being very short, the inspiration loud, crowing and whooping in character. There is generally, however, no cough after inspiration as in whooping-cough—the face becomes cyanotic, consciousness is not totally lost. The patient very suddenly regains his normal condition. Such an attack may last from a few seconds to a minute or two.

A second class of cases are of a severe character. There are sudden paleness and total stoppage of respiration, followed by phenomena similar to those described in the milder attacks. In a third group of cases the spasm of the glottis, initiated as in the first and second classes, is so severe and long in duration that the patient is temporarily asphyxiated. The face and body are cyanotic; the pupils, at first contracted, become dilated; opisthotonos, with some spasms of the whole body and extremities, is in turn followed by clonic spasm and the other phenomena of an attack of a more or less general eclampsia. Such attacks of laryngismus stridulus, however frequent they may be, generally diminish both in severity and in number, as the rachitic disease is diminished and cured. Even in the worst attacks the eclamptic attack is generally followed by the sleep of exhaustion and the patient wakes up in a normal condition. Death from asphyxia very rarely occurs. This, however, does occasionally happen and sometimes in cases in which the attacks have been very mild. The frequency of the attacks varies, some patients having very few, one every four or five days, others one a day or night, while some have as many as forty or fifty attacks of greater or less severity in the course of a day. The severity of the laryngospasm varies with the severity of the auto-intoxication of the particular case. The attacks are generally initiated by some irritation or excitement, by overloading of the stomach or bowels with food, by draughts, by cold water, by fright, indeed by any external irritation.

Sequelæ.—Although in the large majority of eclamptic attacks, of which the patients do not die, recovery is complete, leaving absolutely no sequelæ, the text-books relate a large number of possible injurious effects from eclampsia infantum. A fatal result is far more frequent in these seizures than is generally supposed. Dr. West, in his work on "Diseases of Children," showed that in London the proportion of deaths from convulsions in children under one year, as compared with deaths from other causes, was twenty-one and nine-tenths per cent.; from one to three years, about four per cent.; and from three to five years, about three per cent. Other authorities also agree that eclampsia is the most fatal condition to which children under one year are subject. It is almost impossible, however, to estimate what proportion of infants thus attacked die, since only fatal cases are reported in official statistics, while private statistics are neither sufficiently numerous nor sufficiently trustworthy to be reliable. Of children who recover, many are subject to repeated attacks, even under slight or no provocation, and it becomes a question as to whether the eclampsia so called has not turned into chronic epilepsy. Among the authorities who favor the view that chronic epilepsy is often the result of repeated attacks of eclampsia are R. Demme[15] and E. C. Seguin.[16] It does not seem at all improbable that a frequently repeated convulsion may so disturb the equilibrium of the nervous centres as to render these liable to initiate a convulsion, even under slight provocation, finally terminating in confirmed epilepsy. It is more probable, however, that the cases in which a so-called tendency to eclampsia finally terminates in epilepsy are really at the start epileptic. Some conditions frequently known to give rise to eclampsia may have been present at the time of the first attack, and a diagnosis of eclampsia may have been hastily made; such cases will be cleared up, later on, by the more or less frequent recurrence of the attacks without any cause.

Some of the more immediate sequelæ of an eclamptic seizure are ecchymoses under the skin and conjunctiva. These hemorrhages appear to be the result of the venous congestion which accompanied the tonic spasm at the onset. The violence of the clonic spasm may be so great as to tear muscular fibres or tendons, or even produce dislocations. Indeed, a case has been reported in which a fracture of the sternum was caused by the violent contractions of the rectus abdominis. Temporary paralysis of individual muscles or muscle groups often results from an attack of eclampsia; this seems to be only functional, however, for in a few hours or days power returns, and no trace of the paralysis is left. Temporary contractures may remain in the muscles that have been the seat of the greatest convulsions; thus wry-neck and various contractures have been observed to follow an attack of eclampsia. I recall a case in which the fingers of both hands remained contracted into the palm of the hand for two days after an eclamptic convulsion; but in a short time the patient recovered full use of his hands. I cannot agree, however, with those who claim that a simple attack of eclampsia, not connected with any lesion of the nervous centres, is capable of producing permanent paralysis or contracture. When a monoplegia or hemiplegia seems to result from an attack of eclampsia, I feel convinced that a careful clinical examination will point to the suspicion that the convulsion was the result of a lesion of the nerve centres resulting in convulsions, rather than that the eclampsia had resulted in the paralysis. It is claimed by those who hold the latter view that these paralyses are the result of meningeal hemorrhages over the motor area of the cortex of the brain as a result of the convulsion. It is possible that a monoplegia could occur in this manner, for even a very slight hemorrhage would be sufficient to affect the functions of a limited area of the cortex. A hemiplegia, however, produced in this manner would necessitate the presence of a clot sufficiently large to interfere with the whole of the motor area of one hemisphere; that such a hemorrhage should occur as a result of an attack of eclampsia is scarcely conceivable. Some common congenital deformities, such as club-foot, for instance, have been ascribed to attacks of eclampsia occurring in fœtal life, and resulting in contractures of the muscles, and thus in club-foot, contractured knees, etc. A careful study of these deformities will generally reveal their true etiology.[17] I am disposed to consider all cases of permanent paralysis or contracture (primary or secondary) in children the result of a lesion of the nervous centres, of which the convulsion was only a symptom; in such cases there will generally be found some more rational method of accounting for the production of the paralysis than that which refers it to meningeal hemorrhage as a result of eclampsia.

Diagnosis.—Eclampsia may be confounded with epilepsy, and with convulsions due to disease of the nervous centres. From epilepsy it can be distinguished by a careful examination into the previous history. The older the child, the more often the convulsions have occurred without any recognizable cause, the more probable becomes the diagnosis of epilepsy. In this connection it must not be forgotten that attacks that were originally eclamptic may, after a time, when the convulsive habit has been acquired, become epileptic. Such children must be carefully watched, and should any suspicion of the presence of chronic epilepsy arise, the patient must be treated accordingly.

As far as the attack of eclampsia itself is concerned, there is no way of distinguishing it from an ordinary attack of epilepsy. Convulsions due to disease of the nervous centres have certain peculiarities which distinguish them from ordinary attacks of eclampsia. One important characteristic is that these convulsions are apt to be localized, especially if due to chronic brain disease; lesions of the various motor centres will give rise to convulsions limited to portions of the body supplied with nerve force from these centres. In acute diseases of the brain and spinal cord, and the meninges, there will be other symptoms which will lead to a diagnosis of the disease, of which the convulsion is only one of several manifestations. A differential diagnosis of the different varieties of eclampsia infantum will depend upon a careful physical examination of the patient. If there be fever or toxæmia, they should be recognized together with the

diseases which have produced them. If the convulsion appears to be due to neither of these causes, we should carefully search for some possible peripheral irritant, and, if possible, remove it. Accuracy of diagnosis is apt to depend in these cases upon the broadness of our knowledge of diseases and conditions of infancy. To attempt to treat a case of eclampsia infantum without at least an attempt at an etiological diagnosis, is to submit the patient to the most injurious form of empiricism. The convulsion is a symptom of a disease which the physician must detect. We must search for the pneumonia, the diphtheria, the scarlet fever, the cause, and treat that at the same time that we treat the symptom. From tetany, eclamptic attacks can be distinguished first by the absence of loss of consciousness in tetany and the presence in the latter condition of drowsiness. The presence of Trousseau's symptom will also serve to distinguish the cases of tetany. This consists in the observation by the clinician that in tetany an attack of spasm can be produced by compressing with the fingers the point of emergence of motor nerves from bony canals. The differential diagnosis from hysteria is hardly a factor in young children. Laryngismus stridulus can be differentiated from whooping-cough by the character of the attack and the absence of the short, broken, and rapid cough which follows the long-drawn inspiration of whooping-cough.

Pathology.—Eclampsia infantum, being rather a symptom than a disease, has no pathology. It is true that morbid changes have been found in the nerve centres of children who have died of an attack of so-called eclampsia, but the mere fact that these changes were found precludes the diagnosis of eclampsia, which is essentially a convulsion that does not result from any grave lesion of the nervous centres. It is possible that repeated attacks of eclampsia result in subtle changes in the nerve centres, but if this be the case the changes are not discoverable by any method of examination practised in the present stage of pathological investigation.

Concerning the physiological mechanism by which an attack of eclampsia is produced, it will be proper to say a few words. The subject belongs of right under the head of epilepsy, as it is in connection with epileptic convulsions that it has been more thoroughly studied. Clinically, we have already stated, the eclamptic paroxysm in no way differs from an epileptic attack. The mechanism by which the latter is produced is stated by Reynolds,[18] in his "System of Medicine," as follows:

"The derangement consists in an increased and perverted readiness of action of these organs (medulla and vaso-motor system of nerves), the result of such action being the induction of spasm in the contractile fibres of the vessels supplying the brain, and in those of the muscles of the face, pharynx, larynx, respiratory apparatus, and limbs generally. By contraction of the vessels the brain is deprived of blood and consciousness is arrested; the face is or may be deprived of blood, and there is pallor; by contraction of the muscles which have been mentioned there is arrest of respiration, the chest walls are fixed, and the other phenomena of the first stage of the attack are brought about.

"The arrest of breathing leads to the special convulsions of asphyxia, and the amount of these is in direct proportion to the perfection and continuance of the asphyxia.

"The subsequent phenomena are those of poisoned blood, *i.e.*, of blood poisoned by the retention of carbonic acid, and altered by the absence of a due amount of oxygen."

These are practically the views held by Kussmaul and Schroeder van der Kolk,[3] as to the manner in which a paroxysm is produced; the causes of the primary derangement of the nervous centres will vary with the cause of the convulsions: in one case it is elevated temperature, in another toxæmia, in a third a peripheral nervous irritation, and finally the changes and phenomena due to rachitis.

As I have already stated, only such cases of rachitic eclampsia as are due to a toxæmia depending upon auto-intoxication and the special cases, already considered, of laryngeal spasm, are properly eclampsias. Convulsions due to gross lesions of the brain and of the cranial bones cannot be regarded as eclampsias but rather as symptomatic epilepsies. Thus, for instance, the condition of the skull, known as craniotabes, so frequent in rickets of very young children, renders the brain very much subject to external pressure owing to the pliability of the cranial bones. Convulsions due to such pressure over the motor cortex of the brain are localized epilepsies due to craniotabes. Convulsions of a general character due to hydrocephalus, which is one of the results of rickets, would also be excluded from functional eclampsias, being due to an organic brain lesion. Osteophytic hypertrophies of the inner tables of the skull, causing localized pressure over the brain structure, especially over the motor areas of the cortex, might produce convulsions which would be of the Jacksonian type and not eclampsias, such as we here consider. The general convulsions, however, due to the blood changes resulting from the auto-infection of rickets, as well as such as result from the chlorotic condition of the blood (lower percentage of hæmoglobin, diminished red blood globules) found in rickets, and those due to febrile, reflex, or toxic causes, slight in character and yet producing convulsions owing to the more than usually excited irritability of the nervous centres in rachitic children, such convulsions are of right classed as rachitic eclampsia.

As to why convulsions occur in febrile eclampsia is best understood by discussing some important points in the pathology of fever. Fever itself is a nervous phenomenon, the effect of a primary derangement of the nervous system. Normal temperature is dependent upon the maintenance of a proper proportion between heat loss and heat production. The mechanism which controls this proportion resides in the central nervous system. In fever this mechanism is disturbed. Observers have sought to localize the portion of the nervous system in which this mechanism resides. There is a large literature upon this subject, and although much work must yet be done before we shall know as much of the localization of heat centres as we do of motor-nerve centres, or of the origin, course, and distribution of thermic nerve tracts as we do of motor tracts, yet this much has certainly been established that the heat function of the body is under the control of large nervous centres chiefly located in the brain and probably distinct from the motor centres. The thermic nerve tracts, although separate in the brain from the motor-nerve tracts, are closely connected with the latter at the medulla oblongata.

It would appear, then, that an attack of eclampsia, occurring in the course or at the outset of a disease of such a character that the accompanying elevated temperature is apparently the causative element, so far as the convulsion is concerned, calls into activity two sets of nerve centres—the thermogenic centre or centres, and the convulsive centre or centres.

Hughlings Jackson has shown that what he calls a "discharging lesion" of any portion of the brain may initiate the action of the medulla as a convulsive centre. We do not know the nature of such a "discharging lesion": it leaves no trace which can be demonstrated at the post-mortem table. It may be excessive hyperæmia, or anæmia, or some minute change in structure. We have no means at present of demonstrating the lesion. A similar alteration or lesion occurs in the thermogenic centre. Its effect is fever or rise of temperature; secondarily, it throws into activity the unstable convulsive centre, if I may so term it, in the medulla, especially in infants, in whom, as has been seen, the medulla, together with the motor centres, is in an exalted state of irritation. Furthermore, it is probable that, in the medulla, the motor and thermic nerves are intermingled. What more natural than that an irritation of the one results in an irritation of the other? Of the probable correctness of this hypothesis we have, however, another proof. The same reasons that would lead us to expect that the irritation

of the fever centres should, in proper subjects, cause an irritation of the convulsive centre would also presuppose the possibility of a reversal of this process. In other words, we should expect the activity of the convulsive centre to produce a secondary activity of the fever centre. This is certainly fully demonstrated by practical experience at the bedside. All authorities are agreed as to the enormous rise of temperature which occurs in the course of a so-called "status epilepticus." The epileptic falls into convulsions, repeated at very short intervals, so that the patient does not recover consciousness for hours, or even days. Shortly after this condition has set in the temperature begins to rise, and sometimes reaches an enormous height. Bourneville has seen it as high as 107¼° F.; Reynolds reports equally high temperatures.[19]

Prognosis.—The prognosis of an attack of eclampsia resolves itself into two elements: first, as to the fatal or non-fatal termination of any attack, and secondly, as to the recurrence of the convulsion. Although many infants die in an attack of eclampsia, yet, when we consider the great frequency of such attacks, those that die are comparatively few in number. The prognosis in this respect does not depend upon the severity of the seizure; very severe convulsions will often last but a short time and terminate in complete recovery. The duration of an attack of eclampsia is a more important factor in the prognosis. A series of convulsions of long duration, with short intervals between the individual attacks, renders the prognosis grave; and yet children sometimes recover after lying in "status convulsivus" for one or two days. Convulsions in which one side appears to be far more affected than the other are much more apt to be fatal than those in which the difference between the two sides is not so marked. A fatal termination, as well as a repetition of attacks of eclampsia, will often depend upon our inability to remove the cause which lies at the root of the derangement of the nervous centres. An attack of eclampsia may be repeated either within a short time after the original attack, and is then probably due to the same cause as the original convulsion; or after the lapse of weeks and months, and must then be studied with reference to its etiology as an entirely new seizure. If after an eclamptic seizure a child recovers completely and returns to its normal condition, and especially if the cause of the convulsion has been surmised and removed, the prognosis is good, the convulsion will probably not recur; it is best, however, even in these cases, to be guarded and prepare the patient and friends for a recurrence, rather than the contrary. In the large majority of cases, however, and above all in those cases in which the cause of the convulsion cannot be removed or is unknown, the convulsion is apt to be repeated; the more frequently the convulsion is repeated during a limited period of time, the more unfavorable the prognosis as to recovery. The convulsions are more apt to be multiple in the peripheral and toxic variety of eclampsia than in the febrile. The reason of this is obvious, inasmuch as fever is a condition which, temporarily at least, may be removed, while the former two do not so readily admit of treatment. A child that has already had attacks of eclampsia is far more liable to repeated returns of convulsions, under the influence of proper exciting causes, than one that has never had a convulsion. In the former case the nervous system has already shown its instability, and hence our distrust. In conclusion I would repeat, the prognosis depends to a great extent upon our ability to recognize and treat the cause. In rachitic eclampsia, while the rachitic disease is on the increase, the liability to eclampsia is on the increase. When the rachitis is cured, the eclamptic attacks as a rule no longer recur.

Treatment.—In discussing the treatment of eclampsia infantum, we would repeat what has been urged concerning the necessity of a careful differential diagnosis as to the etiology of the convulsion. We should distinguish whether the convulsion belongs to the febrile, the toxic, or the peripheral group of convulsions. If it belongs to the former, the abnormally elevated temperature should be reduced as speedily as possible. For this purpose rapid and certain means should be employed. Cold sheets, sponging with equal parts of cold water and alcohol dilutus, cold baths, Ziemssen's baths—all of these are excellent methods of reducing the temperature by external means, and our choice should be governed by general principles. Among antipyretic drugs, the best, most trustworthy, and most rapid antipyretic is antipyrin, in proper doses and at proper intervals. I am accustomed to give to children, a year old, gr. v. every hour until two doses have been taken, and to repeat the dose in six hours if the temperature shows a tendency to rise again. The object being to reduce temperature, the drug must be used in sufficient quantity to effect our purpose. If the convulsion be of the toxic variety, the indication is as far as possible to get rid of, or diminish, the toxæmia of the blood; the kidneys, the bowels, and the skin, all the excretory channels, should be brought into play. An enema, together with a cathartic, should be administered; a mustard bath should be given to produce sweating and to make the skin act vicariously for the kidneys; should these organs be at fault diuretics should be administered if not contraindicated by the general disease. Should the convulsion depend upon a peripheral irritant, it must, if possible, be removed. If the stomach be overloaded, an emetic should be administered; if constipation appears to be the cause, the bowels must be emptied. If dentition is the cause, and the gums are swollen, lancing is indicated; the swelling will thus be diminished, the teeth will be more easily cut through, and the irritation removed; if helminthiasis is at the root of the evil it must be treated. Whatever the peripheral irritant may be, it should be removed. There will be many cases that cannot be classified, which must be treated on general principles. In addition to treating the etiological element in any attack of eclampsia, it has been the custom of physicians to administer a class of drugs known as antispasmodics. The most important of these are the bromides and chloral hydrate, and during the convulsions amyl nitrite and chloroform. The bromides should be given in comparatively large doses to produce a beneficial effect. Thus, I am accustomed to give gr. vi. to gr. viij. every three hours to children one or two years old; even larger doses should be given if necessary until the convulsions cease and sleep is produced. Chloral hydrate may be used in small doses, either alone or in combination with the bromides. I have used the amyl nitrite, a few drops inhaled from a handkerchief during the convulsions, with but little success; I have been unable to convince myself of its utility when thus presented to infants. Should the convulsions be very severe and continue for a long time, chloroforming the little patient may be tried, although in cases severe enough to require this measure the convulsions are apt to return as soon as the effect of the chloroform passes away. In cases in which the bromides must be administered during a convulsion, it is best either to inject the drug per rectum or subcutaneously in solution. When given by the rectum the dose should be at least twice as large. I have always considered the application of cold cloths or ice-bags to the scalp as superfluous in ordinary eclampsia; if they accomplish any good at all it is only in cases in which reduction of temperature is an element in the treatment, and in these cases we have more effective means for accomplishing this result.

For the attack Trousseau advised compression of the carotids, either one or both, until the convulsions subsided. The carotids are compressed opposite the thyroid cartilages, by pressing the artery against the spinal column. The corresponding side of the face thereupon becomes pale and then cyanotic. In most cases the convulsive movements gradually cease and the patient falls asleep. Should the convulsion not cease the compression must be stopped as soon as the cyanosis is extreme. The compression may be repeated at quarter- to half-hour intervals; should it then not succeed in checking the convulsions we should not advise its repetition. In conclusion, it must not be forgotten that, whatever may be the disease of which the convulsion is a symptom, our attention must be mainly directed to the treatment of it, and not to the treatment of the convulsion alone.

Henry W. Berg.

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ECPHYMA GLOBULUS.—A name given to a contagious skin disease that manifested itself in Ireland during the famine years of 1847–48 and called by the Irish peasantry "Button Scurvy." Frazer, who described the disease, thought that it resembled frambœsia or yaws of the West Indies. It seems practically to have died out. The eruption, according to Frazer, occurred on any part of the body except the hands and feet, the lesions starting as slight elevations or tubercles. In this stage the skin over the lesions is perfectly normal. Increasing slowly in size the tubercles become discolored, gradually soften, finally forming prominent deep-red tumors looking like raspberries though considerably larger. These persist for an indefinite length of time unless treated, but the free use of nitrate of silver causes them soon to heal.

Charles Townshend Dade.

ECTHYMA.—This form of pustulosis of the skin is not, strictly speaking, a disease separate and distinct. It is rather a secondary or consecutive lesion occurring in many and varied disorders, in which the clinical type remains fairly constant. A predilection is displayed in most cases for the extremities, the lower in particular, but the trunk and neck are often attacked.

In the beginning there is a small pustule which generally takes a rounded outline, although the character of the local injury may give it an unusual appearance; for example, linear in infected scratch marks. When fully developed, the pustule, tense or flaccid, is seated on an indurated base whose redness fades gradually into the surrounding skin. The purulent contents soon dry into a thick, dark crust, adherent, and showing on removal a superficial ulceration of the corium. The base of the ulcer is uneven, due to indolent granulation tissue and of a red or grayish color from the presence of pus. In syphilis, the crust of ecthyma becomes stratified (rupial) like the shells of crustacea, due to spreading of the lesion under its dried cover. The ulcer heals slowly and with the formation of a small scar, which is always evident when the patient is afterward stripped for examination. Development is rather slow, but the individual pustule usually runs its course in a fortnight. It may be single or be accompanied by others, developed either coincidentally or appearing in successive crops. Subjective symptoms are those of burning and pain; itching, if present, is due to previous disease.

Ecthyma gangrænosum may be separated from the mass of ecthyma and given a definite place on account of its etiology and its occurrence in young, cachectic children. The lesions are apt to appear on the buttocks, thighs, and perineum and are extremely indolent. The ulcers are of a deep red color and covered by a dirty, tenacious slough, varying in size up to that of a silver quarter of a dollar. The earliest lesion observed is a brown or dark-red discoloration, which soon becomes necrotic. In Kreibich's case there were twenty-five or thirty ulcers at one time.

Etiology.—As in other conditions, especially those of a purulent character, three factors determine an outbreak of ecthyma: first, a depraved state of health, second, a point of entrance of diminished resistance for (third) the invading organism. The disease attacks all ages and both sexes, but is more common in adult males. Lowered vitality may simply, as in vagabonds, result from insufficient nourishment, but it may be a part of infections like syphilis and tuberculosis (scrofuloderms of Duhring), or of diabetes, Bright's disease, and anæmia. The injury to the tissues is most often due to scratching commonly of louse, bedbug, and flea bites, but also in the course of the pruritus of eczema, diabetes, dermatitis herpetiformis, and senile pruritus. Localization of specific morbid products furnishes a portal of entry doubtless in the granulomata, syphilis, and tuberculosis. The invading organism is in most cases the streptococcus, inoculated by dirty finger nails, clothes, and bedclothes. The other pus cocci of course may be present. The Bacillus pyocyaneus seems to be responsible for ecthyma gangrænosum—I say seems, because it is quite possible that it is inoculated in ulcers perhaps due to another organism, from alvine discharges. At any rate, it has been regularly found by such men as Ehlers, Baginsky, and Kreibich. The infants are invariably cachectic.

Histopathology.—The pathological process begins in the corium in the form of a circumscribed area of purulent inflammation. The cellular infiltration is chiefly of polynuclear leucocytes, the vessels are dilated and congested, and the tissue elements at the periphery are separated by a serous exudate. Later, a central necrosis occurs with complete solution of fibres, vessels, and skin appendages in its area. The epithelium covering the pus collection is much swollen, owing to an intracellular as well as an intercellular œdema. Many of the cells show hydropic degeneration. By specific staining, a network of fibrin can be demonstrated at the centre. The crust is composed of fibrin filaments in whose meshes are entangled epithelial cells and leucocytes; when it separates there is left an ulceration which extends into the cutis and which heals by the usual process of granulation and scar formation. Slight pigmentation may persist for a little time.

Diagnosis.—Differentiation of ecthyma from other pyodermias is a matter of small importance as compared with recognition of the underlying condition. Impetigo contagiosa is superficial with a "stuck-on" crust which leaves on removal no ulceration; pustular eczema is a diffuse process with no epidermal destruction; furuncles show unmistakable necrotic plugs. The ecthyma of syphilis has an indurated base, its crust commonly takes on a rupial character, and the ulceration is apt to be characteristically reniform. In the absence of external factors, careful search for a causative systemic condition—such as anæmia, cachexia, and, too often, starvation—must be made. When the skin shows excoriations and no pediculi or other parasites are to be found, it becomes necessary to decide which of the itching dermatoses is at fault,—an eczema, an urticaria, a pruritus, or a dermatitis herpetiformis.

Prognosis.—The outlook is invariably good so far as ecthyma itself is concerned, but on recovery from the pyodermic condition the prognosis becomes that of the causative, underlying disease. Ecthyma gangrænosum is always a grave condition, merely, however, as a sign of deep systemic depression.

Treatment.—Soap and water, good food and tonics constitute all that is really necessary. Even the pustular syphiloderm and scrofuloderm will get well with them