CRITICAL REVIEW.

Post Partum Eclampsia.

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While all other obstetric complications can now be dealt with on a scientific basis and with definite expectations of success, eclampsia still remains the great exception. Recent advances in medicine have been almost exclusively in the direction of prevention rather than cure, and in this progress obstetricians have not lagged behind. A recognition of the facts and symptoms of the so-called "toxæmia of pregnancy" has now put it within the power of every physician to treat cases presenting the early symptoms of this condition with almost uniform success; and it may be hoped that a wider acceptance of this truth will lead in the future to a great lessening in the frequency of eclampsia.

But where eclampsia has actually developed, we are still in the unhappy position of not knowing any one line of treatment which will of a surety be successful. The methods recommended are legion, but so little confidence is usually placed in any one, that as often as not several are adopted, either together or in rapid succession. The result, whether successful or not, is certainly not calculated to advance our knowledge of the efficacy or of the limitations of any one of the forms of treatment tried.

Especially disconcerting is that form of eclampsia which originates *de novo* after labour is over. Nothing can well be more disappointing to the accoucheur than to see a patient who has been successfully delivered, and who might reasonably be expected to make uninterrupted progress, suddenly develop puerperal convulsions.

The comparative rarity of such a disaster has led to this post

partum eclampsia being somewhat overlooked in the enormous literature of the subject. It is entirely overshadowed in the minds of most writers by the more common, and on the whole more grave, forms of the disease occurring before or during labour. Only a few authorities have made definite endeavour to fit their theories as to the cause of the disease to cases originating post partum. But if eclampsia is one and the same disease, whether it arise before, during or after labour, any theory as to its etiology must explain all three varieties. Indeed, the rarer forms, such as the post partum variety, are just the very ones by which any theory may best be judged; they are the touchstone, as it were, by which the claims of rival theories may best be tested, and put to the proof.

My attention was drawn to the subject by Professor Sir Halliday Croom in connection with a case which occurred at the Royal Maternity Hospital of Edinburgh a few months ago, and which has been recorded by him elsewhere.[1] It seemed desirable, therefore, to try to collect the views and experience of various writers on the subject, and so perhaps to obtain a survey of our knowledge from a different standpoint.

In what follows I have, accordingly, confined my attention entirely to eclampsia originating *de novo* after labour is completed, and have excluded all other cases of eclampsia whatsoever. This was not altogether easy, because many cases recorded as post partum eclampsia are not strictly such, in so far as the fits began before the third stage of labour was completed, and merely continued, or perhaps became more frequent after it was finished. These cases are not really cases of post partum eclampsia, and in no way bear upon the present enquiry, the limits of which I wish clearly to define at the outset.

Frequency. It is impossible not to suspect that there enters into the statistics available in the literature some confusion of the sort just mentioned. In one sense any persistent case of eclampsia might be put under all three headings of ante partum, intra partum and post partum eclampsia. In not a few cases this seems to have occurred. More recent statistics, however, are clear in their distinction, and I have rejected any tables which appeared dubious.

By the kindness of the medical staff of the Royal Maternity Hospital here, I have been allowed to examine the records of the last 5,800 cases in that hospital. I found a total of 126 cases of eclampsia amongst them—a frequency of 2·2 per cent. Of these 126 cases of eclampsia 16 originated post partum—a relationship to the total number of cases of 12·6 per cent.

The following table gives the frequency of post partum eclampsia in proportion to eclampsia in general in thirty-two records in the literature:—

	%		%
Jardine [2]	4·5	Lantos [16]	16·98
Collins [3]	6	Winckel [17]	17
Green [4]	6·6	Goedecke [18]	18·2
Löhlein [5] (2nd series) ..	7·5	Hammerschlag [19]	19
Merriman [6]	12	Meyer-Wirz [20]	19·7
Tietke [7]	12	Verrier [21]	20
Edinburgh Maternity Hospital (collated by myself by kind permission of the Staff.)	12·6	Bayer [22]	20
		Schweder [23]	20·2
		Schröder [24]	20·25
		Brummerstad [25]	21
		Schreiber [26]	21·16
Olshausen [8]	14	Kopetsch [27]	22·5
Geuer [9]	14	Rosthorn [28]	22·72
Feustell [10]	14·25	Löhlein [29]	23
Knapp [11]	14·6	Braun [30]	24
Glockner [12]	15·07	Schauta [31]	26·4
Moran [13]	16·6	Bidder [32]	27
Leopold [14]	16·8	Büttner [33]	32
Goldberg [15]	16·88		

The average of all these figures is a percentage of 17·41.

It will be seen therefore that the text-books are accurate in stating that about 20 per cent. of cases originate after labour.

Time of onset. On this point there is a consensus of experience. The large majority of cases have their onset within a few hours of the completion of labour—certainly within the first twenty-four hours. For example, of twenty-three cases in the Zürich clinic cited by Meyr-Wirz [20] eighteen occurred on the first day, two on the second, one on the fourth, and one on the fourteenth day post partum. These figures are representative of the experience of others. At the same time it is noticeable that the extremes are widely separated. Thus many cases occur within an hour, some within a few minutes of the birth of the placenta, while other cases of *reputed* eclampsia originate in the later weeks of the lying-in period. Most of the cases occurring late are recorded by early writers, and it is impossible not to question the accuracy of the diagnosis.

One is compelled to place under suspicion any diagnosis of eclampsia originating more than seven days after labour. Indeed, a purely theoretical consideration of the phenomena of the puerperal state would lead one to draw the line nearer the end of the first four days. By that time the process of involution is well under weigh, and any little disturbance connected with the beginning of lactation is either past or at least passing off. In short, the puerperium has usually settled down into the even tenor of its way. On the other hand, there are numerous cases recorded as occurring

on the fifth, sixth and seventh days in which the diagnosis, as formed clinically can not be cavilled at, so that one is led to put the dividing line back as far as the end of the seventh day. Any "eclampsia," however, occurring more than a week after labour can hardly be accepted as such.

Cases occurring on the fifty-sixth day are on record by Sir J. Y. Simpson,[34] and Goetze[35] amongst others, while Baudelocque[36] records one on the forty-fifth day. But an examination of the record of Simpson's case makes it evident that it was a uræmic seizure developing in the course of a nephritis, and its relationship to the pregnancy and labour, largely, if not entirely, incidental.

As recently as 1890 a case was read at the Obstetrical Society of London by J. B. Hurry,[37] of "symmetrical erysipelas: premature labour: eclampsia 19 days post partum, 28 fits, recovery." The fits were associated with the sudden appearance of a discharge from one ear. The urine never contained albumen. Unfortunately the discussion on the case was almost wholly focussed on the subject of the symmetrical erysipelas, and Dr. Amand Routh was the only one who called the diagnosis of eclampsia in question. He suggested that the fits were due to a meningitis. This Dr. Hurry denied, but the written record of the case goes far to support Dr. Routh's criticism. This is a typical example of many of the very dubious cases.

Other late cases have been shown to be due to poisoning. Löhlein[38] has recorded a case occurring in the second week in which the cause was clearly carbolic poisoning; and Olshausen[8] had a case on the eighth day of the puerperium due to poisoning with perchloride of mercury in the douche.

Of the sixteen cases in the Maternity Hospital here whose records I have seen, three began within one hour of delivery, five more before twelve hours had elapsed, and three between the twelfth and twenty-fourth hours: four began on the second day, and one on the third day post partum.

Mode of onset and course. In this connection one or two points are worthy of notice. In the first place, in a great many cases the disease occurs without any warning. The patient has been well throughout her pregnancy, the urine remaining normal, and has been delivered without any complication or untoward incident befalling. All at once she is seized with an eclamptic fit or loss of consciousness; or at the most her attendants get a brief warning in the form of sudden headache or restlessness. Even in those cases occurring later in the puerperium there is rarely anything in the way of premonitory symptoms. The puerperium has in most cases been absolutely normal up to the time of the sudden seizure. The only reservation in connection with this statement is in regard to the urine. It is stated in several records that the urine was normal up to the time of the seizure. But in the vast majority of cases there

is no record of the urine *just* before the onset of the eclampsia (with the exception of those cases occurring immediately after labour), inasmuch as it is rare for the urine of a patient to be examined systematically during the puerperium, unless she presents symptoms which suggest the propriety of so doing. This, of course, particularly applies to private practice.

In the sixteen hospital cases that I have referred to, four had presented no symptoms whatever of toxæmia during pregnancy, such as albuminuria or œdema. In no case was there any notable departure from the normal in their labours, and in most of them the eclamptic seizure came on without any warning whatever. In one or two cases there was slight headache. It is very interesting that in one case the only premonitory symptom was severe *epigastric pain*—a symptom, the significance of which as a forerunner of eclampsia, Olshausen [8] was the first to point out.

In regard to the frequency with which the condition is associated with albuminuria, post partum eclampsia is in no way peculiar. The great majority of cases show a large quantity of albumin in the urine, but there are exceptions. As already stated, four of the sixteen cases showed no albuminuria when last tested before the onset of the disease. Of these, three showed the presence of albumin in quantity after the first fit; while in one there was merely the faintest trace of it for a few hours after the fit.

The number of fits is usually small—frequently just one or two. Cases, in which there are more, are very often fatal. In the series of cases which I have been permitted to collect from the records of the Maternity Hospital in this city, of five that ended fatally, all had six or more fits. Of those that recovered, three of them had one fit, three two fits, two three fits, two five fits, and one had as many as fourteen fits. This last case appeared to be moribund when Sir Halliday Croom [1] determined to have recourse to decapsulation of the kidneys. The result was entirely successful.

Prognosis.

Of these sixteen cases, five ended fatally—a mortality of 31·25 per cent. This is rather high. Naturally the statistics of various writers vary a good deal, as is shown in the following table:—

MORTALITY IN ECLAMPSIA PUERPERARUM.

	%		%
Bidder [32]	5·2	Rosthorn [28]	22·72
Green [4]	6·6	Löhlein [29]	23·3
Hammerschlag [19]	9	Schauta [31]	26·5
Schreiber [26]	13·9	Kaufmann [39]	26·6
Feustell [10]	14·25	Edinburgh Maternity Hospital	31·25
Leopold [14]	16·8	(Collated by myself.)	
Lantos [16]	16·9	Buttner [33]	43
Schröder [24]	20·2		
Kopetsch [27]	22·5		

Most authors appear to be of the opinion that it is the most benign form of eclampsia, but there are some who regard it as peculiarly dangerous. Of five cases seen by Sir Halliday Croom only one recovered. Four of these were seen in private in consultation, and it is probable that only the worst cases are seen in this way. Cases which recover after perhaps one, or at most two, fits early in the puerperium are not so likely to come under the notice of the consultant.

Again, of four cases that occurred in St. Mary's Hospital,[40] Manchester, during 1908, only one recovered. These statements, together with some of the figures in the foregoing table, show that it is at least not a uniformly benign form.

Possibly the explanation of the apparent discrepancy between the figures of different authorities is to be found in the time of onset. Olshausen [8] observed in his series of cases that where the post partum attack ensued soon after labour there were seldom more than one or two fits, and the disease was of short duration. In these cases the prognosis was extremely good. Where the onset occurred later, however, the fits were more numerous and the results worse. Thus in his whole series of post partum eclampsias, seven out of twenty-eight died, 25 per cent. In fourteen of these twenty-eight cases the onset occurred within three hours of the end of labour, and of these only two died. Of the other fourteen, in whom the disease began later, five died.

Of the fatal cases in the series I have recorded, three were multiparæ and two primiparæ, although the proportion of multiparæ to primiparæ in the whole number was only five to eleven. In other words, it was three times more fatal in multiparæ than in primiparæ.

Etiology. The question still remains as to how far any of the theories of eclampsia, either old or new, explain these strictly puerperal attacks. Of all these theories the most generally accepted is undoubtedly that of the toxæmic origin of eclampsia. There are, however, as many subdivisions of it as there are conceivable sources of toxin—"*quot judices, tot sententiæ.*"

Until recently, the most usually adopted *working* hypothesis found the source of the poison in the substances which pass through the placenta as by-products of the development and growth of the fœtus, and ultimately reach the liver of the mother. Given any disease or functional weakness of the liver, it can be easily imagined that these substances will not be fully metabolized and will circulate in a state toxic to the mother. One result may be to irritate and damage the kidneys, and so induce an accumulation of the poison in the system by blocking one channel of exit. Another may be to further damage the liver, making it less equal to the demands upon it, and so setting up a vicious circle.

This hypothesis serves as a rational basis for the uniformly

successful treatment of toxæmia of pregnancy in its mild form. That treatment consists of elimination and diet. We stimulate all the functions by which the maternal organism can get rid of the poison circulating in it; and we try to diminish the supply of poison by giving a diet so light that it imposes no work to speak of on the liver and kidneys of the mother, and thus leaves them free to deal with the noxious substances derived from the fœtus.

This hypothesis may also form a satisfactory explanation of eclampsia as it occurs ante partum and even intra partum. It explains why eclampsia becomes more and more common as pregnancy advances, and why it is so often found in association with twins. It is only necessary to postulate the stimulus of any irritation, such as labour pains, acting upon a poisoned nervous system to explain why the convulsive eruption is still more common during labour.

But this theory breaks down entirely in these cases of eclampsia which occur without the presence of a fœtus, that is to say, in cases of hydatid mole. It also breaks down before the problems of post partum eclampsia, and of those rare cases of eclampsia gravidarum in which the onset occurs *after* the death of the fœtus *in utero*.

In the latter case poisons due to putrefactive changes are called on to explain the difficulty. In the former, in which we are more immediately interested, several reasons have been advanced.

Dienst,[41] for example, saw in the process of involution the source of the poisons. No doubt the process of involution must throw into the maternal circulation a large quantity of material which, if only partially oxidized by the insufficient liver, will act as toxins. It may also be true that the fall of the blood pressure, and the slowing of the heart's action after labour, tend to interfere with the elimination of toxins. Dienst claimed that the preponderating frequency with which post partum eclampsia occurs in the first twenty-four hours after labour is in favour of his view. Surely, however, the contrary is the case. The process of involution must be more active, and its products more copious, in the second twenty-four hours after labour than in the first.

Another objection to Dienst's view, as far as it affects cases originating post partum, was that it presupposes damage or inefficiency on the part of the liver or kidneys, which is not easily reconcilable with the fact that so frequently the patient is, or seems to be, in absolutely perfect health until the fit occurs.

Dienst's later views are that all the phenomena of eclampsia are due to an increase of the fibrinogen in the blood, and he regards the source of that increase as being due to the passage of fœtal blood into the maternal circulation. Presumably, in order to bend this theory to the interpretation of post partum cases, we must regard it as possible that during labour a considerable quantity of fœtal

blood passes into the maternal circulation. An examination of the placenta in such cases does not lend much support to this view.

Fehling [42] held the view that the toxins are directly fœtal in origin, and that an accumulation of them occurs in the mother's system during labour. This accumulation theory can certainly claim support from the fact, that so many cases occur just immediately after labour is completed. It does not, however, explain cases where there has been no labour—where the patient has been delivered by Cæsarean section. The occurrence of eclampsia in such cases, a few hours after the operation, has been recorded more than once.[44] In more than one case the Porro procedure was followed,[43] so that in these cases the results of the involution of the uterus could also be discounted. Fehling's theory, moreover, does not explain cases occurring later in the puerperium. There is no doubt that the later the fits occur, the more assuredly must their cause be sought for in the maternal organism.

Büttner [33] seeks the cause of later post partum eclampsia in the return of menstruation. He claims that ovulation at least can occur as soon as seven days after labour, as witness cases where women have been impregnated at such a time. He regards the recurrence of the menstrual molimen as calculated to irritate a poisoned nervous system to the point of convulsions. Of course this presupposes some toxic state as well. He produces evidence which tends to show that eclamptic seizures are liable to occur at times, both ante and post partum, which would correspond to the return of the menstrual wave. The evidence, however, is too meagre to be convincing, and in any case the view applies more to cases occurring so late as to be most probably not true eclampsia at all.

A certain historical interest attaches to Büttner's theory, as Baudelocque [50] vaguely outlined a similar idea as long ago as 1790. It is probable, however, that Baudelocque's cases were, in part at least, more of the nature of menstrual epilepsy.

The placental theory of eclampsia is undoubtedly the one to which most attention is being paid at the present time. The main points in regard to it have been recently noted in this JOURNAL in Dr. Holland's papers in the last three numbers.[51] When one comes to consider them from the point of view of post partum eclampsia the difficulties in the way of their acceptance are by no means diminished. Indeed, the removal of the placenta at the end of labour creates a fresh difficulty. A certain amount of syncytium may possibly be left behind in the sinuses of the placental site, and, as is well known, a certain quantity may be regarded as in almost all cases straying into the maternal circulation during pregnancy. Once the placenta is removed, however, the quantity of syncytium or other placental substance must be very small compared with what is available before the expulsion of the placenta. It is not sufficient

to argue that eclampsia originating post partum may be due to retained portions of placenta, because in the bulk of cases that can be disproved. It is, therefore, difficult to accept Veit's theory that the disease is due to the presence of placental cells in the maternal circulation, and that these cells are directly toxic. Veit argues that any sudden increase in the quantity of these cells in the maternal circulation overcomes the resistance of the anti-bodies formed, and so leads to eclampsia; but it is not easy to see why any such sudden access should occur post partum.

Ascoli's theory lends itself more readily to an interpretation of post partum cases. He regards the anti-bodies which are formed to antagonize the presence of placental cells as the specific toxin of eclampsia, and on this theory any sudden diminution in the quantity of placental cells might be considered to throw a great number of these anti-bodies or syncytiolysins out of employment. That is to say, that these syncytiolysins, being no longer of use in antagonizing the placental elements, are free to act as toxins. It is possible that the removal of the placenta may cause a sudden diminution in the quantity of placental elements circulating in the mother's blood, and that thus in some cases the balance is upset, and the syncytiolysins are enabled to become toxic.

The microbic theory of eclampsia has been generally given up recently—both the idea that it might be due to a specific organism, and the theory of an underlying mixed organismal endometritis or deciduitis. Assuming for the sake of argument the truth of either view, it might readily be understood that the uterine upheaval during labour would cause a rapidly increased disseminaton and absorption of such organisms or their toxins; and the physical conditions of the puerperal uterus are only too notorious as an excellent nidus for microbic growth. It must be admitted, however, that there is no evidence to support this view in regard to post partum eclampsia.

Nicholson [45] and others [46] have looked for the cause of eclampsia in insufficiency of the thyroid gland; and more recently some observers [46] have suggested an insufficiency of the parathyroid. Treatment by administration of the gland products has been moderately successful, but it is open to more than one interpretation. It is difficult to see why any such insufficiency should suddenly show itself for the first time after pregnancy and labour are over. Particularly is this so as applied to the cases occurring just immediately after labour, for, so far as I am aware, there is no absolute proof that the process of parturition uses up any excessive quantity of the thyroid secretion. The experiments of Verstraeten and Vanderlinden [47] point to the probability of this being the case, but by no means prove it.

When we come to cases originating on the second and third day, however, the problem becomes more involved, for there is considerable evidence to show that the thyroid is connected in some way with the function of lactation. An experiment performed by Vassale [48] is suggestive. He performed a partial ablation of the parathryroid in a pregnant bitch. The animal made a good recovery, but was delivered prematurely of two pups which died. Consequently there was no lactation. Later, she again became pregnant and was delivered of seven living pups. She suckled them until on the fifth day she was seized with a very severe attack of convulsions which was cured by the free administration of thyroid extract. Thereafter, she was only allowed to suckle three pups, but again she fell into convulsions and again was cured by thyroid. After that she was allowed to suckle only one pup, and the exhibition of thyroid extract was continued. She made a complete recovery, and the thyroid extract was only stopped when lactation ceased.

Of course, one single instance of this sort is in no way conclusive of anything, but it suggests to the mind the possibility that in some circumstances lactation may put an extreme demand upon the thyroid and parathyroid glands. Numerous other experiments and clinical observations have shown that removal of the thyro-parathyroid organs leads to the onset of fits in pregnant women and animals. Why then should not a purely functional exhaustion lead to the same result?

A further point to be noted in this connection is that Lange [49] and others found that it was the women whose thyroids did *not* hypertrophy in the later months of pregnancy who so frequently developed eclampsia. In these women, there is, therefore, a comparative insufficiency of the thyroid which, although perhaps not marked enough to lead to any complications before or during labour, might very well lead to disaster if the start of lactation causes an extra demand for thyro-parathyroid secretion.

Against these facts and observations, we must, however, put another—namely, that removal of the thyroid and parathyroid may induce fits in animals which are not pregnant nor in the puerperal state. Therefore any fits due to such a cause cannot strictly be classed as eclampsia, unless a subsequent post mortem were to prove that the characteristic lesions in liver and kidneys were present.

It is outside the scope of the present paper to discuss the treatment of post partum eclampsia. It may be noted, however, that the fact that 17 per cent. of eclampsias originate after the completion of labour, appears on the face of it to be an argument against the principle of immediately emptying the uterus in cases of ante partum or intra partum eclampsia. Against this, on the other hand, we must place the fact that in the experience of many authorities,

the post partum variety of eclampsia is the mildest form of the disease, and the one most amenable to treatment. On this controversial point, therefore, whether or not immediate obstetric interference is the best treatment, post partum eclampsia throws no new light.

On the whole, it must be admitted that our knowledge of eclampsia as it originates post partum is, if possible, less than our knowledge of ante partum and intra partum eclampsia. Everything points to the latter forms being toxæmic manifestations, but so far a feasible explanation of post partum cases in this way has not, as I have tried to show, been attained.

Some obstetricians even go the length of regarding post partum eclampsia as a different disease from the other varieties. But this seems simply to be yielding to the difficulty of explaining it. Certainly in those numerous cases, in which it has been preceded during pregnancy by symptoms of pre-eclamptic toxæmia, it must be looked on as the same disease.

It is more than likely, however, that our lack of knowledge of the real meaning and cause of eclampsia leads us to include in it several different forms of disease. This applies equally to pregnancy, labour and the puerperium. It is to be hoped that in the near future an increased knowledge of the chemistry of the metabolism in pregnancy will enable us to make an absolute diagnosis of eclampsia from the laboratory. This will in all likelihood lead to the exclusion of many cases, both before, during and after labour, which are now called eclampsia, but which will then be found and proved to be due either to allied toxæmic states, to pre-existing organic renal disease, or to organic, or even functional, nervous disease. When this advance has been achieved we may hope to understand the exact bearing upon the condition of the functions of such organs as the thyroid and parathyroid, but until then we can only observe and record facts, and from time to time marshal them, and review the situation from different standpoints.

We cannot in these days share the placid satisfaction of Verrier,[21] who, after tracing all post partum eclampsias to the influence of "la puerpéralité, comme ces fièvres puerpérales *sine materia* qui font le désespoir des savants et des practiciens"—naïvely concludes—"mon explication . . . satisfait l'esprit le plus scrutateur en plaçant sous l'influence de la puerpéralité tout accès d'éclampsie survenant jusqu'au retour des couches!"

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