lieved. That it has the power to strengthen uterine contractions cannot be doubted. All these drugs are dangerous in their action and should not be employed without a thorough familiarity with their actions and dangers.

J. Wesley Bovée.

ECLAMPSIA INFANTUM.—(Synonyms: Convulsions in children; acute infantile epilepsy; spasms.) By eclampsia in children is meant a variety of convulsions, more or less general, occurring with or without prodromal manifestations, and not dependent upon any material organic lesion of the nervous centres. Most authorities regard absolute abolition of consciousness as an essential element of eclamptic as well as of epileptic attacks. This loss of consciousness need not persist during the whole attack, but must have occurred at some part of it. It is true that there are attacks of very slight spasms in children in which it is difficult to be positive that consciousness has been lost, by the application of such imperfect tests (irritation of the conjunctivæ, etc.) as these little subjects will admit; but analogous attacks in adults prove that loss of consciousness, however brief, is an essential element of an eclamptic convulsion. An important point in the differential diagnosis between an attack of eclampsia and one of epilepsy is, that in the former the convulsions are not repeated in a series. It is true, attacks of eclampsia may be repeated, but unless the repeated attacks can be shown to have depended upon one or other of the conditions which ordinarily give rise to eclampsia, every repetition of the convulsion renders our suspicion as to its epileptic character stronger.

In describing an eclamptic seizure, it is important to remember that the convulsion differs in no way from an attack of epilepsy. Hence a general clinical picture of the disease will be that of an ordinary epileptic spasm. There may be an aura, a cry, then convulsive movements in some of the voluntary muscles, proceeding rapidly to more or less general convulsions, at first tonic, then clonic in character. There is loss of consciousness, generally, before the tonic convulsions have set in, the little patient may froth at the mouth, the tongue may be bitten, the respiration is that characteristic of epileptic attacks; finally the spasm is often followed by sleep. It is evident, then, that we must not depend upon the character of the convulsion for a differentiation between epilepsy and eclampsia, although a careful study of the clinical history of the case will generally give us the data necessary to a correct diagnosis.

Clinical Occurrence.—Nothnagel[1] and Ozanam[2] discuss the possibility of the occurrence of eclampsia infantum as an idiopathic affection. The latter authority limits the term eclampsia to convulsions of non-epileptic origin, occurring independently of other diseases or pathological conditions. Nothnagel, however, includes under the term eclampsia convulsions which are the reflex expression of some peripheral irritation, such as dentition, etc. The convulsions occurring at the outset of acute infectious diseases and acute inflammations he terms simply epileptiform, a term that might be applied to all varieties of eclamptic attacks. Eclampsia infantum should be considered not so much a disease as a symptom of other diseases; in one case the convulsion may only precede or usher in a pneumonia, in another it may appear at the same time with a brilliant scarlatina; here dentition appears to have been the exciting cause, there the ingestion of indigestible food; in all of these conditions the attacks resemble each other, although in each case the clinical etiology is different. Looking, then, upon eclampsia from an etiological standpoint, it appears that all the possible conditions under which eclamptic attacks may occur in children will come under one of the following subdivisions:

1. Febrile or symptomatic eclampsia.
2. Reflex eclampsia.
3. Toxic eclampsia.
4. Eclampsia of rachitis.

Febrile or Symptomatic Eclampsia.—The most common form of eclampsia occurring in young children is undoubtedly the first group, the febrile or symptomatic eclampsia. By this class is meant that form of eclampsia which occurs, as a rule, at the *outset* of febrile diseases; we say at the outset, to distinguish these initial convulsions from those occurring later on in the course of severe inflammatory conditions, and due not so much to the fever as to cerebral adynamia, or the aggregation of toxic elements in the blood as a result of the disease. So commonly does a convulsion mark the outset of inflammatory disease and acute infectious fevers of infancy, that it would almost appear that in these little patients the eclamptic seizure corresponds to the chill which ushers in similar diseases in adults and grown children. This substitution of a convulsion, an eminently nervous phenomenon, for a chill, corresponds to the exalted irritability of the nervous system characteristic of infants. This excess of irritability is especially marked in the medulla oblongata; and since the studies of most observers, among others Schroeder van der Kolk,[3] point to the medulla as the starting-point of the changes resulting in an epileptiform convulsion, the increased liability to convulsions at this early age is readily explicable. How shall we explain the occurrence of this febrile form of eclampsia? Is the convulsion due to the fever, or to some mysterious infection of the blood by the poison of the disease of which the fever is a symptom? Authorities differ as to this point, the tendency being to individualize and explain the occurrence of the eclampsia in distinct ways for different diseases. In the first place, in inflammations of the brain and meninges the convulsions are not eclamptic, being dependent upon a material lesion of the nervous centres. But the eclampsia introducing an attack of pneumonia, pleurisy, measles, or scarlet fever, how is this produced? Steiner[4] considers the convulsions which precede an attack of pneumonia as the reflex result of the irritation of the peripheral filaments of the pneumogastric distributed to the congested lung. The weakness of this explanation does not lie in the fact to which Soltmann[5] calls attention, that eclamptic convulsions do not occur in all cases of pneumonia in children, but rather in this, that the convulsions are not so frequently repeated as we should expect were this theory true, for the lung remains congested during many hours, while the convulsion lasts but a few minutes, and in many cases is not repeated. We do not think that any further explanation of the eclampsia of the early stage of pneumonia need be given than that it is due to the high temperature which ushers in the disease. The rapid and bounding pulse, characteristic of the febrile process, produces cerebral hyperæmia, the already over-excitable medulla is rendered more excitable, and a convulsive discharge of nerve force takes place; the cause here is evidently simply increased temperature, and its recognition serves as an important indication for treatment.

In the acute infectious diseases, one might readily be excused for supposing that the initial convulsion is most probably due to the altered condition of the blood. The bacteria of scarlet fever, as well as those of measles and diphtheria and other infectious diseases, may well have so changed the blood, even in the opening stages of the disease, as no longer to enable it properly to nourish the nerve centres; accordingly such writers as Soltmann[5] and Henoch[6] consider that the eclampsia, even in the onset of these diseases, is mainly due to the toxæmia; and yet these convulsions frequently do not occur in the most severe cases, and do occur in the mild ones. The toxæmia must be regarded as varying in direct proportion to the severity of the affection, and the same should be true of the convulsions to enable us to accept the view of Henoch. We do find, however, that in most of the cases in which convulsions have occurred, the temperature was very high, irrespective of the severity of the disease. I recall a case in my own experience, similar to one reported by Hunter,[7] in which, with the onset of a varicella, the temperature rose to 105° F., and the child had two eclamptic convulsions; on the succeeding day the temperature had fallen and the little patient was able to run about in the room. One can cause the cessation of repeated eclamptic attacks in a case of commencing infectious disease by the application of a cold bath. For these reasons it would appear that in these cases also the active cause of the convulsion is the increased heat of the body. It must be acknowledged, however, that there are cases in which the specific toxæmia of the disease is so great that the nervous centres are completely overwhelmed. In these cases the convulsions are really toxic in character, and are far more fatal than those depending upon the fever; here, too, we shall often find upon post-mortem examination inflammatory disturbances of the brain and spinal cord. In the vast majority of cases of eclampsia occurring early in the course of infectious diseases, reduction of temperature will result in cessation of the convulsions. The diseases in which this febrile form of eclampsia may occur include almost all of the febrile affections of childhood, that is, all of the acute infectious diseases, among which must not be forgotten erysipelas and typhoid fever; also all of the inflammatory affections of the respiratory organs, such as laryngitis, bronchitis, pleurisy, and pneumonia; and the acute catarrhal, croupous, and diphtheritic affections of the throat and pharynx. Febrile eclampsia, although more frequent at the outset of febrile diseases, may occur later on in the course of these diseases. As already stated, the eclampsia then occurring is, as a rule, dependent upon blood changes; but not necessarily, for although the nervous system of the child seems to become habituated to mere elevated temperature, yet a great and sudden rise of the fever at any stage of the disease may produce a convulsion. Thus, I have seen a convulsion occur in a child suffering from a pneumonia on the evening before the critical day, the convulsion being simultaneous with the highest rise of the temperature curve.

Malarial fever, according to many authorities, frequently has the chill which ushers in the paroxysm in adults, replaced, in children, by a convulsion.

Malarial eclampsia, so called, is most frequent in infants under three years of age suffering from intermittent fever. In oider children the convulsion is absent while the chill is present When the convulsion takes the place of the chill it is said to be exceedingly severe and may last for hours; sometimes each recurrence of the malarial paroxysm is accompanied by a convulsion. Dubrisay[8] reports a case in which the convulsion recurred with eleven distinct malarial paroxysms. These convulsions are probably toxic in character.

Parotitis, when accompanied by high fever, may be an etiological factor in the production of an attack of eclampsia. Dysentery, gastritis, cholera infantum, all of these when accompanied by high fever may produce a convulsion. Here again, however, we must distinguish the febrile convulsions from those which occur in the later stages of these diseases and are dependent upon the cerebral vascular changes, of which the depressed fontanel is so characteristic a symptom. In conclusion, it may be said that any affection accompanied by high fever may result in an eclamptic attack in infants.

Reflex Eclampsia.—By the term reflex eclampsia, which is the second subdivision under which we shall discuss the subject, we mean that form of eclampsia which is the reflex motor expression of some peripheral irritation of sensitive nerves. Such motor manifestations as a reflex result of peripheral irritation are not unfamiliar, even in adult life; as an instance we have the hysterical, and even epileptic, manifestations in females as a result of uterine disease. In infants the inhibitory action of the brain over the spinal cord is still in abeyance, and reflex action is more uncontrolled; there is an exalted irritability of the spinal cord, and hence these little patients are excellent subjects for the full manifestation of the evil effects of a constant peripheral irritant. Among the most constant of these peripheral irritants during infant life is dentition, and this is accordingly the most frequent cause of reflex eclampsia. There are those who believe that dentition, being a physiological process, cannot be connected with the production of so serious a pathological condition as convulsions, but they are of right in the minority. When it shall have been proved that other physiological processes are never productive of serious morbid states, then we shall give some credence to such a view; for the present, it is emphatically the opinion of the vast majority of authorities that difficult dentition is frequently the only assignable cause for a series of eclamptic attacks. The convulsion is often followed in a short time by the appearance of one or two teeth; some children have a convulsion preceding the breaking through of almost every tooth. We deprecate the assigning of dentition as a cause for almost all the diseases to which infant life is liable; but, on the other hand, we place this physiological process as the chief among the peripheral excitants which sometimes result in reflex eclampsia in infants. To account for the occurrence of these convulsions during the period of first dentition, and their almost uniform absence during that of the second, it is only necessary to remember that the condition of exalted irritability of the nervous centres, which is present in infants, is not present in the older children; hence a similar, or even greater irritation will fail to produce a convulsion in the older child, which in the infant might have produced a serious eclampsia. The gastro-enteritis, which is one of the direct results and a concomitant condition of difficult dentition, may cause an attack of eclampsia in teething infants. In this case we would consider the eclamptic attack the result of the toxæmia caused by the auto-infection from the stomach and intestinal tract, and not due to an irritation of the peripheral nerves terminating in the gums. For an excellent discussion of the subject of dentition eclampsia I would refer the reader to Fleischmann[9] and Politzer.[10]

Helminthiasis is another condition which is supposed by many authorities to give rise in some cases to eclampsia infantum. Such convulsions occur more frequently with the larger worms (round worms, tænia). I recall a severe epileptiform attack in an adult, who had never suffered from epilepsy, which preceded the expulsion of a tapeworm. Other authorities give undoubted cases in which the convulsions were the precursors of the discharge of large quantities of worms. It must not be forgotten, however, that the convulsions may depend upon the intestinal catarrh which is the result of the helminthiasis. Here, again, the toxæmia resulting from the auto-infection from the bowels is the cause of the convulsions. The auto-infection may also be caused by the absorption of toxic products specific to the parasite or such as arise from undigested food due to the catarrhal enteritis produced by the presence of the parasite.

The irritation of indigestible food, or food in too large quantities, may be the cause of a convulsion. In these cases, again, the convulsions may be reflex in character as a result of the irritation of the indigestible matter in the stomach, or they may be produced by an acute rise of temperature; finally, the spasm may be the result of a toxæmic condition of the blood. If the attack be the result of the irritation of indigestible food, it generally occurs soon after the ingestion of the offending nutriment. Children vomit so readily that emesis will generally free the stomach from this irritant, and thus prevent a repetition of the attack. This cause of eclampsia has been acknowledged and well recognized as far back as the time of Hippocrates.

I am disposed to class under this head the occasional attacks of eclampsia resulting from præputial irritation and masturbation[11] in children. Where these attacks are frequently repeated, they would with more propriety be classed as epileptic. Other peripheral irritants have been observed, by good authorities, to produce convulsions in children. Soltmann mentions a case in which ascent of the testicles into the inguinal canal resulted in a convulsion. Foreign bodies in the ears have been repeatedly shown to be active in the production of a convulsion. Scalding of the surface of even a small portion of the body, or any other extremely painful, sudden injury, even if of short duration and of slight character, has been sufficient to produce an eclamptic attack.

Toxic Eclampsia.—The group of toxic eclampsia, al-

though least understood, is by far the most interesting. In this class I include all eclamptic attacks which seem to depend upon some blood change which renders it unfit to nourish the nerve centres. We do not know enough of the changes produced by different diseases in the composition and constitution of the blood to enable us to state, with even an approach to positiveness, the exact alteration upon which this toxic action of the blood depends. In some cases it may be that there is a change in the quantity or quality of the constituents of normal blood; in others foreign substances (toxins) may have appeared in addition to the normal ingredients. We cannot here enter upon a discussion of the various kinds of toxæmia and bacteræmia which cause the septicæmia in the different diseases, but the fact is undisputed that the blood in such conditions acts as an irritant to the nerve centres, and this irritation manifests itself in eclamptic seizures. One fact seems to be characteristic of this form of eclampsia, and that is, that the attacks do not occur at the outset, but generally in the course of a disease. To this group belong the convulsions which occur in the course of a pneumonia as a result of the insufficient aëration of the blood[12]; and here also belong the convulsions (Stickungskrampf) accompanying the dyspnœa of severe croupous and diphtheritic laryngitis, and those accompanying the dyspnœa of prolonged attacks of laryngismus stridulus and whooping-cough. As will be seen later on, in every complete epileptiform attack the clonic convulsions are the result of the strangulation produced by the early tonic spasm of the respiratory muscles, and it is therefore not surprising that similar clonic convulsions occur in diseases whose chief clinical manifestation is spasm of the respiratory muscles.[13] An important class of cases belonging to this group is the eclampsia of scarlet fever, which is the result of a concomitant kidney affection with albuminuria. The retention of the urea in the blood is the cause of the convulsion. The occurrence of eclamptic attacks in primary or secondary parenchymatous nephritis is a frequent symptom in these conditions of the kidney. The convulsions occurring in the course of acute diseases which are non-febrile, but which seem to be dependent upon the action of the poison of the disease process, are well recognized, although we cannot define the nature of the toxæmia in each individual case. We must not forget to mention, under this head, convulsions occurring as a result of permitting infants to nurse at the breast of a mother who has undergone some severe fright or mental shock. At the risk of being accused of belief in what is vague and unproven, I must express my conviction that nervous shock certainly does impair and alter the character of the milk secreted by the mother. The experience of ages teaches that a frightened mother should not nurse her child.

Eclampsia of Rachitis.—The eclamptic attacks which occur in rachitic children we place in a separate group, partly because eclamptic attacks are very frequent in rachitic children, owing probably to the especially exalted irritability of the nervous system, which is characteristic of rachitic children; but chiefly because one variety of eclampsia occurs in rachitic children exclusively; this is the condition known as laryngismus stridulus or laryngeal eclampsia.

Although, theoretically, all cases of eclampsia ought to fall under one or more of the four groups into which I have divided the subject, yet clinically there may be cases which will not seem to depend upon any one of the assigned causes, and will have to be classified as a separate and distinct group, termed idiopathic eclampsia—in other words, cases that cannot be classified etiologically. This group will probably diminish with knowledge and improved methods of examination.

Etiology.—It has already been stated that infants have a marked predisposition to the occurrence of attacks of eclampsia. Why this should be so has occupied the attention of many excellent observers, among whom may be mentioned Nothnagel and Soltmann, the latter of whom has written a monograph on the peculiarities of function in the brain and spinal cord of infants. Clinically, we recognize that children under two years of age are more frequently seized with eclampsia than children beyond that age. So frequent is this disease in infancy that it may be counted as the most fatal pathological condition to which infancy is liable. By this is not meant that the prognosis of all attacks of eclampsia is necessarily bad. On the contrary, the prognosis is very good, but so frequent is the condition itself that notwithstanding its comparatively favorable prognosis it still causes the death of more infants than does any other affection. The fact that it occurs most frequently during the period of dentition led many of the older writers to ascribe eclampsia to the dentition process; but the exalted reflex irritability of the spinal cord in infants, as already set forth, is the only element necessary to account for the predisposition to convulsions shown at this time of life. Heredity is an additional factor which helps to augment this natural predisposition. Most physicians in active practice will recall families in which all of the children have "spasms," while in other households such a thing is entirely unknown. A careful examination will generally reveal the fact that in the former class of patients the parents are of a nervous temperament; there may be a history of insanity, paralysis, or convulsions in other members of the family; in other words, these patients give a neurotic family history. It does not appear that either sex shows any predisposition for the disease. Male and female children seem to be equally liable to convulsions; if individual experience is of any value in this connection, I think I have observed more children with convulsions among the male sex than among females. As to the exciting causes, they have been thoroughly discussed in a previous paragraph.

Rickets is a frequent cause of eclampsia, owing to a variety of pathological conditions which, while they are the direct result of the rachitic changes in the tissues and fluids of the body, affect the brain and spinal cord more especially. Such convulsions, when the result of a gross lesion of the brain and spinal cord, are not to be classed as true eclampsia for the purpose of this article. Frequently eclamptic attacks in rachitic children are the result of a febrile, reflex, or toxic condition, insufficient in degree to have produced convulsions in healthy children, yet sufficient to disturb the balance of the delicately poised nervous system of these rachitic patients; so that the presence of rachitis is a predisposing factor in the causation of convulsions.

In addition to this, as we shall see when discussing the pathology of eclampsia, laryngismus stridulus is a form of eclampsia affecting rachitic children exclusively. It is the result in most cases of an auto-infection caused by the absorption of toxic materials produced in the stomach and bowels of rachitic children. This toxæmia thus produced, acting upon the extremely irritable nervous centres of rachitic children, initiates a central irritation passing to the motor nerves of the larynx.

It would, however, appear that such a selective action of the toxic products of the blood upon the central origin of the laryngeal motor nerves must necessarily be initiated and aided by an irritation passing from the respiratory tract, of which the glottis is the entrance, to nerve centres. Such an irritation is found, first, in the catarrhal conditions affecting the respiratory tract of rachitic children; second, in the malformations of the thorax characteristic of rickets; third, in the inflamed, enlarged, and frequently suppurating bronchial glands, which by pressure upon the motor and sensory nerves of the larynx, initiate or aid an irritation which finds its peripheral expression in an attack of laryngo-spasm.

Symptomatology.—A typical attack of eclampsia may be divided into two stages: First, the prodromal stage; second, the convulsive stage.

It is necessary to state that in many cases the first stage is entirely absent, and the second is so brief as to be scarcely noticeable. Many authorities have considered that the symptoms described under the head of premonitory symptoms are simply the marks of the general disturbance, of which the eclampsia is the result. Thus

restlessness, a premonitory symptom to which all writers draw attention, may simply be a sign that there is something wrong with the little patient, without really foreshadowing a convulsion. There can be no doubt, however, that many children who are about to have a convulsion show this by a certain aggregation of phenomena which, to the anxious mother who has already experienced a convulsion in her infant, are the mutterings preceding the coming storm. In one variety of cases the child, which has been in good health, is observed to be feverish. There is a flush in the face, or the countenance may be exceedingly pale. The sleep is restless, the child starts, it grinds its teeth. As it lies dozing it may smile occasionally, or there may be a convulsive twitching of the lips and muscles of the face. The eyes are half closed, the nostrils move irregularly. It throws its arms and legs about, occasionally it sighs deeply, the breathing is irregular, although not stertorous; now and then the whole body starts violently, sometimes waking the patient and causing it to cry fretfully, to be followed again by drowsiness. These cases may be termed the somnolent cases. There are others in which the picture is an entirely different one. The little one is abnormally wakeful, it appears to be more than usually bright, the cheeks are flushed as though with hectic; the eyes sparkle, the pupils are enlarged; the muscles of different portions of the body twitch—now a finger, or arm, or leg, sometimes the eyelids, again the face—the child laughs more than usual, will not rest in the lap of its nurse, seems to prefer activity to rest, and asks to be taken from one attendant to another. The abnormal brightness and restlessness increase, until suddenly the little patient is thrown into violent convulsions. In these cases the parents will relate how bright the child had been before its spasm. There are other symptoms which may well be termed premonitory, although not so frequently observed as the two varieties just described. Thus I recall a little one who, in four out of six attacks of eclampsia (dentition), vomited a few minutes previous to its attack. There are other children who have many of the above-described symptoms without having an attack of eclampsia. In these cases the absence of the complete convulsion is due to a more than usually well-balanced nervous system, which, while yielding to the irritant to a certain extent, does not lose control of the "organ of mind" to a sufficient extent to place the body under the influence of the purely reflex nervous centres. Many cases have a convulsion without any of the above-described premonitory symptoms, or rather without any having been observed. There may not even be a cry, but suddenly the child falls into a more or less general and violent epileptiform spasm. The convulsive stage follows the prodromal symptoms after a shorter or longer period. But the prodromal stage, as has already been stated, may be entirely absent, or so little marked as to escape notice; then the convulsion proper sets in suddenly, often runs a rapid course, and the child recovers without sequelæ, so that before the physician has arrived the child appears bright, and as though nothing had happened. This convulsive stage of eclampsia exactly resembles the convulsive stage of epilepsy. Indeed, most writers have agreed that the attack as such is indistinguishable from an epileptic seizure (Reynolds, Hughlings Jackson, Niemeyer, Brown-Séquard). While this is true for a typical attack of eclampsia, as compared with a typical attack of epilepsy, we shall call attention to several points peculiar to some attacks of eclampsia when we come to consider the subject of differential diagnosis. The little patient, who may or may not have presented the premonitory symptoms above described, is suddenly heard to cry out; the cry is immediately followed by a tonic spasm of the voluntary muscles; the body is thrown into the position of opisthotonus, the head is drawn back, the veins of the neck stand out prominently; the arms are rigid, abducted, and partially pronated; the hands are tightly clinched; there is a gurgling sound heard from the throat, as though the patient were choking; the teeth are firmly closed, the face is at first pale, but gradually grows congested and dark; the pupils are dilated, the eyes being open. This tonic spasm affects muscles of respiration as well as the voluntary muscles, and it is owing to this fact (Schroeder van der Kolk) and the interference with the venous return from the brain that the other clonic stage of the convulsion is due—that is to say, the clonic spasm is the asphyxial convulsion.

Fortunately the severe tonic spasm is but a few seconds or a minute in duration; were this not so, death from asphyxia during the tonic spasm would be more frequent. Even as it is, death sometimes occurs at this stage of the spasm. In the largest number of cases the brief tonic spasm is followed by clonic general convulsions. The extremities are alternately flexed and extended, the arms are pronated, the body is often shaken, and sometimes even raised from the bed in the violence of the convulsion. The eyeballs roll, and the head is turned to one or the other side convulsively. The lower jaw is moved forward and downward, and laterally; the tongue is sometimes caught between the teeth and bitten; there are swallowing movements performed by the larynx and pharynx; even the diaphragm may take part in these clonic convulsions, giving rise to hiccough which Soltmann considers a very dangerous symptom. Consciousness is entirely abolished, the conjunctivæ can be touched without the patient wincing. During this stage the sphincters may become relaxed, so that there is involuntary passage of fæces and urine. The respiration is stertorous, the pulse frequent and intermittent. The temperature is elevated during the convulsion, even when the spasm is of non-febrile origin; in that case, however, it subsides very rapidly, which is not the case in febrile eclampsia. The clonic convulsions may last from a minute to half an hour. In this respect attacks of eclampsia differ from those of epilepsy, which, as a rule, are not of long duration. The convulsions gradually diminish in severity until they cease, although convulsive movements may still remain localized in some muscles of the face or upper extremities long after they have ceased in other portions of the body. The patients return to consciousness gradually; they are at first irritable, soon they become drowsy, and frequently fall asleep. Such is a general description of an ordinary eclamptic seizure. This single attack is followed in many cases by a repetition, either shortly after the first convulsion or at a longer interval depending upon the etiology. If the convulsion is of the febrile variety, the reduction of temperature will generally cause a cessation of the spasm; should it belong, on the other hand, to the toxic or reflex group of convulsions, the frequency will depend upon our success in combating the etiological factor. When the patient falls from one convulsion into another, giving rise to a series of convulsions, he may be said to be in a condition analogous to the status epilepticus. Such a status may continue for days. Eclampsia, as well as epilepsy, may be very slight, consisting chiefly of a brief period of rigidity followed by little or no clonic spasm. There may even be nothing but slight paleness, accompanied by momentary loss of consciousness, and almost immediate recovery. These slight attacks, however, are not so frequent in eclampsia as in epilepsy (petit mal). Localized spasm, well defined in character and unaccompanied by loss of consciousness, cannot be regarded as eclamptic; thus a spasm of one arm or one leg, or of one side of the face alone, even with loss of consciousness, especially if repeated, would point to a local cerebral disease, and therefore cannot be classed in the group of eclampsia, but belongs rather to the class of localized epilepsies.

Laryngismus Stridulus.—The condition known as laryngismus stridulus, of which we have already spoken, while eclamptic in character is peculiar when considered as a convulsion. Cases differ from each other in severity to a very marked extent. In mild cases, the attack, occurring in children affected by active rachitis, consists of the sudden onset of a peculiar crowing respiration, repeated several times. The child turns pale, a look of dread overspreads the countenance, the loud crowing respiration becomes very rapid, the expiratory efforts