It is also probable that unwholesome and improperly prepared food and the general ill-health of this class predispose them to migraine. The men among the poor, however, do not commonly suffer from this disease, because of the outdoor life and great physical exercise incident to their occupations. Here we have an explanation for the fact that women suffer much more frequently from this disease than men. Among the rich and refined, however, those leading a sedentary life and exposed to mental overwork and nerve excitement and commonly given to excesses in eating and drinking, we find the disease almost as common in men as it is in women.

Food.—Excess of highly seasoned foods, coffee, alcohol, meats, and sweets may predispose to migraine, probably through their influence on the functions of the liver and intestinal canal. While excesses in eating and drinking along the lines above indicated are especially harmful, it is important to note that it is the *excess* rather than the character of the food that produces the greatest harm.

II. Direct Causes.—Toxins are responsible for the paroxysms of migraine; of this I think there can be little doubt. As to the exact nature and character of these toxins, and as to whether they are chiefly auto or intestinal, it is still a matter of great uncertainty, and upon these questions the medical profession is by no means agreed. It is my belief, however, that auto-toxins

play the most important rôle in producing migraine and that these toxins are produced by a faulty metabolism of albumins and lack of oxidation of the retrograde bodies, formed by the death and disintegration of the cellular elements of the body. The uric acid bodies, especially the xanthin bases, probably play the most important rôle in this intoxication; at any rate, auto-toxins closely associated in their formation, if not identical, with the uric acid bodies are at least partly responsible for the symptoms of migraine (see chapter on "Auto-intoxications").

Intestinal toxins no doubt also play a rôle in the production of many cases of migraine, since the removal of constipation and resulting intestinal fermentation which are present in so many of these cases is ofttimes followed by a great amelioration of the symptoms (see chapter on "Intestinal Toxins").

Liver Incompetency.—The poisons which produce migraine, whether they be auto or intestinal in their origin, are under normal conditions largely destroyed or converted into harmless products by the liver. These poisons are therefore thrown into the general circulation by all conditions that diminish the functional capacity of that organ, such as indoor life and lack of exercise, and by all conditions that throw increased work upon the liver, such as excessive eating and alcohol and coffee drinking. The liver through its filtering function normally stands guard between the toxins of the intestinal canal and the general circulation, and through its urea-forming function it converts ammonia and the purin bodies into harmless urea. These important functions

of the liver protect against both auto and intestinal toxins, but under pathological conditions, either through a weakened functional capacity of the liver, inherited or acquired, or through excess of poisons produced, the liver is no longer able to destroy these poisons, and a periodic acute functional incompetency of this organ results, thereby throwing these poisons into the general circulation, producing an acute intoxication. Within twenty-four or thirty-six hours, these poisons being excreted and the liver having resumed its function, the attack of migraine is ended.

It is my belief that under aggravated pathological conditions the liver may remain for a long time in a state of chronic partial incompetency, thus allowing a portion of these poisons to filter through into the general circulation, producing a state of either chronic auto or intestinal intoxication. In this condition the liver is commonly enlarged, and the migrainous symptoms, while not so severe as in the paroxysmal attacks, are more or less constant, producing a neurasthenic condition.

Action of Poisons.—The poisons which produce migraine commonly act through both the sensory and sympathetic nerves of a part, producing both pain and vasomotor disturbances. In the young child the vasomotor disturbances are more marked and the poisons are more prone to act upon the sympathetic nerves of the stomach.

In the adult, however, the sensory nerves of the head are commonly attacked, producing a severe hemicrania, and the vasomotor nerves of the same region are also acted upon, but the stomach disturbances are not so severe

or so common as they are in the child. The points of attack which these poisons commonly select are the nerves, both vasomotor and sensory, of the head and of the stomach. It is probable that the vasomotor nerves are primarily attacked and the sensory disturbances are secondary.

III. Exciting Causes.—Eye-strain due to errors of refraction and insufficiency of certain eye-muscles is one of the most common exciting causes of migraine. Cases of this character are relieved by the correction of the eye-strain. This, however, does not prove that the eye-strain was the sole cause of the recurring attacks. It does prove, however, that in some cases the exciting factors are so important that their removal greatly modifies the number and the severity of the paroxysmal attacks, notwithstanding the existence of certain toxic and predisposing factors. The removal of reflex factors, therefore, may lengthen the interval between, and modify the severity of, migrainous attacks, but headaches that are altogether cured by correcting eye-strain are reflex rather than migrainous in character.

Diseases of the naso-pharynx and of the genito-urinary and pelvic organs are among the exciting causes of migraine. In females menstruation is probably the most important of all exciting causes; the headaches in these cases recur with great regularity at or near the menstrual period. It should be remembered, however, that all menstrual headaches are not migrainous in character.

Fatigue, emotional excitement, overtaxation of the nervous system, and overwork at school are important exciting causes of migraine.

Certain foods, such as acid fruits and acid wines, may in susceptible individuals be exciting causes, and digestive disturbances of any kind may precipitate an attack.

SYMPTOMS

The symptoms of a migrainous attack will vary with the part attacked, the nerves involved, the virulence and character of the poisons, and the age and physical condition of the patient.

The attack is at times preceded by certain prodromes such as vertigo, tinnitus aurium, partial vision, bright and dark spots, and flashes of light before the eyes, transient aphasia, with a fullness about the head and a peculiar tingling or burning sensation in some portion of the body, which the individual by experience learns to interpret as the forerunner of an attack. Certain of these prodromes may continue for a number of hours, when the attack is ushered in by a headache, which is, as a rule, unilateral. The headache gradually increases in intensity, sometimes spreading to the opposite side.

The pain is intense, throbbing in character, and continues for a number of hours. It is increased by light, noise, or movement of the body. For these reasons the patient usually lies down in a quiet, dark place.

Nausea, as a rule, occurs early in the attack, and increases in severity until actual vomiting occurs. The vomiting, which occurs some hours after the headache, usually marks the climax of the paroxysm, and from this

time the symptoms gradually abate. The vomited matter contains not only the food content of the stomach, but also bile, considerable mucus, and an excess of free HCl. The hyperchlorhydria which occurs during attacks of migraine is similar to that which occurs in recurrent vomiting.

The pain in the head, which is a characteristic symptom of migraine in the adult, may be almost entirely absent in the young child, and the vomiting, which is rather a secondary symptom in the adult, may be the most pronounced symptom in the child. Frequent vomiting with constant nausea may continue for days, without any pronounced pain in the head, and in this condition we have the symptom group described as "recurrent vomiting" in the next chapter. In the older child and the adult, however, we not infrequently have the persistent vomiting and the severe headache combined. Pain in the stomach of great severity may at times take the place of pain in the head. I have seen cases of migraine go on for years, with the typical symptoms of hemicrania and vomiting, and then, without apparent cause, these attacks would be replaced by paroxysms, characterized by severe pain in the stomach, with nausea and vomiting, but with little or no pain in the head.

During the paroxysm, when the pain is most severe, vasomotor disturbances are present; aphasia and vertigo may occur, and one side of the face may be pale and the other side show red spots on the cheek or ear. The flushing of the ear and side of the face may come and go during the attack, or may continue until the pain in the head has disappeared. These phenomena are more

marked on the side of the face which is the seat of the pain.

Profound sleep, in some instances almost amounting to mild coma, which terminates the paroxysm of migraine, is one of its most characteristic symptoms. The attack may have gone on for twelve or twenty-four hours when the patient, yielding to the sensation of drowsiness, falls asleep, and six or eight hours later awakens free from pain. At times, following severe attacks of migraine, there will be a day during which there is a feeling of mental apathy and disability with partial aphasia, but even in cases of this kind the second day will find the patient entirely recovered and possessed of a keener mental acumen than he possessed before the attack. The storm seems to have cleared his mental horizon.

The temperature in the adult is, as a rule, normal; in children, however, the temperature during the height of the paroxysms is commonly elevated from one to four degrees; later, following the sleep, the temperature may be subnormal. The pulse in the child is rapid and irregular; in the adult it is hard and variable, sometimes slow and sometimes rapid.

Attacks of migraine are self-limited and vary in duration from a few hours to two or three days. Occasionally, however, we have aggravated pathological conditions, producing what may be described as chronic migraine. In these patients there is chronic dyspepsia and more or less continuous depression of spirits, with general nervous irritability and vague fears characteristic of the neurasthenic condition. More or less head-

ache may occur every day, with morning nausea, and this condition, if it continues, soon becomes a pronounced neurasthenia. Chronic migraine, however, is uncommon in childhood.

Migraine does not, like epilepsy, lead to mental impairment. On the other hand, it may be said that children suffering from this condition are nearly all precocious, and the precocity which is manifested early not uncommonly continues through life. At any rate, it is a matter of history that many of the greatest intellects that the world has produced have been sufferers from migraine.

The paroxysms of migraine occur at regular or irregular intervals. Now and then the paroxysms will be observed to recur at regular weekly, fortnightly, or monthly periods. The monthly interval is the most common, since migraine occurs most frequently in women, and since the menstrual period is the most important exciting cause. The fortnightly interval is also common. In these cases we have the menstrual paroxysm occuring at or near the menstrual time, and the inter-menstrual paroxysm occuring half-way between. In many cases in women, however, the return of the paroxysm has nothing to do with the menstrual period, and the interval is quite as irregular with them as it is with men. In children the paroxysm has been observed to recur at weekly intervals.

DIAGNOSIS

One of the important points in differential diagnosis of migraine from other paroxysmal headaches is found

in the urine. The urine in migraine is high-colored, with high specific gravity, and contains an excess of the purin bodies, including uric acid and the xanthin bases. The increase, however, in the xanthin bases is much more marked than is the uric acid increase. The urine contains a diminished amount of urea and an excess of ammonia, which is probably excreted in combination with acids. The urine is increased in acidity and, in some instances, contains acetone and diacetic acid. Occasionally, especially in children and in those past middle life, a transient albuminuria accompanies the paroxysm.

Apart from the urine, however, the differential diagnosis of migraine is not difficult. Recurring hemicrania, associated with nausea and followed by sopor, are not characteristic of any other type of headache. It is only in cases of chronic migraine where these characteristic symptoms are more or less lost in the chronic character of the disease that one is likely to be mistaken. But even in such chronic cases the diagnosis may be made by the early history of typical attacks of migraine, which have gradually merged into the present chronic condition. The hereditary predisposition and the vasomotor symptoms previously described will assist in establishing the diagnosis.

PROGNOSIS

As to Cure.—Many of these cases can be cured, and all of them can be relieved, by proper treatment; that is to say, the severity of the paroxysms may be diminished and the interval between their occurrence prolonged.

As to Complications.—The poisons which produce migraine are most potent factors in producing arterio-sclerosis. Arterial changes come on earlier in the migrainous individual, and, as has been previously said, these changes, by interfering with the elasticity of arteries, prevent vasomotor disturbances; and in this way assist in terminating these paroxyms as age and arterio-sclerosis advance. These arterial changes, however, predispose these patients in later life to cerebral hemorrhage and arterio-sclerosis of the kidney. If, therefore, we recognize in migraine an auto-intoxication which may bring on a premature arterio-sclerosis, we can say that the prognosis, so far as life is concerned, in the untreated cases, is not the best, inasmuch as these patients are likely to die from cerebral hemorrhage or diseases of the kidney years before their allotted time.

TREATMENT

Treatment of Attack.—If large doses of benzoate of soda (60 grains) or bicarbonate of soda (teaspoonful) be given at the onset of prodromal symptoms, the migrainous attack may be aborted, or very much modified in severity; with the soda it is advisable to give two or three grains of calomel. If desirable, the soda and calomel may be given in broken doses: twenty grains of bicarbonate of soda and one-half grain of calomel every hour for four doses. For children under ten years of age the soda in these prescriptions may be diminished one-half. If this treatment does not give relief, the following prescription is a safe, and, if given early in the attack, a very effica-

cious remedy; it will, however, be more effective if the patient on taking it lies down in a darkened room.

℞

Caffeinæ citratæ	2	grains
Sodii salicylatis (gaultheria)	10	"
Sodii bromidi	30	"

Sig.—To be taken in half a glass of carbonated water (dose for an adult, half this dose for a child ten years of age).

This powder when given as here directed is almost if not quite as efficacious as the coal-tar products in relieving an on-coming headache; it may be repeated at intervals with no untoward results.

The use of the coal-tar products, especially acetanilid, for the relief of migrainous headaches is for the most part to be discouraged, since patients suffering from so chronic a disease attended with so much pain may, to their injury, prefer to resort to these remedies for relief during the attack rather than take the trouble to follow the preventive treatment presently to be outlined. There is little doubt that many migrainous patients are assisted on the road to chronic invalidism and neurasthenia by the frequent use of the headache powders (acetanilid) which are so widely advertised as harmless and curative. These powerful headache remedies if taken in large doses at short intervals weaken the heart and destroy red blood corpuscles, and thus, by interfering with elimination, increase the auto-intoxication from which the patient suffers. If, however, in individual cases it is thought advisable under proper restrictions to give the coal-tar products for the relief of migrainous headaches, antipyrin is, in my opinion, the safest and most effective of these

preparations. It may be substituted for the sodium salicylate in the prescription above given. It is, however, rarely necessary to use these remedies in children under ten years of age.

Hot fomentations to the forehead may assist the above remedies in giving relief from pain.

Occasionally an attack of migraine may be so severe as to demand the use of morphine hypodermically. This remedy, as a rule, gives almost immediate relief, and is perfectly safe in the hands of the physician; it is, however, very rarely indicated in children under ten years of age. The giving of morphine hypodermically for the relief of these headaches should never be intrusted to the patient, for fear of establishing the morphine habit.

In the treatment of the attack in young children calomel and soda, followed by caffeine and bromide of potash, should for the most part be relied upon to relieve the headache. It is rarely necessary to use the coal-tar products, and never necessary to use morphine at this age. One or two hours following the calomel and soda a child six years of age may take one grain of citrated caffeine, and five grains of bromide of potash every hour or every half hour for three doses. If this treatment is commenced with the onset of premonitory symptoms, it usually suffices to relieve the attack.

In the child the above remedies not infrequently provoke vomiting, which is, as a rule, a much more prominent symptom of the migrainous attack in the child than it is in the adult. This fact, however, does not contra-indicate the above line of treatment, since with the onset of vomiting the pain in the head generally becomes less in-

tense. At no age is vomiting coming on during the height of the migrainous attack to be considered an undesirable symptom. This act serves the purpose of washing out the stomach, and may, as a rule, be advantageously followed by another dose of bicarbonate of soda dissolved in a glass of carbonated water.

The above line of treatment, which either aborts, shortens, or modifies the severity of the migrainous attack, is very important, and its efficacy should be insisted upon. But in giving attention to this phase of the treatment it should be remembered that the true treatment of migraine is the interval treatment, which has for its object the relief of the underlying constitutional condition and the prevention of these attacks. These desiderata can, for the most part, be realized if the patient will conscientiously follow the medical, dietetic, and hygienic treatment below outlined. The importance of this treatment may be further insisted upon in that it may delay the premature arterio-sclerosis which results from the auto-intoxications that produce migraine.

Preliminary Treatment.—In beginning the treatment of a case of migraine it is all-important that reflex factors which may possibly play a rôle in producing the paroxysmal attacks should be searched for and, if possible, removed. Such reflex factors are most commonly found in ocular defects producing eye-strain. The eye is, in fact, such an important source of reflex irritation to the nerve centers that certain oculists assert that ocular defects are the most potent factors in producing migraine. Abnormal conditions of the nose and pharynx, such as adenoids and hypertrophies, also play a

most important rôle as reflex excitants of the nerve centers.

Diseases of the pelvic organs, in many instances, are unquestionably etiologically related to migraine. Diseases of these organs may, by interfering with elimination, by the formation of toxins, and by acting as a source of reflex irritation, increase the general nervous irritability of the patient, and in that way act as predisposing if not direct exciting causes of migraine. It is therefore important in the early treatment of migraine that the physician should satisfy himself that eye-strain or some disease of the nose, throat, pelvic organs, or gastro-intestinal canal are not important factors in producing the paroxysmal headaches from which the patient suffers.

It is not an uncommon experience to have migrainous attacks almost disappear on the removal of some aggravated pathological condition which is causing intoxication or reflex irritation of the nerve centers. This does not, however, prove that the toxin or the reflex is the sole factor in producing the migraine in these cases. It does prove, however, that these factors are so important that it would be folly to attempt the constitutional treatment of such cases without the removal of these factors.

Migraine is, as I believe, a consitutional disease due to an auto-intoxication, and with this condition there may coexist a cause of reflex irritation to the nervous system so important that it is impossible to completely cure these cases without the removal of the reflex factors.

Medical Treatment for Relief of Constitutional Condition.—Since migraine is a chronic disease, and treatment must be continued over many months, it is absolutely

necessary for permanent success in the treatment of this condition that the medical treatment should be as simple and as palatable as possible. This is true of men, women, and children alike. The busy man cannot, as a rule, be prevailed upon to take over a long period of time three or four doses of medicine each day, and women and children, as a rule, after a few weeks of treatment prefer the disease to taking dose after dose of unpalatable medicine throughout an entire season. The keynote of success, therefore, in the treatment of migraine is in the simplicity and palatability of effective medication. To accomplish these desiderata, some ten years ago I devised a formula, which is here presented with such slight changes as time and experience have suggested:

℞

Sodii sulphatis (dry)	30 grains
Sodii salicylatis (from wintergreen)	10 "
Magnesii sulphatis	50 "
Lithii benzoatis	5 "
Tincturæ nucis vomicæ	3 drops
Aquæ destil. to make	4 ounces

M. S.—Take each morning. Dose for an adult.

This prescription is made in large quantities by a reliable pharmacist, and sent by him to a mineral water factory to be put up in siphons and charged with carbonic acid. These siphons I prescribe under the name of "Siphon C," and direct my adult patients to take from one-quarter to one-half glass of this carbonated medicine each morning on arising, half an hour or more before breakfast. It is important that the dose should be so regulated as to produce a slightly laxative but not cathartic action. Only one dose of this medicine is given in

twenty-four hours, and after the patient is fairly under treatment this is commonly the only medicine used. Children over ten years of age can, as a rule, be induced to take this prescription, but under that age some substitute must be given.

I know from long experience that the above prescription may be given for an indefinite length of time (years, if necessary) without losing its great therapeutic value or producing disgust for it on the part of the patient. I have yet to find a patient over ten years of age who would not continue the taking of this medicine as long as I desired. After the first week or ten days patients grow accustomed to it, and then even the most sensitive no longer object to its use. This medicine, moreover, is not contra-indicated by any condition of the stomach. It is in fact the most valuable formula I have found for the stomach neuroses, and is also of value in the treatment of chronic gastric catarrh and chronic ulcer of the stomach. The condition of the stomach need not deter us, therefore, from prescribing this formula. On the other hand, "a bad stomach" and intestinal indigestion are further indications for its extended use.

I wish especially to insist that the above formula will give better results than the separate use of the various medicines which it contains. The great value of the formula, however, depends upon the fact that it combines palatability and simplicity with efficacy of medication; and these advantages enable one to treat an essentially chronic condition by giving a single dose of medicine in a day.

The siphon formula is put up by Merrill & Co., in the

form of granular effervescent salts, under the trade name "Akaralgia." These granular salts may be used by patients traveling or otherwise so situated that the siphons cannot be had.

It is my habit in the treatment of this condition to continue the above siphon medicine through the winter months only, discontinuing its use about April or May. Patients under this treatment often go through the winter months without a single attack of migraine. On discontinuing the medicine, however, in April or May, when the previous treatment and the climatic conditions make it no longer necessary, I usually advise my patients to report to me again about the first of the following January, or earlier if there is any return of the migrainous symptoms. At this time they are again placed upon the siphon medicine and advised to continue it until the following spring. By this plan of giving the above formula for three or four months in the year I have succeeded in controlling migrainous symptoms in a large number of my patients.

In studying the ingredients of this formula one finds the medicines that have been used for many years in the treatment of this condition. Magnesium sulphate is necessary to overcome the constipation which is present in nearly all of the cases, and by its action we unload the portal circulation and eliminate the poisons through the intestinal canal. Sodium sulphate acts very much in the same manner, plus its cholagogue action. Lithium benzoate acts as an intestinal antiseptic and as an eliminator through the kidneys. The small amount of nux vomica which the formula contains is added largely to cover its soapy taste and thereby make it more palatable. Sodium

salicylate (wintergreen) is the remedy *par excellence* of the prescription. This remedy acts as an intestinal antiseptic, increases the functional activity of the liver, and diminishes the tendency to acid intoxications which is present in this disease.

There are two other siphon formulæ which I occasionally use in the treatment of migraine. They are as follows:

SIPHON B

℞

Sodii sulphatis (granulated)	2 drams
Sodii phosphatis (granulated)	1 dram
Sodii salicylatis (wintergreen)	10 grains
Tincturæ nucis vomicæ	3 minims
Aquæ destil. to make	4 ounces

M. S.—Take each morning. Dose for an adult.

SIPHON A

℞

Potass. bicarbonatis	20 grains
Rochelle salts	1 dram
Sodii salicylatis (wintergreen)	5 grains
Tincturæ gentian comp.	½ ounce
Aquæ destil. to make	4 ounces

M. S.—Take each morning. Dose for an adult.

Siphons B and A may be used in those cases in which Siphon C is too laxative in its action. Siphon A is especially valuable in those cases of migraine suffering in the interval from acid urine, irritable bladder, or muscular rheumatism without constipation. The average adult dose for each of the siphon formulæ—A, B, and C—is four ounces, but the dose must be carefully varied to suit the age of the patient and the degree of the constipation to be relieved. Even when no constipation exists, from one to three ounces of one of these formulæ is to be given

as a necessary part of the treatment. Care, however, must be exercised to prevent excessive cathartic action and consequent intestinal irritation.

In children under ten years of age instead of the siphon formulæ I commonly employ some preparation of phosphate of soda. This may be given in milk or carbonated water, and where the constipation is obstinate Rochelle salts or some preparation of senna in palatable solution should be used. When constipation is not present I prescribe a solution of sodium benzoate and sodium salicylate (wintergreen) in palatable solution. A valuable prescription is as follows:

℞

Sodii benzoatis	1½ drams
Sodii salicylatis (wintergreen)	30 grains
Essence pepsin	1½ ounces
Aquæ menth. pip.	1½ ounces

M. S.—Teaspoonful three times a day for a child six years of age.

In beginning the treatment of every case of migraine, whatever the age of the patient may be, and whether or not the siphon medication is used, I always prescribe one or more of the three following drugs: sodium benzoate, sodium salicylate (wintergreen), and cannabis indica, and I further advise the drinking of water between meals. Sodium benzoate and sodium salicylate (wintergreen) for older children and adults is made more palatable by taking them in carbonated water. Sodium salicylate (wintergreen) may be given to young children in powders, combined with milk sugar; or salol may be used instead of the salicylate of soda; it has no advantages, however, and is probably not so efficacious.

The proprietary preparation known as colchi-sal may be given in place of the sodium salicylate (wintergreen) to older children and adults, one capsule after meals. I have found it a most valuable aid in the treatment of long-standing cases associated with other gouty symptoms.

Cannabis indica, in one-quarter grain doses two or three times a day, is of very great value in controlling the paroxysms of migraine in the adult, and may be used for three or four weeks, until the patient is well under the siphon treatment. I not infrequently combine the cannabis indica in a capsule with sodium salicylate (wintergreen) or salol in the treatment of these cases.

With these drugs to aid the siphon medicine, the paroxysms of migraine may be controlled almost from the beginning of the treatment. After the patient, however, has been under treatment for from three to six weeks, all medication other than the siphon medicine may be discontinued.

In some of the cases, however, it is necessary to give an intestinal antiseptic throughout the treatment of the case, and for this purpose I commonly use a one-grain salol-coated pill of potassium permanganate, taken after meals. This pill, which I devised many years ago and have used continuously ever since, has proven very efficacious in my hands. Dr. M. Allen Starr has modified this pill under the following formula:

℞

Sodii sulphocarbal	5 grains
Kal. permangan.	1 grain
Betanaphthol	1 "

M. S.—One after meals and at night.

This pill is coated with shellac, and is of value in those cases requiring an intestinal antiseptic.

In 1895 [1] I published a paper upon this same subject, in which I recommended the use of the following formula:

℞

Sodii salicylatis (wintergreen)	2 drams
Sodii phosphatis (dry)	4 "
Sodii sulphatis (dry)	10 "

M. S.—A teaspoonful, more or less, to be taken in a glass of seltzer water each morning.

These salts I still prescribe for patients traveling or otherwise so situated as to make it impossible for them to get the siphon medicine.

There is one other drug that has long held a deservedly high reputation in the treatment of migraine, and that is mercury, either in the form of calomel, blue mass, or the gray powder. Either calomel or blue mass is a good drug to begin the treatment of a case of migraine, and even after the patient has been placed upon the siphon medicine it may occasionally be beneficial to supplement this treatment with a few doses of calomel or a dose of blue mass.

The gray powder of mercury, combined with bicarbonate of soda, I have used with great advantage in the treatment of migrainous conditions in children [2] too young to take the siphon treatment. In such patients

[1] *Medical News*, September 7, 1895.

[2] In 1897 I published in the *Archives of Pediatrics* a series of papers on the treatment of this condition in infants and children.

a laxative formula such as the following may be substituted for the siphon medicine:

℞

Sodii sulphatis	2½	drams
Magnesium sulphatis	5	"
Lithii benzoatis	2	"
Aquæ destil. to make	3	ounces
Elixir tarax.	3	"

M. S.—Tablespoonful before breakfast for a child of eight years.

In the treatment of migrainous cases I have occasionally noticed that after a prolonged use of the siphon treatment patients become slightly nauseated, the tongue becomes furred, and there is a dull headache with loss of appetite. In this condition dilute nitro-muriatic acid acts almost as a specific, but during the time that the acid is given compound licorice powder or cascara sagrada may be substituted for the siphon medicine. After a week of such treatment the siphon medicine may be resumed.

Dietetic and Hygienic Treatment.—All that has been said in the way of medicinal treatment will be of little avail unless it goes hand in hand with proper dietetic and hygienic treatment. The most important bit of dietetic advice that can be given to a migrainous patient is that he should not eat too much. Most of these patients are in the habit of eating more than is necessary. It is important, therefore, that any tendency in this direction should be restrained. There are also certain articles of diet which should be partaken of sparingly or not at all—

coffee, alcohol, red meats, and sweets should for a time, at least, be almost if not quite eliminated from the diet.

Patients may be allowed milk, eggs, fish, oysters, the white meat of poultry, cereals, fruits, and well-prepared vegetables, provided a feeble digestion or some idiosyncrasy on the part of the individual does not exclude one or more of the above articles from his diet list. The diet to be prescribed in any individual case will of course depend upon the age of the patient as well as upon his digestive capacity and the character of life he leads.

Exercise in the open air is scarcely less important than diet in the treatment of migrainous cases. All kinds of outdoor athletic sports are to be advised, and among these horseback riding is one of the best. The financial condition, the age, and the inclination of the individual patient will suggest to the physician the character of outdoor exercise to be recommended. General massage is of great value in the early treatment of patients of feeble constitution.

Before closing the subject of the treatment of migraine, I would call attention to the great value of certain hydropathic measures in the treatment of selected cases. The Turkish and vapor baths as well as hot alkaline tub baths are of benefit, especially in patients of rather stout and robust constitutions. These baths are more efficacious when accompanied by large potations of water and followed by general massage.

CHAPTER XVI

RECURRENT VOMITING

Synonyms.—Cyclic vomiting, lithæmic vomiting, periodical vomiting, bilious vomiting. In the present state of our knowledge it is probably best to retain the name "recurrent vomiting," originally used by Gee, in describing these cases.

Definition.—Recurrent vomiting is a symptom group closely related to migraine. It is auto-toxic in origin, and characterized by recurrent attacks of nausea, persistent vomiting, and great prostration.

ETIOLOGY

1. *Predisposing Causes.—Age.*—The great majority of these cases occur during infancy and childhood. The disease may make its appearance as early as the third month, but it is more common between the third and tenth year. The tendency is to spontaneous recovery, but the attacks may continue into adult life or they may be transformed into migraine.

Sex has little influence. A small majority of the cases, however, occur in girls.

Season.—It is somewhat more common in winter than summer.

Heredity is the most important predisposing factor. A family history of migraine or gout is present in nearly every case. A general neurotic inheritance is common,

and a family history of hereditary recurrent vomiting is sometimes noted.

Constipation.—Nearly all of these patients are constipated, and there can be little doubt that this is an important factor in their etiology. The constipation, with the resulting intestinal toxæmia, no doubt contributes to the general irritability of the nervous system in these cases.

Habits of Life.—Mental overwork and nerve excitement, when combined with an indoor life and confinement in ill-ventilated school-rooms, are important predisposing factors.

Station in Life.—Nearly all these cases occur among the hereditary rich and refined. The poor and uncultured are comparatively exempt. This probably means that the hereditary gouty tendency, with the mental overwork and nerve excitement which is so common among cultivated people, are such important predisposing factors that the poor and unrefined, in whom they are rare, are not especially predisposed to this disease.

2. *Direct Causes.*—Nearly all writers are agreed that recurrent vomiting is an auto-intoxication. My own belief is that both auto and intestinal toxins may play a rôle in producing this symptom group, but I do not believe that it is always produced by the same auto or intestinal toxins. I am of the opinion, however, that the largest percentage of these cases is produced by toxins either closely related in their formation to, or identical with, the purin bodies. The close hereditary relationship which exists between this condition and gout and migraine lends strong evidence in support of this

view, and the urine findings elsewhere noted strengthen this opinion.

The acid intoxications which occur during the attack are to be considered rather as effects than causes, and belong, therefore, to the symptomatology and pathology, rather than to the etiology.

Liver Incompetency.—A functional incompetency of the liver is, I believe, an all-important factor. The liver in these cases is probably, by heredity, functionally incompetent, and, in addition to this, it is perhaps called upon by reason of the constitutional gouty taint to do an unusual amount of work in converting ammonia and the purin bodies into urea. Under these conditions we have periods of temporary functional incompetency on the part of the liver, and as a result the auto and intestinal toxins are poured into the general circulation and produce, in some instances, recurrent vomiting, and in other instances, migraine. In a few days, when these poisons have been eliminated and the liver has resumed its function, the acute attack is over.

3. *Exciting Causes.*—Mental and physical fatigue, mental excitement, nervous strain, fright, anger, and disappointment are common exciting causes. Overeating is one of the most potent of the exciting factors, and *acid* fruits, berries, vegetables, and wines may precipitate an attack. Intestinal toxæmia and reflex irritation from the intestinal canal, the eye, naso-pharynx, and genito-urinary organs may also be classed among the exciting causes. These exciting factors are at times apparently so important, and so definitely related to the onset of the attack, that the physician may be inclined

to overestimate their importance, and thus misinterpret the true nature of the disease. It should always be kept in mind that in these patients there is a tendency to the recurrence of auto-intoxications, which are the true causes of the attack, and the particular reflex factor which happens to touch off the paroxysm is not to be considered as the all-important causative factor.

SYMPTOMS

Prodromes are almost always present from a few hours to a few days before an attack. Among the more constant warning symptoms are flushings of the cheek, coryza, general restlessness, nervous irritability, sleeplessness, sallowness of complexion, dark rings under the eyes, general malaise, constipation, coated tongue, a peculiar odor to the breath, and loss of appetite. Not all of these prodromes are present in any one case, but in the great majority of cases the mother or nurse, having observed the onset of other attacks, will recognize, by certain of these warning symptoms, that a paroxysm of recurrent vomiting is imminent. Recently I have especially been interested in vasomotor coryza as an almost constant warning symptom in a number of cases.

Vomiting.—Following the prodromes, from six to forty-eight hours, vomiting occurs. This is the most characteristic and prominent symptom. In the beginning the vomiting may not be severe, food only being rejected; in a few hours, however (six to twenty-four), it becomes very severe, and accompanied by more or less constant nausea; not only everything that is taken into the stomach is rejected, but bile and much mucus some-

times tinged with blood is expelled. In severe cases the vomiting is accompanied by violent retching, and is oft-repeated without apparent cause. The severe vomiting may continue from one to six days, and then, as a rule, disappears as suddenly as it came, and with its disappearance convalescence is established. Following an attack the stomach, as a rule, resumes its functions, and within five or six days the patient is taking his ordinary food without the slightest discomfort on the part of the digestive organs. From this time on the patient rapidly regains his health and strength, and may manifest no gastric symptoms whatever until the next attack, which may recur within a month; but as a rule the interval is from two to six months. Subsequent attacks are very similar in their symptomatology to the initial attack. They may vary, however, very greatly in severity and duration. The first attack is, as a rule, diagnosed as a case of ordinary toxic gastritis, due to ptomain or other poisoning, and every effort is made to discover in the food or vomited matter the cause of the attack. When the second and third attacks follow, in spite of careful feeding and without apparent cause, the physician recognizes their constitutional origin and makes the diagnosis of recurrent vomiting.

While it is the rule that patients who suffer from recurrent vomiting may have in the interval no stomach or intestinal disturbance, yet this is a rule that has many exceptions, especially in patients under five years of age. Many of these are prone to have gastro-enteric disturbances at all times from very slight causes, and many of them during the first three years of life have more

or less trouble digesting cow's milk. In these cases frequent attacks of gastric indigestion with mild intestinal fermentations will now and then be broken in upon by an attack of recurrent vomiting, and following this acute attack the patient again returns to his usual condition of health. These chronic cases are, I believe, analogous to those of chronic migraine described elsewhere, and while they are vastly more common in young infants, they may occur at any age.

Character of the Vomited Matter.—Snow says: "It is to me remarkable, in an illness whose main symptom is vomiting, that so few accurate analyses have been made of the vomited matter. Testing the ejecta would seem to me to be the clew to correct diagnosis and treatment. However, Holt reports the usual findings, as of a fluid, containing mucus and free HCl. In four of my personal cases the fluid vomited was apparently pure gastric juice, containing an excess of free HCl and mucus, and in the fifth case the hyperacidity was due to combined chlorides. It is, therefore, probable that *some* cases of recurrent vomiting are the result of an intermittent form of hypersecretion of highly acid gastric juice."

It appears, however, to me that the hyperchlorhydria which Snow describes in these cases, while it may aggravate the vomiting in some of the cases, is but one of the symptoms of this neurosis, and not its prime etiological factor.

Constipation which precedes the attack becomes, as a rule, very obstinate during the attack, and, owing to the irritable condition of the stomach, which forbids medication, and the arrest of peristalsis, which accompanies

the attack, it is at times almost impossible to relieve it. When the constipation is relieved by cathartics, or by the cessation of the attack, the discharges are putrid. A few of the cases reported have had loose putrid movements throughout the attack, and this, while unusual, is more likely to occur in very young patients.

Thirst.—While there is absolutely no desire for food in most cases, thirst is a striking symptom. The little patients are frequently asking for water, even when it is immediately rejected. When the attacks are prolonged, and when no food or water has been retained for days, the thirst is excessive, and there is usually a parched, dry tongue.

Emaciation is great in the aggravated, long-continued cases. There are few diseases that produce more emaciation in a shorter period of time. As little or no fluid is retained in many of these cases, the tissues are drained of water, and as a result the general emaciation is very rapid. The abdomen is boat-like or flattened, the eyes are sunken, and this, with the anxious expression of countenance, gives the impression of great danger to life.

The prostration in these cases keeps pace with the emaciation. In all cases it is marked, and in some cases so extreme as to demand the most powerful stimulants to tide the patient over the attack.

Fever is present in nearly every case under ten years of age. From this time on fever is less common, until in adult life it is, as a rule, absent. The fever occurs early in the disease, often among the prodromes. It may continue for two or three days, varying in height

from 101° to 105° F. As a rule, after the second or third day the temperature commences to subside, and in the latter stages may be subnormal. At times the fever subsides very early in the attack, with the onset of severe vomiting.

The pulse is irregular, as a rule, and usually rapid.

The tongue in the beginning may be coated, but in the latter stages of severe attacks is dry. The peculiar acetone odor of the breath becomes more marked as the attack progresses. A few of these cases complain of sore throat during the attack, and in these cases the pharynx and tonsils may be irritated.

Respiration may be sighing, or rapid and panting, out of proportion to the pulse and temperature. The respiratory phenomena in this disease are probably due to the direct irritation of these centers by toxins.

Narcotism, which marks the characteristic close of the migrainous attack, is, from my own experience, not uncommon in this condition. Almost nothing is said concerning this symptom in the reported cases. I am, however, of the opinion that in nearly all of the severe cases there is, in the latter stages, a tendency to somnolence, and that a prolonged sleep, not infrequently, is followed by the first indications of improvement. In the earlier cases of this disease which I reported I failed to note this symptom.

Gastric pain is not present during these attacks in children. I believe, however, that in the adult gastric pain of great severity may occur, associated with a severe recurrent vomiting. In some of these cases, at any rate, we have a symptom group exactly similar to that of the

child, plus the gastric pain, and these painful attacks may occasionally alternate with painless attacks of recurrent vomiting or with migraine. These are, perhaps, the cases of periodical vomiting described by Leyden.

Nervous Symptoms.—While patients suffering from recurrent vomiting may be perfectly free from gastro-enteric disturbances during the interval, they are, one and all, nervous, presenting varying degrees of general nervous excitability and restlessness. Snow described a case in which convulsions occurred at the onset of nearly every attack, and I have seen two such cases. Many of these children are precocious, and this precocity, like the precocity of migrainous children, may, if properly guarded and restrained, continue throughout adult life. The precocity of the gouty child, whether the child be subject to any of the gouty explosive neuroses or not, is to be distinguished from the precocity which occurs in tuberculous children. Children of tuberculous type are usually undersized and whimsical, and their precocity, which is coupled with physical inferiority, is fitful and sadly lacking in symmetry. The mental precocity of the gouty child, however, does not necessarily mean physical degeneracy, and it may, if properly treated, be sustained and continued throughout the life of the individual.

Urine.—The urine, in a case described by Holt, resembled that passed during an attack of migraine. It becomes more scanty as the attack progresses. It is very concentrated and strongly acid in reaction. This acidity causes a rather heavy deposit of uric acid and urates, although the percentage of uric acid is not in-

creased. The xanthin bodies, however, are in great excess. Albumin may be present in small quantities during the attack, although this is rather uncommon. Acetone and indican are present in perhaps all of the severe cases. Many observers have found acetone in the urine of these cases, and Marfan published a series of cases which he described as "vomiting with acetonemia," and suggested that acid intoxications may be a phase of this disorder. More recently Edsall found not only acetone, but diacetic acid and oxybutyric acid in the urine of a number of these cases.

DIAGNOSIS

The diagnosis of recurrent vomiting is easily made if the above symptom group is kept in mind. No disease presents exactly the same picture. In the atypical cases, however, and especially in the first attack, there may be considerable difficulty. But after the second and third attacks the nature of the disease is made plain. In the first attack the condition is most commonly mistaken for a ptomain or other toxic gastritis. The intestinal symptoms, however, which develop in gastritis, and the cessation of the vomiting under starvation and proper treatment, should enable one to make the diagnosis.

Intestinal obstruction, as Griffith suggests, may probably offer the greatest difficulty in differential diagnosis, but the absence of pain and bloody mucus in the stools and of any tumor, with the presence of the characteristic symptom group, above described, should be sufficient to clear the diagnosis.

The presence of acetone in the urine with the other

urine findings, above noted, would assist in making the diagnosis.

PROGNOSIS

The prognosis, so far as recovery from the attack is concerned, is good. The vast majority of these cases recover. It should be kept in mind, however, since a number of fatal cases have been reported, that there is a possibility of a fatal ending. The prognosis, so far as the prevention of these attacks, is also good. Most of these cases can be cured, and all of them can be greatly benefited. Under proper treatment the attacks cease, and the child's general neurotic condition greatly improves. This improvement goes on, and as the child grows older its nervous system becomes more stable and the tendency to these recurring attacks is overcome. In the untreated cases these attacks may be transformed into migraine or epilepsy.

PATHOLOGY AND NATURE OF THE DISEASE

Little is known of the pathology of this disease. An autopsy reported by Griffith showed necrotic changes in the mucous membrane of the stomach and intestine, and slight parenchymatous alterations in the pancreas, spleen and kidneys, and fatty infiltration of the liver. Our present knowledge of this condition justifies us in the belief that the disease is an auto-intoxication produced by toxins closely related or identical with the purin bodies, and that a secondary acid intoxication occurs, which may contribute to the symptom group in the later stages of the attack. The auto-toxins in this condition select the vomiting center in the medulla as their point of attack.

The close family relationship which exists between migraine and recurrent vomiting has been previously noted, and I wish here, especially, to note the fact that I have records of four of my own cases where typical attacks of recurrent vomiting were changed into typical attacks of migraine as the children grew older.

In the present state of our knowledge, acetonuria means an excess of diacetic and oxybutyric acid in the urine; we may infer, therefore, that in all of the reported cases in which acetone occurred in the urine these acids were also present. Von Noorden says: "Owing to the fact that this acid (oxybutyric) is so closely related chemically to acetone and diacetic acid, one is justified in suspecting its presence in the urine whenever these two bodies are excreted in considerable quantities. As a matter of fact, one always succeeds in finding the acid under these circumstances." In the light of these observations published reports warrant the inference that acetonuria, with at least a mild degree of acid intoxication, occurs after the onset of the attack in perhaps all of the severe cases. The acid intoxication, however, is in this disease, as it is in many others, a purely secondary pathological process. Von Noorden believes that all acid intoxications produced by the presence in the tissues of acetone, diacetic, and oxybutyric acids are due largely, if not wholly, to an insufficient intake of carbohydrate food, or to some fault in the carbohydrate metabolism. A study of acid intoxications reveals the fact that this form of secondary auto-intoxication very commonly occurs in diseases which produce profound nutritional disturbances. In recurrent vomiting, therefore, we have

all the conditions necessary to produce acid intoxications; first, an insufficient intake of carbohydrate food; second, profound nutritional disturbances, and third, faulty carbohydrate metabolism produced by the functional incapacity of the liver. The acid intoxications in this condition are, therefore, secondary rather than primary. It may further be noted that the characteristic symptom group which this disease presents is not that of acid intoxication, but in the later stages of this disease, when the acid intoxication is more marked, it is possible that the respiratory disturbances, the increased pulse rate, the lowering of the body temperature, and the tendency to somnolence may perhaps be partly due to this intoxication.

TREATMENT

Treatment of Attack.—If seen in the prodromal stage, ¼ of a grain of calomel and 5 grains of bicarbonate of soda should be given every half-hour until 2 grains of calomel are taken. And if the stomach be not too irritable, the calomel should be followed in two or three hours by a saline laxative, and four or five hours later by benzoate of soda in from 3 to 8 grain doses every two or three hours, dissolved in essence of pepsin and peppermint water. No food whatever should be given. Water may be allowed if the stomach will retain it.

After the attack is well on, the nausea and vomiting preclude not only all food, but all stomach medication. The calomel and bicarbonate of soda, however, may be tried at any stage of the attack, and if the nausea and vomiting are not greatly aggravated by them, they may

be continued. At intervals throughout the attack water may be allowed, even though the stomach rejects it; but no food is to be given until the patient is able to retain water in small quantities.

In cases where food and water are not retained by the stomach it is advisable to give, at intervals of every eight to twelve hours, a high rectal enema of physiological salt solution, or bicarbonate of soda solution, a tablespoonful to the pint of water. The tissues, as a rule, are so starved for water that these solutions are absorbed, and the water thus absorbed serves to flush out the various excretory organs and in this way promote the excretion of auto-toxins. The bicarbonate of soda given by the rectum or the mouth serves the purpose of neutralizing acids, thus removing or preventing the secondary acid intoxications which occur in these cases. Edsall's suggestion that very large doses of bicarbonate of soda be given by the mouth is a good one in those cases in which the soda is retained, but my experience is that the cases which need this treatment most are the ones which retain nothing on the stomach. In some cases, however, the soda is retained when all else is vomited, and even in those cases where it is immediately rejected it may perhaps do some good by neutralizing the acids in the stomach.

In the most aggravated cases, where prostration is extreme and stimulation strongly indicated, sterile physiological salt solution may be injected into the subcutaneous tissues. In cases of this kind, also, it occasionally becomes necessary to give morphine hypodermically. This remedy acts specifically in the control of the vomiting, and in the worst cases it is a life-saving measure.

Small doses of from 1-10 to 1-20 of a grain, depending upon the age of the patient, are usually sufficient.

Hygienic and Climatic Treatment.—It will be found that many of these children prefer an indoor life and intellectual pursuits. For habits of this kind, an outdoor life, with moderate exercise in the open air and in a suitable climate, should be substituted. Since these cases occur very commonly among the well-to-do, it is often possible to prescribe an outdoor climate the year round. Our Southern States, and especially Southern California, are admirable winter climates for these children, while the region of the Great Lakes or the sea-coast of our North Atlantic States offer favorable climatic conditions during the summer. Sea voyages are also beneficial.

It should be remembered that while the climatic treatment of many of these cases is important, it does not take precedence over the medical, dietetic, and general hygienic treatment which may be carried out in any climate; and my experience leads me to believe that these cases do better at home during the greater portion of the year, provided the home offers favorable opportunities for carrying out the general treatment here outlined. But even where the treatment is carried out under favorable home conditions a change of climate for a few months during the year is advisable, and by this change the hot months of summer or the cold of winter may be avoided, as the climatic conditions at home may dictate.

These children should, as a rule, be taken out of school, and lead as quiet and uneventful lives as possible. Mental stimulation, nervous excitement, and all forms of mental and physical fatigue are to be avoided for a num-

ber of years, or until the child's physical and nervous condition justifies a return to the ordinary routine of child life.

Dietetic Treatment.—The diet should be carefully restricted, and selected. In beginning the treatment all raw fruits and acid vegetables are to be eliminated from the diet. Strawberries, rhubarb, tomatoes, salads, tea, coffee, beef juice, beef tea, and alcohol are to be avoided, and the child should be allowed to eat but sparingly of beef and sweets. The following foods may be recommended: Milk, cocoa, vegetable soups, cereals, well-cooked vegetables, cooked fruits, eggs, fish, chicken, mutton, and, occasionally, beef. Children suffering from recurrent vomiting have, as a rule, in the interval between the attacks abnormally large appetites. They are therefore to be carefully guarded against taking an excess of food of any kind, and are to be made to cultivate the habit of drinking water between meals.

Medical and other Treatment.—Before beginning the medical treatment sources of reflex irritation on the part of the eye and elsewhere should be carefully sought for and, if possible, removed. Constipation, which is constantly present in this condition, demands our most thoughtful consideration. It must be relieved. This can usually be done by palatable solutions of sulphate and phosphate of soda. These saline laxatives are advisable in the beginning of the treatment. Later, palatable mixtures of rhubarb and cascara sagrada may be used. Enemata are not to be relied upon in the treatment of this condition. Abdominal massage may sometimes relieve the constipation, and where it is necessary to resort

to massage for this purpose, it is advisable to give the patient general massage at the same time.

I am coming more and more to believe that general massage, apart from the influence it may have on constipation, is one of the most valuable remedies we have in overcoming the constitutional conditions which predispose to recurrent vomiting. This is especially true in patients of feeble constitution so situated that an outdoor life with active exercise cannot be had. In beginning this treatment the massage may be given every day, every second day, or two or three times a week, according to the exigencies of the case, and should be continued until the child's physical condition is such that he can lead a strenuous outdoor life without undue fatigue or other untoward results.

In the medical treatment of this condition, however, the wintergreen salicylate of soda and the benzoate of soda, put up in palatable solution in a dose to suit the age of the child, are our most valuable remedies. The following prescriptions will be found to act specifically in preventing attacks.

℞

Sodii salicylatis (gaultheria) . . . . . ½ dram
Sodii benzoatis . . . . . 2 drams
Pepsin essence . . . . . 2 ounces
Aquæ menth. pip. . . . . 2 "

M. S.—Teaspoonful after meals for a child six years of age.

℞

Sodii bicarbonatis . . . . . 60 grains
Hydrarg. cum cretæ . . . . . 20 "

M.—Ft. capsules No. 20.

Sig.—One capsule three times a day.

In the more severe cases one of these prescriptions must be continued, as above directed, for months at a time, and after this is to be given once or twice a day for an indefinite period.

In children over six years of age nothing within the range of my experience acts so well in the treatment of the underlying constitutional condition as Siphon C (see "Treatment of Migraine," page 208). Children above this age can be induced to take early each morning a dose of this siphon sufficient to produce a movement of the bowels during the forenoon. This one dose of medicine each morning will, I believe, with the dietetic and hygienic treatment above outlined, protect the child against future attacks. It will, however, perhaps be safer in the early treatment of the case to give, in connection with the siphon medicine, the salicylate of soda prescription above noted. After a few months of treatment, however, all medication other than the siphon formula may be discontinued. I have found it necessary occasionally to interrupt the alkaline treatment and substitute such tonics as malt and arsenic. In this event, however, it is necessary to give some such laxative as cascara sagrada.

The general treatment here given is largely the same as that outlined by me in 1898, in the "American Text-Book of Diseases of Children," and many years of experience have taught me that under this treatment the prognosis, even in the most severe cases of recurrent vomiting, is good not only as to the prevention of attacks, but also as to permanent recovery.

CHAPTER XVII

EPILEPSY

Definition.—The syndrome which, regardless of its etiology, we call epilepsy is characterized by habitually recurring convulsive seizures, local or general, accompanied by temporary loss of consciousness, and commonly terminated by a narcosis which produces a sleep from which the patient awakens convalescent from the attack.

Epilepsy in its early history was spoken of as the "falling sickness." This loss of equilibrium was thought by the earlier writers to be a necessary symptom. At the present time, however, we recognize a large group of seizures as epileptic in which this symptom is absent, and many of our best medical authorities now assert that neither loss of consciousness nor convulsive movements are necessary to an epileptic seizure. By these writers the syndrome of epilepsy has been deprived of one after another of its symptoms, until there now remains, as a necessary characteristic of the epileptic paroxysm, only the habitual recurrence of attacks, not especially defined as to their nature.

Since the pathology of epilepsy is so obscure and since the term includes a number of conditions differing widely in their pathology, it is manifestly impossible to satisfactorily define epilepsy from an etiological or pathological standpoint. In this dilemma the term epilepsy has come to mean a well-defined symptom group, which

has striking characteristics, notwithstanding its diversified etiology and pathology. It seems, therefore, that until a definite etiological or pathological basis of classification can be decided upon by which we may determine what is and what is not epilepsy, it is most important that a definite syndrome should be recognized as epilepsy; otherwise all is confusion. The characteristics of the syndrome of epilepsy as recognized in this chapter are not only habitually recurring attacks, but loss of consciousness and convulsive muscular action, be they ever so slight.

The great variation in severity of these symptoms, with the addition of a large number of other symptoms, gives great variation to the clinical picture presented by individual attacks of epilepsy, and justifies their separation into rather well-defined clinical groups.

PATHOLOGY

If the literature of epilepsy is agreed upon any one fact pertaining to its pathology, it is that the most important factor in its production is an irritation of the motor neurons of the cerebral cortex. The irritation which causes the violent and rapid discharge of nerve force may be either chemical, mechanical, or reflex.

Epilepsy from a pathological standpoint may be divided into three distinct types, Developmental, Organic, and Toxic.

DEVELOPMENTAL EPILEPSY

Developmental Epilepsy, the synonyms for which are idiopathic, hereditary, and reflex epilepsies, has as its

underlying pathological condition a lack of development of the higher inhibitory centers which control spinal convulsive movements. This lack of inhibition makes it possible for slight reflex causes to precipitate a discharge of nerve force into the spinal motor cells, producing epileptic convulsions. While the reflex factor in this form of epilepsy may be necessary, it is not the most important factor, since reflexes would be impotent if it were not for the lack of inhibition and the irritable nerve centers which make these factors effective. The essential factor, therefore, is a developmental one, the nerve centers of the cortex having failed to acquire the necessary inhibitory control over lower centers.

This form of epilepsy is also spoken of as "hereditary," since the hereditary factor is here more marked than in any other. In more than one-third of these cases there is a family history of eclampsia, epilepsy, or insanity, and not infrequently a number of children in the same family are affected. The hereditary factor is here direct, since all of these disorders are characterized by a lack of inhibition. Feeble inhibition is, therefore, in a large percentage of these cases directly inherited. A family history of other hereditary neuroses is also common. This is the type of epilepsy that is believed by some writers to be occasionally produced by infantile eclampsia. The eclampsia which occurs in the early history of these cases is not the cause of the epilepsy, but both are made possible by the hereditary weaknesses of the nervous system above noted.

Chronic malnutrition is a very important factor in producing this form of epilepsy; it acts by still further

weakening the hereditary defects above noted. (In Chapter II, I have noted the influence of malnutrition in interfering with the development of inhibition in the rapidly developing nervous system of the young child.) One can readily see, therefore, that such diseases as rachitis, chronic gastro-intestinal disorders, tuberculosis, rheumatism, heart disease, the acute infections, and all the diseases of childhood which produce chronic anæmia, and consequently chronic malnutrition of the nervous system, may assist in the development of this type of epilepsy in children who have inherited feeble inhibition.

This group of epilepsies is probably the largest of all, but it is not so large as was formerly supposed, since many of the cases previously classed as idiopathic or developmental are now known to be due to organic diseases of the nervous system.

ORGANIC EPILEPSY

Synonyms.—Symptomatic, mechanical, focal, and Jacksonian epilepsy.

Organic epilepsy has as its essential pathological condition some organic disease of the nervous system, such as porencephalus; microcephalus; cysts formed by a softening of the brain substance secondary to obstruction of the middle cerebral artery or to thrombosis; tumors of the brain and cord due to secondary syphilis or other causes; traumatism producing fracture of the skull or cerebral hemorrhage; lastly, and most important of all, cortical hemorrhages occuring as one of the accidents of birth, or resulting from severe convulsions, or injury to the head in very early infancy.

In the injury to the brain which results from fracture of the skull, hemorrhage, or tumor, mechanical irritation produces a circumscribed menigo-encephalitis, resulting in more or less degeneration of the cells of the cerebral cortex and sclerosis of the neuroglia tissue. These secondary changes explain the continuation of the epileptic paroxysms after the original excitants have been removed. It has long been known that these injuries to the brain are responsible for a large number of epilepsies; but a new interest has been added to this subject by the admirable clinical studies of B. Sachs, who has demonstrated that many obscure epilepsies developed in late childhood are focal epilepsies having their origin in cortical hemorrhages which occurred in infancy. In many of these cases the epilepsy develops long after the paralysis and spastic palsy has disappeared, so that they are commonly classed as developmental, or idipathic epilepsy. In these cases a careful inquiry into the previous history of the child may discover a paralysis in early infancy, and a careful examination of the patient may show an exaggeration of deep reflexes, or an inequality in the strength of the muscles on the two sides. The dynamometer, for example, may show that the muscles of the hand are weaker on the side of the body which presents the exaggerated reflexes. By these signs and symptoms, as well as from the early personal history of the patient, one is often able to make a diagnosis of organic epilepsy in cases that would otherwise be classed as developmental. Epilepsy due to tumors of the brain and cord and to hemorrhagic lesions in the region of the basal ganglia, such as arterial obstruction and

thromboses, apparently prove that cortical irritation is not absolutely necessary to the production of epilepsy. These hemorrhagic lesions may follow heart disease, rheumatism, scarlet fever, pneumonia, and other acute infections, so that in rare cases these diseases may be classed as etiological factors of this form of epilepsy.

Heredity plays an unimportant rôle in the production of organic epilepsy.

TOXIC EPILEPSY

Synonyms.—Migrainous, lithæmic, and leucomain epilepsy.

There is a type of epilepsy which may be classed as toxic, the essential factors in the production of which are auto-toxins. In this type of epilepsy the auto-toxins act upon the convulsive centers at the base of the brain, producing convulsions in the same manner that toxins produce the eclampsia of infancy. The epileptic convulsions produced in this manner are in every way similar to toxic infantile convulsions. All that is necessary to convert an ordinary toxic eclampsia into an epilepsy is to have the intoxications and consequent convulsive seizures recur often enough to establish the epileptic habit. When this habit has been established it is possible that epileptic convulsions may occur, not directly caused by anto-toxins; yet this type of epilepsy is essentially auto-toxic in origin, and for the most part the subsequent convulsive seizures are, as I believe, produced by recurring attacks of auto-intoxication in the same manner that migraine and recurrent vomiting are produced.

A large group of these toxic epilepsies, is, I believe, produced by auto-intoxins, either identical with or closely related in their formation to the purin bodies. This is the form of epilepsy which is so closely related to migraine, and which furnishes the connecting link between these two syndromes.

Toxic epilepsy is not in any way related to the organic form. It may, however, be related to developmental epilepsy in that certain individuals may inherit both feeble inhibitory control of motor nerve centers and a predisposition to migraine. In such cases as these the inefficiency of the inhibitory centers not being sufficient to produce epilepsy, the auto-intoxication which would otherwise manifest itself in an attack of migraine now becomes an attack of epilepsy. The auto-intoxication thus becomes the all-important factor in developing the epileptic habit. As time goes on and recurring attacks of auto-intoxication produce recurring attacks of epilepsy, the inhibitory weakness of the cortical centers may become so exaggerated as to convert the case into a mixture of the developmental and toxic types of epilepsy, so that slight epileptic paroxysms may at times be produced by trivial reflex causes, and at other times severe paroxysms may be produced by the recurring auto-intoxication.

In the development of nearly all of these cases there is a history of attacks of migraine extending over a number of years before the epilepsy appeared. The paroxysms of epilepsy may then be substituted for those of migraine, and thereafter the case may be one of epilepsy, or of epilepsy alternating with migraine. In all of

these cases there is a family history of either gout or migraine.

In one of my cases in which there was no family history of epilepsy, but a very strong family history of gout, migraine, and recurrent vomiting, the patient herself suffered from recurrent vomiting until she was twelve years of age; the attacks were then transformed into migraine, from which she suffered for three years; at the age of fourteen epilepsy began to alternate with migraine, and a few years later, the epileptic habit being established, all of the attacks became true epilepsy. The epileptic attacks in this case were frequently preceded and followed by a severe unilateral headache, giving the symptom complex of both migraine and epilepsy.

The sequence in this case of recurrent vomiting, migraine, and epilepsy was unmistakable, and there seems little room for doubt that all were produced by the same or a very similar auto-intoxication. I have in previous chapters noted the kinship which exists between recurrent vomiting and migraine; the relationship which exists between migraine and epilepsy is a matter of medical history. Concerning this relationship, Landon Carter Gray says: "Some eleven years ago I called attention to the association of epilepsy with migraine, not knowing until some time later that Tisset, Parry, and Liveing had previously observed the same association. In these cases epilepsy alternates with migraine, the migraine disappearing when the epilepsy appears, and the epilepsy returning when the migraine disappears. By this I do not mean to say that all cases of migraine are subject to epilepsy, but I do mean to say that there is a very close

relationship between migraine and epilepsy, and in some cases the relation is so close as to permit of this alternation; indeed, almost all cases of migraine will be found at some period of their lives to have had a loss of consciousness with or without convulsive movements, although generally this fact is strenuously denied."

All recent writers upon this subject speak of the close relationship of migraine and epilepsy, so that I think one is justified in asserting that these two syndromes are not infrequently twin inheritances from the same gouty ancestors. I wish to insist, however, that migraine bears this close etiological relationship only to toxic epilepsy, that it is not in any way related to organic epilepsy, and that it is not related, but may be associated with, developmental epilepsy in the manner above described.

The urine passed by patients suffering from migrainous epilepsy is similar to that passed by patients suffering from true migraine. It is usually concentrated, highly colored, strongly acid in reaction, and has a heavy deposit of urates. The uric acid is not increased in quantity, but the xanthin bodies are.

Intestinal Toxæmia.—Herter and Smith in an admirable research have called attention to the influence of intestinal toxins in producing epilepsy. Their observations show that intestinal putrefaction is very common in epileptics. A large percentage of these cases, as shown by the presence of ethereal sulphates in their urine, were suffering from some degree of intestinal toxæmia. In these cases, however, the intestinal toxæmia was a constant condition and not especially associated with the seizures. The inference therefore is that the intestinal

intoxication in these cases may have aggravated the general nervous irritability of these patients, and in that way aggravated or increased the number of their attacks. It does not appear, however, that the intestinal intoxication was the essential cause of the epilepsy in these cases, and such in fact is not claimed by Herter and Smith. It is important, however, to keep in mind the fact that constipation and resulting intestinal fermentation, which are so common in the toxic and developmental types of epilepsy, may be contributing factors, and therefore justify careful therapeutic attention.

GENERAL ETIOLOGY

Age is an important etiological factor in the various types of epilepsy. The organic type of epilepsy commonly begins before the tenth year; the developmental between the tenth and the twentieth year, and the toxic after the twentieth year. Cases of epilepsy, however, belonging to any of the above types may occur at any of these periods. Gowers analyzed 1450 cases of epilepsy, and found that they occurred regardless of their types as follows:

Under 10 years	422 cases
From 10 to 19 years . . .	665 "
" 20 " 29 " . . .	224 "
" 30 " 39 " . . .	87 "
" 40 " 49 " . . .	31 "
" 50 " 59 " . . .	16 "
" 60 " 69 " . . .	4 "
" 70 " 79 " . . .	1 case

Sex.—Females are rather more liable to be affected than males. This is largely due to the influence of menstruation.

Exciting Causes.—I do not believe that reflex causes are ever wholly responsible for the development of a case of epilepsy. I have, in the chapter on " Reflex Irritation," called attention to the fact that the constant nagging of reflexes may, if continued for a sufficient length of time, produce marked changes in the cells of the spinal cord; changes from which these cells require a long period of rest to recover. Spinal cord cells, subjected to such influences, become abnormally irritable and excitable. They discharge their nerve force fitfully, and under slight reflex provocation. If the chronic reflex irritation, however, be removed, and the cells be allowed a sufficient period of rest, they return to their normal condition, both as to structure and function. In the light of these physiological facts one may suppose that the various ganglia of the brain, or the motor cells of the cortex, may in like manner be so structurally and physiologically changed by strong chronic reflex irritation that they become irritable and discharge their nerve force under slight provocation. If this be true, one can understand how long-continued chronic reflex irritation, from eye-strain and from diseases of the throat and nose, may be very strong exciting causes in the development of epilepsy, and one can understand how these causes, when combined with an hereditary weakness of the inhibitory centers, may be sufficient to produce epilepsy. In such cases, however, the feeble inhibition is the important etiological factor, and diseases of the eye, throat, and nose are the strong exciting fac-

tors without which, in all probability, the epilepsy would not have developed. In the early history of such cases as these the removal of the reflex factor may cure the epilepsy; but later, when the epileptic habit is well established, the removal of the important etiological reflex factor may diminish the number of attacks, but does not cure the epilepsy, since inhibition has now become so feeble in these cases that slight, unavoidable reflex factors may excite a paroxysm. The fact that the removal of the exciting cause cures the epilepsy does not prove that this cause was the underlying factor of the epilepsy. It only proves that the all-important factor of feeble inhibition was not, in this particular case, important enough to produce epilepsy without the aid of a strong and constantly acting reflex factor.

Among other important exciting causes of epileptic paroxysms may be mentioned intestinal irritation from undigested food, worms, or foreign bodies in the intestinal canal, diseases of the genito-urinary tract, such as stricture, adherent prepuce, and stone in the kidney; laryngeal irritation, fright, deficient oxygenation of the blood from remaining in over-crowded rooms, masturbation, sexual excess, heat stroke, and menstruation. Menstruation is, in fact, one of the most important of all of the exciting causes. In many cases there is always a recurrence of the epileptic attack at or near the menstrual time.

SYMPTOMATOLOGY

There are two distinct types of epilepsy. The major attacks, or *grand mal,* are characterized by a sharp cry,

loss of consciousness, a fall, and tonic convulsive movements, quickly succeeded by general clonic convulsions. The convulsive movements last for a few minutes and are followed by a profound sleep, which may continue for an hour or two. From this sleep the patient awakens convalescent from the attack and with little or no knowledge of what has happened.

The minor attacks, or *petit mal,* are characterized by sudden loss of consciousness of short duration, sometimes only momentary, and by slight local convulsive movements, which may be confined to the fingers or face. These convulsive movements are often so slight as to escape attention. The patient recovers himself almost immediately, and is usually conscious that an interval of unconsciousness has passed.

Both unconsciousness and convulsive movements, be they ever so slight, are necessary parts of both clinical types, and the habitual recurrence of these symptom groups stamps the disease as epilepsy. Between these two extreme types we may have great variation in the severity of these two constant symptoms, and these gradations, with the less characteristic symptoms that mark the individual attacks, give great variety in symptom grouping to epileptic seizures.

Aura.—The *grand mal* attacks may occur suddenly without warning symptoms, but as a rule they are preceded by certain prodromes known as aura. The aura in the Jacksonian type of epilepsy may be motor, such as a local spasm of the face, hand, or leg; or they may be sensory, presenting some disturbance or sensation in the same parts of the body. As a rule, a numbness or

tingling sensation precedes the local spasm, and the convulsive movements are first unilateral and then become general.

In toxic epilepsy the aura may be vertigo, hemianopsia, light and dark spots, or flashes of light before the eyes; a sensation of fullness in the head, or nausea.

In developmental epilepsy the aura may be a vague sensation in the stomach, a feeling of numbness or tingling in the extremities, general restlessness, irritability of temper, aphasia, a dazed, dreamy condition, or the ocular phenomena above noted.

The prodromal symptoms, however, to the *grand mal* attacks of epilepsy are so varied that each individual comes to recognize his own particular aura. In many of these cases the patients learn by experience to heed these warning symptoms, and seek safe quarters before the onset of violent symptoms.

Loss of consciousness, which is the most characteristic symptom of the epileptic attack, has strange variations in its manifestations. In certain cases dream-like states with partial loss of consciousness may immediately precede the attack, and may continue for a number of days following the attack. When suddenly the patient recovers consciousness, he may find a number of days or weeks have passed and made no imprint on his memory.

The convulsion, which is the next most characteristic symptom of the epileptic attack, varies greatly in severity and character. The violence of the convulsion may be so great, in rare instances, as to break bones and cause painful bruises, and, on the other hand, it may be so slight in the *petit mal* attacks that the momentary twitch-

ings of the muscles of the face or hands may not be observed at all. The convulsion may be general in character from the onset, as is the rule in developmental and toxic epilepsy, or it may, as in organic epilepsy, be partial, confining itself to one member of the body or to one-half the body, or it may begin as a local and later become a general convulsion.

In severe general epilepsy the pupils are dilated, there is no reaction to light, there is more or less spasm of the muscles of respiration, producing cyanosis and irregularity of the respiratory rhythm; the face may be bloated and distorted; spasm of the muscles of the jaw may result in biting of the tongue, so that the froth which exudes from the mouth during the paroxysm may be tinged with blood; and involuntary passages of urine and fæces commonly occur. At the beginning of the paroxysm the face may be pale; later it may be dark and congested.

In the *petit mal* attacks the loss of consciousness is often so slight that they are mistaken for "spells" of various kinds, such as dizziness, fainting turns, or conscious tricks. But these attacks, be they ever so mild, are none the less serious in character, and are, as a rule, accompanied by a complete change in the child's mental condition; it becomes more irritable, and sometimes a mild form of mania is developed.

As previously noted, the habitual recurrence of these attacks stamps the condition as epilepsy. The recurrence of attacks, however, is in most cases extremely irregular, except in those cases where menstruation is so important an exciting cause as to produce regular monthly

attacks of epilepsy. In these cases we sometimes have not only regular attacks of menstrual epilepsy, but also intermenstrual attacks, occurring half-way between the menstrual periods. In most cases, however, the attacks are very irregular. A number of attacks may occur within twenty-four hours, and then an interval of days, weeks, or months may elapse before another attack occurs.

In those cases in which there is progressive degeneration of mental faculties, there is a tendency to increase in the frequency of attacks. In certain cases the epilepsy may occur during sleep, and never during the waking hours. In these purely nocturnal cases there is sometimes great difficulty in diagnosis, if the patient occupies a bed and room alone. In some of these cases, however, biting of the tongue may produce blood upon the pillow; or incontinence of urine and fæces, followed the next morning by a sense of lassitude, mental dullness and headache, may lead one to suspect nocturnal epilepsy, and the diagnosis may be made by having an attendant sleep with the patient.

Procursive epilepsy is rather a rare form in which the symptoms of *petit mal* are associated with strange running movements.

Mental Symptoms.—In nearly all cases of epilepsy, as the disease progresses, there is more or less mental impairment. In the purely toxic forms of the migrainous type there may perhaps be little or no loss of mental capacity. The mental symptoms of organic epilepsy will depend altogether upon the location and extent of the organic disease. Well-marked mental impairment is the

rule, however, even in the milder cases of this type. Some of the more severe cases are congenital idiots.

In the great group of epileptics belonging to the developmental class, progressive mental impairment, with a tendency to the development of idiocy, melancholia, or mania, is common. As a rule, however, these children fail to develop mentally, maintaining their childish intelligence throughout life. In a large group, however, the mental development may be simply retarded, so that the child is considered backward, but not otherwise mentally deficient.

Associated with the melancholia which develops in some of these cases there may be a peculiar cunning which enables the epileptic to commit acts of violence, even murder, and so cover his tracks as to avoid suspicion.

Associated with organic epilepsy we not infrequently have disturbances of speech and slight degrees of spastic palsy.

DIAGNOSIS

There is little difficulty in recognizing an attack of *grand mal*. These cases can scarcely be confused with anything except hysteria. In hysteria, however, the warning cry is absent, the loss of consciousness is not, as a rule, absolute, the pupils are not dilated, the eyes, instead of being turned upward and inward, stare into vacancy, there is no involuntary passage of urine and fæces, and there is no prolonged sleep following the attack. In some cases, however, we may have a queer combination of hysteria and epilepsy, but these cases

are comparatively rare in this country, although they seem common in France.

In the diagnosis of *petit mal* there is probably greater difficulty, because of the inability or disinclination on the part of the mother to accurately describe these attacks. She is much inclined to minimize these symptom groups, and to speak of them as "spells." The physician, therefore, must attach special importance to the marked change in temperament and irritability which has occurred since these "spells" made their appearance.

Great importance attaches to the differential diagnosis of the various types of epilepsy.

In organic epilepsy there is, as a rule, little difficulty if the physician will carefully search for evidences of organic disease of the nervous system. If Sachs' advice is followed, to test in every case the comparative strength of the muscles of the right and left hand, and to search for an exaggeration of deep reflexes, as well as to inquire carefully into the early history of the child for evidence of disease of the nervous system, many cases that have been classed as developmental will be found to be organic. Partial convulsions, which may or may not become general, also indicate organic epilepsy.

When epilepsy develops suddenly in older children who have been previously healthy, one should suspect, according to Sachs, "the possibility of an intercranial tumor; and a slight weakness of the part convulsed, a possible increase of the deep reflexes in the same part, the presence of headaches and the development of optic neuritis are the symptoms which we must look for in order to establish or to discard the diagnosis of tumor."

Toxic Epilepsy.—In this form the diagnosis is made by the late occurrence of the disease, the family history of gout and migraine, the previous personal history of migraine, the character of the urine, and the stomach and vasomotor symptoms which commonly accompany the attack.

Menstruation is one of the most common of the exciting factors of toxic epilepsy, and all cases of menstrual epilepsy must, therefore, be carefully studied with reference to the possibility of their toxic origin. Mental impairment is not so marked in these cases as in other forms of epilepsy.

Developmental Epilepsy is by far the most common of all types, and all cases that cannot be differentiated as organic or toxic must be included in this group. This form of epilepsy is invariably bilateral, or general in its manifestations. Nocturnal epilepsy belongs to this class. The convulsions occur at night in these cases because the voluntary inhibitory centers are asleep, and the feeble inhibition which is characteristic of these cases is thereby still further weakened. In this form of epilepsy also we, as a rule, have mental stagnation or mental impairment, and some of the stigmata of degeneration are usually present. These are the cases, also, in which we get almost invariably a well-marked neurotic history, and in probably more than one-third of the cases there is a family history of predisposition to epilepsy or some other convulsive disorder. The *petit mal* attacks, for the most part, belong to this class. But when attacks of *petit mal* are associated in the same patient with severe *grand mal* attacks, toxic epilepsy should be suspected.

PROGNOSIS

The prognosis in organic epilepsy is always unfavorable; the severity, the nature, and the location of the organic disease will determine whether any hope is to be offered by operative treatment, as surgery offers almost the only hope for permanent improvement in these cases. A few, however, due to syphilis, may be improved by anti-syphilitic treatment.

In toxic epilepsy, if not of too long standing, the prognosis is much more favorable, since many of these cases are benefited and a few of them cured by proper treatment.

In developmental or idiopathic epilepsy the prognosis is, on the whole, bad; yet a large percentage of these cases may be greatly improved and many of them cured by careful treatment. In cases where the epileptic symptoms have lasted less than a year, and where a potent and removable reflex factor exists, the prognosis for permanent cure is good, and in those cases also where chronic malnutrition is a potent etiological factor the prognosis is not unfavorable.

TREATMENT

Treatment of Attack.—Where the aura precede the attack a sufficient length of time to permit of treatment, patients may be provided with pearls of nitrite of amyl, or with a mixture of equal parts of chloroform and nitrite of amyl, for inhalation as soon as the warning symptoms appear; in this way attacks may sometimes

be warded off. During the attack the patient should be protected from injury. Some foreign body should be placed between his teeth to prevent injury to the tongue, and violent spasmodic movements should not be restrained.

General Treatment.—Epileptics are very favorably influenced by suggestion; this may be a matter of environment, or a matter of medical or surgical treatment. Temporary improvement very commonly follows almost any change. Slight surgical operations, change of locality, any form of counter-irritation, or any new and promising line of treatment, may suspend the attacks or lengthen the interval to months in cases where the interval has been days or weeks.

In beginning the treatment it is important that a careful search should be made for exciting causes, which are usually reflex. Eye-strain should be corrected. Diseases of the nose and throat must have appropriate treatment, and an adherent prepuce or phimosis should be relieved by proper surgical measures. Many cases of epilepsy have been favorably influenced and not a few cases have been cured by the removal of reflex factors having their origin in diseases of the eye, nose, throat, and genito-urinary organs.

Since Herter and Smith called attention to the important rôle which intestinal toxæmia might play as a contributing factor in epilepsy, the profession has recognized the special importance of looking after the digestive tract in the treatment of every case. Constipation must be overcome, intestinal intoxication must, if possible, be prevented, and reflex irritation from the intestinal canal,

such as may be produced by undigested food and worms, must be removed. To do this the diet of the patient must be carefully selected with reference to his age, idiosyncrasies, and digestive capacity.

As a rule, these patients may be allowed a general diet, avoiding alcohol, coffee, tea, sweets, salads, pastry, and an excess of albuminoids. Milk, cereals, vegetables, fruits, and meats in moderate quantities may be allowed. An excess of food is especially injurious.

In menstrual epilepsy, or in those cases in which the menstrual period is the exciting cause, the pelvic organs should be carefully inspected, and any diseases of the ovaries or uterus should be removed by appropriate treatment.

It is of the very greatest importance to correct all forms of malnutrition. This is especially important in the early cases of developmental epilepsy. In cases of this kind of less than a year's duration the correction of nutritional disturbances may result in a cure.

Chronic anæmia, or chronic malnutrition, whether produced by tuberculosis, rheumatism, heart disease, chronic malaria, chronic disease of the digestive organs, hereditary syphilis, repeated attacks of influenza, or other acute infections, must receive appropriate treatment, since these factors are sometimes responsible for the development of epilepsy in predisposed individuals.

The general hygienic treatment must be carefully looked after. As Jacobi says: "The child known to be epileptic must be trained very carefully, both physically and mentally. . . . Feeding with grewsome nursery stories, tight dressing, and early schooling, also horse-

back exercise and swimming, are forbidden. In the interest both of the patient and his schoolmates a public school should not be attended. The child ought to be instructed and trained with a view of preparing him for his future calling, which must not overstrain body or mind, must not be sedentary, nor should it confine him, if avoidable, to the limits and influences of city life and air."

Medical Treatment.—The bromides are the most valuable remedies we have in the treatment of epilepsy. This treatment is not simply palliative, but when combined with the general treatment above noted, it may be, in selected cases, curative. The curative effect of the bromides probably depends upon the fact that the epileptic habit is, by this treatment, interrupted, giving the general treatment, which is always combined with the bromide treatment, an opportunity to remove important factors of the disease. The bromide treatment, therefore, should be continued for a year or more after the paroxysms have ceased, or until nutritional faults are corrected, all exciting causes removed, and the patient's general health so improved that it (the bromide treatment) may gradually be discontinued without causing a return of the paroxysms.

Strontium bromide is perhaps just as effective as any of the bromides, and it is much less irritating to the stomach. For these reasons it is the best of the bromides to use in the treatment of epilepsy in young children. It may also be used in adults where large doses of other bromides have produced stomach or intestinal irritation. Sodium and potassium bromides are, however, thor-

oughly reliable, and it is with these drugs that the bromide treatment of epilepsy has, by long usage, proven its efficacy.

Bromides are to be given in large doses—30 to 60 grains per day for a child of six years. The dose should be large enough to control the paroxysms, where this is possible. It is best that they should be given, as Seguin suggests, in large doses shortly before the expected paroxysm. In nocturnal epilepsy one large dose (one-half to two drams, very largely diluted) taken at bedtime. In other periodic forms the greater part of the daily dose is to be taken shortly before the time of the expected paroxysm. In menstrual epilepsy large doses are to be given just before and during the menstrual period, and smaller doses continued throughout the interval.

Hydrobromate of hyoscine (1-100 to 1-200 of a grain) may be given three times a day with great advantage in connection with the bromide treatment.

Belladonna is to be especially recommended in combination with the bromides in all those cases where there is any suggestion of gastro-intestinal irritation. Borax has also been recommended in 5 to 20 grain doses, combined with the bromides in these cases.

Chloral and antipyrin may be used in connection with the bromide treatment, to get control of the paroxysms in severe cases, but these drugs are not to be continued for any length of time in the treatment of epilepsy.

Fleching advises a combination of opium and bromides for the control of the paroxysms. He begins with one-half to one grain of opium per day, and gradually increases until the patient is taking 10 or 15 grains. After

six weeks the opium is stopped suddenly and large doses of the bromides substituted, and thereafter continued in the treatment of the case as long as sedative treatment is necessary.

Arsenic, in small doses, may prevent or cure the acne which develops from the bromide treatment.

Digitalis may be used when disease of the heart is thought to be a contributing factor in producing the epilepsy.

In the treatment of toxic epilepsy of migrainous origin, in addition to the above treatment the patient is to be given the systematic treatment for migraine as outlined in Chapter XV. In these cases cannabis indica is, next to the bromides, by far the most valuable remedy we have for preventing the paroxysms.

In the treatment of organic epilepsy, in addition to the above treatment, surgical measures may be of value.

The surgical treatment of epilepsy, that at one time seemed to promise so much and attracted such widespread attention, has, to say the least, been a great disappointment. The surgical treatment of organic epilepsy seems altogether rational, and no doubt more cases would be benefited if surgical interference were resorted to earlier. But these cases rarely fall into the surgeon's hands until medical and other treatments have proved inefficient.

Sachs very clearly sums up our knowledge of this subject as follows: "In a case due to a traumatic or organic lesion, an early operation may prevent the development of cerebral sclerosis. If early operation is not done, the occurrence of epilepsy is a warning that secondary

sclerosis has been established, and an operation may prevent it from increasing. Operation must include the removal of the diseased area; here, if all other parts are normal, a cure may result. Under favorable conditions a few cases of epilepsy may be cured by surgery, and many more improved."

Sachs further says: "I consider it important not to wait the actual development of epilepsy; and if the brain has sustained any considerable injury, to remove the injured tissues, which, if allowed to remain, constitute a permanent menace to the future health of the patient. We shall be able to prevent development of epilepsy very much more readily than we can cure it if once established."

CHAPTER XVIII

RECURRENT CORYZA

There is a form of coryza, recurring at irregular intervals without apparent local or external cause, which is self-limited and appears to be closely related in its etiology and pathology to recurrent vomiting; for this reason I have used the term Recurrent Coryza to describe this condition.

ETIOLOGY

Heredity.—There is, as a rule, a distinctly neurotic family history, and there is almost always a family history of gout or migraine. This syndrome is, in fact, often associated in the same patient with recurrent vomiting or migraine. The hereditary factor, therefore, in this condition is very important, and very closely allied to recurrent vomiting and migraine.

Age.—These cases are more common during childhood than during adult life.

Constipation is almost always present, and is an important etiological factor. The constipation probably acts by producing a sluggishness in the action of the liver and a gastro-intestinal toxæmia.

Toxins, either auto or intestinal in origin, are believed to be the all-important causative factors in the production of this neurosis. The auto-toxins of the gouty diathesis which, as I believe, are etiologically related to migraine

and recurrent vomiting may produce this syndrome by their action on the vasomotor nerves supplying the mucous membranes of the nasal passages and eyes. Intestinal toxins, such as commonly find expression in urticaria and other vasomotor phenomena, may also be etiologically related to these attacks of recurrent coryza. What determines this portion of the vasomotor nervous system as the point of attack for these poisons is not altogether clear, since in most instances the exciting causes of the attack are not apparent. In a minority of the cases it may be that a special instability of the vasomotor nerve supplying the parts attacked has been developed by some local irritation in the throat, nose, or eye.

SYMPTOMS

Constipation, loss of appetite, general nervous irritability, and sallowness of skin may be prodromes to an attack of recurrent coryza.

The attack itself comes on with an acute congestion of the nasal mucous membrane, accompanied by a profuse, irritating, thin mucous discharge from the nose, which produces redness and swelling of the lip over which it flows; at the same time there is commonly an acute congestion of the mucous membranes of the eyes, marked by a redness and swelling of the conjunctiva, intense photophobia, and a profuse overflow of tears. These symptoms come on rapidly and produce a state of extreme general nervous irritability. The patient seeks a darkened room and buries her head in the pillows or shields her eyes with her hands when any light is admitted.

These attacks are self-limited. The symptoms continue in the worse cases for four or five days, and then quickly subside. The convalescence is very rapid; within two or three days after the symptoms begin to disappear the patient is quite well, showing little or no evidence of disease of the mucous membranes, which were so recently the site of such extreme irritation. These attacks may recur from time to time at irregular intervals, very like those of migraine and recurrent vomiting, and in the interval between the attacks there may be no evidence of disease of the mucous membranes of the eye and nose.

The above description represents the severe type of this disorder. In milder cases the attack may manifest itself as a more or less severe coryza without the eye symptoms, and may, in this form, occur as one of the prodromes of an attack of recurrent vomiting. Vasomotor coryza is also not uncommonly associated in its clinical manifestations with an urticaria of the skin.

The urine passed during a severe attack of recurrent coryza is highly colored, strongly acid in reaction, scanty, of high specific gravity, and contains an excess of the purin bodies.

Pediatrists have given little or no attention to these cases; they are, however, described by laryngologists under the titles "Vasomotor Coryza" and "Periodic Hyperæsthetic Rhinitis." Lenox Browne speaks of these cases as being caused by "sensitive spots in the nose, with a vasomotor debility and some local irritant as coöperative factors." Kyle says that "they may be due to a local irritant acting from without, usually of botanic origin, or to local irritation from an internal irri-

tant, such as uric acid. . . . The form due to the rheumatic or gouty diathesis is more amenable to treatment than any of the other varieties." And for these cases he prescribes water, sodium phosphate, lithium, and Basham's Mixture.

TREATMENT

Treatment of the Attack.—Local treatment is of little avail; in severe cases, however, a spray of cocaine and adrenalin chloride may be tried. Bromide of potash and tincture of belladonna, in doses to suit the age of the child, should be given throughout the attack. This sedative medication relieves the general nervous irritability and makes the child more comfortable until the self-limited attack has run its course. Medicines, perhaps, have little influence in shortening these attacks. One-fourth grain doses of calomel, combined with five grains bicarbonate of soda, should be given until six or eight doses are taken, and this should be followed by a saline cathartic, preferably the sulphate of magnesia.

In the interval between the attacks the bowels are to be kept open with sulphate or phosphate of soda, which may be dissolved in elixir of teraxicum or some other palatable vehicle. A dose of these medicines sufficient for the purpose may be taken at bed-time or on arising in the morning. In the majority of cases this simple medication will suffice to prevent a recurrence of these attacks. Patients, however, who fail to respond to this treatment may be given, in addition, five to eight grains of benzoate of soda, dissolved in essence of pepsin, after luncheon and dinner. The dietetic treatment is important.

Tea, coffee, sweets, and an excess of red meats are to be avoided, but milk, cereals, vegetables, cooked fruits, chicken, fish, and eggs, and a moderate amount of fresh red meat may be allowed.

These children should also be protected from nervous strain and excitement, and should be encouraged to lead an active outdoor life.

AUTUMNAL CORYZA

Autumnal coryza, or hay-fever, is a form of periodic coryza occurring in the late summer months. It is most severe from the middle of August to the middle of September. The attack, as a rule, lasts from five to six weeks, and during this time the patient either suffers continuously from the coryza or has recurring attacks, the length and severity of these attacks depending upon the exposure to the exciting causes and to the intensity of the hereditary predisposition. This disease is rare in young children; it is seen, however, not infrequently after the tenth year. Its manifestations in the child do not differ in any way from those in the adult, and it is here noted only for the purpose of differentiating it from recurrent coryza.

ETIOLOGY

Heredity is a strong factor in producing this disease. A neurotic or gouty family history is commonly found, and a family history of hay-fever is not infrequent.

Auto-toxins are believed by many writers to play an important rôle in the production of autumnal coryza, but

in the present state of our knowledge we know little of the character of these toxins. Many writers believe that they are closely allied to the auto-toxins of the gouty diathesis.

Diseases of the throat, and especially of the nose, are exciting factors which tend to aggravate, prolong, and precipitate attacks of autumnal catarrh.

There can be no doubt, however, that the most important of the exciting causes come from without, in the nature of irritants received by inhalation, the most important of which are furnished by plant life in the nature of pollen. In this regard hay-fever differs radically from recurrent coryza, which is apparently brought on by toxins formed within the body.

SYMPTOMS

The symptoms of autumnal coryza are very much like those of recurrent coryza. In the former the catarrhal inflammation of the nose, eyes, throat, and bronchi is a more or less chronic condition extending over a period of weeks. The attacks are not self-limited, but depend for their duration and severity upon atmospheric changes and the presence of certain irritants in the inspired air.

TREATMENT

The local treatment of the upper air passages with solutions of cocaine and adrenalin gives great relief. But a change of location to an atmosphere that does not contain the irritants which excite the paroxyms is the

only successful means of controlling the attack. By removal to suitable localities the attack may be entirely relieved or greatly modified. The patient may return home with safety, as a rule, after the first general frost, which is believed to destroy the pollen, or vegetable matter, the presence of which, in the atmosphere of a locality, will excite the disease in susceptible individuals.

CHAPTER XIX

A CLINICAL STUDY OF CASES ILLUSTRATING THE KINSHIP OF RECURRENT VOMITING, RECURRENT CORYZA, TOXIC EPILEPSY, AND MIGRAINE

Migraine is by far the most common of the above-named syndromes, and in previous chapters I have noted the close relationship which exists between each of these symptom groups and the migrainous diathesis. It is my belief, as previously expressed, that true migraine is essentially an auto-intoxication, and that the same auto-toxins which produce migraine may also be responsible for recurrent vomiting, recurrent coryza, and one form of toxic epilepsy (migrainous epilepsy).

I am not prepared to discuss what determines the particular syndrome to be developed in any given case further than to say that these poisons, acting largely through the sympathetic nervous system, may develop any one of the above-named syndromes by attacking different parts of this nervous system. The central nervous system of the same part is also more or less under the influence of these poisons. I do not mean to say that migraine, recurrent vomiting, recurrent coryza, and toxic epilepsy are always produced by the same auto-toxins. It is not probable that any one of these symptom groups has one essential etiological auto-toxic factor without which they cannot develop. It is much more probable that the essential etiologic toxic factor may vary in all of them. But I do

believe that the most potent etiologic factors of a large percentage of the cases of recurrent vomiting, recurrent coryza, and migrainous epilepsy are auto-toxins either identical with or closely related to the toxins which are responsible for most of the cases of migraine.

The following cases are selected from my notebooks for the purpose of illustrating the kinship of these diseases:

Case 1.—Male, aged 8. A strong family history of gout on both sides for several generations. Mother and maternal grandmother suffer frequently and severely from migraine. Father also has migraine, and occasional bilious attacks characterized by pain in the stomach, with nausea and vomiting.

In December, 1896, I saw this patient, then five months old, in a typical attack of recurrent vomiting. At that time I learned from his mother that he had suffered from similar attacks at intervals of four to six weeks since he was two months old. The earlier attacks were thought to be due to bad milk, but as they had recurred under the most careful feeding, and as the same symptom group was repeated each time, I was convinced that the attacks were constitutional in origin, and referred to them as attacks of "Lithæmic Vomiting."

Following the December attack the child took and digested his food perfectly; his stools were normal in color and consistency, and he gained steadily in weight until his next attack, on February 8. This attack having been predicted, its symptoms were carefully noted, and as it is the youngest case of recurrent vomiting I have ever seen, they are here given in more or less detail. (This case

was published in 1897 in the *Archives of Pediatrics,* under the title "Lithæmic Vomiting.")

Feb. 8. Infant refused food, vomited at 1 P. M. Feb. 9, continued to take only small quantities of food, again vomited at 1 P. M. Feb. 10, restless and fretful all night, vomited at 4 A. M. During the day he was nauseated and refused food—evening temperature 102. Feb. 11, nausea continued. He vomited at intervals all night, has retained nothing on his stomach; appears very ill; temperature 102; nausea and vomiting continued during the day; evening temperature 103.5; has had 1-20 of a grain of calomel every hour since morning. Feb. 12, cried all night; took no food; nausea and vomiting continued; breath had acetone odor; temperature 102. He has wasted to a skeleton, and appears critically ill. During the afternoon and evening calomel and water were retained.

Feb. 13. He retained a little dilute cream last night, the first in sixty hours; bowels moved at 4 and 6 A. M.; putrid movements; temperature 101. During the day he retained small quantities of cream mixture. Feb. 14, very much better; temperature normal; took and retained his milk. From this time on convalescence was uninterrupted.

These attacks recurred at intervals of one to six months during the next five years, varying little in character during this time; but when he was about six years of age he commenced to have headache with these attacks of vomiting, and for the last two years he has suffered at intervals of every two or three months with typical attacks of migraine. The headache in these attacks is very

severe, is unilateral, lasts from twelve to twenty-four hours, and is associated with nausea and vomiting.

I have followed this patient's clinical history and observed him frequently during these years, and there can be no question that in this instance *attacks of typical recurrent vomiting have been transformed into attacks of typical migraine.*

Case II. Male, aged 10; a brother of Case I. When about two years of age he commenced to have attacks of recurrent vomiting, characterized by obstinate constipation, fever, nausea, and persistent vomiting. The nausea and vomiting would continue for three or four days, and would then disappear as suddenly as they came, and in a few days all stomach symptoms would disappear. These attacks came and went without apparent cause, and the mother soon learned they were self-limited, and that she might expect their recurrence every six or eight weeks.

When this boy was five years of age these attacks of recurrent vomiting commenced to change into attacks of migraine, and at the present time he still suffers from severe and typical attacks of true migraine; nausea and vomiting always accompany them. Within the last two years he has had two attacks of recurrent vomiting (without headache), lasting four or five days; so that in this boy the attacks of migraine are still occasionally supplanted by attacks of recurrent vomiting.

With this case, as with Case I, I have personally observed the change in the character of the attacks, and I am therefore quite sure that in both of these cases attacks of recurrent vomiting, later in the life of the child, became attacks of true migraine.

Case III. Female, aged 16, neurotic family history; patient herself extremely neurotic and malnourished. She had suffered from attacks of recurrent vomiting since she was a small child, and in the last few years these attacks had occasionally alternated with attacks of severe migraine, in which nausea and vomiting were marked features. I saw this patient in consultation on the sixth day of an attack of recurrent vomiting, in which the nausea was continuous and the vomiting so severe that morphine had been used hypodermically. Calomel and soda were given by the mouth, and high rectal injections of salt water every six hours. On the seventh day the patient commenced to convalesce, and recovered from this attack, as she had from all others, quite rapidly.

Following this attack, which occurred in the early spring of 1900, she went to the seacoast of Maine and there spent the summer. The following winter was spent in Southern California, and when I last heard from her, one year after her attack, she was well and had remained so throughout the year.

Case IV. Female, aged 24, a neurotic and alcoholic family history, suffers severely from attacks of true migraine, nausea and vomiting being prominent features. She gives a personal history of having suffered from attacks of nausea and vomiting during her childhood. These attacks were very severe; they occurred at irregular intervals, and lasted from three to six days. She was well in the interval between these attacks of "gastritis." For the past four years she has not had an attack of recurrent vomiting, but during this time has suffered at intervals from migraine, and she herself associates the

disappearance of the "vomiting attacks" with the appearance of the "sick headaches" from which she now suffers.

Case V. Reported by me in the *Medical Record,* June 22, 1895, under the title "Migrainous Gastric Neurosis." Mrs. P., age 43, mother of four children; her mother and a number of her family have suffered from sick headaches. She has had migraine ever since she was a child. In recent years these attacks have occurred every two or three weeks, and were marked by the characteristic unilateral headache, accompanied by nausea and vomiting. In the interval between the attacks she was well. In 1895, when she was 34 years of age, the attacks of migraine ceased, and were superseded by severe gastric attacks, which recurred every two or three weeks, as the migrainous attacks had previously done. These gastric attacks would come on with pain in the stomach, eructation of gas, and a red spot would appear on the left cheek, with a sensation of burning. These warning symptoms were very soon followed by increase in the gastric pain, constant nausea, and uncontrollable vomiting, but no pain in the head. These symptoms would continue for two or three days, or until they were relieved by hypodermic injections of morphine. After the acute symptoms had subsided, the convalescence was uninterrupted and, as a rule, rapid, so that in a few days she was as well as usual, having no symptoms on the part of the stomach until the next gastric attack, which occurred two or three weeks later.

Attacks of this character continued for about eight months, and during this time she had no migraine. When

suddenly, without apparent cause, the gastric attacks disappeared and attacks of true migraine began to recur every two or three weeks, and they have continued up to the present time.

In Cases III and IV attacks of recurrent vomiting were transformed into attacks of true migraine, and in Case V attacks of migraine were transformed into attacks of Leyden's "periodical vomiting," and the vomiting attacks were again transformed into attacks of migraine. It is not altogether clear to my mind that Leyden's "periodical vomiting" is not closely related to recurrent vomiting. I am rather inclined to believe that attacks of recurrent vomiting occurring in the adult may be associated with severe gastric pain, and thus become the periodical vomiting of Leyden. However this may be, the case above reported is one of many reported instances in which migrainous attacks have been transformed into attacks of "periodical vomiting," and vice versa. And the fact that periodical vomiting (Leyden) and recurrent vomiting may both be transformed into attacks of migraine indicates that these syndromes may be produced by the same etiological factors.

Case VI, which was referred to me by Dr. A. W. Johnstone, in 1899, was one of the most interesting and instructive it has been my good fortune to see. This case at different periods in her life suffered from recurrent vomiting, migraine, and epilepsy, and is here reported in detail:

E. X., female, aged 18. *Family history.*—Tuberculosis in one of the grandparents. Her grandmother on the mother's side suffered from recurrent gastric attacks,

which continued for many years and were called "bilious." They were characterized by nausea, uncontrollable vomiting, and severe pain in the stomach. They would come on suddenly, completely prostrating her, and for five or six days she would not be able to retain anything on her stomach. She would then gradually improve, but would not be entirely well for four or five weeks. Then would follow a period of perfect health, during which time she ate all kinds of food and had perfect digestion. She would continue well for four or five months, and then become prostrated with another gastric attack having the same symptoms as before. She continued to have two or three of these attacks a year for nine or ten years, and during this time her physicians predicted that she would be better after the menopause. This prediction proved true, and for nine years she did not have an attack. At the age of 58, however, she had a severe gastric attack similar to the one previously described. This was followed after two years by another one, which caused her death. This death occurred at the time the mother of our patient was pregnant with the child whose history I am now relating. This and other domestic troubles caused the mother to be very nervous during her pregnancy, and probably increased the attacks of recurrent vomiting from which she also suffered. The mother of our patient continued to have paroxysmal gastric attacks of nausea and vomiting at intervals of a month or six weeks during the whole time of her pregnancy. Our patient, therefore, has the remarkable family history of "recurrent vomiting" in both the grandmother and mother.

On the father's side there is a very strong rheumatic family history. Two of his brothers are now suffering from "chronic rheumatism," while his father and one of his brothers died from chronic Bright's Disease. The father himself has pronounced gout, having attacks which are quite typical in character. During these attacks he suffers intensely from pain in the toes of both feet, especially the big toes, and the joints are swollen and tender. He is confined to bed for two or three weeks at a time, and is then able to go about in perfect health until his next attack, some six or eight months later. His big toes are deformed with gouty deposits.

Previous History.—Patient commenced to suffer from attacks of recurrent vomiting when she was a child. These attacks would come on without apparent cause, and would last three or four days, and be followed by rapid convalescence. From the description, they coincided in every particular with the description which I have previously given of recurrent vomiting. When she was about eight years of age these attacks commenced to be associated with pain in the stomach, and later with headache, and gradually they became attacks of true migraine, the headache being severe and the narcotism pronounced, while the gastric symptoms were not very marked, and after a time disappeared altogether. The migraine continued until she was about thirteen years of age, when the menstrual function appeared, and about this time the epileptoid attacks commenced. A year later the epilepsy was fully established, and the migraine had almost entirely disappeared. These epileptoid attacks in the beginning were very mild. It was at first noticed

that she commenced to lose consciousness with her migrainous attacks, and gradually these attacks came to resemble true epilepsy. Epileptic attacks have continued up to the present time, and have no relation whatever to her menstrual periods. They occur every four or five days, and are characterized by a loss of consciousness, severe clonic spasm of the muscles, and frothing at the mouth. The patient sleeps for a few hours following the attack and then appears dazed for the remainder of the day. These attacks are now not associated with pain in the head, but they are at times accompanied by nausea or vomiting just before or after the attack. Within the past year these epileptic attacks have occasionally alternated with an attack of true migraine. She has been under constant medical treatment for five years, and during this time has taken a large amount of bromide of potash, and under this treatment has grown steadily worse, so that for the last two years she has been taught to believe that she is a confirmed invalid and has been treated as such. When she came under my care all medication was stopped and she was kept under close observation.

Patient's Present Condition.—October 14, 1899. She is poorly nourished, undersized, and undeveloped. A physical examination by Dr. Arthur W. Johnstone revealed a general lack of development of the pelvic organs, but no organic disease.

October 16. Her aunt, with whom she is now living, reported that she had an unusual appetite and took food in quantities more than sufficient to sustain a laboring man. I advised that her food be restricted in quantity, but otherwise nothing was done to ward off an attack.

October 24. At 2 A. M. she had a severe epileptic attack and was found lying on the bed partially dressed, with a large quantity of blood and mucus exuding from her mouth. When spoken to, however, she recovered consciousness and wished to get out of bed and have her breakfast. During the convulsion she lacerated her tongue quite badly and discharged a large quantity of urine involuntarily, completely emptying the bladder, and saturating her clothing and the bed. At 10 o'clock she drank a glass of milk, while still in bed. Half an hour later she complained of nausea, which was followed by a second epileptic convulsion. Following the convulsion she vomited a very sour, semi-solid mixture containing milk and other food. She remained in bed, but took no food. At 2 P. M. she had a third epileptic seizure. This was also followed by nausea and vomiting of half a pint of very acid, greenish fluid (bile). Following this third seizure there was considerable headache and a period of somnolence, such as followed the preceding convulsions. At 9 P. M. she had another convulsion, less severe than the others, but it was followed by a longer period of narcotism. She slept heavily for more than an hour, and awoke with nausea. Then followed a period of pronounced hysteria, which alarmed the aunt of the patient very much. She crawled about the bed, was very restless and nervous, and continued to be more or less excited until she fell asleep at midnight, and slept quietly until morning.

October 25. This morning I found her willing and anxious to get up. A saline cathartic, followed by an enema, had produced a free evacuation of the bowels;

very constipated. I ordered that she be kept in bed during the day, given milk to drink and a saline cathartic the next morning.

October 26. Five P. M. I was called to the house by the aunt of my patient, who was greatly alarmed because she thought the girl was "going crazy." I learned that all of yesterday afternoon and all of to-day she had been in an extremely hysterical condition, and had alarmed her aunt by refusing to talk or to understand anything that was said to her. She would remain in bed apparently in a semi-conscious condition so long as her aunt would remain in the room. If left alone, however, she would get out of bed and either talk incoherently or refuse to speak at all. She was caught, however, listening at the keyhole of her room to a conversation concerning her which was being carried on in the next room. When I saw her she was easily brought out of her hysterical condition, and was the next morning, October 27, sent to a hospital.

November 8. The patient has now been in the hospital eleven days, and during this time she has remained perfectly well, except for a slight attack on November 3. This attack lasted only a few minutes, during which time the patient says she was unconscious. She was not, however, convulsed. Immediately afterwards she got out of bed and seemed as well as usual. The nurse who witnessed the attack said that it did not last more than three minutes. Apart from this there have been no hysterical or other abnormal symptoms since she has been in the hospital, notwithstanding the fact that she has passed through a menstrual period while here. On going to the hospital she was given the following treatment: Milk

and bread diet at every meal, with the addition of an egg at breakfast, soup at dinner, and a baked apple at supper. The medical treatment has been a saline cathartic each morning, containing the sulphate, phosphate, and salicylate of sodium, and one-quarter of a grain of cannabis indica three times a day. During the eight months this patient was under treatment she improved very much, both mentally and physically, and the epileptic attacks were less frequent and less severe.

She returned to her home in a distant State, August, 1900, and since that time I know little of her history except that the epileptic attacks have continued.

This is the only case that I have ever seen presenting the three syndromes of recurrent vomiting, migraine, and epilepsy. The association, however, of epilepsy and migraine is so common, and so well recognized, that it would be a waste of time to narrate cases in which attacks of migraine have been transformed into attacks of epilepsy. In the *Medical Record* of June 22, 1895, I reported a case of this kind under the title "Migrainous Epilepsy." This case suffered from typical attacks of migraine for thirty or forty years, when the migrainous attacks ceased and epileptic attacks took their place, and they were continued for about ten years up to the time of her death, and during the period in which she suffered from epilepsy she had no attacks of migraine.

Case VII. Male, aged 8, family history on father's side gouty, and on mother's side alcoholic and neurotic; one other child, a sturdy phlegmatic boy of five.

Personal History.—Has had several severe attacks of gastro-intestinal trouble, and has always been nervous,

malnourished child. At five years of age had his first attack of recurrent vomiting, which was ushered in by a convulsion, associated with high fever; temperature during the first day of the attack ranged from 103 to 105. This attack lasted four days and was followed by a slow convalescence. These attacks have recurred at intervals of from two to six months up to the present time, and are, as a rule, marked by a single convulsion, which occurs during the first twenty-four hours of the attack. In the intervals between the attacks the patient is nervous, anæmic, and has feeble digestion. The child is mentally precocious.

Case VIII. Male, aged 6. Neurotic family history on maternal side. The mother herself has suffered from migraine for years, and is markedly neurasthenic.

Personal History.—The child has a poor physique, is intensely neurotic, and is below the average in mental development. Has been ill a great portion of his life. All of the many acute illnesses from which he has suffered since infancy have been marked by high temperatures, and, as a rule, by convulsions. He had many attacks of eclampsia during the first three years of his life. When about three years of age he had his first attack of recurrent vomiting, during which he had three convulsive seizures. Since then has had two or three attacks of recurrent vomiting each year, and they have always been associated with one or more convulsions and high fever, occurring during the first twenty-four hours of the attack; after this the temperature subsided and the convulsions ceased, but persistent nausea and vomiting continued for from three to five days. With the disappearance of these

symptoms the child convalesced rapidly, and was as well as usual in two or three days.

In Cases VII and VIII we have eclampsia associated with attacks of recurrent vomiting. A case of this kind was reported by Snow in 1893. *It is well, therefore, in the treatment of such cases, to keep in mind the kinship above noted of migraine, recurrent vomiting, and toxic epilepsy. It is possible that the recurring auto-intoxications in these cases, producing repeated attacks of eclampisa, may finally establish the epileptic habit, and in this way transform the attacks of recurrent vomiting into epilepsy.*

Case IX. (The corrected and completed history of a case reported by me in *American Medicine,* July 27, 1901.) Female, aged 12. Her father suffers from migraine; her mother died of diphtheria when patient was but a few weeks old.

Personal History.—There is no previous history of any serious illness, but she has always been nervous, and since she was five years old she has been subject to attacks of nausea and vomiting, coming on at intervals of two or three months.

The nausea was continuous, the vomiting uncontrollable, and the convalescence from these attacks of recurrent vomiting was rapid and complete. Besides these attacks of recurrent vomiting the child, since she was six years of age, has had attacks of intense coryza. It was in one of these attacks that I first saw her in January, 1899. I found her in a darkened room suffering so intensely from photophobia that I could not admit sufficient light to make a satisfactory examination. She was in a

state of extreme nervous irritability, which added to the difficulty of inspecting the case. I managed to see, however, that the eyes were swollen, the nostrils intensely irritated, and that an abundant watery secretion was running from both eyes and nose, producing considerable irritation of the lip and other parts over which it ran. I learned that the child had been taken suddenly ill with this attack about noon of the previous day, when she commenced to complain of photophobia and nasal irritation, went to bed at once and remained in a darkened room. At the time of my visit, thirty hours after their onset, the symptoms had not abated in the least. I learned also that the many similar attacks from which the patient had suffered had lasted two or three days, at the expiration of which time she would get well as quickly as she got ill. All pain, irritation, and hypersecretion from the eyes and nose would rapidly subside, and within a few days she would be at school again quite as well as before the attack.

In the intervals between these attacks there was no trouble with the eyes and nose, and apart from being a nervous child, suffering somewhat from constipation, she was not considered unhealthy. She was quite equal to all the outdoor exercise incident to childish play, and went through her school work as easily as the average child. Of late these attacks have been more frequent and more severe, occurring at intervals of two or three weeks, while formerly, especially in summer, several months had elapsed between seizures.

This very clear history of self-limited paroxysms of coryza, occurring in a young patient who had suffered

from frequent attacks of recurrent vomiting, and who had a family history of migraine, led me to the belief that the paroxysms of coryza were but another manifestation of the auto-intoxication which at times found expression in recurrent vomiting, the difference in the symptom groups produced being dependent upon the portion of the vasomotor nervous system attacked.

Following this first attack I advised that she should have as much exercise in the open air as possible, and should avoid tea, coffee, sweets, and an excess of meats. That she should drink milk and eat cereals, vegetables, fruits, chicken, fish, eggs, and a moderate quantity of fresh meat. Her bowels were to be kept open with a mixture containing sodium sulphate, sodium phosphate, and lithium benzoate. Three weeks later the patient had a slight attack of coryza which lasted less than twenty-four hours. From that time to the present, more than five years, she has been kept under observation, and during this time has not had a severe attack of coryza. She has, however, suffered from a number of slight attacks, some of which were associated with mild attacks of recurrent vomiting, and recently she had one quite severe attack of recurrent vomiting, which was ushered in by an attack of coryza.

Case X. Female, aged 7. *Family History.*—Mother has migraine, and one aunt on father's side had epilepsy.

Personal History.—She was very well up to one year of age, when she weighed twenty-one pounds and ten ounces. Her first severe gastric attack occurred at this time, and lasted nine days. It was characterized by intense irrita-

bility of the stomach, no food, medicine, or water being retained. From that time to the present she has had similar attacks at intervals of from three to four months.

At the present time her mother recognizes the approach of an attack by the child's general nervous irritability, obstinate constipation, and facial pallor, with dark rings under the eyes. These symptoms are commonly accompanied by a slight coryza and whistling bronchitis (asthma). The first symptoms, on the part of the stomach, to appear are eructations of gas, and very soon thereafter the nausea and vomiting begin, and everything that the stomach contains is discharged. Intense nausea, with periodical attacks of vomiting, continue from four to nine days, and during this time no food, water, or medicine is retained; everything is rejected by the stomach almost as soon as it is swallowed. Throughout the attack there is a tendency to somnolence, and during the last days she sleeps most of the time. Following the cessation of vomiting convalescence is rapid, and in twenty-four hours all stomach irritability has disappeared, and she is again taking malted milk and other light foods.

In one very severe attack last winter she vomited considerable blood, enough to color all the vomited matter, and the retching and vomiting were so severe that she was at times profoundly cyanosed; but just when she seemed utterly exhausted, and when her life was almost despaired of, the stomach irritability suddenly subsided and convalesence from the attack was soon established.

The constipation which preceded these attacks continues for a number of days, notwithstanding the calomel and enemata that are given, but towards their close the bowels

move, and for a few days there are two or three putrid discharges daily.

There are two symptoms belonging to these attacks to which I wish to call especial attention. One of these is the somnolence which lasts throughout the greater portion of the attack, being especially prominent after the second day. The child from this time on sleeps not only all night, but also nearly all day. The sleep is, as a rule, not a heavy one, but becomes deeper towards the close of the attack, and a prolonged heavy sleep usually precedes the beginning of convalescence.

The other symptom to which I wish to call attention is a slight coryza and whistling bronchitis which mark the beginning of nearly all of these attacks. These symptoms, as a rule, come on with the general nervous irritability, and precede the vomiting by one or two days. They, however, subside within two or three days after the vomiting begins. These symptoms are so pronounced that the physicians in attendance for a long time thought that the child had each time "taken cold," and that the medicines given for the coryza had produced the "gastritis"; but as time went on it was evident that "the cold" was a part of the attack, and the coryza and whistling bronchitis are now recognized as ominous prodromes presaging an attack of recurrent vomiting.

This patient is the daughter of a physician, and has been reared under good hygienic conditions. She has lived an outdoor life in country air, she has been put to bed at 7 P. M., and has slept all night; she has been protected from nervous strain and mental overwork, and notwithstanding these favorable conditions she has con-

tinued to suffer during her whole life from very severe attacks of recurrent vomiting. During this time, however, her physicians, not recognizing the true nature of her malady, have treated her for gastritis due to "errors in diet" or "cold," and in their efforts to protect her stomach they have dieted and underfed her until they have added innutrition to the malnutrition from which she suffers. In the last few years her outdoor life has been greatly interfered with by the slow convalescence from the severe attacks and by the general feebleness of her constitution, which was thought to unfit her for exposure to any but the most clement weather.

October 10, 1904. I saw this patient for the first time to-day, and obtained the above history. Her last severe attack was two weeks ago, and since that time she has been living on soup, toast, and malted milk.

Present Condition.—Age 7, weight 52 pounds. She is thin, malnourished, precocious, and intensely nervous. Her thin, pale face, large bright eyes, sprightly temperament, quick, nervous, restless movements, emaciated body, rapid heart action, and general feebleness of constitution mark her as a very ill child.

The parents had come to look upon the child's condition as hopeless, and were therefore much surprised when I made an uncompromisingly favorable prognosis. The following treatment was ordered:

An outdoor life with a moderate amount of exercise, a minimum amount of mental work and all possible protection from nervous excitement; light general massage, using plenty of lanoline, followed by one hour's rest in bed, every second afternoon. *Diet:* malted milk, cere-

als, eggs, stewed fruit, well-cooked vegetables and meat, either chicken or beef, at least once a day.

Medical Treatment.—Phosphate of soda or Kutnow's Carlsbad Powder before breakfast each morning in a dose sufficient to move the bowels, and the following prescription to be taken three times a day:

℞

Sodii benzoatis	ʒ iii
Sodii salicylatis (wintergreen)	ʒ iss
Essence of pepsin	ʒ vi

M. S.—Teaspoonful in water after meals.

A glass or two of water was to be taken between meals, and the child was to go to bed at 7 P. M., after a light supper. If prodromal symptoms appeared, indicating an approaching attack, one-quarter of a grain of calomel and five grains of bicarbonate of soda were to be given every half-hour for six or eight doses, and followed two hours later by a dose of calcined magnesia sufficient to move the bowels.

October 25. Is much improved in every way. She has gained three pounds in weight, and is much less nervous, has a good appetite, and is taking a sufficient quantity of the prescribed foods. The first massage treatments were followed by a sensation of fatigue and general nervousness, which lasted a greater part of the next day, but the recent treatments have had a tonic effect.

November 8. Four days ago the mother thought she recognized signs of an approaching attack in the loss of appetite, constipation, odor of breath, and increased nervous irritability. She accordingly gave calomel, soda

and magnesia, as above directed, and these symptoms disappeared. Child continues to show improvement in her general condition, and has gained one pound. Treatment continued.

December 3. Patient for two days has suffered from an acute coryza, such as almost always precedes her attacks of recurrent vomiting. This has been the only warning symptom of the attack, which commenced this morning with nausea and vomiting. Calomel, one-quarter grain, and sodium bicarbonate, six grains, were given every half-hour for eight doses; during this time, however, the vomiting occurred at intervals, so that perhaps little of the medicine was retained.

December 6. The nausea and vomiting have continued, no food or water has been retained by the stomach. Bicarbonate of soda has been given every day, and to-day the calomel was again tried, but it is a question whether the stomach has retained any of the soda or calomel. The bowels have not been moved since December 2, notwithstanding the numerous enemata that have been given. Every day two or three high rectal enemata of one pint or more of normal salt solution or bicarbonate of soda solution have been given. These solutions for the most part have been absorbed. The water and salts absorbed in this way have kept the kidneys more active, prevented great loss of weight, and otherwise favorably influenced the course of the attack. The urine examined on the 6th contained acetone, diacetic and oxybutyric acids, and the vomited matter contained free hydrochloric acid.

December 7. Vomiting ceased to-day and stomach retained some water and a little malted milk.

December 8. Bowels moved to-day following a dose of Epsom salts.

From this time on convalescence was rapid. Within one week the patient had gained the four pounds she had lost during the attack, and was in every way as well as before the attack. As soon as convalescence was established she resumed in every detail the interval treatment above described.

January 24. Is better than she has been for years, weight 60 pounds, and looks like a well child. Treatment continued. Massage, diet, outdoor life, and medication.

March 24. Has remained well and has continued to gain in weight; now appears to be a perfectly normal child.

May 1. Continues to gain in weight, health, and strength, and has, up to the present time, had no further attacks.

Case XI. Male, aged 7. *Family History.*—Father and one uncle had neuritis; father has "bilious headaches" characterized by severe hemicrania and accompanied by nausea and vomiting. Mother has a gouty and "rheumatic" family history.

Personal history obtained from Dr. Collins H. Johnston, Grand Rapids, Michigan. This boy has always had more or less trouble with his digestive organs, suffering at intervals with constipation, coated tongue, and lack of appetite. He has had at intervals nocturnal incontinence of urine, and has also suffered from habit-spasm. The attacks of habit-spasm consisted in twitching of the muscles of the face, blinking of the eyelids, and raising the

eyebrows, all of which were made worse when attention was called to them. Associated with these attacks there was considerable nervous irritability and disturbance of articulation. Neither the incontinence of urine nor the habit-spasm has been continuous; they would disappear when the boy's general health was improved, and would return when his nutrition was markedly impaired.

He has suffered from attacks of naso-pharyngeal catarrh throughout his life. When he was five years of age he was operated on for enlarged adenoids, and two months later he had a severe attack of influenza, marked by severe catarrhal inflammation of the throat and nose, and complicated by a suppurative otitis media. During this attack he had gastro-enteric symptoms and severe vomiting. Since infancy he has had occasional attacks of vomiting thought to be due to indigestion. During his fourth year he had three of these attacks, about three months apart, each lasting two or three days, and one year later he had another vomiting attack lasting four days; following this the next vomiting occurred in February, 1903, with the influenza attack above noted. In September, 1903, he had another attack, lasting four days, preceded as were nearly all of his attacks by *acute catarrh of the nose and throat* and considerable fever. This attack was followed by another, one month later (October, 1903), lasting four days. In December, 1903, occurred an attack of vomiting, which nearly terminated his life. This attack began with nasal congestion, listlessness, loss of appetite, and constipation, followed a few hours later by nausea, and twenty-four hours later by vomiting, and for ten days the nausea was continuous

and the stomach irritability was so great that not a particle of food or water was retained. During this time every effort was made to control the vomiting; food was given at intervals and again withheld; thirst was excessive. The highest temperature reached, 100.8, occurred in the beginning of the attack. After the sixth day the patient seemed so dangerously ill that nutrient enemata were given. They were followed, however, by an irritation of the large intestine, which prevented the giving of food and medicines in this way. The patient's condition was now, on the eighth day of the attack, very alarming, the nausea was continuous, and the vomiting occurred at intervals without apparent exciting cause; pulse 140, feeble and intermittent, respiration sighing, restlessness extreme, abdominal distress, extremities cold, finger nails blue, eyes sunken, skin cyanotic, and emaciation and prostration extreme. Dr. Johnston now gave hypodermically one-twelfth grain of morphine, combined with atropin and strychnine, and subcutaneously three ounces of salt solution. The boy improved at once under this treatment. The morphine had to be repeated a number of times, but from this time on the stomach became less irritable and he began to take and retain liquid foods. Convalescence was slow but uninterrupted. He was confined to his bed for one month, and was then taken to Florida in a private car. In Florida he slowly regained his usual health, and had another attack on April 11. This attack was very severe, lasted six days, and was finally controlled by morphine. His convalescence from this attack was slow. As soon as he was well enough he was taken to his home in Michigan, where

he had another severe attack about the first of August, 1904.

August 25 I saw this patient for the first time, and although it had been three weeks since his last attack he was very nervous, emotional, anæmic, emaciated, feeble, and confined to his bed most of the time. I advised the following treatment: Light general massage every day, out-of-door life with as much exercise as the boy's strength would permit, and a diet and medication exactly similar to that prescribed in Case X, above reported.

October 20, nearly two months later, I saw this boy for the second time. The treatment prescribed had been continued; the improvement was very remarkable. He was better than he had been for years. He had gained eight pounds in weight, and was able to indulge in all kinds of outdoor play with boys of his age. He had lost much of his nervous irritability, but was still quite emotional. He had a voracious appetite, which had to be somewhat restrained. His digestion was good; he was eating largely of the full diet prescribed two months before. Treatment continued and outdoor life insisted upon.

January 1, 1905. The father writes me that the boy is now strong and well.

June 6. More than nine months have passed since the beginning of the treatment, and during this time the patient has had no attack of vomiting. He has gained gradually in health, strength and weight, so that at the present time he weighs fourteen pounds more than he did nine months ago, and has apparently the strength and endurance of the average child, but he is perhaps abnormally nervous and emotional.

From January 15 to the present time the patient has taken, early each morning, a small portion of "Siphon C" (page 208), just sufficient to produce an evacuation of the bowels during the forenoon, and this has been accompanied at times by tonics containing arsenic or a diastase. The prescription containing benzoate and salicylate of soda (page 233) has, during this period, been used on two occasions for a week or ten days at a time, and these occasions were determined by the presence of certain symptoms which indicated that an attack of recurrent vomiting might be impending. At such times bicarbonate of soda, grains five, and calomel, grain one-quarter, was given every half-hour for eight doses, and followed for a week or ten days by the benzoate and salicylate of soda prescription above referred to. Under this treatment the prodromal symptoms quickly disappeared, and the patient continued his practically uninterrupted convalescence and return to health and strength.

Cases IX, X, and XI make a very instructive group, and their study clearly indicates the close relationship which exists between recurrent coryza and recurrent vomiting.

Case IX was one of recurrent coryza, these attacks at times being complicated by or alternated with recurrent vomiting. There can be little question that in this case these two syndromes were produced by the same auto-toxins acting upon different parts of the vasomotor nervous system, the primary point of attack determining whether the syndrome of recurrent vomiting or of recurrent coryza was to predominate in the attack.

Case X illustrates also the close connection between these syndromes. In this patient, attacks of recurrent vomiting were almost always preceded by coryza, and sometimes by whistling bronchitis. The association of these symptoms with those of recurrent vomiting was so close that in nearly all of her earlier attacks she was treated in the beginning for "cold in the head and bronchitis," and the medicines used in the treatment of these symptoms were thought to bring on the secondary "gastritis," which continued for days after the "cold" had disappeared. It later became evident to her parents that the coryza and whistling bronchitis were a part of the attack. This child has had eighteen to twenty attacks of recurrent vomiting, and in the great majority of them the syndromes of recurrent coryza, whistling bronchitis, and recurrent vomiting have been blended. This is not a coincidence, and can only be explained on the theory that the auto-toxins produce this combination of symptoms by their action on different parts of the vasomotor nervous system.

Case XI shows this same association of symptoms. In this boy, who has suffered from a large number of very severe attacks of recurrent vomiting, nearly all of these attacks have been preceded or accompanied by a more or less marked coryza, and at times by an irritation of the throat and bronchial mucous membranes.

These three cases, therefore, establish the fact that the syndromes of recurrent vomiting and of recurrent coryza are not uncommonly blended, and may be produced by the same auto-toxins.

A further interest attaches to Cases X and XI, in that

they were very severe cases of recurrent vomiting that, from the beginning, yielded promptly to treatment.

Case XII. Male, aged 6. Mother has migraine and grandmother on mother's side is gouty and intensely neurotic. Father is of a "bilious temperament" and has bilious headaches. Sister, three years of age, has had two attacks of recurrent vomiting.

Patient, when two years of age, had his first attack of recurrent vomiting, and since then has had two attacks each year up to one year ago, when he was put under treatment. The attacks from which this boy suffered were typical and moderately severe, lasting from four to six days. They, for a long time, were attributed to errors in diet, but their similarity and regular recurrence led the mother to the belief that they were constitutional and responsible for the marked nervous irritability from which the child constantly suffered. This case is reported for the purpose of calling attention to the interval condition of the child. Notwithstanding the fact that these attacks occurred but twice a year, he was in a deplorably nervous condition all the time. When awake he was never quiet. This nervous restlessness was very much exaggerated at times, and these exacerbations of restlessness were associated with a loss of appetite, coated tongue, canker sores in the mouth, and a sallowness of complexion, all of which symptoms his mother grouped under the term "biliousness." Under calomel these symptoms would disappear. These "bilious attacks" occurred every three or four weeks, and twice a year they were the prodromes of an attack of recurrent vomiting.

This child was never well, and his constant restlessness, which showed itself in his arms, legs, and head, gave the impression that he was below normal in mental development. I saw the patient for the first time in October, 1903, and put him under the treatment outlined in Case X, without the massage. More than sixteen months have now elapsed since he was put under treatment, and he has not had an attack in this time. His "bilious attacks," his general restlessness, and nervous irritability have almost disappeared, and he is now almost if not quite physically and mentally a normal child.

Case XIII. Female, aged 12. *Family History.*—An own cousin of her mother, Case VI, had recurrent vomiting, migraine, and epilepsy. Her father is gouty and suffers from gall stone attacks.

Personal History.—When six years of age she almost lost her life in a very severe attack of scarlet fever, which was followed by a middle ear infection, and when seven years of age she was operated for mastoid disease. These illnesses left her weak, anæmic and nervous. When six years of age she had her first attack of recurrent vomiting. These attacks recurred at intervals until she was ten years of age, at which time I saw her and put her under treatment. From this time up to six months ago she had no attack of recurrent vomiting and she had steadily improved until her general appearance indicated fairly good health, but she was still much more nervous and emotional than the average child.

About six months ago, April, 1904, she contracted measles, which was complicated by one of the most violent attacks of recurrent vomiting it has ever been

my misfortune to see. This attack in the beginning was thought to be an uncomplicated case of severe recurrent vomiting. I was led to this opinion because this attack began very like her other attacks. The nausea was constant, the vomiting very severe and exhausting, and no water or medicines were retained by either the stomach or rectum; the large intestine seemed almost as intolerant as the stomach. The symptoms increased in severity, and on the fifth day of the attack a well-defined measles rash appeared, and I then realized that the catarrhal irritation of the bronchial, nasal, and conjunctival mucous membranes, which had been present for two or three days, were symptoms of measles. The measles rash was typical and remained out for three days, disappearing on the eighth day of the attack, but during all this time the nausea and vomiting continued and no food or water was retained either by the stomach or large intestine. The prostration was now extreme, the pulse was feeble and rapid, the temperature 96 F., and the child was delirious. At this time I gave one-eighth grain of morphine hypodermically, and threw under the two breasts one pint of sterile physiological salt solution. The effect of this medication was magical; the nausea and vomiting stopped at once and the child slept for three hours. From this time on the stomach retained water and liquid foods in small quantities, but the morphine had to be given at intervals of six or eight hours for the next two days to prevent a return of the vomiting. After this her convalescence was rapid, and she is now, twelve months later, in better condition than she has been for years.

The above case is one of extreme interest, and is here reported because it is the only instance I have ever seen in which an attack of measles, or other acute infection, precipitated an attack of recurrent vomiting in a child which has been subject to these attacks. It is an interesting question in this case whether the measles poison, by its action on the nerve centers, was the cause of the vomiting, or whether this poison was assisted by a complicating auto-intoxication, viz., the same that had produced the previous attacks. I rather incline to the latter opinion.

Case XIV. Female, aged 5. Mother comes from a gouty family, and has for many years suffered from migraine, and for the last two years had nervous prostration. Patient is the youngest of four children; all the others are strong and well.

July, 1903. Saw this patient for the first time in a well-marked attack of recurrent vomiting, which lasted four days. On the third day of the vomiting the somnolence which had been present throughout the attack became more marked, and the mother, who had observed the child in many attacks, predicted that she would be better when she awoke from this deep sleep, as she knew by experience that a prolonged and profound sleep preceded the beginning of convalescence. The high enemata of bicarbonate of soda solution which were given in this case may have had some influence in shortening this attack.

One week later, when this patient had recovered from the attack, I found her to be very nervous, precocious, and attractive. I then prescribed the same treatment above

described in Case X, and since that time, now eighteen months, she has had no attacks of recurrent vomiting, and her general condition has greatly improved.

Case XV. Female, aged 3. A sister of Case XII. Had a severe attack of vomiting one year ago. In this attack the vomiting lasted three days, and during this time everything taken into the stomach was rejected; the stomach then suddenly became tolerant, and convalescence was rapid.

During the past year this child has, on the whole, been well. She has had, however, a number of "bilious spells," in which for a few days she would lose her appetite, become nervous and irritable, have a coated tongue and bad breath, and the constipation, which is habitual with her, would at these times become very obstinate. These attacks would yield to calomel, and the child would in a few days be as well as usual.

About four weeks ago the mother called my attention to the child because of certain nervous symptoms that had developed, which were associated with the most obstinate constipation; she was nervous and irritable during the day, and had had attacks of night-terrors for the last five nights. The constipation yielded only to strong doses of cathartic medication. A few days later a typical attack of recurrent vomiting began. The vomiting lasted four days, and was followed by a rapid convalescence. The urine contained acetone and diacetic acid. This patient slept almost continuously for the last forty-eight hours of the attack. During this time she could be readily aroused, but would quickly fall asleep again. When she was awakened from this profound sleep she was nauseated,

and would vomit when anything was taken on the stomach. On the morning of the fifth day the drowsiness passed away, the stomach lost its irritability, and convalescence began.

Cases XIV and XV are reported for the purpose of again calling attention to the narcotism (noted in Case X) which sometimes marks the close of attacks of recurrent vomiting, just as it does of attacks of migraine.

CHAPTER XX

CHOREA

Synonyms.—St. Vitus' dance, St. Anthony's dance, chorea minor, Sydenham's chorea.

Definition.—Chorea is a syndrome characterized by involuntary, inconstant, incoördinate, and jerky muscular contractions involving a part or all of the voluntary muscles, and occurring only when the patient is awake.

MORBID ANATOMY AND PATHOLOGY

In the present state of our knowledge it seems probable that chorea may be produced by a large number of organic lesions of the nervous system, and by the bacteria or toxins of certain acute infections, as well as by nutritional changes and functional derangements of the cerebral cortex. The widely varying pathological conditions which may be responsible for chorea make it expedient that this condition should be described as a syndrome rather than as a definite disease.

Organic Chorea.—If the large number of organic lesions of the nervous system which have been found to be associated with this disease are accepted as pathological factors of this syndrome, then it may be caused by inflammatory and degenerative lesions of the optic thalmus, corpus striatum, lenticular nucleus, and cerebral cortex, as well as by other diseases of the central nervous system.

Meynert and Elischer found hyaline degeneration in the nerve cells of the basal ganglia and cerebral hemorrhage and capillary emboli in the brain cortex. Dana, in a fatal case of chorea that had lasted for more than twelve years, and that apparently conformed to the Sydenham type, found a chronic lepto-meningitis of the cerebrum, meningitis of the upper part of the spinal cord, hyaline bodies in the brain cortex, and degenerative changes in the arterial walls, with dilated lymph spaces in the internal capsule, corpus striatum, and optic thalmus. Accompanying these changes were noted degeneration of the nuclei of nerve cells. A number of authors have reported congestions, hemorrhages, embolism, and softening of the brain tissue. These lesions are more commonly found in the lenticular nucleus, optic thalmus, and motor portions of the cerebral cortex. In a number of cases minute hyaline bodies have been found in the lenticular nucleus.

Among other lesions of the cerebrum that have been noted, on autopsy, as being associated with chorea may be mentioned cysts, tubercules, trauma from depressed bone, cicatrices from all hemorrhages, and all brain injuries producing hemiplegia.

If in the cases reported the above injuries have been responsible for the syndrome of chorea, it is very evident that it may be produced by a large number of widely varying pathological conditions. Organic chorea therefore has no definite pathology, and it is questionable whether these cases, which are for the most part chronic and incurable, should be included in a description of Syndenham's chorea. These cases, however, except for their chronicity and incurability, present the same clinical

picture, and have therefore by all writers been described with the toxic and idiopathic cases.

The embolic theory, which holds that chorea is commonly produced by capillary emboli washed from the vegetations which occur in endocarditis into the capillaries of the brain, is a theory not in keeping with pathological findings. It therefore deserves no further consideration.

Toxic Chorea, or chorea due to acute bacterial toxæmia, has within recent years attracted a great deal of attention, and there can be little question that at least a considerable proportion of the cases of chorea belong to this type. This type of chorea, however, does not include those cases which are produced by inflammations of the brain or its membranes, even though bacterial findings may be present. They have been classified above among organic choreas.

The toxic choreas include only those cases produced by the direct action of bacterial or other toxins on the nervous system, which slowly disappear when these toxins have been eliminated. The cases of chorea which occur during or immediately after the acute infections, including rheumatism, probably belong to this class.

Cesares-Demel, by injection of pathogenic microorganisms and their toxins under the dura mater, has succeeded in producing a symptom group similar to chorea.

Idiopathic Chorea.—Organic lesions of the brain and acute and chronic bacterial toxæmias are responsible, perhaps, for more than half the cases of chorea, but a large minority of the cases of ordinary chorea are, from

a pathological standpoint, yet to be accounted for. These are the great group of so-called idiopathic choreas that are believed to be due to nutritional and functional disturbances of the brain.

GENERAL ETIOLOGY

Predisposing Causes.—Age.—Chorea begins as a rule between the ages of six and fifteen, but the largest number of cases occur between nine and thirteen. It is rare to see the disease in children under three and a half years of age. Cases, however, have been reported as occurring in infancy, and old age is not exempt from this disease.

Heredity.—A neurotic family history is very common, and in not a few cases there is a direct family history of chorea. It is not uncommon to find two or more cases of chorea in the same family at different times, so that imitation could play no rôle in precipitating the attacks. Chorea, epilepsy, and migraine not uncommonly occur in the same families, and all of these neuroses may occur in the same patient at different periods of life. A family history of gout arthritism and migraine is fairly common in these cases.

Sex.—Chorea occurs in females three times as commonly as it does in males. This proportion is given by Gowers, Sinkler, and other writers.

Season.—All writers agree that chorea occurs most frequently in the spring; it, however, may occur at any season of the year. Morris J. Lewis examined 1383 cases of chorea with reference to the beginnings of the attacks, and found that of these 106 occurred in January,

101 in February, 172 in March, 159 in April, 160 in May, 150 in June, 126 in July, 106 in August, 76 in September, 74 in October, 54 in November, 99 in December.

The frequency with which chorea occurs during the months of March, April, May, and June has been variously explained by different writers. Some believe that the prevalence of rheumatism and other acute infections during these months is the explanation; others that the strain of school life and school examinations is the cause. In a former publication[1] I expressed the belief that the prevalence of chronic anæmia, from a multitude of causes, is in part responsible for the frequency of chorea at this season.

Race.—It is very uncommon in the negro race, but is very prevalent, according to Dana, among the Germans, Hebrews, and Portuguese of New York. Other writers have noted its prevalence among the Jews. Sinkler says that in Philadelphia it is "more common in children of American parentage than in foreigners." On the whole, however, it is probable that race in and of itself has little influence.

Climate has little influence so far as heat and cold are concerned. It may, however, be stated that a climate which is unfavorable for outdoor life will predispose to chorea and other neuroses.

Social Station.—Chorea is alike prevalent in all stations of life. Chronic anæmia and malnutrition, as etiological factors of chorea among the poor, are offset by the arthritic diathesis and nervous strain so common among the children of the rich.

[1] *Medical News.*

DIRECT CAUSES.—*Rheumatism.*—There is perhaps no fact better established in medicine than that the poison of rheumatism is responsible for about 25 per cent. of all cases of chorea. The relationship between chorea and rheumatism has been recognized for many years, but even at the present time there are great differences of opinion as to the importance of the rheumatism poison as a factor in producing chorea. Many writers assert that from 50 to 60 per cent. of all cases are due to this cause, and others hold that as few as 15 or 20 per cent. of these cases are rheumatic in origin. Osler found in 554 cases, 88, or 13.8 per cent., were due to rheumatism; he also found that 15.5 per cent. of these cases had a rheumatic family history. F. M. Crandall analyzed 111, and found a definite history of rheumatism in 63, or nearly 60 per cent. Holt believes that evidences of rheumatism may be found in 50 or 60 per cent. of all cases. Sinkler examined 927 cases and found that "there was a history of rheumatism, acute or chronic, in 187 cases, or 20.1 per cent.; and in addition to this there were 38 cases in which the children were said to have "growing pains"; in 79 cases, or 8.5 per cent., there was a distinct history of acute articular rheumatism; the intervals between the rheumatism and the attack of chorea varied from six years to immediately preceding; in 38 cases the attack of rheumatism had occurred within one year of the chorea; in 8 cases acute rheumatism immediately preceded the chorea, and in 7 the two affections were coincident."

Starr analyzed 2476 cases and found evidences of rheumatism in 26 per cent. The following table is taken

from "The American Text-Book of Diseases of Children":

TABLE I.—Showing the Relationship of Chorea, Rheumatism, and Endocarditis (Starr).

Author.	No. of Cases Chorea.	Rheumatism.	Cardiac	Reference.
Groendal	52	37	Majority	Wien. Med. Woch., Mar. 26, 1891
Meyer	121	11	15	Berl. klin. Woch., July 14, 1890
Koch	267	48	37	Arch. klin. Med., 1886
Peiper	30	14	6	Deut. Med. Woch., July, 1888
Sée	196	134	....	La Med. Moderne, October, 1891
Leroux	80	5	5	Rev. Mens. des Mal. de l'Enf., June, 1890
Dale	20	3	8	Lancet, October 31, 1891
Herringham	80	37	20	Lancet, January 12, 1889
Garrod	80	36	45	Lancet, January 12, 1889
Cheadle	84	62	....	Lancet, May 4, 1889
Brit. Col.Invt.Com	439	116	141	British Med. Journ., Feb. 28, 1857
Gowers	100	24	40	Dis. Nerv. System, Vol. II., p. 550
Sachs	70	8	12	Keating's Cyclo. Child. Dis. Vol. IV., p. 843
Dana	130	7	8	Arch. of Pediatrics, April, 1888
Sinkler	279	37	82	Pepper's System of Med., Vol. IV., p. 44
Starr	448	83	83	
	2476	662	502+	
		26 per cent.		

The variation in percentages presented by different writers depends upon the individual writer's idea of what constitutes rheumatism. Almost all writers at the present time agree that rheumatism is an acute affection. The term, however, is used very loosely. By some writers it is spoken of as a definite disease, by others as a syndrome. If by rheumatism we mean broadly a symptom group characterized by fever, arthritis, and, commonly, endocarditis, then the term is a very broad one, and covers the rheumatism syndrome, not only of true rheumatism (acute inflammatory rheumatism), whose specific cause is unknown, but also the same syndrome when it accompanies or follows such acute or chronic infections

as tonsilitis, septicæmia, tuberculosis, scarlatina, influenza, diphtheria, typhoid fever, measles, gonorrhœa, and syphilis. The syndrome of rheumatism is very commonly produced by an infection which enters the body through the tonsils, and is often preceded by a quinsy or an ulcerative tonsilitis. F. A. Packard has called special attention to the relationship which exists between tonsilitis, endocarditis, and rheumatism.

The syndrome of rheumatism being produced by such a large number of micro-organisms cannot in any direct sense be hereditary. Individuals, however, may inherit arthritism or a susceptibility to inflammations of serous membranes. This hereditary taint is in many instances related to the gouty diathesis. Such individuals by reason of this inheritance would be more susceptible to the syndrome of rheumatism. That is to say, they would be more likely to have arthritis, endocarditis, and their accompanying symptoms and sequelæ than those individuals who had not inherited this diathesis.

Patients having once suffered from the syndrome of rheumatism would be predisposed to second attacks by reason of the fact that the micro-organisms which produced the first attack may remain in a latent form in or near the joints, so that exposure to cold, dampness, and various other exciting causes might precipitate an attack. Chorea may be a part of the rheumatic syndrome, whatever its origin. This is especially true when this syndrome includes both arthritis and endocarditis. All of the acute infections mentioned above may also produce chorea without the appearance of the rheumatic syndrome.

If the term rheumatism is used, therefore, to include all cases that present the rheumatic syndrome, however ill defined it may be and whatever may be its origin, then it is more than probable that rheumatism is associated with 50 or 60 per cent. of all cases of chorea. If, however, we eliminate the vague cases of "growing pains" and all those produced by acute infections other than true rheumatism, then the percentage will fall to between 20 and 25 per cent.

True rheumatism is a definite and distinct disease characterized by fever, arthritis, acid perspirations, and, commonly, endocarditis, and yielding, in a degree, to the alkaline and salicylic acid treatment. The difficulty has been, and still exists, that this disease is probably produced by an unknown infectious agent, and therefore cannot be differentiated by bacteriological or pathological findings from the rheumatic syndrome which is produced by a large number of other infectious agents.

The clinical picture, however, of rheumatism is clearly enough defined in most cases to make the clinician fairly sure of his diagnosis. Confusion, however, will continue to exist so long as the exact pathology of rheumatism is unknown, and so long as the term is loosely used in describing a syndrome instead of a disease.

It is important, however, in studying the relationship which exists between rheumatism and chorea, that we should differentiate those cases that are produced by true rheumatism from those cases which are associated with the syndrome of rheumatism produced by other forms of infection. Rational therapeutics, in many of

these cases, will depend upon our ability to make this differentiation.

Heart Disease.—Chorea is very commonly associated with some disease of the heart; this may be an endocarditis, pericarditis, or merely a weakening and irritability of the cardiac muscles. Endocarditis may manifest itself in a systolic murmur, soft in character and commonly heard at the apex, and not infrequently heard at the base. Aortic murmurs are rare, but are occasionally observed. In some cases it is difficult to say whether the cardiac murmur is due to a mild endocarditis or to a malnourished and irritable heart muscle, with possibly a low grade of myocarditis. Cardiac sounds, however, associated with chorea, are to be classed as organic until it can be definitely proven that they are not so. Irregularity in the force and rhythm of the heart's action may be found without any evidence of endo- or pericarditis. These cases are, as a rule, very anæmic and malnourished. Pericarditis may produce well-marked friction sounds followed by an increased dullness in the cardiac area.

The association of heart disease and chorea is graphically shown in Starr's table, page 308. In 2476 cases, 25 per cent. had heart disease, and the collective investigation of the British Medical Association, which is included in this table, found heart disease in 32 per cent. of 439 cases of chorea.

All writers are agreed that there is a close relationship between arthritis, endocarditis, and chorea, about 25 per cent. of all choreic cases presenting at some time in their history well-marked symptoms of arthritis and about 25

per cent. showing previous disease of the heart. Writers, however, are not agreed as to the exact relationship which exists between these three conditions, some believing that the endocarditis, rather than the arthritis, is the important factor in producing the chorea, and others believing, with Cheadle, that the arthritis, endocarditis, and chorea are but different manifestations of the rheumatic poison, and that the syndromes of arthritis and endocarditis have nothing to do directly with the production of chorea. The symptom group which the rheumatic poison may produce depends altogether upon whether it attacks the joints, the heart, or the nervous system, and the order of appearance of these symptom groups when they occur in the same individual will depend upon whether the nervous system, the heart, or the joints is the point of first attack. In some instances the chorea precedes both the rheumatism and the endocarditis. In others it may follow either one or both of these symptom groups. My own opinion accords with these views, that rheumatism is a specific poison which may produce chorea by its action on the nervous system, and endo-, peri-, and myocarditis by its action on the heart, and arthritis by its action on the joints. One or more of these syndromes may occur in the same individual, and the order of their sequence is not important.

The association of endocarditis with chorea in other acute infections, such as streptococcic infections of the tonsils, scarlet fever, etc., does not prove that the endocarditis is etiologically related to the chorea. The chorea here may also be an expression of the action of the specific poison on the nervous centers. The common

association, however, of endocarditis and chorea produced by a number of the acute infections has suggested to medical writers the possibility of the endocarditis being a factor in the production of chorea. The theory of the embolic origin of chorea, which has now been discarded, had its origin in this association. At the present time, however, some medical writers believe that the feebleness and irregularity of the capillary circulation of the brain which may result from heart disease may be a factor in producing chorea.

Chronic Lymph Node Tuberculosis, which is one of the most potent factors in producing anæmia and malnutrition in childhood, is one of the most important etiological factors of chorea. I came to this conclusion from careful studies in the children's clinic of the Medical College of Ohio extending over a period of ten years.

The family histories in such records are necessarily incomplete, and "negative" or "good" is sometimes written when a family history of tuberculosis exists. The long-continued association, in the medical mind, of chorea, rheumatism, and endocarditis makes it altogether probable that the histories in public clinics record every semblance of these diseases, and the fact that tuberculous malnutrition and chorea have never been closely associated in the medical mind makes it also very probable that these histories do not record all the cases of tuberculosis. I have elsewhere called attention to the fact that the diagnosis of tubercular malnutrition is very commonly overlooked, and one is justified in making a provisional diagnosis of concealed tuberculosis in every well-marked chronic malnutrition occurring without apparent

cause in young children in whom there is a family history of tuberculosis, or exposure to the tuberculous contagion.

When we investigate our cases of chorea as carefully for evidences of concealed tuberculosis as we now do for vague signs of rheumatism and heart disease, we will find that at least 25 per cent. of the cases of chorea occurring in public clinics have tuberculosis in an active enough form to produce profound anæmia and malnutrition. An examination of 91 cases of chorea from my clinical records includes 28 tuberculous cases. In these cases cod-liver oil and iron, with good food and open-air treatment, gave the best results.

Chronic Malaria and other diseases which produce profound anæmia and chronic malnutrition may be etiologically related to chorea. In Chapters VI and VII I have called attention to the blood changes which are responsible for the profound anæmias and malnutritions which diseases of this character produce, and to the potency of these factors in producing an irritable and unstable condition of nerve centers, thus predisposing to functional diseases of the nervous system, such as chorea.

Exciting Causes.—*Fright,* which by nearly all writers is classed as one of the most important exciting causes of chorea, is responsible for the onset of the attack in about 20 per cent. of all cases. The fright, however, in these cases is made potent only by the presence of other very important etiological factors, such as profound malnutrition, heart diseases, or inherited neurotic tendencies. In studying the etiology of functional nervous diseases

such as chorea, it is important to keep in mind the tripod of etiological factors upon which these diseases rest. First, the predisposing factors, such as heredity, age, sex, and social condition. Second, the blood state, such as may be produced by acute and chronic infections and chronic malnutritions of all kinds; and, lastly, the exciting causes which, in individuals made susceptible by the two preceding factors, act in touching off the particular nervous syndrome. Fright acts in this manner in producing chorea in susceptible individuals.

Among other exciting causes which act in this way may be mentioned gastro-intestinal diseases, worms, delayed menstruation, eye-strain, diseases of the nose and pharynx, phimosis, masturbation, pregnancy, and imitation.

Duration.—The average duration is about ten weeks. Mild cases may get well in two or three weeks, and severe ones may continue for months. The attacks that continue longer than this are, as a rule, very severe throughout their course and are dependent upon grave etiological factors. Cases with severe cardiac lesions and grave nutritional disturbances may continue for six months or more, and cases in which the chorea continues for years are usually dependent upon organic disease of the nervous system. Such chronic cases, however, are rare even in organic chorea.

Recurrence.—Children who have had chorea are to be kept under observation for a number of years, in order to prevent a recurrence. Attacks may recur at the same time of the year, until the etiological factors which produced the first attack have been removed or until age

confers immunity. Recurrences are not common after fifteen. During the period of susceptibility to chorea, subsequent attacks may be produced by any of the above-named exciting causes.

Second and third attacks occur in about one-third of all cases, and girls by reason of their predisposition to neurotic diseases are more apt to have recurrences. Cases suffering from profound nutritional disturbances which do not yield readily to treatment, and children suffering from chronic diseases of the nose and throat predisposing them to acute infections, and those suffering from severe organic disease of the heart, are more likely to have subsequent attacks, and these attacks are, as a rule, similar in their course and severity to the first attack.

PROGNOSIS

The prognosis is good. When death occurs it is due to the organic disease of which the chorea is a symptom, and in the few cases that become chronic, while the organic disease is not severe enough to produce death, it is irremediable and severe enough to continue indefinitely the paroxysm of chorea.

Urine.—The urine in these cases does not assist in the diagnosis or prognosis. It very commonly is of high specific gravity, and the phosphates and urates are increased.

SYMPTOMS

Before the characteristic symptoms of chorea develop, the child, as a rule, is anæmic, nervous, and irritable. At school the teacher may observe his inability to sit still and a clumsiness in the handling of objects. The drop-

ping of pencils, books, and other objects brings reproof, under which the child's restlessness increases. Very soon twitchings of the muscles of the shoulder, face, or hand suggest the fact that the child is ill, and a physician is consulted.

In the early history of mild attacks the child may be able to partly control these irregular movements, but muscular spasm may be detected by directing the child to perform very slowly some rather delicate movement, such as threading a needle, or lifting a pin from a smooth surface, or by asking the child to remain in a standing position with both arms extended for a number of minutes. Under this strain the choreic movements are manifested, and an early diagnosis is made. The early awkwardness of choreic children may sometimes be noted by their tripping, stumbling gait or by peculiar muscular contractions which momentarily distort the face. Very soon, however, following these early symptoms, unmistakable and more or less general choreic movements develop, and then the diagnosis may be made at a glance.

There is probably no more clearly defined or more characteristic symptom group than that of well-marked chorea. The involuntary, inconstant, incoördinate, jerky muscular contractions involving a whole or part of the body, and aggravated by efforts to control them, present an unmistakable syndrome.

These irregular muscular movements vary greatly in severity. Mild as a rule in the beginning, and confined perhaps to one member of the body, in a short time they extend to the whole or half of the body and increase in severity, until at the end of the second week they have

reached their maximum severity. At this time in severe cases the muscular movements are almost constant, and the whole body may be undergoing bizarre movements which twist or distort the body to such an extent that the patient may be unable to maintain an upright position. The limbs are jerked and twisted in more or less constant movement, and every voluntary effort increases these incoördinate muscular contractions.

In these severe cases the patients are kept in bed with great difficulty owing to the twisting spasmodic movements, which toss the child in various directions. These movements may be so severe that unless the patient is carefully protected, by constant watching or padded sides to the bed, he is likely to receive painful bruises, or worse injuries from being thrown to the floor, or knocked against hard objects.

In the less severe cases the child may be able to go about as usual and have limited control of the spasmodic muscular movements, so that he is able to pick up a pin, button his clothes, or make letters with a pencil, but all voluntary movements of this kind are made after a few moments of deliberate preparation, and then the act is carried out with great rapidity. If, however, the movement requires any extended control of muscles, it usually fails, except, of course, in the mildest cases.

Speech is commonly involved, the choreic movements extending to the tongue and muscles of the jaw. In these cases the articulation is imperfect and jerky. The patient hesitates and then speaks rapidly. The control, however, of the muscles of articulation may be lost in the middle of a word or sentence. In severe cases articu-

lation may be impossible, and in mild ones there may be little or no trouble in this regard. The muscles of deglutition may also be affected, producing difficulty in swallowing. The muscles of the larynx may in rare cases be affected, producing great irregularity in the tone, pitch, and volume of the voice. An effort to speak in these cases may produce a whisper, a barking sound, and other unusual noises.

The diaphragm and other respiratory muscles may occasionally be affected, producing irregularity and loss of rhythm of respiratory movements.

In severe cases of chorea the muscles become so exhausted by constant movement that they appear to be paralyzed. This extreme weakness of muscles, however, is not common, and there is little or no change in their electrical reaction. An increased response to the faradic and galvanic currents has been claimed by some authors.

Sleep quiets the choreic movements and gives time for the tired muscles to recover.

Choreic movements become, as a rule, general. In about one-fourth of the cases, however, they are confined to one side of the body, but there is no apparent preference for the right or left side. These cases of hemichorea do not differ materially in any other particular from those cases which involve the whole body.

Choreic children are, as a rule, precocious, but the precocity is not infrequently associated with malnutrition, and is not therefore supported by a strength of body which will enable the child to undergo the mental and nervous strain into which its natural precocity leads it.

Among the children who hold the highest honors in school are to be found some of poor physique, who break down under the work and develop chorea and other neuroses in the spring of the year. Precocity therefore, coupled with physical inferiority, is commonly found in choreic children.

Children of this type, even before the chorea has developed, tire easily, are irritable, emotional, and suffer from headaches and general nervous irritability. After the chorea has developed all of these symptoms are exaggerated.

Mental symptoms leading up to acute mania and melancholia have been observed in a few cases. In not a small percentage of the cases the child's disposition is so changed, its mental irritability so greatly increased, and its moral sense so blunted, that it is not responsible for many acts of disobedience. These facts should be impressed upon the parents.

The tendon reflexes in chorea are, in the majority of cases, normal. According to Sinkler, "In quite a considerable number the knee jerk is either absent or may be described as capricious; that is, the knee jerks may be absent at one moment and at the next an involuntary movement of the child causes a reinforcement, and the response to a tap upon the patella tendon is prompt and energetic; there are some cases, however, in which the knee jerk cannot be developed under any circumstances."

Anæmia.—Well-marked anæmia is a very common symptom of chorea, and when present it is most important that it should be properly interpreted, since it

indicates, as a rule, that severe nutritional disturbances are present, and are probably potent factors in the production of the chorea. Pronounced anæmias, due to severe disturbances of general nutrition, are present in at least a third of all cases. These cases may suffer from purpura, large purple bruises occurring over the body on slight injury; or they may have sores, slight wounds of all kinds being quick to suppurate and slow to heal. The skin may be dry, harsh, and sallow, and the patient's condition cachectic. Sufficient stress has not been laid upon the importance of the profound anæmias and malnutritions which are associated with chorea. This cachectic condition is to be looked upon as a most potent etiological factor, rather than as a secondary symptom.

Heart Symptoms.—In every case of chorea the heart must be watched throughout for evidence of cardiac disease. A systolic or diastolic murmur may indicate endocarditis. At any rate, when murmurs are discovered the case is to be treated as one of endocarditis complicating chorea. In a few of these cases the murmur appears to be due to a weakness of the cardiac muscle, and in others it is probably purely hæmic. Osler, however, has called attention to the fact that many of the cases that are diagnosed as hæmic are later found to be organic. The symptoms of pericarditis may also appear during or following the attack.

TREATMENT

Treatment of the Attack.—It is to be remembered that an attack of chorea is, as a rule, self-limited, and that mild cases can for the most part be satisfactorily treated

with very little medication. Our efforts in the beginning should be directed towards the control of the spasmodic muscular contractions, and to shortening the duration of the paroxysm.

In the beginning it is of the utmost importance that a most careful search for reflex exciting causes should be made. Eye-strain, adherent prepuce, and diseases of the throat, nose, and genito-urinary organs very commonly act as exciting factors in developing an attack of chorea, and these organs should therefore be carefully examined and abnormal conditions be corrected. Above all, the intestinal canal should receive most careful consideration; a cathartic should be given, preferably calomel, followed by a dose of castor oil, to remove worms, foreign bodies, and undigested food. Intestinal fermentations should be corrected by proper food and medication.

The diet in all cases is important. The food should be carefully selected safely within the range of the child's digestive capacity. Milk is an ideal diet, unless intestinal disease or an idiosyncrasy forbids its use. Chicken and beef in small quantities may be allowed, and cooked fruits and easily digested vegetables may be given to the milder cases. Among the articles of diet to be avoided may be mentioned coffee, tea, strong beef soups, sweets, and all indigestible food.

Rest both of body and mind is necessary to the successful treatment of an attack of chorea. In the milder cases it is possible to get on fairly well without putting the child to bed, provided he is kept moderately quiet and not allowed to engage in any severe physical exercise,

Ordinary childish sports with other children are to be forbidden or carefully supervised and curtailed. In the severe cases the child should be put to bed and kept there until the paroxysm commences to subside, and thereafter, until convalescence is established, should spend the greater portion of the time in bed. In the most severe cases confinement to bed is absolutely necessary for a period of three or four weeks, and in these cases the railing about the bed should be high and well-padded to prevent the convulsive movements from throwing the child out of bed, or from otherwise injuring him by knocks against hard objects. The bodily rest which is so important in the treatment of uncomplicated chorea is even more important when there is a concurrent endocarditis; even the milder cases with this complication should be kept in bed.

Mental rest is quite as important as bodily rest. Nervous strain and mental work, which are ofttimes important factors in the production of chorea, should be reduced to a minimum in the treatment of these cases. The child should be taken out of school, and should in every way be protected against all forms of mental excitement. The tactful mother and nurse, when properly directed, will be able to interest the child without tiring or irritating him.

Medical Treatment.—Arsenic is the most valuable remedy we have in the treatment of the attack. It exercises, in many cases, considerable influence in shortening the attack. In giving arsenic one should commence with small doses, three minims, three times a day. After a few days of treatment, when it has been ascertained

that the arsenic will be tolerated, the dose is to be gradually increased, one or two drops a day, until at the end of the second week the patient is taking 15 to 18 minims three times a day, or until the characteristic signs of arsenic poisoning are produced. These symptoms are headache, an irritable stomach, diarrhœa, and puffiness of the face; and on the appearance of any one of these the arsenic is stopped for a few days, and then continued in smaller doses throughout the attack. There is no way of judging beforehand whether an individual attack of chorea will be benefited by the arsenic treatment. In only a small percentage of the cases does it act specifically in controlling the attack. In a larger percentage, however, while its action is not so pronounced, it apparently exercises a favorable influence on the duration of the attack. My own impression is that it acts better in the cases previously described as idiopathic chorea. It is not necessary, however, to continue these heroic doses of arsenic in a case where such treatment has made no impression upon the attack. If good is to be had from giving arsenic to the point of arsenical intoxication, the improvement will be made manifest by a single course of this kind of treatment. In those cases where this treatment fails to improve the patient, the arsenic should either be discontinued altogether or continued in small doses for the possible influence it may have upon the anæmia.

Strontium salicylate and sodium salicylate (wintergreen) and salol may be used in the treatment of an attack of chorea produced by rheumatism. These drugs are of undoubted value if symptoms of rheumatism

coexist with chorea, or if the attack of rheumatism has but shortly preceded the attack of chorea.

In the treatment of all forms of toxic chorea occurring during, or shortly following, rheumatism and other infections, it is important that the child be put upon a milk diet and confined to bed, however mild the attack may be. Warm baths are indicated, and mildly laxative medication, preferably sodium phosphate, should be used.

Idiopathic chorea associated with profound anæmia and malnutrition is to be treated with iron, cod-liver oil, and good food. Meat, eggs, and milk are especially indicated. As I have previously noted, many of these cases are due to concealed tuberculosis; but whether or not there be evidences of this disease, the iron is a very valuable remedy. It may be given in the form of saccharated carbonate of iron, or in any one of the palatable and efficacious modern preparations. Under good-sized doses of iron, which may be combined with small doses of arsenic, many cases rapidly improve upon which the heroic arsenic medication has made no impression.

Quinine is of value in those cases in which the chronic anæmia is due to malarial intoxication.

By the treatment above described, attacks of chorea can, as a rule, be brought to an early and successful termination. In the very severe cases, however, sedative medication, which we avoid when possible, is absolutely necessary for successful treatment. Chloral hydrate, trional, and potassium bromide may be used to produce sleep, and hydrobromate of hyacine hypodermically, and chloroform by inhalation, may be used to control

the severe muscular contractions. Morphine hypodermically is absolutely necessary in the treatment of a few of the most violent cases. Jacobi says: "Very bad cases must be kept sleeping eighteen out of twenty-four hours by means of mild opiates or chloral hydrate, with or without bromides. Sometimes large doses are necessary, but the effect *must* be obtained. I have met with cases in which an occasional inhalation of chloroform was also required. Meanwhile, the symptomatic measures adapted to the average case should also be attended to."

Treatment of the Interval.—Following an attack of toxic chorea of rheumatic or other origin, the patient should have his nose and throat carefully investigated and any disease of these organs removed by proper treatment. Tonsils and adenoids enlarged by disease are portals through which infections enter the body, and they should therefore be removed to prevent second attacks of rheumatism or other infections from producing a return of the toxic chorea. Rheumatic cases should also be carefully guarded by such hygienic measures as are used for the prevention of a return of this disease.

In the treatment of those cases in which there is an underlying profound anæmia and malnutrition, the syrup of iodide of iron, cod-liver oil, fresh air, good food, and appropriate hygienic measures should be resorted to, in order to restore the patient to perfect health.

In no instance should a case of chorea be dismissed as soon as the attack has disappeared. The attack should rather suggest to the physician's mind the importance

of searching for the disease of which the chorea is a symptom, and when the underlying disease has been determined, the physician should insist that appropriate treatment should be resorted to for a sufficient length of time to effect a cure, if this be possible. In this way second attacks of chorea may be prevented, and the general health of the patient ofttimes vastly improved over that which preceded the attack.

When cardiac disease is associated with the attack of chorea it is, of course, of the greatest importance that the heart trouble should receive most careful attention after the attack of chorea has disappeared.

Chorea, being a symptom group due to some grave constitutional disorder, may be considered a blessing in disguise, since it calls attention to and leads to the diagnosis and successful treatment of the underlying constitutional condition. If one look at chorea from this standpoint, one is sure to attach the greatest importance to the constitutional treatment of this disease.

CHAPTER XXI

HYSTERIA

Definition.—Hysteria is a psycho-neurosis due to functional disturbances of the cortical centers. It is characterized by defective will-power, emotional excitability, and the control of body and mind by perverted notions and fixed ideas, which are not uncommonly produced by suggestion.

PATHOLOGY

Hysteria has no morbid anatomy, and its pathology is not definitely known. It is a real, not a simulated, disease of the cortical centers of the brain, the functions of which are perverted or wholly or partially lost. The lack of inhibition which results from the impairment of cortical centers diminishes or destroys the restraint which these centers normally exercise over the lower motor centers of the brain and cord, thus permitting them to be thrown into a state of wild excitement from slight causes. There is in hysteria, also, a lack of mental inhibition, which leads to the most violent mental excitement and emotional explosions from apparently trivial causes.

The failure of voluntary (the will) and involuntary inhibitory centers to exercise normal restraint over both mental and motor acts is believed to be the important

underlying pathological condition in hysteria. This defective development of inhibitory centers is made more potent for evil by the fact that in hysteria there is a hypersensitiveness and increased excitability of sensory and motor centers throughout the nervous system, due to heredity and nutritional disturbances.

That the profound disturbances of the nervous system which occur in hysteria are functional, and not organic, is believed by all writers, and the transfer of motor and sensory disturbances from one part of the body to another lends strong support to this view. Apart, however, from the changes which nerve cells exhibit under fatigue (see chapter, "Reflex Irritation") and malnutrition there is no definite pathology for this disease other than that above outlined.

Hysterical symptoms, however, may be produced by organic diseases of the nervous system, and by all organic diseases which produce profound nutritional disturbances. The mechanism by which the hysteria is produced in these cases is probably the same as that above outlined, and is not due to any specific lesion. In organic diseases hysteria is a complication which may lead to great confusion in diagnosis.

ETIOLOGY

Age.—Hysteria is rarely met with before seven. From ten to fifteen it is common, but not so much so as in the adult. The milder forms of hysteria are seen more commonly than the severe in children, but if the diagnosis is made, as it should be, upon mild hysterical

manifestations, the disease is not so uncommon in childhood as one is led to believe by the literature. Hysteria, however, is not a disease of childhood. It is not only much more common, but much more severe, in the adult. In late childhood, however, from thirteen to seventeen, we may have the most pronounced and severe types of hysteria.

The feeble inhibition of mental and motor acts is much more pronounced in the child than in the adult, but this factor does not become so active in producing hysteria until the mental and emotional centers are sufficiently developed to require inhibitory control. The cells exhibiting mental energy are very slowly developed (see chapter "Physiological Peculiarities of the Nervous System in Childhood"), so that it requires the full period of twenty or twenty-five years to give them functional maturity. It is during the period from twelve to twenty-five, which sees the most rapid functional development of mental cells and of emotional faculties, that hysteria is most common and most severe. Hysteria occurs frequently in middle life, and may even continue into old age. These cases, as a rule, however, have begun in earlier life, and the manifestations in later life are either a continuance of or a relapse from these earlier attacks. Hysteria, however, may appear *de novo* in middle life. Clopatt gives the following table of the relative frequency of hysteria at different ages during childhood:

	Girls.	Boys.	Total.
In early childhood. . . .	19	1	20
3 years	—	1	1
4 "	1	1	2
5 "	4	2	6
6 "	3	2	5
7 "	15	4	19
8 "	16	6	22
9 "	15	7	22
10 "	18	15	33
11 "	24	17	41
12 "	22	13	35
13 "	27	16	43
14 "	12	8	20
15 "	—	3	3
	176	96	272

Briquet found, by the analysis of statistics, that in the female one-fifth of the cases occur before puberty, one-third between the ages of fifteen and twenty, and that after twenty the frequency of hysteria rapidly diminishes up to twenty-five years. From twenty-five to forty there is no diminution, but after forty the disease is infrequent.

Heredity.—Excessively irritable mental, motor, and sensory centers, under feeble inhibitory control, which are the all-important pathological factors of hysteria, are in most cases largely a matter of heredity. That is to say, heredity is the great predisposing cause of hysteria. A strong neurotic taint is present in the family history of

most of these cases. There may be a family history of hysteria, chronic alcoholism, epilepsy, insanity, chorea, or other neuroses. The worst cases occur in families that are mentally degenerate.

English, Germans, and Americans are believed to be less susceptible to this disease than the Latin races, and the Jews are especially inclined to hysteria. This is probably a matter of both heredity and environment.

Sex.—Hysteria occurs much more commonly in females, but the preponderance of females is not so great in children as in adults. According to hospital statistics of French writers, hysteria in the male is as common as it is in the female. This, of course, applies only to those of the lower classes, among whom the struggle for existence has proved a failure, as is evidenced by the fact that they are in charity hospitals. In America, hysteria, even among the poor, is much more common in women than in men.

Malnutrition of nerve centers is by far the most important direct cause of hysteria. The term malnutrition is here used very broadly, not only to include the innutrition of nerve cells which results from lack of sufficient food, but also the condition which results from a partial starvation of nerve cells, from their being supplied with blood deficient in some important ingredient, such as hemoglobin or oxygen. It also comprehends the condition of nerve cells which results when they are fed with blood containing auto or intestinal toxins. Malnutrition of nerve centers, therefore, comprehends not only the condition which results from poor blood, but also that

which results from bad or poisoned blood. The blood state of all hysterical individuals demands the closest investigation, since the most important etiological factors of this disease are to be found there. In the chapters on "Malnutrition," "Auto-intoxications," "Intestinal Toxæmias," and "Bacterial Intoxications" I have discussed the blood conditions which may, in susceptible individuals, be etiologically related to hysteria.

Chronic tuberculosis is, on the whole, more closely related to hysteria than any other chronic disease. In some sections of the country chronic malaria is an important factor. All acute and chronic diseases which produce profound nutritional disturbances of the nerve centers, or irritate and poison these centers, over a long period of time, with auto or bacterial toxins, may, especially in individuals who have inherited defective will-power and feeble control of the emotional centers, produce hysteria.

Chronic poisoning from alcohol, tobacco, lead, and mercury may be etiologically related to hysteria. In such cases it is possible that these poisons may act by producing general malnutrition, organic disease, or chronic irritation of nerve centers.

Environment is the most important exciting cause of hysteria. Hysteria is more common in the city than in the country, not only because of the impure air and bad hygiene, but also because of the noise, the rush, and the strain of life in a large city. In the country the child may have, for a portion of the day, solitude and mental rest, both of which are necessary for the normal development of the nervous system. In the city he is subjected

to the constant excitement and mental activity with which our social order has surrounded him.

The strain of school life and school examinations is a very important factor in developing hysteria. Children in our public schools must conform to a routine in confinement, school work, and school examinations which the average child is able to withstand without material damage to his nervous system. Those children, however, below the average either in physical or mental ability have more or less trouble in keeping up with their classes, and are subjected to very great nervous excitement and mental strain by the periodic examinations, which may force them to acknowledge to their little world and their home circle that they have been reduced to a lower grade, and that they are not the equals of their fellows of the same age. This strain of school life and school examinations falls with greatest force on those that are least able to stand it—on the neurotic, malnourished child of poor physique. The routine of school work cannot be changed to suit the weaklings, the system must go on like a great machine, and must be adapted to the mental and physical capacity of the average child. If the weaklings are not saved from this mental and physical grind, in which they may develop hysteria, chorea, or some other neurosis, it is not the fault of the machine, but rather of parents, guardians, and superintendents of schools, who should see to it that neurotic, malnourished children, if they go to public school at all, should be placed in a grade below that to which their age and mental capacity would admit them, thus putting their school work easily within both their physical and mental

capacity. In small private schools the routine is not so rigid, and there is a better opportunity on the part of teachers to give personal attention to the individual, protecting precocious, neurotic children from overwork, and stimulating dull, vigorous children to increased mental work. For these reasons, children who are below the normal average, either in mental or physical ability, do best under home instruction or in small private schools.

Lack of home discipline, which allows self-indulgence and free play to the emotions, may prepare the child for the development of hysteria. On the other hand, home training and school discipline, which teach the child to control his emotional nature, and which protect him from influences that excite the emotions and harass the mind, and which educate him not to act precipitately in the heat of passion or under emotional excitement, but to withhold his resentment until his passions and emotions are well under control, may prevent the development of hysteria even in children more or less predisposed to this disease by heredity and malnutrition. In the chapter on "Excessive Nerve Activity" I have more fully discussed the relationship of school life to hysteria and other neuroses.

Excessive nerve activity and mental strain are potent factors also in developing hysteria in adults. Business and household cares and worries, when long continued without periods of rest, may in susceptible individuals develop hysteria. The close association of members of a neurotic family creates a nervous atmosphere very conducive to the development of hysteria; under such conditions the principle of imitation may produce an

epidemic resulting in a number of cases in the same family. Business misfortunes, plunging families from positions of influence to dire poverty, necessitating an entire change of surroundings and the giving up of associations that seem necessary to happiness, and the facing of trials incident to a struggle for existence, may be exciting causes of hysteria. Great grief, such as follows the loss of one who has been the mainstay of a family, with the cares and responsibilities which follow, may, in the physically weak and irresolute, produce hysteria. Disappointment in love and religious excitement are not uncommon exciting causes. The excitement, gloom, privations, mental anxieties, and nervous strain incident to great wars may be a widespread cause of hysteria and other neurotic diseases.

Fright, such as may result from fires, cyclones, earthquakes, lightning, panics, or the witnessing of some awful catastrophe may develop hysteria in those predisposed to this disease by malnutrition or heredity.

Trauma.—Severe forms of hysteria may be produced, or perhaps it may be better to say developed, by injuries resulting from explosions, railroad accidents, and other causes likely to produce severe nervous shock. In such cases it is difficult to say whether fright or shock is the exciting cause of the hysteria.

SYMPTOMATOLOGY

Extreme selfishness and dependence masquerading under the cloak of self-sacrifice are common manifestations of hysteria.

The hysterical patient is very exacting of all around her, and in narrating her own sufferings she tells of the sacrifices which she makes for the comfort of others, when in truth she does not hesitate to call upon those around her to sacrifice themselves to administer to her apparently trivial ailments. The selfishness of hysteria, however, is a part of the disease, and not within the control of the patient. The selfishness, therefore, being more apparent than real, can hardly be spoken of as true selfishness. It is a defect in will-power which makes the hysterical patient so dependent upon those around her. She is often controlled by fixed ideas with reference to her inability to act and think for herself. She cannot do those things which others have been in the habit of doing for her because a perverted notion to that effect controls her.

One of the most peculiar and characteristic examples of the control which fixed ideas have over hysterical patients is shown in the symptom group known as astasia-abasia. This is one of the most common of hysterical manifestations, and is produced by the fixed idea in the patient's mind that she cannot either stand or walk. She may have perfect control of her legs when lying down, moving them at will in any direction, and not manifesting any muscular weakness, but the minute she is placed upon her feet her legs give way; or they may stiffen, the patient losing her equilibrium; or she may stand upon her feet and not be able to walk, making wild, incoördinate movements of the legs when she attempts to do so. In other instances, the fixed idea may confine the patient to bed for months or years, or it may

cause her to avoid light, remaining constantly in a darkened room. Some perverted notion or fixed idea is a large factor in producing symptoms in almost every case of hysteria.

Perhaps the next most characteristic symptom group of hysteria is that produced by emotional excitability. Hysterical patients are very emotional; fits of crying and laughing may follow each other without apparent cause. They are moody, irritable, and are easily thrown into states of great nervous excitability. In extreme cases the mental excitability and sleeplessness may pass into a state of acute mania, with absolute loss of reason. These severe mental symptoms not uncommonly follow attacks of hysterical convulsions.

The hysterical patient may lose her temper, may pass into a state of ecstasy, may pass into a state of gloom, may be wildly excited, or made pallid with fear from causes that would produce no such results in a well-balanced nervous system under proper inhibitory control.

Suggestion is one of the most potent factors in developing symptom groups in hysterical patients. The susceptibility to this influence marks one of the most important characteristics of the hysterical mind. Syndromes may be suggested to hysterical patients by the questions of the examining physician, and at the next visit symptoms may be present which the patient has learned might develop. The story of another's symptoms and sufferings may suggest the same symptoms to the hysterical mind, and they promptly appear. In a thousand ways these suggestions may come not only from without but also from within. The hysterical

patient may come out of a dream, a convulsion, or a trauma, with hallucinations which may be productive of paralysis, anæsthesia, loss of voice, or in fact almost any of the multitude of hysterical symptoms.

Paralysis is a common manifestation of hysteria in the adult, but it is comparatively rare in young children. In late childhood, however, it is not infrequently observed. The slighter forms are more common in the child than the severe types so frequently witnessed in the adult. The paralysis may be flaccid, with diminished reflexes and occasionally an absence of the knee jerk. The spastic form, however, associated with contractures and exaggeration of deep reflexes is much more common. The exaggeration of reflexes, however, is not so marked as in spastic paralysis of organic origin.

The paralysis may vary in form from a slight weakness of a few muscles to complete paralysis of almost all the voluntary muscles. It may be hemiplegic, paraplegic, monoplegic, or it may be irregularly distributed, involving only certain groups of muscles. Hysterical paralysis is rarely complete. As a rule, it is partial, and accompanied by muscular contractions, which give rise to a great variety of symptoms.

Paralysis of the muscles of the face may produce a lack of symmetry in the two sides; of the foot, club-foot; of the wrist and hand, wrist-drop, and various contractures; of the neck, torticollis; of the back, curvature of the spine; of the mouth, dropping of its angle and drooling; of the larynx, aphonia.

Hysterical aphonia, which is frequent in childhood, is one of the most common and easily recognized symptoms

of this disease. The voice may be lost very suddenly and may return as quickly. The aphonia may continue for days, months, or years. It may disappear under a strong faradic brush applied over the trachea, or it may resist all forms of treatment. Complete paralysis of the vocal cords and laryngeal muscles, resulting in absolute mutism, may occur in the child, but not so frequently as it does in the adult.

Paralysis of the tongue may produce disturbances of speech; of the eye, squint; of the diaphragm, singultus; of the respiratory muscles, cough, dyspnœa, and other disturbances of respiration; of the œsophagus, dysphagia, regurgitation of food, and globus hystericus. The globus hystericus is perhaps the most common of all hysterical manifestations, in children as well as in adults. The hysterical cough, persistent, dry, harsh, and easily excited by suggestion, is a very common and very troublesome symptom. To those constantly associated with hysterical patients the cough and the hiccough are perhaps the most trying and exasperating of symptoms.

Tremor is a peculiar motor symptom, which occurs not infrequently in traumatic hysteria, and may also occur in hysteria from other causes. Tremor may persist for years, and is a very distressing symptom. The tremor is manifested especially in the hands, but may be more or less general.

Incontinence of urine and fæces are very rare in hysteria in the adult. In the child they are not so uncommon. When they do occur, however, they are intermittent, and not constant, as in organic disease.

Hysterical paralyses in their duration, development,

and disappearance follow no rules. They may last for days or years; they may occur very suddenly or they may be very slowly developed. They may disappear almost instantly or there may be gradual recovery; they may come, go, and again return; they may shift from one part of the body to another, not following the rules of organic paralysis.

Diagnosis of hysterical from organic paralyses can, as a rule, be made very readily. Hysterical paralysis, as above noted, does not conform to anatomical laws of distribution. Hysterical hemiplegia, which so closely resembles in distribution the organic form, may not be associated with aphasia, paralysis of the tongue, an exaggeration of deep reflexes, and is accompanied by more marked sensory changes than those occurring in organic hemiplegia.

In the flaccid palsies of hysteria there is no change in electrical reactions. The reflexes, as a rule, are not lost; the sensory disturbances are very marked, and other hysterical symptoms are present.

Anæsthesia, which is one of the most common hysterical manifestation in the adult, is not so common in the child. It occurs, however, not infrequently in older children, and is, as a rule, associated with paralysis of the part affected.

The distribution of the anæsthesia is one of the strongest aids in the differential diagnosis of hysterical from other anæsthesias. It is commonly confined to one-half the body, preferably the left side. This hemianæsthesia is profound, confined strictly to one-half the body, and comprehends not only absolute anæsthesia to all forms

of ordinary sensation, but is also accompanied by loss of hearing, seeing, smelling, and tasting on the affected side. The line of demarcation between the anæsthetic and the normal sides is sharply defined, extending from the top of the head to the feet, involving general sensation and the special senses. The anæsthèsia is not confined to the skin, but extends to deeper tissues. The patient is not always conscious of the location, extent, and character of the anæsthesia, showing that conscious impressions are not necessary to its development. Hemianæsthesia, however, may be transferred from one side of the body to the other under influences of suggestion. The anæsthesia, as a rule, returns to the side first affected.

The anæsthesia and hysteria may also be regional, confined to a limb or to small portions of the body. Anæsthesia of a limb is, as a rule, associated with paralysis, and is sharply defined by a line running around the limb. In its disappearance this line may slowly pass down the limb, or the anæsthesia of the whole limb may suddenly disappear. Small spots of anæsthesia may occur; these islands are usually round or oblong, and may vary in size from a few inches to a foot in diameter. In rare instances hysterical anæsthesia may involve almost the entire body. An absence of tactile, thermic, or painful impressions, or a loss of the muscular sense—any or all of these may constitute hysterical anæsthesia.

Hyperæsthesia is one of the most common of the sensory disturbances in children. It is most commonly observed over the spine, ovaries, breasts, and abdomen. The slightest touch or injury to the skin over these areas

may produce pain, convulsive disorders, and other hysterical manifestations. In children, however, the milder hysterical phenomena follow pressure in these hysterogenic zones.

Painful joints, simulating inflammatory diseases, is one of the most common of the hyperæsthesias of childhood. Hysterical disease of the hip or knee joints is not uncommon between the ages of ten and fifteen, and occasionally occurs in very young children. The similarity between hysterical and organic diseases of joints is so great that mistakes in diagnosis are frequent. The pain on motion and the tenderness on pressure are greater in the hysterical joint, but the deformity, contracture, and apparent shortening disappear when the patient is anæsthetized, and these facts, together with the presence of paralysis of the part affected, or other hysterical manifestations, suffice to make a differential diagnosis.

Closely associated with hysterical joint disease are the hysterical contractures, which may be confined to one limb or may involve a number. A contracture may be so strong that no movement of the joint whatever can be produced. In other instances there is limited movement, not allowing complete flexion or extension. These contractures may involve the muscles of the face, tongue, and neck, as well as those of the body and extremities. The thighs may be flexed upon the abdomen, or the arm upon the forearm, and any attempt at overcoming these contractures may produce great pain. Some of these cases are very puzzling as to diagnosis, but as they are usually associated with other well-marked hysterical symptoms which lead us to suspect their nature, an

anæsthetic under which these contractures subside suffices to differentiate them from organic contractures.

Disturbances of special senses are not infrequent in hysteria. It was above noted, in speaking of hemiplegia, that the special senses on one side may be completely or partially lost, while on the other side they remain normal. In this condition the patient may be blind in one eye, deaf in one ear, and in one-half the tongue the sense of taste may be absent, and in one nostril the sense of smell may be gone, and over the skin of one-half the body the sense of touch may be lost. Besides these unilateral disturbances of the special senses, there are others affecting the special senses of both sides, and not necessarily associated with hemiplegia and hemianæsthesia. On the part of the eye there may be photophobia, color blindness, and absolute or partial loss of sight. Complete blindness is rare and transient, but partial blindness, produced by peculiar and irregular contractions of the visual field, is not infrequent. Hysterical disturbances of hearing, smelling, and tasting are much less commonly independent of a general hyperæsthesia and anæsthesia than are those of sight. An exaggerated acuteness, as well as diminution or obliteration of the special senses, may occur on one or both sides, entirely apart from other sensory disturbances.

Eclampsia is the most striking of the motor manifestations of hysteria. The hysterical fit or convulsion has been commonly spoken of and described under the term hystero-epilepsy, from the fact that the paroxysm may somewhat resemble that of true epilepsy.

Hysterical eclampsia is usually heralded for a number

of days by some of the psychic symptoms previously noted, and is commonly followed by sensory disturbances. Immediately preceding the paroxysm of eclampsia a group of symptoms peculiar to the individual announce the onset of the fit. Among such aura of hysterical convulsions may be mentioned a sensation of suffocation, severe headache, abdominal pain and vomiting, globus hystericus, ringing in the ears, or an increased sensitiveness over the ovaries, or over some of the other hysterogenic areas, pressure over which may start the chain of hysterical symptoms, culminating in eclampsia.

Hysterical eclampsia is very uncommon in the child. It does occur, however, in older children, and about puberty is not so rare. The seizure may be ushered in by a cry, and during the attack the patient may scream or make other noises. The convulsion is at first tonic, producing oposthotamus, the back and limbs stiffening and curving like a bow. This stiffening gives way to clonic convulsions, and the body is jerked and tossed about by violent muscular contractions. In a short time, from five to ten minutes, the convulsive movements cease and the patient is relaxed, and often falls into a light sleep, to awaken shortly in a state of emotional excitement. This stage gives the impression of conscious deception by its strange talk and bizarre movements. In the final stage there may be a period of semi-consciousness or delirium.

In other cases the convulsive movements are immediately followed by a profound sleep or trance lasting for hours, from which the patient may awaken with

paralysis, contractures, or anæsthesia of all or part of the body. The emotional element before, during, and after the attack is much more marked than in epilepsy, and the loss of consciousness is less profound. Hysterical convulsions may, in some cases, be modified or stopped by pressure or electricity applied to some of the hysterogenic areas; this is not true of epilepsy. Incontinence of urine and fæces does not occur, the tongue is not bitten, and the patient's subconsciousness seems sufficient to protect him from injury; he falls softly; he does not toss himself against hard objects, although he seems on the point of doing so. This strange subconsciousness that protects the patient in hysterical convulsions often leads to the unwarranted conclusion that there is an element of conscious deception in the attack.

While the severe convulsive seizures above described are comparatively rare in the child, mild convulsive attacks, with partial loss of consciousness and characterized by strange and apparently purposive movements, are not uncommon. During such attacks the patient may continue to perform some special movement, such as the flexing of an arm or leg, or retraction or rotation of the head; or he may jump about the bed in mimicry of some animal; he may bark, bite, and snarl like a dog as he tosses the bed clothing; but he does not injure himself or others. In other cases the patient may lie in one position, dazed or semi-conscious, with eyes open and fixed. There may be localized spasm of almost any muscle or group of muscles, producing "chorea major" or localized movements. Some of these movements of voluntary muscles seem to be purposive, but that they

are not so is indicated by the fact that localized convulsive movements occur in involuntary muscles. Spasm of the diaphragm may produce hiccough, which may be a very distressing and troublesome symptom; spasm of other respiratory muscles may produce very rapid breathing and dyspnœa; spasm of the esophagus may produce difficulty in swallowing and globus hystericus: spasm of the muscles of the intestines may produce diarrhœa.

The emotional element is great in all hysterical attacks, and they not unusually terminate in fits of laughing or crying. The more profound mental disturbances, such as catalepsy, lethargy, trance, and ecstasy, which may occur in the adult, either associated with or independent of the hysterical convulsion, are rare in the child.

Hyperpyrexia is sometimes observed, and fever is not unusual in juvenile hysteria. Some remarkable cases of hyperpyrexia have been reported. Jacobi reports one in which the temperature reached and continued above 110° F. for days.

Visceral Symptoms.—Anorexia nervosa is a classical symptom group produced by hysteria. In this condition the patient may go for weeks without being seen to retain any food; the sight of food may produce nausea, or all food taken may be vomited, and sometimes with a conscious effort. The severity of these symptoms may vary from slight nausea to a nausea so profound that all food is refused or rejected after being taken and the patient brought to the point of starvation.

Paralysis of the bowels may produce constipation. In-

creased peristalsus may cause diarrhœa. Enormous distension of the stomach and bowels may occur; phantom tumor of the abdomen, produced by gaseous distension, is not uncommon.

A very large quantity of light-colored urine of low specific gravity may be passed by hysterical patients. Anuria has also been noted.

TREATMENT

In beginning the treatment of a case of hysteria it is most important that all physical causes that may have contributed to the production or the continuance of the disease should, if possible, be removed. A careful search should be made for causes of reflex irritation to the nervous system. Eye-strain, diseases of the nose, throat, reproductive and genito-urinary organs should receive appropriate treatment. While these factors, if they exist, may not have been of prime importance in the development of the disease, there can be no doubt that they may exercise an influence in continuing the hysterical condition, and in precipitating hysterical paroxysms. In the child, eye-strain, and, in the adult, diseases of the reproductive organs are the most common sources of reflex irritation, associated with and etiologically related to hysteria.

The next step in the treatment comprehends a search for and the removal of the underlying causes of the chronic anæmias or malnutritions so commonly found in hysterical patients. If marked malnutrition exist, it is one of the causes of the extreme excitability of the nervous system which is an important factor in pro-

ducing the hysteria. The malnutrition factor is especially important in the hysteria of childhood, and may be produced by a concealed or lymph node tuberculosis, a chronic malaria, some form of chronic auto-intoxication, or chronic intestinal toxæmia; or it may be purely a question of improper food, impure air, and unhygienic surroundings. At any rate, it is most important in the treatment of hysterical patients presenting evidences of nutritional disturbances that every attention should be given to improving the physical condition of the patient. In order to do this it is not only necessary to prescribe medicines suitable to the individual case, such as iron, cod-liver oil, arsenic, quinine, or some tonic that will stimulate the appetite and improve digestion, but it is of even greater importance that diet and general hygiene should be as carefully prescribed.

There is, of course, no diet belonging to hysteria proper, but one can say, in a general way, that alcohol, tea, coffee, concentrated sweets, salads, pastries, rich and highly seasoned dishes, are to be avoided, and a diet simple, wholesome, and nutritious prescribed, suitable to the digestive capacity of the patient and the character of the malnutrition from which she suffers. In addition to this, the hysterical patient should live as much as possible out of doors, away from the whirl, noise, and excitement of a large city. Moderate exercise and congenial surroundings are also important. In brief, every attention should be directed towards improving the physical condition of hysterical patients, since the mental condition is largely a reflex of physical disabilities.

The mental condition, however, of hysterical patients must also be carefully and tactfully treated. Whatever may have been the surroundings under which the hysteria developed, a complete change is to be recommended, not only for the purpose of avoiding the etiological factor that produced the hysteria, but also to get the benefit of the marked and not altogether understood curative influence which a change of surroundings has on these cases. All mental stimulation must be stopped at once, school life, as well as home instruction, for mental development must be discontinued, and the patient (child or adult) should, if possible, be separated from her family. This is especially imperative if other members of the family are strongly neurotic, as in the majority of cases they are. The removal from the nervous atmosphere of a neurotic household, the stopping of all mental stimulation and avoiding nervous excitement, are important factors in the cure of hysterical patients, but the removal from home comprehends not only these curative influences, but also the powerful influence which is exerted by placing the patient under entirely new conditions. If, for example, the patient is sent to a hospital, the going to bed, the presence of trained nurses, the routine of treatment, which may include hydrotherapy and massage, the regular visits of the tactful physician, and all the machinery which moves as he directs, makes a powerful mental impression upon and inspires confidence in the patient, which is the first and all-important step in the cure.

The new surroundings which are thus produced by change, when tactfully used by the physician, constitute

a form of *suggestion,* and this is, after all, the most potent agent we have for the cure of hysteria. As previously noted, suggestion is one of the most powerful factors in developing hysterical paroxysms, and it is also, probably, the most powerful agent we have for controlling these same paroxysms. The hysterical patient should be under the influence of a nurse or companion whom she loves and in whom she has confidence.

This attendant should be of good physique, of strong will, of sober mind, and full of tact; and she should have sufficient intelligence to study the peculiarities of her patient's mental condition so that she may tactfully avoid touching upon topics which, by suggestion, may influence her patient unfavorably, and so that she can utilize the fads and idiosyncrasies of her patient in such a way as to help her keep her mind from dwelling upon her own troubles. The majority of hysterical patients desire to get well, and they desire to be surrounded by people and by influences which help to convince them that they are going to get well. The successful treatment, therefore, of hysterical patients will depend largely upon the ability of the physician to so control the surroundings of his patient that she will be constantly influenced by wholesome suggestions—suggestions that she is improving from time to time, and that her early recovery is assured. The influence of change is so potent in the treatment of hysterical patients that it is necessary that radical changes should be made in the surroundings from time to time. The wholesome surroundings of a new location after a certain length of time become mere

routine, and routine wears upon the nervous system of hysterical patients.

In beginning the treatment of severe cases of hysteria the Weir Mitchell Rest Cure is often of great advantage. The confinement to bed, massage, forced feeding, isolation, and striking change of surroundings which this treatment comprehends act not only by suggestion upon the mind of the patient, but the treatment itself is especially adapted to many cases.

Hydrotherapeutics, in some form, is applicable in the treatment of nearly every case of hysteria. The cold tub-bath or the cold douche to the spine will often bring a patient out of a severe paroxysm of hysteria. This treatment, however, is applicable not only in overcoming severe symptom groups of hysteria, such as trance, paralysis, and mental despondency, but in many cases it acts as a tonic to the nervous system, and should be continued as a part of the routine daily treatment. The alternate hot and cold bath is applicable in some cases. The hot bath, followed by general massage and an alcohol rub, is of very great advantage in many cases. The operator must be carefully selected and carefully instructed in these cases; she must have explained to her the powerful influence of suggestion in the treatment of hysteria, so that she, by her manner and conversation, may strengthen the patient's confidence in her physician, and convince her that just this particular treatment has cured many other cases exactly like hers.

Electricity is one of our most valuable agents in the treatment of hysteria, and it acts largely by suggestion. This fact should be kept in mind in giving electricity,

and all legitimate means to make the suggestion as strong as possible should be used. The method of application, the size of the electrical apparatus, and the preparation of the patient's mind for the treatment may be influences that work for good or evil in the giving of electricity; but after all it is the tact and personal magnetism of the operator that counts for most in these cases.

In the treatment of aphonia and paralyses of various kinds it may be necessary to use the electric brush, at the same time impressing the patient that, while the operation is painful, it is wonderfully efficacious in the cure of the condition from which she is suffering. The electric brush performs wonderful cures in some of these cases. Apart, however, from the power of suggestion and the dread of treatment, it is not exactly clear how it may act upon the hysterical mind.

Surgical operations and injuries of any kind may exercise a temporary wholesome influence upon hysterical symptoms. Blistering the skin for the relief of pain and cauterizing the spine for tenderness are of benefit in some cases.

Pressure over the hysterogenic areas, such as the ovaries, may sometimes cause sudden recovery from a severe hysterical paroxysm.

Sedatives play a very unimportant rôle in the treatment of hysteria, and it is doubtful whether they ever do any real good. The bromides and valerian, however, may be used for the relief of symptoms, but they should not be continued for any length of time. The hysterical patient, under no conditions, should depend upon seda-

tives for the relief of nervous symptoms. They aggravate, rather than control, the underlying pathological conditions of the disease. Hysterical patients, however, may be given a tonic suitable to their condition, and they may be impressed with the idea that this tonic has remarkable sedative and curative powers. The medical treatment, like the other forms of treatment, largely depends for its efficacy on suggestion.

CHAPTER XXII

HEADACHES

ETIOLOGY

Age.—Headaches are very uncommon in children under five years of age, but when they occur they are, as a rule, due to some intercranial organic disease or to disease of the internal ear. After five years of age headaches become more frequent, so that between the ages of eight and fourteen they are very common, but even during this period they are nothing like so common as they are between the ages of twenty and forty, this being the period of selection for neuralgic, neurasthenic, and migrainous headaches.

Sex is a very noticeable factor in producing headaches after the fourteenth year of life, women suffering more commonly than men, in the ratio of 3 : 1; but in early childhood sex has little influence.

Heredity.—A neurotic inheritance predisposes to headaches. This is especially true of migrainous, neurasthenic, and neuralgic headaches. These cases commonly have a family history of gout, migraine, neurasthenia, hysteria, or general nervous instability. Feebleness of constitution due to chronic diseases in the parents may be inherited by the child and predispose it to reflex, toxic, anæmic, and other varieties of headache.

Anæmic Headache.—Anæmia and malnutrition are such potent factors in producing headaches that all writers upon this subject discuss anæmic headaches. All of the

various forms of malnutrition so common in infancy and childhood, produced by tuberculosis, hereditary syphilis, gastro-intestinal diseases, constipation, rheumatism, malaria, bad hygiene, and improper food, have as one of their most characteristic symptoms a profound anæmia and a general instability and irritability of vasomotor and other nervous centers. This condition is a very powerful predisposing factor to headaches and the other neuroses of childhood. Malnourished, anæmic, neurotic children may have headaches from such slight exciting causes that these causes may be overlooked. It is perhaps better to consider anæmia as a predisposing rather than as an exciting cause of headaches, since anæmia is but one of the signs of the general malnutrition of nerve and other tissues which predispose these children to headaches, and since this condition is commonly associated with important exciting factors which otherwise may be overlooked. In the treatment of headaches in anæmic children, however, it must always be kept in mind that these cases cannot be successfully treated unless the underlying malnutrition is removed. The removal of the exciting causes in these cases may relieve the headaches, but it does not remove the predisposing cause.

Neurasthenic Headaches.—Neurasthenia is, like anæmia, a powerful predisposing cause of headaches. This factor is, however, more commonly found in older children. The neurasthenic condition in children is largely dependent upon malnutrition of the nervous centers and upon neurotic inheritances, and is developed by subjecting this kind of a nervous system to mental overwork, nervous strain, and emotional excitement. These factors

bring about an exhaustion and functional incapacity of nerve centers which we call neurasthenia, and which has as one of its characteristics the development of headaches from slight exciting causes.

Among the exciting causes which may develop headache in anæmic and neurasthenic children are nervous and emotional excitement, nervous and physical fatigue, nervous shock, fear, anger, mental overwork, the strain and confinement incident to school life, as well as the ordinary toxic and reflex factors presently to be discussed. Such causes as these may in neurotic and malnourished children with vasomotor instability produce fluctuations in the blood supply of the brain, thus producing congestive or hyperæmic headaches. In older girls the menstrual period may act in a similar way in producing very severe headaches. Congestive and other types of headache occurring in anæmic and neurasthenic children are usually vertical, and the pain is dull and boring in character. They are commonly associated with vertigo or a feeling of faintness, and may be relieved by the application of cold to the head and nerve sedatives, such as the bromides.

Reflex headache is perhaps the one form of headache which is more common in childhood than in adult life, and this is because the immature and unstable nervous system of the child responds more readily to reflex stimuli than does the stable and mature nervous system of the adult.

Between the ages of six and fourteen eye-strain is a very common cause of headache. Errors of refraction and strabismus are very frequent in young children, and

are commonly overlooked until the child goes to school. Here sooner or later, if there be any marked ocular defect, the eye-strain will make itself known by a headache at times so severe as to make it impossible for the child to use his eyes sufficiently to do his school work in a satisfactory manner. Under these conditions he becomes nervous, irritable, dislikes his school, and suffers from a headache more or less severe and chronic in character. It is located, as a rule, in the forehead or between the eyes. It may, however, involve other portions of the head. It comes on after using the eyes for some length of time, and grows more severe towards the close of the school day. It disappears or at least is very much better in the morning, after the eyes and the nervous system have had a night of rest. It should be remembered, however, that while eye-strain is a very important factor in producing headaches in children,—so important, in fact, that it is at times the only apparent cause of this condition,—yet in the majority of instances where this factor is present it is assisted by other and more important factors. In every instance, therefore, where eye-strain is found to be present as an apparent cause of headache, a careful search should be made for other possible causes. Anæmic, neurasthenic, and toxic headaches may be developed or greatly aggravated by eye-strain and other reflex factors. In the chapter on Migraine I have called special attention to the secondary rôle which reflex factors may play in precipitating and aggravating this particular type of headache, and here again I wish to insist that a neurotic inheritance and chronic malnutrition producing an instability of vasomotor and other nerve centers is, as a

rule, the basis of reflex headaches, and plays quite as important a rôle in their production as the reflex factor itself. Among the causes other than eye-strain for reflex headaches may be mentioned adenoid growths and other diseases of the naso-pharynx, phimosis with adherent prepuce, and foreign bodies and undigested food in the intestinal canal.

Toxic headaches are very common in childhood, especially those of gastro-intestinal origin. Undigested food and the irritating and toxic products produced by gastro-intestinal fermentations are perhaps the most important exciting causes of headaches during childhood. Headaches of this character are commonly located in the front or top of the head, and are frequently associated with rise in temperature and other symptoms of gastro-intestinal fermentations, such as nausea, vomiting, flatulency, diarrhœa, constipation, and coated tongue. They are, as a rule, acute, especially in younger children. They may, however, in older children assume a chronic character, continuing from day to day so long as the chronic intestinal toxæmia exists. The relief which follows cathartic medication, diet, and intestinal antiseptics assists in the differential diagnosis of this form of headache.

Toxic headaches are also produced by systemic bacterial poisons acting on the nerve centers. This type of headache occurs in all the acute infectious diseases, and is especially severe in influenza. Headaches of this type are, as a rule, most severe during the invasion of the organism by the toxins, and therefore are among the early symptoms. In older children, headaches from this cause are more common and more severe. Headaches,

however, due to acute systemic bacterial toxæmias quickly declare their origin by the appearance of other signs and symptoms which announce the character of the infection.

Uræmia may in the child, as in the adult, produce severe toxic headaches, but headaches due to this cause are nothing like so severe in the child as they are in the adult. Uræmic headaches are commonly located in the occipital region, and are associated with disturbances of vision, vertigo, nausea, and other symptoms of Bright's Disease. An examination of the urine in these cases readily differentiates this type of headache.

Neuralgic headaches are commonly toxic in origin, and may be produced by malaria, influenza, gout, and rheumatism. Malaria is a very common headache producer, but plays this rôle somewhat less commonly in the child than in the adult. Malarial headaches declare themselves by their periodicity, and, as a rule, by their neuralgic character. The diagnosis of malarial headaches may also at times be confirmed by the presence of the plasmodium in the blood, and by other characteristic signs of malaria. Periodic neuralgias may also be produced by influenza. A favorite location for these periodic neuralgias is in the supra- or infra-orbital nerves, which may remain sensitive to touch in the interval between the neuralgic headaches. It must also be kept in mind that infections involving the antrum of Highmore, frontal sinus, and other bony cavities of the face may produce very severe and very persistent periodic neuralgias of facial nerves. The periodic character of these neuralgias commonly leads to the mistaken diagnosis of malaria or influenza, until more serious symptoms announce the infection of these bony cavities.

It is important, therefore, in the treatment of all severe, persistent periodic neuralgias of the supra- or infra-orbital nerves or other nerves of the face to make sure that the bony cavities of the face are not involved.

Auto-toxins play, in the child as in the adult, the rôle of producing the most important of all the syndromes in which headache is the central symptom, viz., migraine. A separate chapter has been devoted to this form of toxic headache and it will not be here discussed.

It should be remembered that both auto and bacterial toxins, whether of systemic or intestinal origin, act, like other exciting causes, more powerfully in producing headache in nervous, anæmic, malnourished children.

A neurotic inheritance, anæmia, general malnutrition, and neurasthenia may one or all be underlying causes of headaches which may be excited by reflex, toxic, or other exciting factors. The etiology of headache is, as a rule, complex, and a diagnosis of anæmic, neurasthenic, reflex, or toxic headache may, therefore, be incomplete, since, as a rule, more than one of these factors are operative.

Organic Headaches.—Headache may be a symptom of disease of the ear or of organic intercranial disease. Earache due to disease in the internal ear is perhaps the most common form of pain in the head occurring in very young children. Persistent pain in the head in young children should always excite the suspicion of disease of the internal ear. These cases occur so commonly in children during the first year of life, before the child is old enough to assist in the location of the pain, that they are commonly overlooked unless the physician is on the lookout for this one great cause of headache during infancy.

The rarity of other forms of headache during this period and the frequency of this type should lead to an examination of the ear in all young children who seem to be suffering from severe pain, the location of which is not apparent. A very young child will occasionally, by lifting the hand to the ear or by the position which it takes in protecting that portion of the head, direct attention to the location of the pain. In children old enough to declare the location of the pain the diagnosis is of course very readily made; the tenderness of the external ear and of the mastoid, with an examination of the internal ear, will determine the cause of the pain.

Headaches due to organic disease within the cranium may be produced by meningeal inflammation, tumors of the brain due to syphilis and other causes, cerebral abscess, and traumatic lesions. Headaches, however, of this character can scarcely be mistaken for non-organic headaches. They are more severe, persistent, and localized, and are accompanied by other signs of the organic disease of which they are a symptom.

TREATMENT

The successful treatment of headaches comprehends, of course, the differential diagnosis of the various etiological factors and their relative importance. A search should first be made for reflex factors, with special reference to eye-strain. Such reflex factors as may be found should, if possible, be removed. Attention should next be directed to the gastro-intestinal canal. It is good practice to begin the treatment of all kinds of headache in children with some form of cathartic medication, such

as calomel, followed by castor-oil. This will clear out the intestinal canal and assist very materially in determining the importance of the rôle which gastro-intestinal factors play in producing the headache. If the results of this treatment and the character of the headaches and other symptoms justify the diagnosis of toxic headache of intestinal origin, then the further treatment will consist in such diet and medication as will remove the exciting cause. If, however, the headaches are produced by some acute systemic bacterial toxæmia, they may be relieved by cathartic medication, cold to the head, and the specific treatment of the acute infection of which they are the symptoms. In these acute conditions one is justified in using sedative medication to relieve the pain in the head. For this purpose the bromides of strontium, sodium, and potash, put up in essence of pepsin or some other palatable vehicle, are especially serviceable. Citrate of caffeine in one-grain doses every hour or two, until the headache is relieved, is also a valuable remedy; the caffeine may be combined with phenacetin or antipyrin in doses suited to the age of the child. My own experience teaches me that children bear these coal-tar products very well, and I have never seen any ill effects from their judicious use in the treatment of headache in children when the headache was dependent upon an acute systemic intoxication, and I have seen very good results from their judicious use in the treatment of the headaches of influenza and other acute infections. The coal-tar products, however, are not to be recommended in the treatment of headaches due to chronic systemic intoxications (see "Migraine").

The treatment of toxic headaches, whatever may be the origin of the toxins, also comprehends a depurative or eliminative treatment. This is accomplished by elimination through the intestinal canal by the use of proper cathartics, preferably saline in character, and by warm baths to facilitate the action of the skin. When high temperature accompanies a headache, an ice-bag to the head and cold bathing to reduce the body temperature will at times act specifically in the relief of the headache.

When the exciting cause of the headache is some emotional or nervous excitement brought on by fear, anger, or nervous shock, or when the headache is associated with extreme nervous irritability or other hysterical or neurasthenic symptoms, cold applications to the head and good-sized doses of bromides act kindly in its relief. Periodic headaches of malarial, influenzal, or other origin are to be treated by iron, arsenic, and quinine. The following is an excellent formula:

℞

Quininæ sulph.	30 grains
Ferri reducti	30 "
Acid arseniousi	½ grain

M.—Make capsules No. 20.

S.—One after eating, for a child eight to ten years of age.

This formula is almost a specific in periodic neuralgic headaches, and is also of value in all forms of anæmic headaches. The salicylates are also valuable in the treatment of neuralgic and neuritic headaches.

It should be remembered, however, that after all reflex factors have been removed, and all sources of intoxication looked after, there may yet remain to be considered

and treated the neurotic condition which is the underlying cause of the headache. In some cases it is true this factor is happily absent, and the removal of the exciting cause, toxic or reflex, establishes a cure; but in most instances even after these factors have been removed there remain to be treated the constitutional causes of the general nervous irritability which underlie these headaches. It is not, however, within the scope of this chapter to discuss the treatment of these conditions. This treatment comprehends not only proper hygiene, suitable and wholesome food, and well-directed medication, but also the intelligent direction of the whole life of the child, so that he may be properly nourished, his constitutional and local diseases eradicated, and his nervous system so protected that it may recover its normal tone and powers of resistance. Chronic headache not organic and not wholly dependent upon removable exciting factors indicates some more or less profound nutritional disturbance of the nerve centers, and should therefore be the warning sign to direct the physician's attention to the disorder of which it is a symptom.

CHAPTER XXIII

ASTHMA

Asthma is a bronchial neurosis characterized by recurrent attacks of spasmodic dyspnœa or sibilant bronchitis without fever, but associated with or followed by discharge of mucus from the bronchial tubes.

PATHOLOGY

The pathology of asthma is not definitely known. It is believed to be a neurosis which has as its underlying factors an instability or irritability of the nuclei or ganglia which control the pulmonary branches of the pneumogastric and sympathetic nerves. The readiness with which these nervous mechanisms respond to irritants, reflex and toxic, in certain individuals constitutes the asthmatic tendency or predisposition. In such individuals comparatively slight exciting causes, acting through the pneumogastric, may produce a spasmodic contraction of the muscular fibers of the smaller bronchi, or, acting through the sympathetic, may produce a vasomotor turgescence of the mucous membranes of these same bronchi, and thus so reduce the lumen of the small bronchial tubes as to seriously interfere with the intake of air, and produce an attack of bronchial asthma.

For a number of years the medical profession has very generally accepted the theory that a large proportion of

the cases of asthma was produced by direct (toxic) or indirect (reflex) stimulation of the nuclei of the pneumogastric or its terminal fibers distributed to the unstriped muscular fibers of the smaller bronchi. Brodie and Dixon have recently furnished convincing experimental evidence that a narrowing of the lumen of bronchial tubes and dyspnœa may be produced in this way. They found that direct stimulation of the pulmonary pneumogastric and reflex stimulation of the same fibers, produced by irritating the nasal mucous membrane, would constrict the small bronchi and diminish the intake of air into the lungs, thus confirming the observations of Lazarus, made eleven years before, that electrical stimulation of the nasal mucous membrane would produce a contraction of the small bronchial tubes. These researches confirmed the observations of clinicians that diseases of the naso-pharynx may be important factors in producing attacks of asthma, and demonstrated the important rôle that reflex factors may play in this disease. Brodie and Dixon also demonstrated that certain drugs (toxins), such as pilocarpine, muscarine, digitalin, and carbon dioxide gas, will diminish the intake of air by contracting the bronchi, and these experiments sustain the generally accepted view that certain toxins may produce asthma by their action on the pulmonary pneumogastric. These same observers found that certain drugs, such as atropin, hyocin, lobelia, and morphia, relieve asthmatic attacks, either by stimulating the broncho-dilator fibers of the pneumogastric or by paralyzing the bronchial endings of this nerve.

Another important group of spasmodic asthmas is pro-

duced by irritations (toxic and possibly reflex) of the pulmonary sympathetic. In these cases the lumens of the bronchial tubes are diminished by congestions and swellings of the bronchial mucous membranes, and these swellings are probably due to a vasomotor paresis.

Hay-fever asthma is an example of this type of asthma. Of this condition Osler says that he fully agrees with the statement of Sir Andrew Clark, that "if the structural changes occurring in the nasal mucous membrane during an attack of hay-fever were to occur also in the various parts of the bronchial mucosa, their presence there would form a complete and adequate explanation of the facts observed during a paroxysm of bronchial asthma."

In susceptible individuals, not only the pollen of plants, but irritating vapors, dust, and peculiar odors, by their contact with the nasal mucous membrane, may excite an attack of asthma. In some instances these attacks seem to be excited by a toxin (pollen) to which the patient is especially susceptible, and in others reflex irritation seems to be the exciting cause.

Attacks of asthma due to vasomotor turgescence of the bronchial tubes may also be produced by certain auto or intestinal toxins to which the individual patients are peculiarly susceptible. These include the cases of so-called urticaria of the bronchial mucous membrane. In such individuals there is a peculiar idiosyncrasy or susceptibility of the pulmonary vasomotor system which makes it respond, in an asthmatic attack, to the unknown auto and intestinal toxins which commonly find expression in urticaria of the skin.

The above outline of pathological factors is believed to present a rational explanation of the syndrome of asthma, and from this outline it is evident that the term asthma, as here used, includes at least two distinct pathological conditions, the one finding expression in a functional disturbance of the pulmonary pneumogastric nerves and the other in a functional disturbance of the pulmonary sympathetic nerves. It is also evident from this outline that the etiological factors of asthma may act, in the first place, by producing the instability of these nervous mechanisms which constitute the susceptibility to asthmatic attacks, and, in the second place, they may act as exciting factors. The exciting factors include inflammatory, reflex and toxic causes, which act upon the mucous membranes of the nose and pharynx; local inflammations which act upon the terminal filaments of the pneumogastric and sympathetic nerves in the bronchial mucous membranes, and auto and intestinal toxins which act upon the nuclei of the pneumogastric, or the ganglia of the sympathetic nerves, or perhaps directly upon their terminal filaments in the bronchial tubes.

ETIOLOGY

Predisposing Factors.—Age.—Asthma may occur at any period of life. My own experience leads me to believe that during infancy and early childhood sibilant bronchitis, which may be classed as a mild asthmatic manifestation, is quite common, but that typical attacks of spasmodic asthma are comparatively infrequent. In older children, however, between the ages of six and twelve years, the adult type is very commonly seen.

Sex.—In childhood there is the same preponderance of males over females that occurs in later life.

Heredity.—An hereditary neurotic constitution is believed by all writers to be an important factor in a majority of the cases of spasmodic asthma. There is not uncommonly a family history of asthma, and there is almost always present a neurotic family history of some kind. These patients very commonly inherit a gouty, rheumatic, or migrainous diathesis, predisposing them to attacks of auto-intoxications and indirectly to attacks of asthma.

Rachitis and diseases of the gastro-intestinal canal and other chronic anæmia producers may, by causing a malnutrition of nerve centers, increase the predisposition of the individual patient to asthmatic attacks.

Exciting Factors.—Auto-toxins of the gouty or lithæmic diathesis play a not unimportant rôle in the etiology of asthma. The toxins in these cases are closely related to or identical with those which produce migraine and recurrent vomiting. Jules Comby classes among the respiratory manifestations of lithæmia in childhood spasmodic coryza, sibilant bronchitis, and asthmatic attacks. The close relationship of these syndromes is evident, and it is also clear that the same poisons, acting through different parts of the pulmonary vasomotor nervous system, may produce either coryza, sibilant bronchitis, or asthma. This type of bronchial asthma may have among its etiological factors constipation, excessive eating, and an inactive indoor life.

The auto and intestinal toxins which sometimes find expression in an urticaria of the skin may excite

asthmatic attacks. F. A. Packard, in a paper on urticaria of mucous membranes, called attention to the fact that sharp attacks of asthma and sibilant bronchitis may be due to urticaria of the mucous membranes of the respiratory passages. Asthmatic attacks of this character are preceded or followed by urticaria of the skin, and have the same etiological factors.

Bronchitis, whooping cough, influenza, and measles are very common exciting causes of asthmatic attacks. They may act by irritating the nervous filaments of the pneumogastric or sympathetic nerves in the bronchial mucous membrane, or they may act, as does tubercular and other pulmonary inflammations, by enlarging the bronchial lymph nodes, which, by impinging on the recurrent laryngeal nerve, may reflexly excite an attack of asthma.

Diseases of the naso-pharynx, such as enlarged tonsils, adenoids, and hypertrophied turbinated bones, may be reflex factors of sufficient importance to excite asthmatic attacks in especially susceptible individuals.

The pollen toxin may be the exciting cause in hay-fever patients. In other specially susceptible individuals attacks of asthma are sometimes produced by a great variety of comparatively simple exciting causes, such as an overloaded stomach, intestinal indigestion, fright, or emotional excitement of any kind, dust, irritating vapors, emanations from animals, as the dog, horse or cat; the aroma of certain medicines, and the odor of certain flowers. Atmospheric and climatic conditions are important exciting factors in a large percentage of cases; peculiar localities may excite the disease in one individual and not in another.

SYMPTOMS

Asthma is an afebrile condition. The bronchitis or influenza, however, which is present as the exciting factor may produce an elevation of temperature, but the fever itself does not belong to the syndrome of asthma.

Asthmatic attacks resembling the adult type of this disease may occur at any age, but they are much more common in older children. They recur at irregular intervals, weeks or months intervening. When the paroxysm is on, severe attacks of dyspnœa may recur nightly for a time, or in other instances the dyspnœa may continue with marked severity for twenty-four or thirty-six hours, and then gradually subside into convalescence. Typical attacks of asthma, as a rule, come on rather suddenly. They usually begin at night with a wheezing respiration, which soon becomes a marked dyspnœa. The child sits up in bed, fixing his shoulders or arms so as to bring all of the accessory muscles of inspiration into play in the attempt to force air into the already distended air vesicles. This increases the emphysema which accompanies these paroxysms, and gives a barrel-shaped appearance to the chest in the latter stages of the attack. Expiration is prolonged and accompanied by sonorous and wheezing rales, and the vesicular murmur is ofttimes scarcely discernible. After a number of hours the dyspnœa gradually subsides, and is, as a rule, followed by a cough, with more or less mucous expectoration. The cough and mucous expectoration, accompanied by wheezing and large moist rales, may continue for a few hours or days, and then subside into rapid convalescence.

In infants and younger children afebrile sibilant bronchitis with slight dyspnœa is much more common than the typical asthmatic paroxysm above described. The dyspnœa in this condition is not very great. The number of respirations, however, is markedly increased and sibilant, and wheezy bronchial sounds occur, and in some instances persist for five or six weeks. There is no pain and comparatively little discomfort—these patients often go about the house and amuse themselves without complaining of feeling ill.

Holt calls attention to another type of asthma which occurs in infants and resembles capillary bronchitis. He says: "These cases are rare, but may be seen even in infants. The onset is sudden, with moderate fever, incessant cough, severe dyspnœa, and sometimes symptoms of suffocation . . . cyanosis, prostration, and cold extremities. The chest is filled with sonorous, sibilant, and soon with subcrepitant rales. Instead of running the usual course of bronchitis of the finer tubes, the symptoms may pass away very rapidly, and in forty-eight, and sometimes in twenty-four hours, the patient may be quite well. It is only by the course of the disease, and by recurring attacks, that their true nature can be recognized. In infants this form of asthma may be fatal."

LaFetra calls special attention to the eosinophilia which occurs in asthma. He says: "The leucocytes are usually, but not always, increased, as in bronchitis; but a differential count of the white cells shows what does not occur in bronchitis: a constant and usually marked increase in the number of poly-eosinophiles. The cases examined for me at the Vanderbilt Clinic by Dr. Ira

Wile showed an eosinophilia from 6 to 18 per cent. Cabot reports, in adults, a mean eosinophilia of 7 per cent., in a range from 0 to 53.6 per cent. This eosinophiliæ is greatest for any given patient at the height of the attack. It disappears in the interval, but in sub-acute cases a low grade of eosinophilia exists. Thus the differential count of the leucocytes is of diagnostic and prognostic value, so far as the attack is concerned." This eosinophilia, LaFetra thinks, indicates the toxic origin of the disease.

PROGNOSIS

Patients rarely die of asthma, and the prognosis, so far as recovery from the asthmatic constitution or susceptibility to these attacks, is also good, provided these patients are so situated that they can take advantage of the means that are offered for the cure of this condition. To accomplish a cure, however, years of careful medical supervision are, as a rule, necessary. Chronic cases, however, which have gone on to the development of a chronic emphysema, do not yield readily to any form of treatment.

TREATMENT

Treatment of the Attack.—Inhalation of the fumes of stramonium leaves and niter paper may relieve the paroxysm; if these fail, chloroform by inhalation will temporarily arrest the attack. In severe cases, especially in older children, a hypodermic of one-tenth of a grain of morphine may be given; this remedy acts specifically in cutting short the paroxysm. Atropin, 1-1000 of a grain, combined with nitroglycerin, 1-200 of a grain, may be

given hypodermically for the control of the paroxysm, and, if necessary, this dose may be repeated in two or three hours. An emetic will sometimes cut short a paroxysm of asthma, even when the gastric contents have little to do with exciting the paroxysm; syrup of ipecac may therefore be given for this purpose. Tincture of belladonna combined with bromide of potash, chloral, or antipyrin, in doses suited to the age of the child, are valuable remedies for modifying, shortening, and especially for preventing, the development of an impending attack.

Asthmatic attacks due to vasomotor turgescence of the bronchial mucous membrane are best cut short by local applications, by means of an atomizer to the respiratory passages of a solution of cocaine and adrenalin chloride. The 1-1000 solution of adrenalin chloride may also be used in one to three minim doses in deep hypodermic injections. These remedies at times act specifically in controlling this type of asthma.

The Interval Treatment.—The nose and throat should be carefully examined for causes of reflex or toxic irritation, and all such factors carefully removed. Adenoids, large tonsils, nasal hypertrophies, and all diseases of the rhinopharynx should receive appropriate treatment.

Bronchitis, whooping cough, measles, influenza, and all diseases which produce catarrh of the bronchial mucous membrane should be studiously avoided, or if present should be carefully treated until all bronchial irritation has disappeared.

If the patient has a well-marked lithæmic history, the

treatment in the interval should be similar to that recommended in the chapter on Migraine. If no such history exists, or if the patient fail to respond to this treatment, then careful attention should be given to general hygiene, with reference to removing such nutritional disturbances as may possibly be predisposing factors of this neurosis. Iodide of potassium and syrup of hydriodic acid are valuable remedies in these cases, and by many writers are believed to exercise a specific influence in preventing asthmatic attacks. These remedies, therefore, should always be given a trial, unless some other line of tonic medication looking to the correction of some special nutritional disturbance is indicated. Cod-liver oil, iron, and arsenic are of value in many of these cases, and if there be any suspicion of malaria, quinine may be given.

Patients suffering from asthmatic attacks associated with urticaria of the skin are to be treated for the urticaria. In such cases a preliminary cathartic of calomel and soda is to be followed every day for a week or more by a dose of phosphate of soda of sufficient size to keep the bowels thoroughly evacuated. It is advisable also to give these patients, for a number of days following the attack, either benzoate of soda or bicarbonate of potash in some such palatable vehicle as essence of pepsin. The interval treatment of these cases is largely dietetic, and consists in avoiding such articles of diet as are commonly believed to be responsible for urticaria, or, more specifically, the particular food which, in an individual patient, seems etiologically related to these attacks.

Change of climate or change of locality is after all

the most important factor in the cure of these cases; but in this respect it is difficult to lay down rules, since asthmatic patients, above all others, have the strongest idiosyncrasies with reference to certain localities and certain climates. A climate or locality that may benefit one individual may fail to give relief to another. These patients, as a rule, however, do well in high and dry altitudes, unless they have chronic emphysema. Experience alone will determine the best locality for the individual asthmatic patient. It is a good rule, however, to avoid the locality in which the attack developed, especially at the season of the year when attacks are liable to occur. If the attack has developed in the city, a change to the country is advisable. If the attacks are worse in winter, or if they are precipitated by recurring attacks of bronchitis, it is advisable to spend the disagreeable months of the year in some such climate as that of Southern California or Florida.

CHAPTER XXIV

DISORDERS OF SLEEP

Sleep is the physiological rest which the tired organism demands to repair the fatigue changes incident to the physiological activity of cells, especially those of the nervous and muscular systems. The physiological activity of all the organs of the body alternates with periods of relative repose. This repose is absolutely necessary to the vital activity of cells. In the higher animals the central nervous system rests at least once in twenty-four hours, and this condition of rest is called sleep. Normal sleep is characterized by loss of consciousness, loss of voluntary inhibitory control of motor and mental acts, and more or less complete loss of all the special senses. Sight goes first, probably taste and smell next, and finally touch and hearing disappear as sleep becomes profound. During sleep all of the higher functions of the brain are held more or less in abeyance, and the involuntary inhibitory control of motor and mental acts is also partially lost. The discharge of nervous stimuli to all the organs of the body is greatly diminished, and as a result there is more or less relaxation of the muscular system, and a feebler functional activity of nearly all the important glands.

During sleep, however, the capacity of the central nervous system to react to peripheral stimuli is not altogether lost. But the more profound the sleep the stronger must the peripheral stimulation be to make any impression

upon the nerve centers. In the very beginning of sleep the nervous system may respond very actively to slight external stimuli, producing muscular twitchings of the body, which may be severe enough to arouse the individual with the knowledge that this spasmodic contraction has occurred. These phenomena, however, are more likely to occur in highly nervous individuals, the nervousness being produced by unusual activity of the brain before going to bed, or by an excitable condition of the higher nerve centers produced by toxins. While this condition of increased reflex excitability at the beginning of sleep can scarcely be said to be physiological, yet it is made possible by the fact that the higher nerve centers, which exercise inhibitory control over the lower, are the first to lose their functions under the influence of sleep; and as sleep becomes more and more profound the entire nervous system gradually sinks into a condition of more or less complete repose, the motor centers at the base of the brain and the reflex centers of the cord being the last to come under its sedative influence. When the entire nervous system has come under the influence of profound sleep, the reflex centers of the brain and cord are not so readily excited to action by peripheral stimuli as they are in the beginning of sleep, when the inhibitory centers are in repose, and the motor centers have not yet lost their normal excitability. During the first hour sleep becomes more and more profound. At the end of this time the higher nerve centers are very profoundly under its influence, and it requires comparatively powerful stimuli to bring the individual back to consciousness. During the second hour sleep becomes gradually less profound, and

from this time on a comparatively slight stimulus is sufficient to awaken the individual. The profound sleep of the first two hours has been likened to a condition of narcotism, which slowly passes off, leaving the individual still unconscious, but easily aroused. The lower motor centers of the brain and spinal cord maintain about the same degree of irritability from the beginning to the close of sleep. They are apparently not influenced, as the higher centers are, by the narcotism of the first and second hours of sleep.

The healthy new-born infant sleeps nearly all of the time, at least twenty out of the twenty-four hours. During the first month the normal infant is awake about four hours in the twenty-four. From this time on the child requires slightly less sleep, so that at six or eight months he is sleeping sixteen hours in the twenty-four, and at the age of one year he sleeps from twelve to fourteen hours. During the first few days of life sleep is heavy, owing to the fact that the organs for receiving and carrying peripheral stimuli to the central nervous system are not yet fully developed. From this time on during the next month sleep becomes less profound, and from the end of the third month to the end of the second year sleep is not so deep as it is after the third or fourth year, when the heavy sleep of childhood is seen. It is at this time in the life of the individual that the profound narcotism of the early hours of sleep is most noticeable.

The most common disorders of sleep are night-terrors, somnambulism, and insomnia. Of these the most important is night-terrors, or pavor nocturnis.

Pavor Nocturnis.—Night-terrors is a neurosis depend-

ent upon an abnormally irritable nervous system, easily excited by reflex stimuli having their origin in distant parts of the body or in the cortical centers themselves. It is characterized by a night-terror which finds expression in the child's screaming or crying out in a panic of fright during sleep.

ETIOLOGY

Heredity is a very potent etiological factor. In the most severe cases there is commonly a well-marked neurotic family history, and such neuroses as epilepsy, hysteria, chorea, migraine, and neurasthenia not uncommonly occur in the family histories. This strong hereditary taint predisposes these children to reflex and convulsive neuroses of all kinds. The particular defect of the nervous system which is inherited is a feeble inhibitory control of mental and motor acts. This may explain the relationship existing between epilepsy, infantile eclampsia, and night-terrors which appear to be present in some families. Beyond this there is perhaps no direct connection between these neuroses. While a neurotic family history resulting in an extremely irritable nervous system under feeble inhibitory control is present in many of the more severe cases of night-terrors, this factor is by no means so well marked in the milder types of this disorder. In some instances the excitable nervous system seems to be wholly dependent upon other factors entirely foreign to hereditary influences.

Malnutrition is an important factor in developing irritability of the nervous system in young children, and the common causes of malnutrition, such as lymph node tuber-

culosis, chronic diseases of the gastro-intestinal tract, chronic malaria, hereditary syphilis, and rachitis, with improper food, impure air, and bad hygiene, may therefore be important predisposing factors of night-terrors.

Mental overwork and nerve excitement, when coupled with physical inferiority, are most potent factors in producing the highly excitable state of the nervous system which makes possible the development of this syndrome. School life, therefore, in a child of feeble constitution may, with its mental grind, increased nerve excitement, close confinement, and eye-strain, be a factor in the development of night-terrors.

Exciting Causes.—The normal irritability of the nervous system of the child having been exaggerated by heredity, malnutrition, mental overwork, or nerve excitement, makes it possible for certain reflex exciting causes to develop an attack of night-terrors. The intestinal canal is one of the most important sources of this reflex irritation; undigested food, improper food, excess of food, intestinal worms, and intestinal fermentations, with the intestinal toxins which they produce, may all either directly or indirectly act as exciting factors of night-terrors. Adenoids, enlarged tonsils, and nasal obstructions that interfere with normal breathing during sleep may either act as reflex factors or they may act by producing a partial asphyxia, and thus excite an attack of night-terrors.

In many cases, however, the reflex factors are absent, or perhaps it might be better to say are so slight that they cannot be readily discovered. In these cases the attack is apparently excited by a horrible dream, which has its

origin either in some alarming occurrence of the previous day or in the overstimulation of the emotional centers produced by blood-curdling tales or exciting fairy stories just before going to bed. These cases, which are cerebral in origin, belong to the class previously described as strongly neurotic. The nervous systems of these extremely neurotic children may be so excited by punishment, by fits of anger, and by fright that they fall asleep with the incidents of the day still impressed upon their nervous systems, and, as a result, the cortical centers do not come profoundly under the reposeful influences of sleep, and in the paroxysm of night-terrors which supervenes the horrible vision which presents itself to the child in his night-terror is but an exaggerated reflex of some mental impression which he received during the day.

SYMPTOMS

Silbermann divided night-terrors into two rather distinct clinical types, which for the most part have been recognized by recent writers. One of these he called Idiopathic Night-Terrors, and the other Symptomatic Night-Terrors. The idiopathic type is of central or cortical origin, and the symptomatic of peripheral origin. In the description which follows these two types will be recognized.

Central or Idiopathic Night-Terrors has for its most important etiological factor an extremely excitable nervous system under feeble inhibitory control which has been inherited from neurotic parents. In the family history of these cases, hysteria, neurasthenia, and the convul-

sive neuroses, all of which are largely dependent upon feeble inhibition, are common. The inherited neurotic condition in these cases may also be aggravated by malnutrition and improper training. There can be little doubt, however, that even in these cases peripheral irritation plays a part in touching off the paroxysm; but the central nervous system is in such a state of excitability, and under such feeble inhibitory control, that a slight peripheral irritation produces a maximum result, and for these reasons it is commonly disregarded or overlooked.

Idiopathic night-terrors occur in the great majority of instances between the ages of two and eight years. This is the period of life when feeble inhibitory control of cortical and other centers is responsible for many of the graver neuroses, such as eclampsia, epilepsy, and chorea.

THE PAROXYSM

A neurotic child, with its nervous system unusually excited by the incidents of the day, falls asleep, and after an hour or two suddenly starts in its sleep with a cry of terror which alarms the household. A moment later he is found apparently wide-awake, sitting up in bed, or crouching on the floor in a state of wild excitement, staring and pointing at some horrible imaginary object which he seems to see with great distinctness. He trembles with fear and gesticulates wildly, calling for assistance, but when spoken to fails to recognize his mother or nurse, who are vainly endeavoring to arouse him to consciousness. He may call out the name of some man or animal who he thinks is about to do him injury. After

a few minutes of this agonizing fear the attack seems to spend its force, the excitement gradually passes away, and the little patient may fall back upon the pillow and become quiet in sleep, which may continue without further disturbances until morning. In many instances the child will go through an attack of this kind without recovering consciousness. In other words, the whole attack occurs during sleep. In other instances the strenuous efforts of the attendants may arouse the child to a vague consciousness, or, rather, semi-consciousness, during which, in a dazed way, he recognizes his surroundings, and then quickly drops asleep, and the next morning has little or no recollection of what has occurred during the night. According to Silbermann, Coutts, and other observers, the seeing of visions is the most characteristic feature of these attacks of central or idiopathic night-terrors. In this condition similar attacks may occur for a number of nights in succession, or there may be an interval of weeks or months between them, but they always present very much the same clinical picture, although they vary in intensity.

Incontinence of urine may occur during these attacks, or the child may at the close of the attack make known his wants, and after seeking the commode pass urine or have a movement from the bowels, as though he were entirely conscious of his actions, and yet give no other evidence of being conscious of his surroundings, returning to bed and continuing his sleep, and the next morning having no recollection of these occurrences.

This central type of night-terrors is believed by many writers to be closely related to epilepsy, and quite a num-

ber of cases of epilepsy have been reported in which night-terrors occurred as a part of their early history. Concerning this relationship, however, I am quite in accord with the opinion expressed by Charles Putnam in his excellent paper on this subject in the "Cyclopedia of the Diseases of Children." He says: "Altogether, the connection between night-terrors and epilepsy, in so far as they are separate diseases, is no clearer than that between any two of the neuroses, and yet, inasmuch as attacks closely resembling night-terrors are occasionally only symptoms of epilepsy, it is well to watch carefully for a time before deciding that epilepsy is not present."

Symptomatic Night-Terrors are more common in childhood, but they may occur at any age. This type is much more common than idiopathic night-terrors. In symptomatic and peripheral night-terrors the essential etiological factor is outside the nervous system in some peripheral excitation. Children suffering from this symptom-complex have, as a rule, unstable and irritable nervous systems, but this nervous instability, instead of being hereditary, is, as a rule, acquired. Chronic malnutrition and other factors capable of producing an unstable nervous system in an otherwise healthy child may commonly be observed. The reflex factors above noted as having their origin in the intestinal canal, nose, throat, and other organs are present, and can, as a rule, be very readily discovered.

The Paroxysm.—The child falls asleep and may toss restlessly for an hour or two before the reflex irritation to the nervous centers culminates in an attack of night-terrors. The patient screams with terror, sits up in bed,

or runs about the room. He is wildly excited, trembles with fear, and exhibits a very marked but as a rule undefined terror. He sees no visions and hears no noises, and responds to the efforts of his attendants to arouse him. He recognizes his attendants and seeks consolation from them. His nervous fears, however, are soon quieted, and he falls asleep to awaken the next morning with perhaps a vague recollection of the occurrences of the night.

Silbermann, and after him Coutts, have called attention to the differences in the clinical pictures portrayed in the two types of night-terrors. Coutts uses the term night-mare to describe the class of cases which Silbermann speaks of as symptomatic or peripheral. As Coutts puts it, the chief distinction between these two symptom groups is that the one suffering from idiopathic night-terrors "sees visions," while the one suffering from symptomatic night-terrors merely "dreams dreams." Silbermann expresses the same idea by saying that the former is characterized by objective terror and the latter by subjective terror. It may be added also that in the idiopathic form the terror is more real, the mental excitement greater, and the condition of unconsciousness more profound.

Notwithstanding the differences in the clinical pictures which the two types of night-terrors present, I am not prepared to say that they are distinct clinical entities. I am rather inclined to believe that the idiopathic type of this disorder presents the aggravated clinical picture as it may occur in highly neurotic children whose mental and motor mechanisms are under feeble inhibitory con-

trol. Between this extreme type and the milder attacks of symptomatic night-terrors, due almost wholly to strong reflex excitation of an almost normal nervous system, there is indeed a wide difference in the clinical pictures presented, but certainly not more so than there is in epilepsy or other neuroses. In this regard I quite agree with Putnam, who says: "It is hard to convince one's self that there are two classes so definitely separated from each other. It is true that between two individual cases there may be a vast difference in all the particulars mentioned by Silbermann, but taking all cases together, the degrees of difference are so slight that it is almost, or quite impossible, to draw a line of demarkation."

PROGNOSIS

In the symptomatic form the prognosis is very good, because it is produced by etiological factors which can readily be removed by appropriate treatment. In the idiopathic form the prognosis is not so good, and depends largely upon the gravity of the underlying hereditary taint. All of these cases, however, should yield to appropriate treatment, but idiopathic night-terrors should call attention to, and demand treatment for, the underlying hereditary condition.

TREATMENT

In beginning the treatment of all of these cases the intestinal canal must be carefully scrutinized and all possible reflex irritation from this source removed. A preliminary cathartic followed by a carefully regulated diet

with a light evening meal should be a part of the treatment in every case. It is impossible to lay too much stress upon the rôle which disorders of the gastro-intestinal canal play in these cases. It is incumbent upon the physician, therefore, to thoroughly satisfy himself that the intestinal canal of the child is no longer a source of irritation or intoxication to the nervous system, and in doing this he must remember that severe intestinal toxæmia may be present without any pronounced symptoms on the part of the gastro-intestinal tract. Enlarged tonsils, adenoids, and nasal obstructions of all kinds, as well as all other discoverable causes of reflex irritation, should be removed.

The child's general health should be carefully looked after. A diet should be selected with reference to the character of the malnutrition present. Tonics, such as iron, arsenic, cod-liver oil, or a malt containing diastase, may be indicated in individual cases. An outdoor life, with an abundance of sunshine and fresh air, is also important. With all these measures, looking towards the removal of reflex irritation and the underlying constitutional factors, must be combined careful protection of the nervous system. In the idiopathic cases the child's nervous system should be as carefully shielded from mental strain and nerve excitement, as if she were suffering from one of the graver neuroses. The medical treatment of these cases consists in giving the bromides of strontium, or potassium, in five- or ten-grain doses at bedtime. It is best to combine with this a dose of tincture of belladonna suitable to the age of the child (one to four minims). The bromide of potash and bella-

donna will, as a rule, readily control the paroxysms, and after four or five nights all sedative medication may cease. In some cases, however, it is necessary to give this prescription for weeks at a time for the control of paroxysms in the severe cases. It is best, however, to discontinue all sedative medication as soon as the paroxysms are controlled.

INSOMNIA

Prolonged insomnia, as it occurs in the adult, lasting through the greater portion of the night, is uncommon in children, and when it does occur is a symptom of some more or less serious disease.

Disturbed or unrefreshing sleep, with possibly a few hours of wakefulness, is common in childhood, and it is this condition rather than true insomnia which here interests us.

ETIOLOGY

Disturbed sleep is produced by very much the same etiological factors as night-terrors. A general nervous irritability is probably the most important underlying factor, and this irritable condition of the nervous system may be a matter of heredity, or it may be produced by chronic malnutrition, or it may occur in the convalescence from acute infections. This irritable condition of the nervous system may be very greatly exaggerated by more or less constant nerve excitement. The mental stimulation and strain of school life, with night study and the anxiety which sensitive children have concerning the lessons of the following day, may in older children

be causes of disturbed sleep. In infancy nervous excitement is also a cause of restless sleep. The habit of constantly entertaining infants, and constantly attracting their attention, and bringing them into the whirl and excitement of the living-room, where they may be observed and commented upon, cannot be too severely condemned. Filling young minds with exciting stories before they are put to bed predisposes to dreams and disturbed sleep.

Lack of proper training is, in the young infant, the most potent of all causes of insomnia. Rocking infants to sleep and lifting and fondling them every time they make an outcry, with feeding at night, will bring about the habit of insomnia and disturbed sleep.

Disturbances of digestion are the most important of the direct exciting causes of insomnia. Over-feeding and improper feeding may develop in the intestinal canal important reflex and toxic factors which, by their action on the nervous system, may disturb sleep. In infants intestinal fermentation may, by the development of gases, produce colic. This may also occur in older children, but as a rule constipation, with a more or less obscure intestinal toxæmia, is with them a more important factor of nocturnal restlessness. In very young infants hunger may be a cause of sleeplessness.

Poorly ventilated and overheated rooms, with lack of fresh air, heavy and uncomfortable bed-clothing, dentition, otitis, adenoids, enlarged tonsils, and nasal obstructions, may cause restlessness at night.

As a rule, more than one of the above-named factors are present in the production of insomnia, and individual

cases must be carefully studied, with all of these possible factors in mind, in order to ferret out the responsible factors in any given case.

TREATMENT

The prophylactic treatment, which should begin when the child is born, is of the utmost importance. This consists in carefully regulating the life of the infant, shielding it from excitement, feeding it at regular intervals, and insisting from the beginning that the night shall be devoted to sleep. It is a comparatively simple matter to establish a routine regularity which will firmly engraft upon the infant the habit of sleeping profoundly throughout the night. This habit, when once established and closely adhered to, will do much to overcome the nervous irritability which the infant may have inherited. As the child grows older this regularity in eating and sleeping should be carefully adhered to, and the child should be given a light evening meal and put to bed soon afterward.

Treatment of the Condition.—When the habit of insomnia is once established, the treatment consists in attempting to establish the regularity above referred to, and which a lack of proper training has interfered with. An effort should be made to discover the essential causes of the sleeplessness. Disturbances of the intestinal tract should be carefully treated, and all possible causes of reflex irritation, whether they occur in the nose, throat, or elsewhere, should be removed. The child should sleep in a well-ventilated and not overheated room, and the bed-clothing should be properly adjusted to the season

of the year. If the child suffers from cold feet, a warm bath at night with a hot-water bottle to the feet may assist in overcoming the sleeplessness. Over-pressure at school and mental excitement of all kinds, especially just before going to bed, should be avoided.

Insomnia, occurring as an acute condition in an otherwise healthy infant, should lead one to suspect acute intestinal disturbance. Intestinal pain produced by colic, which is such a common cause of restless sleep, may be relieved by an enema. A child that has fretted and tossed for hours may fall asleep after this procedure.

The use of medicines to promote sleep in children is rarely necessary, unless the restlessness is produced by some acute febrile condition. Bromide of potash and strontium are perhaps the most justifiable remedies under these conditions. Other hypnotics which are so valuable in the treatment of insomnia in the adult are of doubtful value in the child.

SOMNAMBULISM

Somnambulism, or sleep-walking, is a disorder of sleep having very much the same etiological factors as night-terrors and insomnia.

The somnambulist, soundly asleep and apparently perfectly unconscious, with his special senses in abeyance, may rise, walk, or run about in the dark, avoiding objects and performing difficult and apparently purposive acts quite as dexterously as he could when awake. When aroused from this state he is perfectly unconscious of what has transpired.

Somnambulism is not uncommonly observed in chil-

dren, but the marvelously complicated movements which have been accredited to adult sleep-walkers have not been noted in the child. Children, however, may get out of bed and walk or run about the room in the pursuit of some object, or with a definite purpose suggested by a dream, which the child is acting.

Sleep-talking may be combined with sleep-walking. I once witnessed a performance of this kind in a child seven years of age. This child during the day had been much interested in seeing his dog Towser catch and kill some rats as they were one by one liberated from a trap. In the early hours of the night he sprang from bed and ran in the dark through the house, calling to his dog, "Rats! Towser, rats! Towser, here they are!" and for some minutes, avoiding furniture and directing his movements with great accuracy, he led the chase until he was finally captured by his mother and in his half-dazed state led back to bed and to sleep. The next morning he knew nothing of the occurrence.

The treatment for this condition is the same as that above outlined for insomnia.

CHAPTER XXV

NYSTAGMUS AND ASSOCIATED MOVEMENTS OF THE HEAD IN INFANTS

W. B. Hadden, under the title "Head-nodding and Head-jerking in Children, Commonly Associated with Nystagmus," described a not uncommon neurosis characterized by rotary, lateral, or vertical movements of the head, commonly associated with rotary, lateral, or vertical movements of the eyes.

CHARACTER OF THE MOVEMENTS

Peterson described, under the term "gyrospasms," a rotary movement of the head from right to left and left to right. These head movements may also take the form of "head-nodding"; in these cases the head moves with a vertical nodding motion. In other cases the movements of the head are horizontal. These vibratory movements of the head are, as a rule, rhythmical and rapid, two or three vibrations occurring to the second. The same movements, however, do not always persist. Any one of these movements may be replaced by or alternate with either of the others, or the three movements of the head—vertical, horizontal, and rotary—may all occur at different times in the same patient.

Hadden says that pure nodding is rare, but this movement is commonly combined with or alternates with the lateral or rotary movements.

In some cases these movements may cease when the child's attention is firmly fixed on some object, but as a rule the movements are increased when the child is under observation. During sleep the movements cease, and they are not so well marked, and commonly disappear when the child is lying down and quiet in a darkened room, and they may sometimes cease when the eyes are covered.

Nystagmus is commonly associated with these head movements, and the eye movements may be rotary, vertical, or lateral. The movements of the eyes, however, are more rapid than the movements of the head, the vibrations in some instances being as rapid as six to the second. These involuntary vibrations of the eye are, as a rule, rhythmical. The horizontal movement is the most common, but it may alternate with or be replaced by vertical or rotary movements, and rarely, according to Mills, "the vertical and horizontal oscillations may alternate regularly or irregularly, or a vertical movement may be present in one eye and a horizontal in another. The commonest form of nystagmus is that in which the movement is bilateral, horizontal, and consentaneous."

Hadden also notes that there is a "relation between nystagmus and the position of the eyes, or even the ocular state. In one case the nystagmus was exaggerated on extreme conjugate deviation to the right. In two instances the nystagmus was chiefly evident when the eyes were directed upward, and in one of these it was generally horizontal and tended to become vertical when the eyes were turned upward. The nystagmus

may vary in direction apart from this; in two instances it was sometimes vertical, sometimes horizontal, and sometimes rotary."

The movements of the head and eyes do not always correspond. Any form of eye movement may be combined with any form of head movement; for example, head-nodding may be combined with lateral nystagmus, or we may have nystagmus of one eye associated with any form of head movement. In short, any number of combinations of the various head movements and eye movements are possible, but it should be remembered that in perhaps a majority of cases the head and eyes move in the same direction.

The various head movements above described, while commonly associated with nystagmus, may occur without the nystagmus, and on the other hand the nystagmus may occur without the head movements. Nystagmus is not associated with any abnormal condition of the eyes, although in some instances it is associated with strabismus. Head movements are sometimes associated with strabismus, without nystagmus. On the whole, however, the association between strabismus and the syndrome above described is not very common.

ETIOLOGY

This syndrome, as a rule, occurs during the first year of life, commonly between the second and twelfth months. During the second year of life it is not infrequent, but after that it is very uncommon, except as it is associated with organic disease of the nervous system, insanity, or congenital idiocy. In this chapter, however,

we are interested only in this syndrome as a manifestation of a not-uncommon neurosis which occurs almost exclusively between the beginning of the third and the end of the twentieth month of life. The fact that this condition almost never occurs before the end of the second month and is very rare after the twentieth month is an evidence that the condition is a developmental one. Before the second month the centers which control the eye and head movements are not sufficiently developed to respond to reflex and other excitations, but after these centers have developed and before the eye and head movements are under proper inhibitory control we may have developed the syndrome as above described. Later, however, when the spinal accessory and motor oculi centers are under inhibitory control from cortical centers, these movements become impossible, and this neurosis disappears. Age is, therefore, above all the great predisposing cause of this neurosis.

Sex has little influence. Most writers state that the condition is more common in females.

Heredity is perhaps an important predisposing factor. In many of the cases there is a bad neurotic family history, epilepsy, chorea, hysteria, and other neuroses which are characterized by feeble inhibition having been noted.

Rachitis and gastro-intestinal disease, with improper food, impure air, and bad hygienic surroundings, are also very important predisposing causes of this neurosis. These are the great factors which produce malnutrition in infants, and the malnutrition of the nerve centers, which are a part of these conditions, is probably the

predisposing factor. All writers are agreed that there is a close association between rachitis and this neurosis, some believing that rachitis is almost an essential factor and others that it is present as an etiological factor in a minority of the cases only. It is not probable that there is any specific relation between rachitis and this neurosis; if so, it would be much more common than it is. It is more probable that rachitis acts as a predisposing factor by interfering with the development of inhibitory centers and increasing the irritability of the nerve nuclei involved.

Exciting Causes.—We know little or nothing of the exciting causes of this condition. Peterson believed that trauma, or head injury of some kind, mild or severe, producing concussion of the brain, may be found in most of the cases to be the determining factor. Other writers, however, have not laid much stress upon trauma as an exciting cause. Henoch believed that dentition is an exciting factor; the reasons, however, for this belief are not very clear. It is true that some of these cases occur during the time of dentition, and it is also true that dentition in rachitic, malnourished infants may produce rather pronounced nervous symptoms; but there is no more direct evidence than this that dentition is an exciting factor of the syndrome under discussion.

PATHOLOGY

The pathology of this condition is largely a matter of speculation. Hadden expressed the opinion that this syndrome is produced by an instability of cortical motor

centers having control of the nuclei in the spinal cord and fourth ventricle. The young infant gradually acquires certain voluntary or purposive movements of the head and eyeballs, and these movements, not being thoroughly under the control of the cortical inhibitory centers, are not directed or restrained, and there results the involuntary oscillations above described. This view of Hadden is concurred in by Mills and other writers, who believe this the best explanation of a very obscure condition. My own view is also in accord with Hadden's, as I above outlined in explaining the important rôle that age plays as an etiological factor.

PROGNOSIS

The prognosis is, as a rule, good. This syndrome, however, in one or more of its manifestations may continue for months, but under proper care recovery finally occurs. The head movements, as a rule, disappear before the nystagmus.

In making the prognosis in an individual case it is important that the neuroses above described be carefully differentiated from the same head and eye movements occurring in certain organic diseases of the brain, as well as these same movements occurring with the so-called imperative movements of defective children. These imperative movements in feeble-minded children very commonly take the form of a salaam, or repeated movements of the arm, trunk, or leg. If such movements as these are associated with the syndrome under discussion, the prognosis is not so good.

TREATMENT

The treatment is largely a matter of improving the child's general nutrition. Rachitis and the underlying gastro-intestinal disease, if present, must be carefully treated by diet and proper medication. A carefully selected diet, suitable to the age and digestive capacity of the child, is absolutely necessary; fresh air and wholesome hygienic surroundings should be insisted upon. Cod-liver oil and some palatable and easily assimilated preparation of iron may be of value. Under this treatment the child's malnutrition gradually disappears, the nervous centers are better nourished and become less irritable, and the inhibitory centers of the cortex gradually assume more perfect control of the lower centers, and as a result the syndrome disappears.

Sedative treatment is also of value in beginning the treatment of some of these cases. Bromide of strontium, or some of the other bromides put up in essence of pepsin or some other palatable solution that will not disturb the stomach, may be given for the purpose of controlling the nervous symptoms. These bromides may be given in from three to five-grain doses three or four times in twenty-four hours, but they should be discontinued unless there is evidence that they are of decided value in the treatment of the case.

CHAPTER XXVI

HABIT-SPASM AND OTHER HABIT-NEUROSES

Habit-spasm is a pure neurosis characterized by sudden and quick contractions of certain groups of muscles. These spasmodic movements are most common in the muscles of the face, neck, and shoulders.

ETIOLOGY

This syndrome is sometimes spoken of as habit-chorea. This name is a misnomer, not only because it leads to confusion, but also because the two syndromes, chorea and habit-spasm, are not in any way related. The clinical pictures which the two conditions present are so different that they can scarcely be mistaken one for the other, and their etiological factors are not the same.

Heredity is an important predisposing factor. These patients, as a rule, belong to families having strong neurotic tendencies, and have themselves inherited unstable and easily excitable nervous systems.

Chronic auto or intestinal intoxications, or any of the forms of malnutrition which either irritate or malnourish the central nervous system, may be important predisposing factors of the habit-neuroses.

Age.—According to Weir Mitchell this syndrome is most common between the ages of seven and fourteen.

The prevalence of the disease during this period may perhaps be largely explained by two important etiological factors which are potent at this time in the life of the child, viz., the development of the reproductive organs and school life, both of which may aggravate the instability of the nervous system of neurotic children.

School life, in my opinion, brings to bear on the irritable nervous systems of neurotic children the etiological factors which are most important in the development of habit-spasm. The mental training, the confinement, restraint, and enforced quiet, the unhygienic surroundings, the anxiety to excel, the fear of punishment, and the increased eye-strain which school life entails may all be factors in aggravating the neurotic tendencies of nervous children. The precocity which is common in children suffering from habit-spasm may encourage a degree of mental training that leads to the exhaustion of nervous energy, and thus may produce neurotic disease in a rapidly growing child. Rapid growth of body is not to be combined with rapid mental development in nervous children.

The failure to properly protect the nervous systems of neurotic children during the functional development of the reproductive organs may also lead to the development of habit-spasm and other neuroses. The physical condition of the child may also be important during this period of development, but general malnutrition does not appear to play so important a rôle in the development of this neurosis as it does in many others.

Exciting Factors.—With the nervous system of the

child prepared by the etiological factors above noted, it is easy to understand how apparently unimportant exciting causes may play a rôle in developing habit-spasm. These factors are, of course, made potent only by reason of the irritable and unstable condition of the nervous system which has resulted from the more important factors. Among the exciting causes, however, which play a rôle in the production of habit-spasm, *imitation* is perhaps the most powerful, and, when once the habit has been developed, sympathy for the child and attention to the spasm are very important factors in aggravating and continuing the spasmodic movements.

An extremely precocious and highly neurotic little patient of mine, two years of age, who from the tenth to the eighteenth month of her life practiced the habit of thigh-friction, recently came in contact with a child who had habit-spasm of the muscles of the face. Very soon my little patient was noticed to have contracted the identical habit-spasm which the older child had. The spasm consisted in a drawing downward and outward of the left corner of the mouth with a quick, sudden contraction. On seeing the child, I was able to elicit the spasm by talking of it or calling attention to it. For this reason I forbade the nurse or any member of the family to again notice or speak of the spasm. Under this treatment the contraction became less frequent, and after about ten days ceased entirely. In this case the spasm was controlled before the habit became fixed.

Eye-strain, diseases of the nose, throat, and pharynx, and all other sources of reflex irritation should be care-

fully searched for and removed, since such factors may be etiologically related to habit-spasm.

SYMPTOMS

The child is nervous, restless, quick of movement, and, as a rule, bright of mind. But the characteristic symptom is a spasm of one or more groups of muscles in the face, neck, or shoulders. These muscular contractions most commonly occur in the facial muscles. There may be rapid winking or blinking of the eyes, with the drawing of the mouth downward and to one side, distorting the face. The eyebrows may be raised or the brow lowered, as in frowning. A sudden twisting of the head and shrugging of the shoulders are very characteristic movements. A peculiar inspiratory sniff, with a lifting of the alæ of the nostril, occurs in some cases. Habit-spasm of muscle groups in the arms and legs may also occur, but they are not so common. These habit-movements may occur at short intervals, and especially when the patient is under observation. Attention to and discussion of these symptoms always increases the number and violence of the contractions. These movements may almost or quite disappear during the vacation months, especially if these months are spent in the country under good hygienic conditions, and with the return of the child to school the movements may become more aggravated.

The worst cases, as a rule, are seen in the latter part of winter or the early spring months. Associated with habit-spasm there is not uncommonly a hyperæsthe-

sia of some portion of the face or neck. In a little patient of nine that I now have under treatment there is a marked sensitiveness of the ears demanding great care on the part of the mother or other attendants lest they should touch them when they assist her in dressing her hair or putting on her clothes.

Habit-spasm may continue for many months or even years; as a rule, however, the prognosis is good, provided the hereditary taint is not too strong and the child can be placed under the most favorable conditions for recovery.

TREATMENT

As above indicated, the treatment should begin with the removal of all abnormal conditions which may possibly be a source of reflex irritation. The child should be taken out of school, and should receive such mental training as is thought necessary at home. Care should be taken to protect it from all forms of mental excitement, and its surroundings should be such that attention would never be called to the spasm. In very young children the attendants should deny, if necessary, in the presence of the child, the very existence of the spasm. In older children rewards are sometimes efficacious.

An outdoor life, with exercise, a carefully selected, nutritious diet, and such medication as may be indicated to remove the particular anto-intoxication or malnutrition that may be a basic factor in the individual case is in every instance a part of the general treatment. It may, for example, be necessary to treat a migrainous diathesis or a tubercular anæmia; or it may be necessary to re-

move some source of chronic reflex irritation before any progress can be made in the treatment of a habit-neurosis.

THUMB-SUCKING

Thumb-sucking is a habit-neurosis which has its origin in the animal instinct of self-preservation, which causes the infant to suck everything that comes in contact with its lips. The child by instinct conveys to its mouth everything that touches its hands, and when nothing happens to be in the hand the child places its thumb, finger, or some other portion of its body in its mouth. In this way the injurious habits of sucking are gradually developed. In the beginning the act of sucking some portion of the body or some foreign substance is done in response to normal instincts, but after a time the sucking habit is gradually formed, and then the infant, during the greater portion of its waking moments, indulges this habit, and seems to get comfort and satisfaction from the act. In indulging this habit the infant does not, as in the beginning, suck promiscuously anything that happens to come in contact with its mouth, but confines the habit to some particular object, such as the thumb. Among the objects commonly selected by the infant for sucking are the thumbs, fingers, toes, tongue, a rubber nipple, a piece of cloth, or some toy.

The habit of sucking does not produce any notable constitutional disturbances, and does not apparently influence the growth or development of the nervous system, and the infant is allowed to form this habit because the mother or the physician does not believe it is worth

while to try to prevent the formation of a habit which gives the child a pleasurable occupation and does not seriously interfere with its development.

The sucking habit, however, does produce certain deformities of the part sucked, and may also lead to irregularities in the development of the mouth. The deformities of the mouth, thumb, and fingers may, in aggravated instances, be so pronounced that they are noticeable when the child grows up. It is, therefore, for the purpose of preventing these deformities that the sucking habit should be prevented, and this can best be done before the habit has become thoroughly formed.

If the child is allowed to indulge in the sucking habit for months or years, it is then a very difficult matter to overcome it. In such cases the habit can only be broken up by some mechanical device which makes it impossible for the child to continue it. In some instances, where the habit has been indulged in for only a short period of time, good may result from covering the thumb or fingers or part sucked with solutions of quinine or aloes. These bitter solutions, however, are of little value where the habit is well formed.

The mechanical means which may be used to prevent the continuance of the habit vary with the individual child and with the part of the body sucked. Splints may be used which will prevent the child from bending the elbow, and thus make it impossible for it to get its hand to its mouth. Mittens, gloves and bandages for the hands may be tried in suitable cases. The difficulty which the physician experiences in overcoming the habit of sucking should lead him to give more careful

attention to preventing the formation of this habit in other children who may come under his care. Punishment does not, as a rule, correct the sucking habit, but rather teaches the child habits of deception.

Older children may sometimes be influenced by rewards or by appealing to their sense of shame.

The sucking habit is always more difficult of treatment in nervous and malnourished children, and for this reason malnutrition and other causes of nervousness should be carefully treated before an attempt is made to break up the habit of sucking by mechanical restraint.

THIGH-FRICTION (INFANTILE)

Thigh-friction is a habit-neurosis not infrequently observed in infants. It is commonly accomplished with the child lying on its back; the thighs are flexed, crossed, and pressed tightly together, closely embracing the external genitalia; in this position the infant makes an up and down body movement or rubs the thighs together. These movements are apparently attended by a pleasurable excitement, and there are flushings of the face and an increase in the general nervous tension. Following this act, which continues for a few minutes only, there is a general relaxation, accompanied by mild perspiration and an apparently quiet contentment. This act may be accomplished by the infant in a variety of ways. At times it is done by rubbing the inside of the thighs against some object, such as a pillow or other portions of the bed.

This act, by reason of the pleasurable excitement it produces, is repeated from time to time until the habit be-

comes engrafted upon the nervous system. The habit once formed, the infant may practice it many times in the twenty-four hours, especially if left alone. In the beginning it will indulge in thigh-friction quite as openly and innocently as it indulges in thumb-sucking or nail-biting; but after being restrained from accomplishing this act, and finding itself watched with this purpose in view, it becomes very secretive, and indulges in the practice when not observed.

Thigh-friction is commonly described as a form of masturbation, and very closely resembles this act as practiced by older children. Thigh-friction, however, as practiced in infancy differs in some particulars from the masturbation habit practiced in later childhood. In the older child masturbation is a very pernicious practice, which has its primary origin in the newly awakened sexual instinct which accompanies the development of the sexual organs, and in the altogether new and tremendously intense and pleasurable sensations which accompany the gratification of this instinct. These indulgences may be led up to by a neurotic constitution and by local irritations; but after a time the masturbation habit is formed. This habit is hard to overcome, and eventually produces an instability and irritability of the local nervous mechanism involved, and at times profound functional disturbances of the central nervous system—absent-mindedness, mental depression, neurasthenia, and even insanity may result. The tendency of the masturbation habit, when excessively indulged and long continued, is to produce physical and moral degeneracy.

Thigh-friction, or this so-called type of infantile masturbation, presents an altogether different picture, and is from a clinical and prognostic standpoint a different condition.

Thigh-friction in infancy, because of the rudimentary condition of the sexual organs, is not and cannot be accompanied by the intensely sensual sensations which accompany masturbation in later life. It is purely a habit neurosis, similar in its etiology to habit-spasm and thumb-sucking. The sensations, however, in thigh-friction are more intense than they are in the other habit-neuroses, because it involves the excitement of that portion of the nervous system which is later to control the fully developed sexual organs. But these sensations surely do not compare in intensity to, and are perhaps very different in quality from, those that are afterward produced by exciting the same nervous mechanism in an older child in whom the sexual organs are sufficiently developed to respond with their physiological function.

Thigh-friction, like other infantile habit-neuroses, disappears under proper treatment before the child is four years of age. In certain cases where the predisposing and exciting causes are not removed, it may persist for a longer time; but in my experience there is no connection between this habit and masturbation in later childhood; the one does not lead up to and does not predispose to the other. Thigh-friction is common in infancy. In early childhood both this condition and true masturbation are rare. In later childhood masturbation is very common, and thigh-friction very rare. Thigh-friction is vastly more common in female infants. All of my cases,

strangely enough, have been of this sex. True masturbation is much more common in boys, and with them the habit becomes more firmly fixed and produces more deleterious results.

The diagnosis of thigh-friction is readily made if the physician's attention is called to the symptom group. In many instances, however, the diagnosis is never made because the infant is not intelligently observed, or because the act is considered a trick or innocent habit of not sufficient importance to be considered seriously. It is my belief that most of these untreated cases are cured by the mental and physical development of the child. Between the third and the fifth year of life there is a tendency to spontaneous recovery from this and all other infantile habit-neuroses. In children, however, who have inherited an intensely neurotic constitution, or in whom there persists a profound malnutrition or a local genital irritation, the habit may continue indefinitely.

ETIOLOGY

As above noted, age and sex are important predisposing factors. The great majority of these cases occur in female children under four years of age.

Heredity.—This is a very powerful predisposing factor. Nearly all these children inherit a neurotic constitution. Gout or tuberculosis is also commonly found in the family history.

Auto or intestinal intoxications are important predisposing factors. Anæmia and general malnutrition produced by gastro-intestinal diseases, rickets, lymph node

tuberculosis, malaria, improper food, impure air, and bad hygiene aggravate the general nervous irritability of the child and increase the tendency to this and other habit-neuroses.

Exciting Causes.—Local exciting causes commonly exist in these cases. Of these Holt says: "The most frequent are long or adherent prepuce, phimosis, balanitis, vulvo-vaginitis, eczema of the labia, thread worms, and tight clothing. A urine which is irritating because of excessive acidity, or the presence of crystals of uric acid, may be a cause. Any irritation may lead the child to rub the parts in some way, and, a pleasurable sensation being excited, this action is repeated until a habit is formed." In my own experience a mild vulvo-vaginitis and recurrent attacks marked by a hyperacidity of the urine are the most common exciting factors which lead to the habit of thigh-friction in infants.

PROGNOSIS

The prognosis in thigh-friction is very good. If properly cared for, children will lose the habit and all danger of relapse before they are four years of age. The prognosis in masturbation as its occurs in older children is, however, a very different matter. These cases are very difficult to treat, and the habit, once formed, is kept up to a greater or less extent throughout childhood and into adult life.

TREATMENT

In infantile thigh-friction the first and all-important step in the treatment is to place the patient under such careful observation or such ingeniously devised mechani-

cal restraint as to make the continuance of the habit an impossibility. The accomplishment of this purpose in some instances is a matter of the very greatest difficulty. In the great majority of these cases the act is performed only when the infant is lying down and when the thighs are flexed. In these cases the infant when awake is to be kept in a sitting posture, or when lying down is to be carefully watched by a thoroughly trustworthy nurse. When it is taken for an outing, if it is old enough to sit up, the go-cart is to be preferred to the baby-carriage. During sleep the infant should either be carefully watched or should be held by some mechanical contrivance in such a way that the act will be impossible. If the infant sleeps in pajamas, the heels of this garment may be fastened by safety pins to the mattress in such a manner as to hold the legs apart and prevent the flexion of the thighs; at the same time the child's body is prevented from slipping down in the bed by a ribbon stretching from the back of the pajamas to the head of the bed. I have used some such device as this many times with great success, and I have found it more satisfactory than nightly vigils over the sleeping infant. Many mechanical devices have been recommended, all of which have in view the forcible prevention of thigh-friction. The profound sleep of the young child lends itself to this mode of treatment, and the patient quickly becomes accustomed even to such cumbersome appliances as the double thigh splints with a separating footboard which have been recommended in troublesome cases. No special device, however, is suitable to all cases, but if the physician is sufficiently impressed with the necessity for this method of

treatment, the particular mechanical device by which the end is to be accomplished may be left to his ingenuity.

In treating older children for the masturbation habit the above methods do not apply. Forcible restraint in these cases does more harm than good. Neither is corporal punishment of value. These are the cases that are most difficult and discouraging to treat. There is little that the physician can do except to remove, if possible, all predisposing and exciting factors. The malnutrition may be treated and the cause of local genital irritations removed. The common sense, pride, and judgment of the child are also to be appealed to, but this is usually better done by the mother than by the physician. But even when all these measures for the control of the masturbation habit in the older child are carried out, the results are far from satisfactory, since the majority of these cases are little influenced by these measures.

In this chapter, however, we are more especially concerned with the infantile habit-neurosis thigh-friction, in which the prognosis is most favorable, and in which the control of the habit by mechanical measures is a long step in the cure. This interruption breaks into and helps to destroy the habit which has been engrafted on the nervous system, and in that way makes for the permanent cure of the affection. The habit interrupted, the next most important step in the treatment is the removal of all possible sources of genital irritation. The preputial hood should be separated from the clitoris, and vulvo-vaginitis, if it exist, should be carefully treated. Phimosis, prepucial adhesions, pin worms, and other causes of genital

and rectal irritation are to be carefully sought for and treated. The clothing of the infant is to be so adjusted as not to irritate the genitals. Infants having a tendency to increased acidity of urine should be given benzoate of soda put up in palatable solution. Either this or some other alkali should be given daily for months to prevent the recurring attacks of increased acidity of urine which are present in many of the cases of thigh-friction.

Lastly, but of not less importance, is the treatment looking to the removal of the nutritional disturbances and general nervous irritability which are such important predisposing factors in many of the cases of thigh-friction. This treatment embraces an out-of-door life free from excitement and mental stimulation, a carefully selected diet, and medicines suitable to the form of intoxication or malnutrition from which the infant suffers.

CHAPTER XXVII

PICA, OR DIRT-EATING, IN CHILDREN

Pica is a habit-neurosis which manifests itself in a perverted appetite. Patients having this disorder eat all kinds of indigestible and innutritious substances, such as plaster, clay, sand, cinders, ashes, and dirt, which to a normal appetite would be repulsive or disgusting. The term pica is taken from the Latin name of the jay or magpie, because of its supposedly greedy appetite. The peculiar perversions of appetite which occur in this condition have some analogies to the well-known gastric neurosis—bulimia. Bulimia, however, is an exaggeration of the normal appetite, and is characterized by an almost insatiable craving for food which for the most part is wholesome. The patient suffering from this condition may eat at short intervals enormous quantities of food, as much as twenty or twenty-five pounds in twenty-four hours, without immediate discomfort and often without any bad after-effects.

In pica, on the other hand, the appetite is so perverted that normal food in any quantity does not satisfy the unnatural cravings, which demand not large quantities of food, but unwholesome and repulsive substances which have, as a rule, little or no food value.

Perversion of appetite similar to pica, as it is manifested in man, also occurs in such animals as dogs, sheep, and goats. Young lambs sometimes manifest this ten-

dency by eating wool, hair, and dirt in preference to the grass of the rich pasture upon which they are wont to graze.

A clear conception of this neurosis, as it is manifested in children, can only be had by a careful study of the great variety of etiological factors which have been accused of producing pica.

GENERAL ETIOLOGY

Predisposing Causes.—Insanity and feeble-mindedness are predisposing causes. Pica occurs as a pure psychosis in about 15 or 20 per cent. of insane and feeble-minded individuals. This class of patients, however, are not inclined to select certain substances which they take instead of food, but they fill their stomachs with all kinds of materials that may come in their way, such as pieces of dress, bedding, sand, or more disgusting materials, and on another occasion these same patients may fill their stomachs with entirely different things. This condition, therefore, as it manifests itself in the insane, is not so much due to a perverted appetite as it is to a condition of mind in which judgment and discretion are lacking, and in which the animal instinct of self-preservation so predominates that they instinctively put everything into their mouths with which their hands come in contact. In these patients this practice does not produce a desire for certain definite materials as it does in the young child, and should not, therefore, be classified under the neurosis we are now describing.

Age.—Cases of pica occur at all ages. A mild form of this condition is very commonly seen in infants, and

the more severe types are seen in children and young adults. In later life the condition is rarely observed.

Sex.—The large number of cases that occur in infancy are about equally divided as to sex. In early childhood there is a slight preponderance of females, and the cases occurring in adults are almost wholly confined to this sex.

Heredity.—There is, as a rule, a strong neurotic family history. Writers upon this subject have also recorded that in many cases there is a direct inheritance of the dirt-eating habit. It is more probable, however, that in these cases the child, having inherited the neurotic temperament, contracts the dirt-eating habit by imitation.

Malnutrition is a very potent etiological factor in a large percentage of the cases. In the infantile cases, rickets, intestinal disease, and lymph node tuberculosis, combined with bad hygiene, lack of sunshine, and improper and badly prepared food, are etiological factors of importance.

In children and young adults chlorosis, well-marked anæmia, and more or less profound nutritional disturbances are almost always present, and are believed to be very common and very powerful predisposing causes of pica.

Menstrual disorders and hysteria are very commonly associated with this condition in older children, and in young adults.

Pica is not infrequently an hysterical manifestation, and in many cases seems to be closly related to disturbances of the menstrual function. Cases of amenorrhœa and menorrhagia occurring in hysterical girls are often

associated with dirt-eating. These cases are, as a rule, anæmic and malnourished. Samuel Wright reports in the *Medical Times,* 1847, the case of a young girl, aged twenty, who was malnourished, had not menstruated for four years, and was employed as a glass polisher. She acquired the habit, as did one of her companions employed in the same business, of eating the Fuller's Earth which she used in polishing. He estimated that she swallowed "in one year and a half no less than twelve hundred ounces of aluminous earth." Under careful treatment she slowly convalesced.

Edward Rawson in the *Medical Press* of 1881 reports the case of a girl, aged eighteen, very anæmic, profoundly malnourished, marked nervous symptoms, and had not menstruated for three years. She came into the hospital suffering from an abdominal tumor, and in response to a powerful cathartic a large bucketful of rags came away. Among them were ribbons eighteen inches long, pieces of velvet, handkerchiefs, and large pieces of cloth.

A. M. Gould in the *Boston Medical and Surgical Journal,* 1876, reported a case of pica, aged forty-three, female, anæmic, has dyspnœa, and suffers from menstrual disturbances. "For two years she has had a longing for innutritious articles. At first she ate charcoal; at present fine sand and gravel. She asserts that she has eaten 'nearly a bushel of sand, and takes daily from a tablespoonful to a cupful.'"

The medical literature is full of cases of this description that appear to be etiologically related to hysteria, menstrual disturbances, anæmia, and general malnutrition.

Exciting Causes.—Imitation and mimicry are important exciting factors. In many instances the individual has the practice of dirt-eating suggested to him by contact with others who have the habit. Following this suggestion in imitation or in a spirit of mimicry, he begins the practice, which afterwards becomes a habit. This to my mind explains the fact that pica has occurred endemically from time to time in almost every quarter of the globe. Dr. Foot, in describing and explaining the prevalence of pica among the natives of Jamaica, says: "Negroes have been overheard urging their companions to indulge in the habit of dirt-eating." Among neurotic and anæmic girls working at trades in which they have to handle chalk, Fuller's Earth, sand, and clay, pica is not uncommon. Propinquity and imitation are responsible for beginning the practice of dirt-eating under such conditions. Chalk-eating among school children, having a like origin, is not infrequently the beginning of a habit which develops into pica. Imitation and mimicry are especially strong characteristics of the childish mind, and they are therefore potent factors in the development of many of the neuroses of childhood. This is especially true of certain of the habit-neuroses, such as pica and habit-spasm.

The animal instinct of self-preservation is a very important factor in developing this neurosis in infants. It is this instinct which causes them to put everything that touches their hands into their mouths, and is therefore largely responsible for developing the habit, the after-indulgence of which constitutes the infantile type of pica. Plaster from the wall, dirt scraped from boots or the

floor, ashes and cinders from the fireplace, and sugar and candy from the nursery, are the most accessible materials. The infant's hands coming in contact with one or more of these articles, they are conveyed to the mouth and swallowed. This instinctive act of the infant is repeated time and again, until a habit is engrafted and a desire created, the gratification of which gives pleasure or satisfaction, and when once the habit is formed of taking one of these substances, the number of innutritious things which the child swallows may be gradually enlarged until it contains the whole available list.

It will thus be seen that *propinquity and opportunity,* so far as the child's contact with materials is concerned, have much to do with the development of this habit in infancy. And this is also true with older children and adults. It not uncommonly happens that predisposed individuals begin a practice which leads to the habit of dirt-eating by reason of the fact that they are handling daily in their occupations certain materials, such as chalk, Fuller's Earth, sand, or clay; the practice of taking into the mouth any of these articles may lay the foundation for the dirt-eating habit.

Pregnancy is an exciting cause which may develop pica in nervous, malnourished, or anæmic individuals who are predisposed to this condition. Pregnant women may have perversions of appetite which create a desire for unwholesome and innutritious articles, such as chalk, cinders, coals, etc. This condition as it is manifested in pregnant women is a true neurosis, perhaps reflex in its origin and similar in its etiology to the vomiting which occurs in this condition. It is perhaps dependent upon

some neurotic disturbance of the functions of the stomach, producing a burning, gnawing, or disagreeable sensation which is relieved by the taking of such articles as chalk, ashes, and cinders. The pica of pregnancy, however, passes away with the condition which produced it, and does not become a fixed habit.

Functional disturbances of the stomach, producing a burning, gnawing, or aching sensation which is relieved by taking into the stomach food or other absorbents and diluents, is a very important factor in developing pica in many cases. This local manifestation of a general neurotic condition is very commonly associated with hysteria, menstrual disturbances, chlorosis, and the anæmia and general malnutrition which results from rachitis, tuberculosis, chronic intestinal disorders, and other chronic anæmia producers.

Foot says, in the *Dublin Quarterly Medical Journal* of 1867, that "in the dyspepsia with which the negroes of Jamaica become infected when exposed to hardships and privations, a prominent symptom is a tormenting gnawing pain in the stomach, and it is for the relief of this uneasy symptom that the sufferer betakes himself to eating some absorbent earth which affords temporary relief."

Dr. Mason, who studied the endemic pica of Jamaica, is inclined to consider that "this habit, as observed among these negroes, instead of being a disease, or the cause of disease, is actually a remedy prescribed in a rough way, the absorbent earth made use of being only injurious from the many impurities they contain."

Intestinal irritation from worms, indigestion, or catarrh

is very commonly associated with the dirt-eating habit in infants and young children, and is believed by medical writers to be etiologically related to this neurosis.

Many of these cases are associated with worms in the intestinal canal. Bacot, in the *Australian Medical Gazette* of 1892, speaks of an epidemic of pica that occurred in and around Cairns in North Queensland, the principal characteristics of which were an inordinate appetite for red clay. This epidemic occurred among children, several of whom died. He reports two of these cases, one of which, Lucy H., four years of age, commenced to eat red clay, and this was followed by wood ashes and dirt scraped from the floor. She died some months later, and the post-mortem records the following: "The body was bloodless, mesenteric glands enlarged, and the duodenum, jejunum, and upper part of the ileum contained multitudes of round worms adhering to the mucous membrane, and many pin worms in the cæcum."

Dukes, in the *Lancet,* 1884, reports the case of a child aged five years who was brought to him to be treated for round worms. Some time later the mother noticed that the child ate the soil in the garden. She said she ate the earth to relieve the gnawing pain in her stomach. So urgent was the demand for this soil-eating that the child would, if she could not get earth, eat sand and mortar. This habit continued for two years, and during that time she passed about one hundred large, round worms.

While the literature of this subject affords ample evidence that many cases of pica are associated with intestinal worms, and while it is also possible that the presence

of worms in the intestinal canal may be responsible for perversions in the functions of the stomach which might be etiologically related to pica, yet it is not by any means certain that the worms are not the result rather than the cause of the dirt-eating in these cases. But even if we assume that the worms are the result, rather than one of the primary causes, one may yet believe that once the worms have taken up their habitat in the intestinal canal they may increase the morbid appetite, and thus produce a vicious circle becoming a secondary etiological factor.

Intestinal catarrh, or intestinal disturbances of some kind, is one of the most constant accompaniments of pica in children under two years of age. Here again it is difficult to decide whether the intestinal disturbances are a cause or a result of the pica. Children of this age are very subject to pica in a mild form, and one is frequently called to see children suffering from intestinal disturbances who have been addicted to the habit of plaster- or dirt-eating for many months. These cases may be found to suffer from more or less constant indigestion, or in other instances the child will recover for a time from both the intestinal catarrh and the pica, and after an interval of weeks or months both the pica and intestinal disorder return. Whether or not the intestinal disturbance in these cases is a cause or a result of the pica, it seems plain that the intestinal irritation exaggerates the child's general nervousness and increases its morbid appetite.

Habit is by far the most important of all the etiological factors of pica. This condition is in fact a habit-neurosis. That is to say, whatever predisposing or exciting factors may have been active in starting the practice which leads

up to dirt-eating, it is the habit which is formed by these practices which impels the patient to continue to satisfy this perverted appetite. The influence of this factor is illustrated in the following case:

E. F., male, seven years of age. Had always been nervous, but had never suffered from any severe illness. Some years ago he developed an unusual appetite for sweets. This appetite grew by indulgence, until at the time I first saw him he was living entirely upon candy and sugar. His father, who is a physician, stated that for three or four weeks he had not taken a mouthful of any other food. He was thin, pale, and nervous, but still had a good deal of endurance. He was on his feet the greater part of the day, played with other children, and ate candy and sugar at short intervals during his waking hours. In trying to break up this pernicious habit we attempted to starve him into taking other foods, giving him all the water he wanted, but withholding sweets. He could not, however, be forced to take other food, and as he became very weak we were forced at long intervals, twelve or twenty-four hours, to give him some candy to eat. We then resorted to rectal feeding, and to the introduction of milk and other foods into the stomach through a tube. These methods were so disagreeable to him that he gradually came to drinking milk and eating bread. After months of careful supervision the father's perseverance was rewarded by seeing the boy's appetite for wholesome food returning. But his convalescence was assured only by a total abstinence from sweets and a constant insistence on the taking of milk and bread at proper intervals.

A somewhat similar case is spoken of by Dr. Foot, who

says: "Among the cases of pica observed by Sir D. Corrigan is one in which this depraved condition of the appetite was traced to the acquired habit of eating sugar. On this child having been weaned and transferred to the nursery, the nurse in charge gave the child lumps of sugar, with the object of keeping her quiet at night. The morbid appetite for sugar increased to such a degree that the child would at last take no food, not even broth, unless loaded with sugar. From a desire to have a lump of sugar in the mouth, the child then turned to other substances, and was never contented unless she had something in the mouth. Clay came most easily to her, as she was frequently in a small garden; and when in the house twine was the next favorite for sucking and swallowing.

Pica, as it occurs in infancy, has more or less decided characteristics. These cases are milder than those that occur in childhood and adult life; they are, as a rule, complicated by or associated with some gastro-intestinal disturbance, and while habit becomes the important etiological factor in these cases, the habit is not so firmly fixed but that in nearly all of these cases it passes off under mild restraint before the child is three years of age. The mental development of the child, which makes it more amenable to discipline and which places its appetite and desires under better inhibitory control, is perhaps an important factor in the cure of these cases. John Thompson, in the Edinburgh Hospital Reports, 1895, expresses the opinion that all these infantile cases manifest a tendency to spontaneous recovery in the third or fourth year of life.

Samuel Wright, in speaking of a case of pica in a young woman twenty years of age, clearly illustrates the rôle that habit may play in the development of these cases in older children and in adults. He says: "She assured me that she never in her life had the least desire to put anything not eatable into her stomach, until it occurred to her one day, she knew not why, to bite a piece of Fuller's Earth.

"She occupied herself some time in chewing it and turning it about her mouth, and at last, when liquefied, swallowed it. This led to the taking of another piece, and to another, and so on, until the practice became agreeable as a mode of pastime. From this it grew into a pursuit of gratification, and at last the indulgence created a positive appetite. The desire became so strong, and the necessity for its satisfaction so urgent, owing to usage, that even the eating of substantial food did not atone for the absence of the filth longed for. Thus, whenever the inclination grew dominant it was answered by an immediate partaking of the material sought; and thus, also, was the morbid appetite increased and confirmed."

Lack of proper training and proper supervision may be mentioned as accessory factors in the production of this neurosis, since it is fair to presume that under proper supervision, especially in infants, this habit could not be formed.

Fright, anger, home-sickness, grief, and other emotional causes may aggravate the habit of dirt-eating, or may cause the individual to return to the habit, if it has been discontinued for but a short time.

SYMPTOMS

A detailed account of the symptoms which constitute and which are associated with the syndrome of pica has already been given in the previous pages. Patients with pica have strange perversions of appetite which lead them to forego wholesome, appetizing food for such innutritious and indigestible things as plaster, sand, gravel, chalk, Fuller's Earth, dirt, clay, ashes, cinders, coal, soapstone, slate pencils, paper, rags, and sometimes such disgusting materials as their own excrement. In some instances these individuals will give up all other food except sweets, such as candy and sugar. This sugar-eating habit not uncommonly leads to dirt-eating and the development of troublesome and disgusting types of pica.

Many patients who practice the habit of dirt-eating may also take for a considerable time a sufficient quantity of nutritious food. The tendency, however, is to gradually increase the quantity of dirt taken and to gradually diminish the quantity of wholesome food. In such cases the patients' general health suffers. They become anæmic, malnourished, emaciated, and more nervous than before. These patients are, as a rule, constipated by reason of the accumulations of dirt in the large intestine. The constipation is sometimes so obstinate that it results in obstruction of the bowels, and threatens or takes the life of the patient. Profound nutritional disturbances are much more commonly associated with pica, as it occurs in the older child and adult, than in the infant.

Dr. Foot in speaking of pica, as it occurs among the negroes of Jamaica, says: "Whatever the motive may

be that induced them to begin the practice, it soon proves fatal if carried to great excess. There are instances of their killing themselves in ten days, but this is uncommon, and they often drag out a miserable existence for several months, or even one or two years. On many estates half the number of deaths on a moderate computation are due to this cause. The negroes subject to pica almost always complain of incessant pain in the stomach. On examination of the body after death there are frequently found in the colon large concretions of the earthy matter which they have swallowed, lining the cavity of the bowel and almost completely obstructing the passage." There are also many reported cases where death has occurred from perforations of the stomach or intestine from the soapstone or other hard materials which have been swallowed.

The infantile type of pica, however, which has been previously described, and which is common with us, bears little resemblance to this severe type of the disease, which has occurred endemically in almost every quarter of the globe. Infantile pica is, for the most part, a mild habit-neurosis somewhat analogous in its etiology to such habit-neuroses as masturbation, habit-spasm, and thumb-sucking. This condition is very commonly associated with gastro-intestinal disturbances and worms. They also suffer from more or less marked nutritional disturbances. Many of them have complexions that are dull and murky, and they may be thin, anæmic, or even cachectic. Many of these cases, however, especially before the habit is well formed, show very slight nutritional disturbances.

The prognosis in pica is good. This is especially true

in the infantile cases, as all of them get well under proper treatment before they are four years of age. The average duration of these cases has been estimated at twenty months.

TREATMENT

The first step in the treatment is to so place the patients under such supervision that it is absolutely impossible for them to continue the habit. It is futile to attempt to overcome this habit, especially where it is strongly intrenched, by persuasion, by rewards, or by punishment. These measures, as a rule, fail. It is advisable, therefore, especially in older children and in young adults, to begin the treatment in a hospital or some other institution where they can be kept under proper control. The change of surroundings is a mental factor which assists these patients in giving up the habit. In young infants it is advisable to place them in the hands of a thoroughly competent nurse. If the habit is thus forcibly broken up, it gradually loses its hold upon the nervous system, and this measure is, therefore, of itself a curative one.

The next important step is to prescribe a proper dietary which is suitable to the age and digestive capacity of the patient. The food problem is especially important in the treatment of infantile cases, since these cases are commonly complicated with gastro-intestinal disturbances, and the first step in their treatment comprehends the removal of all gastro-intestinal irritation and the restoration to a normal condition of the digestive functions.

In older children and young adults the treatment comprehends the removal of the predisposing causes where

this is possible. The causes which produce anæmia and general malnutrition are to be carefully searched for and treated, and in short the object of the treatment is to improve the general health of the patient and to overcome his nervous tendencies. There is no specific medical treatment indicated which will apply to all cases of pica, but medicines are sometimes of great value where the morbid appetite is associated with a burning, gnawing, nervous sensation in the stomach. In these cases alkalies, such as bicarbonate of potash or bicarbonate of soda, may in the beginning of the treatment be of decided value in giving relief to this sensation. Bitter tonics and hydrochloric acid may also in some cases be beneficial in modifying the stomach sensation which is associated with the perverted appetite. For the relief of the anæmia and general malnutrition, iron and cod-liver oil are of value.

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