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Compliments of Author

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PUERPERAL ECLAMPSIA.

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Some two weeks since I was requested by the committee to prepare a paper for this meeting, upon some subject connected with obstetrics or gynecology. The short notice, and the little time at my disposal, would, at first, have led me to decline, particularly as I was aware that reports much more satisfactory would be made by others. But while age may have its privileges, among others that of rest, it also has its duties; and, with the reminiscences of nearly half a century, during which I have been a member of this Faculty, and with the heartfelt appreciation of what the profession has been to me and conferred upon me during that time, I did not feel at liberty to withhold my mite, however small and unworthy that mite may be.

There are a number of subjects, both in obstetrics and gynecology, which, in our best text-books, are not and cannot be thoroughly worked up. This is shown in the number of text-books which are constantly appearing, and still more in the monographs which are yearly issuing from the press. There is no text-book, however modern or however full, which gives a full and complete history, in all respects, of puerperal fever, hemorrhage, or eclampsia.

I have thought that it would not be uninteresting or uninstructive, while of course making no claim whatever to originality, to bring before you some gleanings with regard to one of these most important accidents, offering you only "corn from the sheaves of science and stubble from mine own garner."

In what fitting terms can I bring to your notice a complication of labor, so sudden and startling in its attack, so terrible in its expression, so fatal in its results, until of late years so uncertain

in its seat and pathology, and so empirical in its therapeutics as to render it, through the greater part of its history, truly an *opprobrium medici?*

Startling and dangerous as are other complications of labor, there is no accident, incident to the puerperal state so frightful to behold and so distressing as puerperal eclampsia or convulsion. Never are we able to eradicate from our mind, or entirely forget the sad and deep impression made by our first introduction to the scene where this terrible malady displayed its powers. The young wife, who has just entered upon a new existence, for which her whole previous life had been but one preparative scene; who had now formed those ties sanctioned by the purest affections of the heart, to which she has been wont to look for all the joys and pleasures of her future days; who with throbbing heart and blissful expectation has felt, for the first time, the moving life within her, and experienced the powerful yearnings of maternity not yet realized; who regards the time to come as but to her, one long, bright day-dream of trust and confidence, and hope and love, here lies before you stripped of all her beauties of mind and person.

The features, but a few moments since calm and placid in all the fullness of her accomplished hopes, now distorted with pain and anguish; the frothing mouth, the wildly rolling eye, the writhing form—all betoken the struggles of the deadly enemy within, and at the same time call upon you for the exertion of your most strenuous efforts, and strike despair into the chilled hearts of surrounding friends.

They can bear to part from those nearest and dearest, whose thread of life resistless fate has severed one by one, and whose progress to the land of spirits they have watched day by day, and hour by hour; they can see the old, who have performed all the duties of life, go down to the quiet tomb, and deem it right and proper; they can commit the yet untainted infant to the care of Him who said, "Suffer little children to come unto me," not without grief, but with a sweet and soothing consolation in their sorrow. But to see one thus loving and beloved, happy and making happy, around whom all their hopes and joys are brightly clustered, thus hurried in anguish and torture to an early grave, seems strange and unnatural,—some terrible dream, they scarce can realize. "The silver chord is loosened and the golden bowl

is broken," and that not gently or with preparation, but rudely and almost ferociously.

While the symptoms and the external characters are so strongly marked as when once seen never to be forgotten, and always immediately recognized, its history has been so obscured by false pathology, by hypothetical assumption of its nature, by narrow and partial views on the one hand, and by too sweeping generalizations on the other, that for its proper understanding it requires a full and thorough review of the subject in all its aspects.

The first great necessity is to determine, definitely and positively, the entity with which we have to deal.

The pregnant and parturient woman is liable to any of the convulsive diseases which may occur in other conditions.

During pregnancy, or parturition, she may have hysteria, epilepsy or apoplexy, precisely identical with these diseased states in the unimpregnated or non-parturient, in their nature, history and therapeutics, and too often have these all been included in the consideration of eclampsia.

But under this title we embrace properly only those which, in a certain sense, are peculiar to these states, entirely different in every respect from hysteria or apoplexy, more nearly allied in its external expression to epilepsy, epileptiform in character, but without the history, the aura, or the cry of that well-marked disease.

Is it, then, a special or specific malady, found only in connection with pregnancy or parturition, and peculiar thereto?

It certainly is not; it occurs in other and widely-separated conditions, and by recognizing this fact we can more readily trace it to its cause, establish its essential seat, investigate its pathology, and thus, and thus alone, work out a rational therapeutics.

Fortunately, tolerably rare, (occurring, according to different statistics, once in 350 to 500 cases), its mortality, until the past few years has been excessive, both to mother and child, 1 in 3 or 4 of the former, and nearly 1 in 2 of the latter.

Barker found, in 1855, a mortality of 32 per cent. in cases occurring before and during labor, and 22 per cent. in those after labor. In Dorhn's collection of 747 cases, the death-rate reached 29 per cent. In 104 cases collected by Hofmeier, in Schroeder's Clinic, the mortality was 32.4 per cent. Braun makes a better showing from Vienna, in ten years, from 1869 to 1878, inclusive,

73 cases with 20 deaths (26 per cent.); still later it has fallen, under proper and judicious treatment, to 14 per cent.; according to Charpentier to 11 per cent., and I know, from personal experience, with care and judgment in therapeutic and obstetric management, can be reduced to a still lower figure.

Symptoms.—While the convulsive seizure may, and does at times, come on suddenly and unexpectedly, in most cases there are some premonitory symptoms which can be, with watchfulness, detected. This is particularly the case with convulsions during pregnancy. These are cephalalgia, pain in the head (at first it may be intermittent, later continuous), irritability of temper, drowsiness, vertigo, despondency. Again we meet with disturbances of vision, varying almost indefinitely in different cases, from mere dimness, *muscæ volitantes*, double vision, to absolute blindness. In others epigastric pain, at times very intense and acute, may precede the attack. But that which most often attracts attention is œdema of the lower extremities, the hands and the face, which should at once call forth our watchfulness, and direct attention to the renal condition.

When first occurring during labor, it may come on with the rapidity and suddenness of lightning, or for a moment the woman, in the midst of her pains, may assume a perfectly placid expression of countenance, a deceitful lull before the storm; the eyes present a far-away look, as though gazing into space.

Almost instantaneously the perfectly characteristic convulsive seizure, varying in different cases but little, save in intensity and duration, sets in with rapid contractions of the face, the eye-lids and of the eye-balls, which seem to roll in their sockets. The eyes move rapidly from side to side, or roll upward, while the pupils dilate and lose their sensitiveness to light. These twitching movements, which give to the countenance a most painful expression, presently give place to tonic contractions of the same muscles and of the neck. The mouth is first twisted toward the left, and the face is slowly turned toward the shoulder of the same side. The upturned eye-balls show, through the half-closed eye-lids, the inferior segment of the sclerotic. After being slowly turned toward the left, the face, by a movement in the contrary direction, turns toward the right shoulder. From the head the convulsive phenomena rapidly extend to the other parts of the body. The extensors of the trunk, thrown into violent contrac-

tion, tend to bend the spinal column backward (*opisthotonos*). The trunk becomes perfectly rigid; the legs are equally so and generally extended. The hands close with force, the thumb being bent inward upon the palm, and grasped by the other fingers. Occasionally the predominant action of the flexor muscles has the effect of fixing the different segments of the superior extremities in a semi-flexed position, so that the arm sometimes takes the attitude which is given to it to protect the head from a menaced blow. Finally the diaphragm and the respiratory muscles become involved. Respiration is suspended; the face becomes livid, and the tongue, if projecting from the mouth at the commencement of the fit, is seized and lacerated by the spasmodic closure of the jaws, and the blood, which escapes from the wound thus produced, tinges the saliva which flows from the lips. The muscles of the larynx, and possibly those of the throat, being strongly convulsed, close those orifices, consequently the air, compressed by the convulsive constriction of the thorax, can only escape with great difficulty, and produces a peculiar, interrupted, hissing expiration.

There is observed at the same time a complete loss of consciousness and of all sensation—the patient neither sees nor hears; and if we pinch or bruise the skin, she makes no attempt to withdraw from an irritation, of which she does not seem to have the slightest perception.

Clonic convulsions, affecting the whole muscular system, soon succeed the tonic variety. Jerking movements of the head, trunk and limbs take the place of the general rigidity of the preceding period. Frightful contortions of the countenance are the result of irregular movements of the mouth, eye-lids and eye-balls. Respiration, which up to this time is almost completely suspended, becomes gradually re-established. The expiratory act is interrupted and stertorous; a frothy and often bloody foam is forced from between the lips; the movements of the trunk and limbs consist generally of twitchings, so trifling in extent as merely to move the body without displacing it, so that there is not the same necessity for restraint as in some other convulsive diseases.

The pulse, if strong and full at the commencement of the fit, is rapidly accelerated under the influence of the muscular and respiratory disturbances, and becomes extremely feeble toward the height of the paroxysm.

It sometimes happens that the contents of the bladder and rectum are voided during the fit, either by paralysis of the sphincter, as some have supposed, or by convulsive action of the diaphragm, or the muscles which form the abdominal walls, and again at times the child is suddenly and unexpectedly expelled from like causes.

As the fit passes off all these symptoms decline; the balance of the respiratory and circulatory functions is restored; the color of the surface becomes natural, the movements of the trunk and limbs become feebler and less frequent, and finally cease.

In a word, the convulsive manifestation of eclampsia may be divided into two distinct periods: The first, which is characterized by tonic convulsions, seldom lasts more than 15 or 20 seconds; the second period, that of clonic convulsions, lasts much longer—from one to two minutes—seldom more.

The gradual restoration of the respiratory function during this second period prevents any special danger to life, and it is, therefore, during the first, or tonic period only, that there is immediate risk.

When the fit has entirely ceased the patient remains in a comatose condition, the depth and duration of which are in proportion to the intensity of the paroxysm, so that she may regain consciousness in a few minutes, or after the lapse of many hours. A dull languor, or a compressed feeling, with head-ache, is then very generally complained of, and it may thus be some time before she completely recovers. This is, of course, supposing she has but one attack, or that a considerable interval occurs between them.

In extreme cases the tonic phenomena are such in intensity and duration that the patient's life is at once sacrificed; and in those cases in which the fits succeed each other with great rapidity, she has, as it were, no time to regain her consciousness, and she remains in a condition of complete coma, which is only disturbed by the recurrence of the dreaded paroxysm, and which persists until the case terminates either in recovery or death (Braun).

Now what is this picture? What is the self-evident character of the attack?

It is essentially *irregular, inco-ordinate, uncontrolled muscular spasm*, which is of itself the very essence of convulsion.

If this be so, and it cannot be otherwise, are we to stop here? The muscular system, while it gives expression to the disease,

cannot of itself be the morbid entity. We have, as we have seen, complete loss of consciousness and sensation, and with this general spasm of the whole muscular system of animal life, and of that of organic life also.

We must look, then, to that portion of the organism which controls and calls into play this muscular system. This can only be the nervous system, and we must seek the portion of the latter which may and can alone be its seat, and search for its condition, and for the causes which produce and light up its perverted expression.

Thus if we wish a plain definition of puerperal eclampsia in a few words, we can find none better than that of Braun: "*Eclampsia puerperalis is an acute affection of the motor function of the nervous system, characterized by loss of consciousness and of sensibility, by tonic and clonic spasms,*" and if he had stopped here he would have covered the whole ground clearly; but taking too narrow a view, he goes on: "It occurs only as an accessory phenomenon of another disease, generally of Bright's disease, in an acute form, which, under certain circumstances, spreading its toxcemic effects on the nutrition of the brain and the whole nervous system, produces these fearful accidents."

If we concede that there is no other cause for eclampsia than albuminuria, then the above is perfectly true. But while we know perfectly well that in a large proportion of cases, as we shall hereafter see, the renal condition is the exciting cause of the seizure, we are equally well aware that in many instances this is not the fact, and even where it is, it does not in itself, and of itself, thoroughly or sufficiently enlighten us as to its pathology. It does not establish the pathological seat, nor does it give us the direct indication to the therapeutics of the seizure.

Where, as is the fact, we constantly meet with eclamptic attacks, whose symptomatology is identical with these, whose course and natural history cannot be, by any means, distinguished from the former, and whose results are precisely the same, but where there is no renal trouble, it would be folly to say, with many of the followers of Braun and others, that only those cases which depended upon albuminuria were true eclampsia, and all others were false or spurious.

No one, obviously, can take a clear and comprehensive view of the pathology of puerperal eclampsia who does not freely admit that there are cases in which no uræmic poisoning exists.

For a long period of time it was believed and taught that cerebral congestion was its true pathological cause, the theory being supported, to some extent, by the appearances of external congestion, by the apparent results of *post-mortem* examination, without determining whether the condition of the brain after death was the cause or the effect of the fit, and by the curative results at times of venesection, sometimes cutting it short utterly and entirely.

In 1842 Dr. J. C. W. Lever (Guy's Hospital Reports), reported 14 cases of eclampsia, in 10 of which he examined the urine. Albumen was found in greater or less quantity in nine (9). These were followed by like observations by Simpson, Cormack, Garrod and Rees, in England, and in France by Cohen and Delpech, by Desvilliers and Regnault.

The similitude between these attacks and the seizures in Bright's disease, known as uræmic convulsions, necessarily at once attracted attention. When Braun and Frerichs, in 1851, published their cases, and when still further, in 1857, Braun brought out on this subject, his most meritorious treatise, the whole drift of professional opinion was at once established in this belief, and though Babington, Bright, O'Rees, Christison, Frerichs, Schöttin, Hoppe, Segallas, Gallois, Brown Séquard, Claude Bernard and Oppolzer proved that urea is inoffensive, and the theory of Wilson (who first in 1833 created the word uræmia), was overthrown by the experiments of Claude Bernard, who, by injecting urea into the veins without producing convulsions, showed that urea was incapable of producing the nervous accidents of albuminuria and eclampsia, yet even to the present hour a large part of the profession still hold to the terms uræmia or uræmic convulsions.

While the excess of urea in the blood during eclamptic attacks has been proved by analysis, recent researches upon the temperature in eclampsia overthrow the theory of uræmia, for the temperature in uræmia, progressively and notably lessens, while in eclampsia, on the contrary, it is generally elevated, and that continuously. We may also add that in cholera, where we find an enormous quantity of urea in the blood, we do not observe eclamptiform convulsions.

Even Frerichs was forced to see that urea was not the essential poison in the blood, and assumed that there was some ferment in the blood by which the urea was converted into carbonate of

ammonia, and thence termed this condition ammoniæmia and the convulsions ammoniæmic, which was, however, completely disproved by the experiments of Claude Bernard.

While, then, it is true that the condition or constitution of the blood in and dependent upon albuminuria is one, and one of the most frequent and important causes of convulsions, we cannot trace it to urea or ammonia; it may be other constituents of the urine, which with this, retained, produce the result, hence the condition has been aptly termed urinæmia.

In opposition to this narrow theory of uræmia or urinæmia, as the direct essence of eclampsia, we have later the classical experience of Seyfert, of Prague, Director of the Maternity Hospital, second only in size to the great Maternity of Vienna.

Seyfert states specifically that—

1. Convulsions may occur without albumen.

2. The albuminuria is in many cases the effect and not the cause of the convulsions.

3. In many fatal cases the kidney lesions are almost or wholly insignificant.

4. Convulsions are rare in chronic Bright's disease, which had existed prior to pregnancy.

5. In true uræmia, such as necessarily is produced by the suppression of urine when, in uterine cancer, the ureters are invaded, convulsions do not occur.

This last position has since been confirmed by the pathological investigations of Cornil and Ranvier. In a very large proportion of women who had died from uterine cancer, the ureters were found occluded, with attendant dilatation, and in somes cases with hydronephrosis. The histories of these patients showed that in not one instance had convulsions taken place.

Seyfert reports over 70 cases where women suffering from Bright's disease became pregnant. Only two of these had convulsions. Of 46 cases, chronic in character, reported by Hoffmeier, only one-third of the patients had eclampsia. Including acute and chronic cases (Bright's) together, Braun himself estimates that only 60 in 100 develop uræmic convulsions. Bamberger reports 152 cases of Bright's disease in puerperal and pregnant women, viz.: 80 acute cases, 56 chronic cases and 16 cases of atrophy. Puerperal convulsions occurred in only 23. These facts are still further supported by others. Imbert Goubeyre

found that out of 164 cases where albuminuria was developed during pregnancy, 95 had no eclampsia, and Blot, out of 41 cases, found that 34 were delivered without untoward symptoms. It may be taken as proved, therefore, that albuminuria is by no means necessarily accompanied by eclampsia.

Cases were also observed in which the albumen only appeared after the convulsions, and in these it was evident that the retention of urinary elements could not have been the cause of the attack, and it is highly probable that in these the albuminuria was produced by the same cause which induced convulsion. Special attention has been called to this class of cases by Braxton Hicks, who has recorded a considerable number of them.

He says that the nearly simultaneous appearance of albuminuria and convulsion—and it is admitted that the two are almost invariably combined—must then be explained in one of three ways:

1. That the convulsions are the cause of the nephritis.

2. That the convulsions and the nephritis are produced by the same cause, *e. g.*, some detrimental ingredient, circulating in the blood, irritating both the cerebro-spinal system and other organs at the same time.

3. That the highly congested state of the venous system, induced by the spasm of the glottis in eclampsia, is able to produce the kidney complication.

We thus find that albuminuria or renal insufficiency cannot be considered as the sole cause of eclampsia, and even if it were, whether the original or the exciting cause of the seizure, that it would not give us its anatomical seat or its essential nature.

For these we must trace it still further back, and as I before said, we must look to the nervous centres for its seat and its essence.

Another theory, which has much to support it, both in physiological experiments and clinical observation, is that which refers the seizures and their cause to anæmia. We know perfectly well, that in animals bled to death in the shambles, convulsions, epileptiform in character and similar to those of which we are speaking, are almost invariably present, and we are equally aware that eclamptic convulsions occur before or at the moment of death, in cases of fatal puerperal hemorrhage. During the period when venesection was

employed so persistently and so profusely in English practice in nearly all forms of disease, as almost to justify the opprobrious epithet of *vampyrism* on the part of their continental confrères, both Travers and Marshall Hall report, that after excessive bleeding convulsions were by no means uncommon.

In addition, as we shall hereafter see, our therapeutics afford us an *experimentum crucis*, with regard to the actual condition and the effect of treatment, in cases where benefit, more or less decided, has followed the use of the lancet, but where a repetition *coup sur coup* of the venesection is at last by the anæmia produced, followed by its aggravation, and the very remedy which at first may have been curative in its effects is, if too far pushed, at last lethal-

The experiments of Kussmaul and Tenner unequivocally established the fact in the lower orders of animals, that anæmia of the cranial contents, brain and medulla oblongata, whether produced by hemorrhage or by ligating or compressing the carotid and vertebral arteries, was invariably followed by such seizures. Brown Séquard showed that an anæmic condition of the nerve centres preceded an epileptic attack.

Following these, and observations of a later date, we have the theory of Traube and Rosenstein, who referred them solely to anæmia; adopting the results of the researches of Andral and Gavarret, they start with the hydræmic condition of the blood in pregnancy. If albuminuria co-exists, this of course increases the depravation of the circulatory fluid, rendering it still more hydræmic. Accompanying this condition of the blood, there is increased tension of the arterial system from the hypertrophy of the heart, which we know to be a normal ocurrence in pregnancy. The result of these combined states is a temporary hyperæmia of the brain, which is rapidly succeeded by serous effusion into the cerebral tissues, exerting pressure upon its minute vessels and consequent anæmia. An anæmic condition of the hemispheres would, it was predicted, produce coma; while convulsions would result if the condition extended to the motor centres. The brain changes would then be œdema, anæmia and flattening of the convolutions.

Lusk says, that although he at first favored these views, he later failed to find these changes after death, and in 19 examinations Löhlein reported these alterations in but a single case, while Spiegelberg entirely repudiates them.

The recognition of the state of the blood in pregnancy, the frequency of albuminuria and our knowledge of its influence on the blood in the same direction, the recognition of cardiac hypertrophy, and the necessarily increased arterial tension from and during the pains of labor, render this theory attractive and apparently satisfactory, and unquestionably the convulsive seizures may be and are at times thus produced. But they cannot account for all cases. The absence of the post-mortem changes so frequently, the presence in many instances, found by so many observers of cerebral congestion after death, and the existence of this latter condition (congestion) supported by the condition of the circulation, the state of the vessels and of the general system during life, forbid us to ignore the observations of Von der Kolk, Levret, Broussais, Blot, Peter and others.

In 1878 Angus McDonald reported, that in the examination of the brain in eclamptic persons, he found the meninges congested and the venous sinuses filled with blood, while at the same time there was anæmia in the deeper layers of the brain structure. The ventricles, instead of being empty, as should have been the case, according to the Traube-Rosenstein theory of œdematous swelling, were found filled with serum. In place of secondary compression, he expressed his belief that the anæmia resulted from arterial contraction, due to irritation of the vaso-motor centres.

Granted, however, as it must be, that this theory is true in some cases, although for the reasons assigned, we cannot hold it to be the universal key to the mystery, yet even then we must go, as in the previous instance, a step farther back and look to the nervous centres for their pathological seat and entity.

Still further, if we examine the old doctrine, that the albuminuria, uræmia or urinæmia was due to the pressure of the enlarged uterus upon the renal veins, thus producing mechanically renal congestion, or of still later date, the doctrine of renal insufficiency, or the views of Braun, as to the obstruction of the ureters, or of Löhlein, who found in the records of 32 autopsies made upon eclamptic women, that in 8, or 25 per cent. of the entire number, dilatation of one or both ureters co-existed with renal disturbances, and who, therefore, inquires how far simple mechanical obstruction of the ureters may explain this apparent development of uræmic manifestations in certain cases, without the warning furnished by the albumen in the urine, in each and all, it is evident, we must still go behind these to the nervous system.

Finally, in those cases in whose history we cannot find any of the preceding causes, and where, from their course, their concurrent symptoms and the result of therapeutic or obstetric treatment, we are obliged to recognize a peripheral irritation as its direct exciting cause, as we shall hereafter see, in each and every one, we are compelled to fall back upon the nervous centres, without which it will remain a mystery, and only by a knowledge of their physiological functions and activity can we alone find the light and determine intelligently their pathological seat, their nature, character and treatment.

If, as is then evident, we must look to the nerve centres for an explanation and a rationale of eclampsia, we require to determine whether we can fix upon any part or parts which alone can produce and originate the attack of themselves, and whether, while we locate the seat and character of the attack, we are forced to look behind such part or parts for some predisposing cause.

In the first place, as to the pathological seat—

The nervous centres consist of three distinct parts, intra-cranial and intra-vertebral; the brain (including cerebrum and cerebellum), the medulla spinalis, or spinal marrow, and the medulla oblongata.

The attack essentially consists, as we have seen, of loss of consciousness and sensibility, and of general, we may say universal tonic and clonic spasms.

We can at once exclude the spinal marrow proper, as we know that injuries, unless severe, to the cord, are followed by unilateral results, and if severe by paralysis, and not by convulsions. In the brain an effusion of blood in cerebral apoplexy, produces only an unilateral effect, giving rise to so-called unilateral paralysis or hemiplegia. The same is true in the spinal cord. There also unilateral lesions, or irritations, if they are not so violent as to give rise to general excitement, exercise only an unilateral effect upon the powers of motion.

It is true, that after section of the cord by Brown Séquard convulsions did follow, but not until three weeks afterward, when the irritation or inflammation, the result of the injury, had had time to travel up to the medulla oblongata, and then did not evince themselves in parts supplied with nerves by the injured portion of the cord, but first in the face, then extending to the neck, later to the respiratory muscles, and finally to the general muscular

system, thus showing that the direct seat and source of the attack was the medulla oblongata. And, further, proving the same fact, the spasms were on both sides, while the spinal and cerebral effects are, as we have seen, unilateral, and the medulla oblongata is alone bilateral in its action.

Still further, in the experiments of Kussmaul and Tenner, when anæmia of the spinal marrow was induced, it was never attended with convulsions, always with paralysis.

Now, loss of consciousness and of sensibility must of necessity be referred to alteration in the hemispheres, the non-excitable parts of the brain, lying above and beyond the excitable portions, the motor centres, the portion which is undoubtedly the seat of the mental operations—of sensation, of consciousness, of will, of judgment.

In addition, where we find the irregular, inco-ordinate, uncontrolled muscular spasms, constituting the essence of convulsions, we are forced to refer it to the medulla oblongata.

The brain centres are then all involved, either as cause or effect, but when we recollect that convulsions arise at times in the midst of the deepest coma, when we learn that certain poisons produce precisely similar seizures, which poisons affect only the spinal centre, not the brain proper, and that in those circumstances, as well as in some neuroses, these convulsions occur and continue while the functions of the brain are intact, when we see that equally can this be induced in decapitated animals, when the brain is entirely removed, we have only the medulla oblongata to which to trace their seat and cause.

Finally, in the experiments of Kussmaul and Tenner, before referred to, producing convulsions by anæmia, either by hemorrhage or by compression of the carotid and vertebral arteries, they removed part after part of the hemispheres, as far back as the posterior portion of the optic thalami and the pons, and still after their removal the same convulsive results, and in the same manner, followed their manipulations.

While, then, we refer the unconsciousness and the insensibility to the hemispheres, we are equally forced to refer the convulsions to the medulla oblongata, and then follows the question, which is the essential point and from which does the attack start?

Now, whether we examine these tissues and structures anatomically or physiologically, or base our views upon clinical observations in this disease, we arrive at the same conclusion.

The spinal marrow is comparatively simple in its construction. We have the sensitive and the motor nerves, and the medullary portion of the cord with which they are continuous, so far only conductors in the one direction or the other of sensations or impressions, and of motor power or influence, to or from the brain. In addition there is the central spinal ganglion, or cineritious neurine, wherever found a centre of nerve power, which in conjunction with the former, the nerves, constitutes the true spinal system, the excito-motor system of Marshall Hall.

In the cerebral hemispheres we have the gray, or cineritious neurine, displayed on the surface in the convolutions, with the rayed or fan-like arrangement of the medullary fibres, the conductors merely of whatever sensations or impressions may be conveyed to that great ganglion or centre of nerve power, being subjected there to consciousness, to will, to judgment, and the resulting influences thence transmitted.

But when we examine the medulla oblongata we find there an arrangement differing widely from both.

Independent of the anterior and posterior, or motor and sensitive columns of the medulla spinalis *en route* to the hemispheres, cerebrum and cerebellum, and in addition to the spinal ganglion, or continuation upward of the central gray neurine of the cord, we find here a perfectly unique organization (Von der Kolk).

The nuclei or ganglionic groups, whence the nerves arise, are here more distinct from one another; the nuclei for motion, as those of the hypoglossus, the accessory, the facial, and the small branch of the trigeminus (the fifth pair), the nucleus of the abducens (still uncertain), the nuclei for the nerves of sensation, which are first seen in the medulla oblongata, as the portio-major trigemini, the vagus, the glosso-pharyngeus and auditory, and in addition auxiliary ganglia or accessory nuclei, each of which has its own functions. We find here also a system of fibres, descending from the brain and passing into these various nuclei, or ganglia, as conductors of the orders of our will, or for the communication of the impressions of sensations to the brain. Filaments arise from the nuclei of the sensitive nerves, which convey the received impression to parts situated higher up. Besides this, there exists in the medulla oblongata a system of transverse fibres (fibræ arcuatæ), which serve to unite the two halves most inti-

mately, and to produce a bilateral action so eminently characteristic of most nerves of the medulla oblongata, and such as occurs in no other part of the body, being seen in the bilateral action of the face, the tongue, the voice and respiration.

Filaments run down to these various nuclei from the brain; filaments run up to them from the spinal marrow. They give origin, as we see, to the cranial nerves; they are intimately united together by these transverse fibres. There is no other portion of the nervous centres which offers such complexity of structure, none which contains, in a like space, so many ganglia, or centres of nervé-power, none whose functional activity is consequently so great or so varied. So great is the intricacy of the arrangement, by means of nerve fibres bringing these centres into close and intimate relation with each other, that even to the present day, and with all the labor which has been expended upon it, the most astute and industrious anatomists and microscopists have not been able to trace them thoroughly, or elucidate all their connections. But the physiologist, with the anatomist, have traced pretty satisfactorily its relations and functions, extensive and active, and consonant with this its vascularity is excessively and richly displayed. According to Von der Kolk, "Nowhere is the quantity of capillary vessels so great and presenting such a densely interwoven tissue as in the corpora olivaria. It affords one of the most beautiful capillary net-works to be met with in the system, in much greater number than in the gray cornua of the spinal cord itself. The other centres in the medulla oblongata are also uncommonly rich in blood-vessels. More arterial blood flows to and in them than in the gray horns of the spinal cord, and it is therefore evident that the vascularity and quantity of arterial blood supplied to these centres are directly related to the intensity of their action." The motor and sensitive columns of the spinal marrow enter into the medulla oblongata, and partially, at least, pass on to the hemispheres, the cerebrum and cerebellum; filaments pass up from it to the brain above, the cranial nerves arise from it, its nuclei or centres are closely united by cross-fibres, and from these various centres arise the nerves of motion — the hypoglossus, the accessory, the facial, and the small branch of the trigeminus (the fifth pair), the nerves of sensation, the portio-major of the trigeminus, the glosso-pharyngeus, the auditory, and the vagus controlling respiration.

Let us return, for a moment, to the experiment of Brown Séquard, before noticed, in which, after section of the spinal cord, convulsions followed, but not at once, only in his cases, after three or four weeks, when the consequent irritation, or inflammation had had time to travel up the cord and reach the medulla oblongata. If the left side of the cord was cut through between the seventh and eighth dorsal and the third lumbar vertebræ, it almost invariably followed, that stimuli applied to the left side of the face occasioned convulsive movements, while if applied to the right side, they by no means had this effect:—the epileptogenous zone. If only one side of the column was cut through, only one cheek was capable of exciting them, but if both were cut through, the symptoms were occasioned by irritating either one or the other cheek. The first convulsive movements which arose were confined to spasms of the face and of the eyes; some days after this first attack, the muscles of the larynx, neck and chest were affected with convulsions, and finally, the muscles of the trunk and extremities. The stimulus is here received by the second and third branches of the trigeminus, conveyed to the medulla oblongata, and thence reflected to the muscles, a true reflex convulsion. If this local irritation is severe, the head is drawn strongly by the convulsion to the one side by nearly all the muscles of the side (probably most strongly by the sterno-cleido-mastoid); the mouth is opened by the action of the depressors of the lower jaw; oftentimes a loud hoarse cry is produced by the muscles of expiration and the convulsive vibrations of the chordæ-vocales, or rather of the muscles of the larynx, which put these on the stretch.

The convulsive spasms now manifest themselves over the whole body, except in the paralyzed limbs. The head moves alternately from one side to the other (bi-lateral), so characteristic of the action of the medulla oblongata; the muscles of the face and eyes successively contract (facial); the non-paralyzed parts are thrown into violent convulsive movements; the fœces are expelled, and discharge of urine also often takes place.

We see here the exact picture, says Brown Séquard, of a violent attack of epilepsy with all its symptoms, in the order in which they occur in an epileptic patient, and we can further and equally say, of eclampsia in an eclamptic patient.

We see then from all these sources, we are forced to refer the seat of these attacks to the medulla oblongata.

No other part fills the requirement. Dieters has pointed out that in the pons exists the first *central* termination of the motor-fibres, which come from the periphery; at the floor of the fourth ventricle lie united the gray nuclei for the vaso-motor cerebral nerves; moreover, in the medulla oblongata is found the so-called respiratory centre; and finally, also the vaso-motor centre, which is concerned in the genesis of the cerebral symptoms.

Von der Kolk states the groups of gray matter for the cranial nerves are situated in the floor of the fourth ventricle and in the substance of the medulla oblongata. Nothnagel calls the medulla the "convulsive centre," and in these locations agree partially Kussmaul and Brown Séquard, entirely Echeverria and Reynolds.

Besides thus referring the convulsive seizure to the medulla oblongata, we can also find here the primary cause of the cerebral anæmia so often existing. The vaso-motor centres are present in the medulla and co-ordinate with the motor centre, and we can very readily understand how and why they should act together. The irritation while impressing the motor-centres produces the convulsions, when acting on the vaso-motor centre produces contraction of the arterial coats and consequent anæmia, most frequently together, but sometimes separately.

So, as we see, anatomical structure and physiological experiment both point to the medulla as the starting point of the attack.

You may now ask, if this be so, does pathological anatomy throw any light upon it? and are there any lesions of the medulla, which found after death, permit us positively to determine such cause in any structural alteration or organic change?

As in epilepsy, so also in eclampsia, while a great number of gross macroscopic changes and some microscopic alterations have been and are from time to time found, they are almost universally effects and not causes, and there is not a single one, which is found in every case.

If to produce the more marked and fixed disease, epilepsy, which is chronic, no disorganization, no change appreciable to the eye or the touch, even with the microscope, is necessary, how much stronger is the case with eclampsia, which is not so fixed, which is acute, which is short-lived, and which, instead of continuing for years, terminates at once either in recovery or death. We must refer it then to functional excitation of this great centre.

We are perfectly aware, that while there are certain poisons which act specifically in exciting or increasing the irritability of these centres, there are also certain neuroses which act in the same manner and produce the same effect. We can thus combine our experimental researches with the results of clinical observations.

If we poison a frog with strychnia, the animal is destroyed with tetanic convulsions. If we decapitate the animal and poison him with strychnia, any irritant or excitant applied to the surface, lights up the same convulsions, showing that the brain has naught to do with the seizure. If he be left entirely at rest, with no peripheral irritation he will die, but without convulsions; but the touch of a feather or the shaking of the table induces the tetanic attack, showing that the convulsions are reflex, and as we have seen that the spinal marrow is not their seat, and as the brain has been removed, we have only left the medulla oblongata.

As showing still further their reflex character, if we cut all the posterior or sensitive roots, or those conveying sensation or impressions from without inwards, and strychnize him, as in an experiment of Claude Bernard, we cannot induce convulsions, because the conveyers of the irritation or excitement have been severed.

This then would only be explained by an increased or exaggerated irritability, excitability or impressibility, induced in the medulla by the action of the drug.

So likewise if you take a case of tetanus, the purest of the spinal neuroses, we constantly find that we require a peripheral irritant to excite the spasm. The patient may lie perfectly still, until a movement of the bed-clothes, the opening of a door, the jarring of the bed acts upon the excitability of the medulla, not the hemispheres, for the mind remains perfectly clear and intact. It is evidently not the hemispheres which are here affected, and equally, as in the previous experiment, the convulsions are, in a large majority of instances, reflex.

Now, still further, and recollect that I have already said, there is nothing specific in puerperal eclampsia; practically, we know how frequent are convulsions in childhood, and how easily they are excited.

And why? Simply and entirely from the influence and great excitability predominant in the nervous system at that age, when precisely similar convulsive seizures are of common occurrence, on the application of a sufficiently exciting cause. They are truly

cases of eclampsia, and differ not in their character or their essence, from those we have been considering.

From all that we have said then it results—that the pathological seat is not the hemispheres (the cerebrum or the cerebellum), but the medulla oblongata, that there is not any structural lesion or organic change; such lesion could not exist, for the attack once over, the mind and body both present fully their normal condition.

We are forced then to the conclusion (already conceded by Schroeder, Von der Kolk, Brown Séquard, Reynolds and Nothnagel), that their pathological seat is the medulla oblongata; the pathological entity, the true pathology of the disease, is increased or exaggerated excitability, irritability or impressibility of this great nerve centre, which in this condition has been likened to an overcharged Leyden jar.

Now, unquestionably, and this we cannot fail to recognize, and indeed we must acknowledge it, from the continued and persistent irritation of an important class of incident nerves in pregnancy, those of the genital apparatus, there is produced an increased or augmented excitability, irritability or impressibility of this great centre, similar to that which we find to exist in the child, and which is unquestionably the pathological cause.

The medulla oblongata is the pathological seat, the increased or exaggerated irritability of its cellular or gray neurine, the pathological entity.

Now, inasmuch as we have seen this to be an excito-motor-centre, we can appreciate and are forced to recognize, that these convulsions may be either *centric* or *eccentric*—in other words, in the convulsion we have to consider two factors—the nervous centre, with its augmented excitability, and an irritant or excitant, which may be applied either directly to the centre itself, or through some of the incident or excitor nerves.

The centric causes must then be intra-cranial. Any condition which may mechanically or physiologically excite such centre, would act as such cause. Thus, as before said, congestion of the cranial vessels, whether by merely increased vital stimulation from increased amount of blood, its true physiological excitant, or from pressure from over-distended vessels, may thus induce the fit.

We know positively, from what has been before said, that the precisely opposite condition, or anæmia, often acts in the same manner.

Now it may appear paradoxical that two directly opposed conditions—too much blood in one case, and too little or impoverished blood in another—shall produce precisely identical results. But we must bear in mind, that these nuclei have but one mode of expression; the vaso-motor centre, to induce contraction of the arterioles, whence the cerebral anæmia, the respiratory centres to control respiration, and the motor centres, to send out the motor influence and produce muscular contraction, which, when inco-ordinate and uncontrolled, constitutes muscular spasms or convulsions. They have but the one mode of expression, and, therefore, when irritated, it matters not what the character of the irritant, it can only act according to its kind.

Now, not only may congestion or anæmia thus act, but we can equally and readily understand that blood which is poisoned, which is loaded with excrementitious matter may produce the same effect.

From the alterations in the secretions during pregnancy, from the constipation almost natural and habitual, from the interference with the respiration, from the pressure upward of the diaphragm, and still more from the fact, that the excrementitious results of fœtal nutrition make their way into the maternal blood, to be thence excreted, we have a fruitful source of blood depravation. We recognize asphyxia as a fruitful source of convulsions, the blood becoming loaded with an excess of carbonic acid.

But of all these causes of blood poisoning there is none so frequent, none so important as those cases of albuminuria, with uræmia or urinæmia, to which I have before so fully referred, and in which whether we term it uræmia, ammoniæmia, or urinæmia, whether the result of albuminuria or only of renal insufficiency, they act as such centric cause, irritating by their noxious properties these centres. The changes in the blood in the inception of various fevers may also thus act.

These causes are purely physical, but we can readily perceive what clinical observation has again and again proven, that psychical causes, whether joy, or grief, or shame, may and do act in the same direction and manner.

Besides these centric causes, we have the peripheral causes, or causes of eccentric convulsions, which are equally or even more numerous. Of course, the most frequent and important of this

class, where the irritation is first impressed upon the incident excitor nerves, and by them conveyed to the medulla, where they produce their effect, we refer to the generative organs, the uterus and vagina. We find this last cause evident to us in many cases, and not to be mistaken. The greater frequency of eclampsia in primipara points decisively in this direction, which is abundantly proved by the relief, partial, or it may be entire, which so often follows the completion of delivery. According to Braun—"after delivery in thirty-seven (37) per cent. the convulsions cease entirely, in thirty-one (31) per cent. they become feebler, while in thirty-two (32) per cent. they continue with undiminished severity." We see it in the greater frequency and intensity of the attacks in many instances, when and while the head of the child is traversing the pelvis after leaving the uterus, and is pressing upon the vaginal walls, which are far richer in their supply of incident nerves, than are the walls of the uterus. We see it in cases of hydramnion, and also, in ordinary cases without this complication, where after all other means have failed, I have more than once completely arrested the seizure, by simply rupturing the membranes, and evacuating the waters; we see it as the result of the irritation from a dead fœtus; we see it where the convulsions having once occurred, the slightest irritation of the organ has been followed by their recurrence or their increase. We have in this connection the classical case of Denman, who says: "When the os internum began to dilate, I gently assisted during every pain, but being soon convinced that this endeavor brought on, continued or increased the convulsions, I desisted, and left the work to nature;" and a like instance is related by Dr. Heming; we see it still further where it has attended the operation of turning; we see it where, as has not infrequently occurred, it has been produced by the attempt to remove the placenta. Dr. Ingleby, relates one of this kind, "where at the moment when the operator's hand had reached the organ, my own hand making counter-pressure on the abdomen, the patient became violently convulsed, and died in less than a minute." In still another, Dr. Ramsbotham states: "The moment I had passed my hand completely into the uterine cavity, the patient turned upon her abdomen, and without uttering any expression of pain went into a convulsion." Spiegelberg has frequently seen convulsions awakened in the placental period by the mechanical irritation of the uterus during the employment

of Crédé's method of expression; and Tyler Smith says: "He has seen it more than once produced by the careless and improper use of the binder after labor." In the same category of eccentric causes, or causes due to peripheral irritation, we must also study the contents of the cranium. While we have already spoken of centric causes and their mode of operation, we must also recognize and acknowledge that irritation of intra-cranial nerves may also thus act, as from the exostoses, at one time thought to give us a clue to the whole disease, meningeal inflammation, etc. So we must study the cranial contents, as both centric and eccentric causes; under different circumstances, as a reflex as well as a direct cause.

As we find in children, so also here, when the excito-motor system, from the stimulus of pregnancy, labor, or the puerperal state, is in the condition of increased irritability, which I have described, and which is universally acknowledged, the stomach loaded and oppressed with a full meal, or with indigestible food, or the bowels irritated with indurated fœces or other cause, may be the immediate exciting cause of the seizure.

In all these we see the *modus operandi*, and we can only bring the isolated facts into a consistent whole, we can only determine its etiology, its nature and character, its pathological seat and entity, by referring to the medulla oblongata.

We have the increased irritability, excitability or impressibility of that great centre, due to pregnancy, labor, or the puerperal state, when, by the action of any stimulant or irritant, centric or eccentric, the convulsion is lighted up.

Treatment.—If this be so, and I do not see how we can come to any other conclusion, or fail thus to render all the isolated facts bearing upon it, whether anatomical, physiological or clinical, it renders the treatment perfectly plain and precise.

As we have been forced, by direct observation, to divide these cases according to their exciting cause into *centric* and *eccentric*, so we must study here two great classes of remedies: those which act upon the nervous centre and those which act upon the seat of the peripheral irritant.

Of all sedatives to the nervous centres, in appropriate conditions, there is not one which is so direct, so immediate and so powerful as blood-letting, V. S. As in the case of all therapeutic means which are active and positive, as powerful as they are for

good, when properly timed and administered, equally powerful are they for evil when improperly used.

When venesection, as was once the case, was considered our sheet-anchor in this disease, while in some instances its use was followed by the most beneficial results, it must be confessed that the rate of mortality was much greater than it has been since it has fallen into general disuse. The disease was treated, as it were, by name; it was thought to be ever due to cerebral congestion, and, therefore, if it was continuous, the lancet was used again and again. I believe there are cases in which it is more powerful for good than any other remedy. Where we have all the evidences of active congestion, the swollen, livid face, the turgid neck, the deeply-congested eyes, the cord-like tension and fullness of the external veins, and the full, hard, resisting (sledge-hammer) pulse, he must be a bold man, indeed, who would withhold the lancet, if he had ever seen it used in such a case. And the more would we be inclined to use it when the attack occurred in a woman, strong, vigorous, well-nourished and plethoric. I have used it, and seen it used too often in appropriate cases, to doubt for a moment its efficacy.

Some years since I was stopped on the street by a medical friend, who asked me if I would like to see a case of eclampsia. I told him that I would prefer never to see another. On visiting his patient I found a young woman, precisely filling the above picture, who had been having convulsions for eight hours. She had been treated with chloroform and chloral, she had a large dose of calomel followed by a strong purgative, she had enemata repeatedly, and she had been twice leeched, but without any effect. I asked if she had been bled, and was told not. I opened a vein with a free orifice in each arm, and permitted her to bleed until the lividity and congestion of face and neck were subdued, and the pulse was rendered soft and compressible, with entire and permanent relief.

We have, however, another thing to be considered here; we have seen that the brain proper, cerebrum and cerebellum, was to be studied in this connection in two phases, as the seat of certain centric causes of convulsion, and also of a set of eccentric causes, through irritation of the intra-cranial excitor-nerves.

But besides this, the brain enters into yet another relation with these convulsions. Independent of primary congestion, which may or may not exist, the convulsive attack, with the disturb-

ance of respiration, with the compression of the returning veins of the neck, by the muscles, trachelismus, and the spasm of the glottis (laryngismus), induces secondary cerebral congestion, which may be fatal. As in epilepsy, the patient, at times, dies apoplectic, so also does this repeat itself in eclampsia.

Here, also, V. S. may afford the most prompt relief, and snatch from death a life otherwise lost. We see whence the lethal arrow speeds, and meet it accordingly. It may have been, from its success in thus warding off death, that it so long retained its place as the *summum remedium* in this disease.

However this may be, I am assured, that it still has and should have its place in proper cases, in their therapeutic management. And I believe still further that *veratrum viride* which has from time to time of late years been pretty largely used, and with some success, acts in the same direction as a vascular sedative, though far inferior, and that where it has been used and with benefit, V. S. properly timed, would have been more active, more immediate and more beneficial.

While in such cases we can appreciate its advantages, we can as readily understand how, on the other hand, it would be absolutely injurious. And here again, we can see how its exclusive advocates may have been misled and induced to persist in its employment, even if injurious. Even if we adopt, *in toto*, the Traube-Rosenstein theory, that it is always dependent on anæmia, we know that then, as we have seen, we have vascular tension, and so at first V. S. may for the time, lessen this condition and give apparent temporary relief. But we equally well know, that the vessels will soon again refill by the absorption of serum, but that while the quantity of the circulating fluid may be thus restored, its quality is being steadily deteriorated, and we are rendering it more and more hydræmic.

So again, when we have in operation an eccentric cause, the lancet may for a time overcome the central irritability, but the eccentric cause remaining, the convulsions will return again and again, and if deceived and deluded by the apparent effects of the first bleeding, we repeat it *coup sur coup*, we produce or increase the anæmia, and the means which at first had a curative effect, become at last the cause of the fatal seizure, as undoubtedly, from their histories, often occurred when such treatment was pushed beyond its proper limits.

But as I have before said, under this old spoliative treatment, the mortality was terrific, 1 in 3 or 4, said by some to be exceeded in fatality, but by one other of the accidents of parturition, rupture of the uterus.

Fortunately, we have had placed in our hands, a remedy applicable to a much greater range of cases, not spoliative; and when properly used, free from danger—that giant of the pharmacopæia, chloroform. It stands at the head of the list of nervous sedatives, and since its employment, with our more precise knowledge of the pathological seat and entity with which we have to deal, the mortality has been reduced to 11 per cent., or as I before said, with skillful therapeutic and obstetric management, even below this figure.

Its *modus operandi* is evident, it sedates the nervous centres, it controls the spasms, it prevents the spasmodic closure of the glottis, with its most deleterious effects. By some, it has been used continuously, but I am convinced, we obtain all its benefits by administering it freely upon the approach of each paroxysm, and withholding it in the intervals.

But at times, its action thus is not sufficiently continuous and permanent. We may check partially the paroxysm or abate its intensity, but it will still return. We can then, and often with decided benefit, resort to chloral, preferably, and if unconsciousness persists, necessarily, per rectum, 60 grs., and repeated in two, four or six hours. But should this too fail, we are by no means of necessity foiled or without resource, for we still have the *donum dei*, in form of morphia, administered hypodermically.

I suppose this is to most of you, an old and oft-told story; but the more frequently you have seen it, the more willing will you be to acknowledge, that we do meet with cases in which we have to run through the whole category, one after another, before we succeed.

But while we are employing these *centric* remedies, it is equally imperative that we seek for any *eccentric* cause, and if possible, remove it. The catheter is said to have succeeded, when the lancet and other means had failed (Tyler Smith).

Of course, the genital organs, the uterus and vagina, are the first and by far the most important seat of peripheral irritation to demand our attention, and here, where we would *a priori* expect perfect unanimity of opinion, we find authorities on most points reliable, here widely disagreeing. Without entering here upon the question of the induction of premature labor, where eclampsia

is threatened during pregnancy, we will speak of obstetric management during the paroxysms. Gooch dismisses the subject by saying, "attend to the convulsions and leave the labor to take care of itself," and Schroeder equally curtly asserts that "especially no kind of obstetric manipulation is required for the safety of the mother."

But we cannot so summarily dispose of this, which we consider a most important practical point, and this interference or its mode must depend upon the stage of labor.

In the first place, recognizing the extreme irritability of the nervous system, and the readiness with which, as we have seen, even slight irritations elicit the convulsions, we should avoid, as far as possible, all sources of reflex irritation, and above all, unnecessary examinations or manipulations.

In the second place, although speedy delivery may be thought necessary, we should choose those which are least likely to excite muscular action. In the words of Tyler Smith, "the point to aim at should be, never to produce more irritation than we remove, and not to destroy the patient by an excessive temporary irritation instituted for her permanent relief." We should not, for instance, resort here to turning, unless in those cases where turning would be absolutely demanded, by the position, etc., if no convulsion existed, and under all circumstances, by Braxton Hicks' method, when possible. The irritation from turning would exaggerate immeasurably the dangers of the case.

If the convulsion occur in the earlier stage of labor, with the os still undilated, we would not hesitate to rupture the membranes and evacuate the waters. I have several times in consultation, when all other means had been used in this stage without avail, found the convulsions materially abated, and in a great number entirely aborted by this simple means. It is true, we may thus increase uterine action, but we lessen the size of the uterus, the pressure which it exerts, and the irritation which it excites. The partial evacuation of the organ seems to act, as does the partial unloading of the stomach by an emetic, or of the bowels by a purgative, in other classes of eclampsia.

When this has been done artificially or spontaneously without avail, and prompt delivery is required, with the os undilated, we can use Barnes' fluid dilator, with but little irritation; with the os dilated or perfectly soft and dilatable, I would at once resort to

the forceps, under anæsthesia of course, as the least irritant means we could employ, and I can unhesitatingly assert, that I have never had occasion to regret their application under such circumstances.

In an extreme case, where I deemed speedy delivery absolutely necessary for the safety of the mother, and the forceps failed, I would rather resort to craniotomy than to turning. This on the ground that the mother's life should always be preferred to that of the child, and that in this crisis we can never be certain that the child will survive; we have seen, one in two children—and by some statistics a larger portion are lost in these cases of eclampsia, and we know further that although born alive, the child is liable to die soon after birth.

Even more discrepant have been the views and opinions of obstetricians as to the propriety of inducing premature labor where eclampsia is threatened during pregnancy, or has already occurred. It is true that, at all events, until lately, if not to the present day, the majority, as to number, oppose this dernier resort, from the danger to the mother, and partly from the dread of sacrificing the child. And they add still further, that not unfrequently from the act of convulsion itself, uterine action is lighted up, and, therefore, the intervention of art for this purpose is not required.

As to the latter point, there is some show of truth. It is true that nature not rarely responds and completes the process, but we know, on the other hand, that this does not always ensue, and when it does, it may not be in time to save the life of the child, or ensure the life or the subsequent safety of the mother. In addition we know, as we have seen, how often the child is lost in these cases, and we are the more justified, therefore, in disregarding it.

With regard to the mother, we understand her danger continuing and constantly increasing while we permit this state of things to go on, and the almost certain relief which follows prompt delivery.

Albuminuria existing during pregnancy, we all know the benefits of strict milk diet (Tarnier), the advantage from attention to all the secretions, the use of the natural purgative waters, as the Freiderickshall, the Hunyadi, the employment of the Buffalo Lithia, the Vichy, the Seltzers and the Poland waters, alkaline and exerting some diuretic properties, the resort to the warm bath for action upon the skin and the administration of the tincture of chloride of iron freely for its hematinic and diuretic effects.

When cerebral symptoms once threaten we can then resort to rectal injections of chloral, or this combined with bromide of potassium, 30 grains each, to check nervous irritability.

The results of free catharsis are here often very apparent, unloading the blood of urea, diminishing arterial tension and relaxing the arterioles, by means of calomel alone, or combined with jalap, of elaterium, of podophyllin, or if there is insensibility, croton oil, by placing a drop or two upon the tongue.

If there are evidences of active congestion or full, hard pulse, swollen veins, flushed face, I would precede these means by a moderate detraction of blood.

Above all things, when this condition is threatened, we must place our patient at perfect rest, physical, mental and emotional, and at the same time abstain religiously from all digital or other interference, save such as is absolutely necessitated by the emergencies of the case. If these means suffice, then all is well, but if they fail we cannot go on repeating them, it may be for weeks, it may be months, and we must then resort to premature delivery. Indeed, we may almost take it as a rule, that when grave cerebral symptoms manifest themselves, and are not promptly averted by ordinary means, this last resort should be kept constantly before us.

Besides the uterus, however, other organs are at times the seat and starting point of eccentric or peripheral irritation, and although of far less frequency and of minor importance, should not be neglected. As in eclampsia in children, the stomach from repletion, or from the presence of indigestible substances, or the intestines from constipation and the presence of scybala may thus act, and must receive attention. If the stomach be overloaded, if the convulsion come on after a full meal, an emetic should be administered, and that of the direct class as sulphate of zinc.

Here, however, I would give one caution, that when there are evidences of active cerebral congestion, the emetic should, for safety, be preceded by V. S. Blood-letting itself will not unfrequently be followed by emesis, while if we first give the emetic, the mere act of vomiting may seriously and dangerously add to the already existing congestion.

As I incidentally mentioned before, the catheter is said to have arrested convulsions where the lancet had failed, and with a view to the coma and unconsciousness, the condition of this viscus should always be interrogated.