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THE TOXEMIAS OF PREGNANCY, AND THE TREATMENT OF ECLAMPSIA*

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In this paper I shall consider only the toxemias of pregnancy as they occur during the last third of pregnancy, and particularly the treatment of eclampsia. I use the plural rather than the singular for the reason that the term toxemia is employed very loosely, and is used by many to include a group of conditions that vary from a trace of albumin in the urine without clinical symptoms to actual eclampsia, or even to acute yellow atrophy of the liver. Indeed, many go so far as to claim that the several conditions merely represent stages in the evolution of a single disease process.

For years, I have contended that such a conception is erroneous and tends to complicate rather than to clarify the question, as I believe that several distinct entities have to be dealt with and that any attempt to gather them into a single group will retard the eventual discovery of their respective causes. In the last edition of my textbook, I treated the subject under the following headings: nephritic toxemia, preeclamptic toxemia, eclampsia, and presumable toxemias. For years, I have studied these conditions with the greatest care both from the clinical and the pathologic point of view, and for the last seven years my associates, Everett D. Plass and H. J. Stander, have devoted the greater part of their time to the chemical and metabolic investigation of the problem, in the attempt to discover means of differentiating clinically between the several types, and if possible to obtain a clue as to their cause. Something has been accomplished along the former, but nothing, I am sorry to say, along the latter line.

Except when the patients come to autopsy, differentiation between the various types is not always possible during the stay in the hospital. Consequently, it occurred to me that valuable information might be obtained by restudying the patients at intervals after their discharge; and for the last five or six years we have attempted to get back as many of them as possible a year after delivery, and to follow their subsequent course as long as feasible after that. In 1924, Harris reported the results obtained and, generally speaking, found that a year after the eclamptic attack practically all of the women were normal so far as blood pressure and manifestations of renal involvement were concerned. On the other hand, the patients who had been discharged with a clinical diagnosis of chronic nephritis or nephritic toxemia uniformly presented signs of the disease at the return examination. Such observations were in accordance with our expectations; but when we came to analyze the results in the patients who had been discharged with a diagnosis of preeclamptic toxemia, we were surprised to find that a considerable proportion (60 per cent) presented signs of chronic nephritis, notwithstanding the fact that on discharge the blood pressure had fallen to normal and the urine was free from abnormal constituents. Up to that time we had regarded the so-called preeclamptic toxemia as the forerunner of eclampsia, and had held that its detection and treatment was the ideal method of preventing the development of the latter, and yet we found that the end-result of what we had considered the less serious process was actually graver than that following the more serious one. Such observations could only mean that we know less about preeclamptic toxemia than we had believed, and made it probable, in many instances in which such a diagnosis had been made, that we were actually dealing with chronic nephritis in a mild or larval form.

With this in mind, we have studied our cases of preeclamptic toxemia still more intensively, and have reached the conclusion that we had formerly included under that heading several conditions of radically different significance, and that closer analysis might enable us to differentiate between them more successfully. In May, 1926, Stander and Peckham gave the results of the clinical and chemical study of 120 toxemic patients recently studied in our service, and suggested the following classification: eclampsia, preeclampsia, chronic nephritis, eclampsia superimposed on chronic nephritis and low reserve kidney. Nothing need be said concerning the clinical characteristics of eclampsia or chronic nephritis, as they are well recognized clinical entities, but the other terms require some explanation. It might, however, be stated that eclampsia is distinguished at autopsy by the presence of characteristic liver lesions in the shape of necroses beginning in the periportal spaces and encroaching on the periphery of the lobules. Moreover, it should be recalled that when recovery ensues the return to normal is very prompt, and that the disease is unlikely to recur in subsequent pregnancies. After chronic nephritis, on the other hand, recovery is tardy, if it ocurrs at all, and the process tends to undergo exacerbation earlier and earlier in each successive pregnancy, so that it frequently offers an indication for contraceptive advice or even for sterilization.

Stander and Peckham have greatly limited the scope of preeclamptic toxemia, or preeclampsia, as they prefer to call it, and have transferred the great majority of cases, which were formerly so designated, to another category—that of low reserve kidney. They limit the former term to the relatively small group of cases in

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which the patient is acutely ill, presents a very high blood pressure and albumin content of the urine, suffers from amaurosis and epigastric pain, presents a blood chemistry picture identical with that of eclampsia, and appears on the verge of convulsions, whose outbreak can be prevented only by the promptest possible termination of pregnancy consistent with safety. In other words, it is essentially eclampsia before the development of convulsions and coma. Defined in this way, preeclamptic toxemia is a relatively rare condition and does not exceed 5 per cent of all late toxemias, while the great bulk of the cases that were formerly so designated fall into the group of low reserve kidney.

In the latter condition, which may be due to congenital causes or to such factors as may have lessened the number of functioning glomeruli without producing a chronic nephritis, the kidney reserve has become so reduced that, while it suffices for the usual conditions of life, it breaks under the strain of the last months of pregnancy. That it does not lead to chronic nephritis is shown by the fact that the condition may or may not recur in subsequent pregnancies; and if it does recur, the symptoms are usually no more severe than on the previous occasion. In patients presenting this condition, the blood pressure is not greatly elevated, rarely exceeding 150 systolic and 90 diastolic, the albumin content varies between a trace and a couple of grams per liter, and, while the patient may present edema, headache and more or less malaise, the condition improves with rest in bed and a restricted diet, and rarely if ever eventuates in eclampsia. Following delivery, there is a prompt return to normal and any trace of the condition is not detectable until a subsequent pregnancy occurs, when it may or may not become manifest in the latter months. Change does not occur in the chemical constitution of the blood, and the phenolsulphonphthalein output is not altered.

The category eclampsia superimposed on chronic nephritis means what its name implies, and is made up of a small group of patients suffering from chronic nephritis who later develop eclampsia. In general, such patients are more ill than those with uncomplicated eclampsia; they present an unusually high blood pressure and an excessive amount of albumin. They are particularly interesting from two other points of view: first, that chemical examination may give evidence of marked retention of nonprotein nitrogen, and, second, that after recovery from the acute attack the symptoms of renal insufficiency persist. Generally speaking, it is safe to conclude that, whenever after an eclamptic attack the blood pressure remains permanently elevated and a trace of albumin persists in the urine, one has had to deal with this combination and not with a chronic nephritis originating from the eclampsia.

It is only fair to say, however, that while this new classification has helped materially, it has not solved all our problems, and with the patient in bed before us it is not always possible to make the differentiation immediately, nor is it always easy at autopsy. Generally speaking, the diagnosis of well marked preeclampsia is readily made from the presence of characteristic symptoms. On the other hand, it is not always possible to differentiate between the low reserve kidney and chronic nephritis before delivery. In such cases, the previous history may be of great assistance, while the demonstration of nitrogenous retention in the blood or the existence of albuminuric retinitis speaks decisively for chronic nephritis; but when such signs are absent, differentiation may be impossible until the end of the puerperium, when a very gradual fall in blood pressure and the persistence of a trace of albumin and casts in the urine will speak strongly against the low reserve kidney.

Similarly, it is not always possible to distinguish between uncomplicated eclampsia and one engrafted on a chronic nephritis until recovery ensues or an autopsy is had. In general, it may be said that the absence of albuminuric retinitis and of nitrogenous retention in the blood speak in favor of the former, but the latter cannot be excluded until several months later, when we find that the patient is either perfectly normal or presents signs of chronic nephritis. It is for this and similar reasons that we have recently inaugurated the custom of having our toxemic patients return to the clinic for a day two months after delivery, when they are carefully restudied with particular emphasis on the clinical signs and the chemical changes of the blood and urine. It is as yet too early to state what may be accomplished by such reexaminations, but we have already learned that it frequently necessitates a radical revision of the original diagnosis, and we hope that it will eventually throw still further light on our problems.

TABLE 1.—*Usual Range of Certain Constituents of the Blood*

	Normal Nonpregnant	Normal Pregnancy	Nephritic Toxemia	Eclampsia
Nonprotein nitrogen...	30-35	25-30	35-100	25-35
Uric acid.....	2.0-3.5	2.0-3.5	3.5-9.0	4.0-12*
Blood urea nitrogen...	13.4	13.3	13	13.4
Sugar.....	70-100	70-100	70-100	120-185*
Lactic acid.....	20-35	20-35	35-80	50-200*
Inorganic phosphorus.	1.5-3.0	1.5-3.0	1.5-3.5	2.5-3.5*
Carbon dioxide.....	55-65	40-50	40-50	15-55

* Radical changes.

Repeated reference has been made to the chemical changes in the urine and blood, and the time has come to say a few words as to their significance. Generally speaking, it may be said that the most important features in the examination of the urine consist in ascertaining its amount for the twenty-four hours, the determination of its albuminous content in grams per liter, and the presence or absence of tube casts. We have learned comparatively little of value from its accurate chemical analysis; and while we still determine its total nitrogen, the percentage of the urea and ammonia nitrogen to the total nitrogen, and occasionally the chloride content, it must be confessed that it is done rather for the sake of completeness than for any definite information it affords. In our earlier work, such estimations were undertaken in the hope that they might reveal the retention of certain substances which would give a clue to the mode of production of the toxemias and some indication for their treatment; but such hopes have long since proved illusory.

Likewise, in the early days the chemical study of the blood was undertaken, first, to ascertain the changes which its constituents undergo during normal pregnancy, and secondly, in the hope that some form of nitrogenous retention would be discovered which would facilitate diagnosis and be of prognostic value in toxemic patients. Table 1, which shows the usual ranges for certain constituents of the blood as found in our laboratory, will give an idea as to what extent these ideals have been realized. The first two columns show the normal conditions, and on comparing the nonpregnant and pregnant woman, respectively, it is seen that there is a slight diminution in the amount of nonprotein nitrogen and a slight decrease in the carbon

dioxide combining power in the latter. There is, however, no evidence of nitrogenous retention, but rather the reverse, and this is associated with a moderate acidosis, as indicated by the lowered combining power for carbon dioxide, to which Hasselbalch and Gammeltopf first directed our attention.

No figures are given for the low reserve kidney, but as it is accompanied by no appreciable change, the data in the normal column are applicable to it. Passing to the third column, it is seen that radical changes are present in nephritic toxemia, and that the most important consists in a marked retention of nonprotein nitrogen, which is accompanied by a considerable rise in uric and lactic acid. If, in the normal pregnant woman, one constructs a fraction in which the blood urea nitrogen is the numerator and the nonprotein nitrogen the denominator, one obtains a constant which represents the normal relation but which becomes lower and lower, the greater the retention of the nonprotein nitrogen.

The fourth column represents the conditions in eclampsia, and the five items marked by a star present radical changes, while the remaining two are unchanged. It will be noted in the first place that there is a marked increase in the uric acid content, which is followed by equally striking changes in the amount of sugar and lactic acid, and by a lesser change in inorganic phosphorus, while at the same time in extreme cases the carbon dioxide combining power is reduced to such an extent as to offer a distinct menace to life from acidosis. In general, it may be said that the changes noted in eclmapsia are very striking and do not in any way bear out the original supposition that it is associated with nitrogenous retention, but on the other hand indicate the existence of a profound disturbance of metabolism. It would lead too far afield to attempt to theorize concerning the significance of these changes, and even if I possessed the ability to do so, it is doubtful whether my deductions would meet general approval. I shall therefore merely state that the figures will be referred to again when the question of treatment is taken up.

Thus far, it must be admitted that with the exception of the nephritic type, neither pathologic study nor chemical analysis of the blood and urine gives any clue to the ultimate cause of eclampsia or any of the other forms of late toxemia of pregnancy, and that in all probability it is due to the formation of some chemical substance (toxin?) which leads to a profound disturbance of metabolism, accompanied by certain organic lesions; but what it is, or whether it originates primarily in the child, the placenta or the mother is not known. That the supposititious substance leads to an increase in the permeability of the smaller vessels and capillaries is probable, especially in view of the almost constant presence of edema; and such a view receives additional support from Starling's theory that the rise in blood pressure is due to anemia of the vasomotor centers at the base of the brain, resulting from edema of the surrounding tissues. Furthermore, it is possible that the quantity of toxin produced, or the resistance offered to its action, must be susceptible to pronounced variations, as is evidenced by the occurrence of so-called intercurrent eclampsia, and the occasional normal progress of pregnancy after its cessation.

Whatever the cause may be, it is apparent that it will not be discovered by theorizing, but solely by the patient and intelligent application of approved means of research by devoted investigators. Furthermore, it is clear that the treatment must remain empiric until the problem is solved, and in the meantime it is our duty so to study the natural history of the disease and its manifestations that we may treat it with the least possible danger to the patient, as well as with the least possible personal pride.

TREATMENT

This being the case, the most effective treatment of eclampsia must consist in prevention. For that reason the greatest possible extension of intelligent prenatal care is essential to the early recognition and treatment of the several varieties of the toxemia of pregnancy. This is not the place to insist on the necessity for the frequent routine examination of the urine, the determination of the blood pressure, and the general supervision of the physical condition of the patient. Suffice it to state that if this duty is conscientiously performed, the incidence of and mortality from the toxemias will become enormously reduced. I am not, however, sufficiently optimistic as to assert that eclampsia can be done away with entirely, and an experience during the past year has served to confirm this conviction. In this instance, a patient had been conscientiously followed during an apparently normal pregnancy and all tests were negative at her last visit; yet she was admitted to the clinic three days later in eclampsia and died undelivered. Notwithstanding this experience, no one is a more enthusiastic advocate of prenatal care than I; and since the physical accommodations of our clinic have been adequate for the hospitalization of all patients requiring prophylactic treatment, the incidence of eclampsia has been greatly reduced, and, when it occurs in our own patients, it is usually of so mild a type as to be readily amenable to treatment; on the other hand, practically all of the serious cases occur in neglected patients who have been sent to the clinic by physicians or midwives after the outbreak of the disease.

Although the main object of this part of the paper is the discussion of the treatment of actual eclampsia, a few words may not be amiss concerning the treatment of the other types of toxemia. As has already been indicated, the most serious of these is the preeclamptic variety, or preeclampsia. In this, the outbreak of convulsions and coma is imminent, and the part of wisdom is to forestall the eclamptic attack, if possible, by the earliest termination of pregnancy consistent with the safety of the patient. In such circumstances the choice of procedure will depend on the condition of the cervix. If it is soft or already partially dilated, the modified Stroganoff treatment is begun, and as soon as the patient has come under its narcotic influence, a bag is inserted without anesthesia. On the other hand, if the cervix is rigid we do not hesitate to resort to cesarean section with the limitations concerning the choice of anesthesia to which reference will later be made. At this point, it may be added that it is my conviction that only in such circumstances should this operation be considered in the treatment of the toxemias, as I have come to regard it as greatly increasing the danger of actual eclampsia.

In the treatment of nephritic toxemia, or the complication of chronic nephritis by pregnancy, I have become more and more radical, and advocate the termination of pregnancy as soon as the condition becomes serious, unless for the sake of the child the patient is anxious to face the serious risks which delay may entail. My reason for this is fourfold: (1) Rest in bed and dietetic treatment rarely have the desired

result; (2) the child usually dies before the period of viability is reached; (3) the patient is frequently exposed to serious immediate danger, and (4) the continuance of pregnancy greatly adds to the gravity of the underlying renal condition, and thereby materially shortens the patient's life should she recover from the immediate attack. Consequently, if rapid improvement does not occur under conservative treatment, I terminate the pregnancy by the introduction of a bougie, and afterward give such contraceptive advice as may be necessary; and if it seems inadvisable or proves ineffectual, I do not hesitate to terminate a subsequent pregnancy by operative means, and at the same time to effect sterility by the method best suited to the individual case.

On the other hand, since we have learned that the low reserve kidney rarely eventuates in eclampsia, responds fairly satisfactorily to conservative treatment, and after the conclusion of pregnancy has no effect on the future health of the patient, we have become more and more conservative in its treatment. In such cases, the patient is put to bed, placed on a bland diet and watched. In many instances this is followed by such marked improvement that the patient can be discharged and eventually is delivered at term. Other patients respond less satisfactorily, and, after showing a certain amount of improvement, tend to remain in statu quo. In such circumstances the pregnancy can usually be carried to such a stage that the induction of labor offers a fair prospect for the birth of a child reasonably fitted for the contest of life. On the other hand, an occasional patient shows no improvement, but on the contrary the albumin content of the urine remains constantly at 2 or 3 Gm. to the liter. In such cases, unless the blood pressure presents an alarming rise, it is usually permissible to allow the pregnancy to progress to the period of viability, and then to induce premature labor. If this is not done, the patient may pass into the preeclamptic state with its accompanying dangers.

I approach the discussion of the treatment of actual eclampsia with considerable hesitation, for the reason that my views concerning it are still in a state of flux, and on the whole are negative rather than positive. My material consists of 275 cases which have occurred from the opening of the service to March 31, 1926. These are divided into two series of 110 and 165 cases, respectively, according as they occurred prior to or after 1912. Up to that date, in accordance with the prevailing practice, the primary object in treatment was the promptest possible delivery of the patient, with the result, if the cervix was not fully dilated, that accouchement forcé, or vaginal or abdominal section was frequently resorted to. Chloroform was still employed as the anesthetic, and delivery was followed by the employment of such therapeutic procedures as liberal bleeding, sweating, drastic purgation, the free use of morphine, and the attempt to dilute the suppositious poison by the subcutaneous or intravenous administration of saline solution.

The results were not satisfactory, and when I came to analyze them in the eighty-five cases of antepartum and intrapartum eclampsia, I found that one out of every four women in the first series had died (24.89 per cent). For some years previously, Stroganoff had been reporting wonderful results obtained with morphine and chloral, and by waiting until the cervix had become fully dilated before resorting to delivery. At that time, I could not bring myself to believe that his results were attributable to the treatment employed, but I was obliged to admit that they were better than ours, and I was inclined to attribute them, if true, to the avoidance of the trauma and shock incident to radical operative delivery.

Since 1912, our practice has become more conservative, and we have tended more and more to defer operative intervention until the cervix has become completely dilated, when labor is completed by version and extraction or by low forceps. Furthermore, we gradually restricted our therapeutic activities and came to rely more and more on free venesection (from 500 to 1,000 cc.) and morphine. Such a practice was easy to follow in the relatively mild cases, but became more difficult if prompt improvement did not occur, for with seriously sick patients it was difficult to control the activities of energetic associates and assistants. Consequently, accouchement forcé and cesarean section continued to be done occasionally, until I was prepared to lay down the strict rule that all cases must be treated absolutely conservatively. This was not done until 1922, but since then printed regulations have hung on the wall in the delivery suite, which no one has authority to change except myself and the associate responsible for the care of such patients. Moreover, my associate Stander was personally able to follow the work of Stroganoff in Petrograd three years ago, and while he was not convinced that the results were all that the latter claimed, yet he felt that his method, with certain modifications, was worthy of trial, and the following regulations were put into effect in October, 1924, and are still in force:

1. On admission:
   - (*a*) To be placed in a quiet darkened room and disturbed as little as possible.
   - (*b*) To have a special nurse continuously until definitely out of coma.
   - (*c*) To have one-fourth grain (16 mg.) of morphine hypodermically immediately.
   - (*d*) To be catheterized, examined medically and obstetrically, and bled for 200 cc. under nitrous oxide anesthesia, if conscious.
   - (*e*) To be placed on one side, with the foot of the bed elevated so long as coma persists. Mucus to be swabbed from the pharynx as it collects.
   - (*f*) To have water freely while conscious. If the patient cannot drink on account of coma or lack of desire, the intravenous administration of 500 cc. of 5 per cent glucose solution should be considered.
   - (*g*) Not to be delivered until after the cervix is fully dilated, and then by the simplest operative means, unless spontaneous delivery seems imminent.
   - (*h*) No chloroform to be used.
   - (*i*) Chemical assistants to be notified as soon as the patient is admitted, so that the necessary observations can be made.
2. One hour after admission:
   If comatose, 2 Gm. of chloral hydrate to be given in 100 cc. of physiologic sodium chloride solution and the same quantity of milk by rectum. If conscious, the chloral can be administered by mouth in 100 cc. of milk.
3. Three hours after admission:
   One-fourth grain (16 mg.) of morphine hypodermically.
4. Seven hours after admission:
   Two grams of chloral hydrate as above.
5. Thirteen hours after admission:
   One and five-tenths grams of chloral hydrate as above.
6. Twenty-one hours after admission:
   One and five-tenths grams of chloral hydrate as above.

7. General directions:
   (*a*) While eclamptic patients are under treatment, the assistants and nurses must insist on the greatest possible quiet.
   (*b*) Catharsis, sweating, or venesection in excess of 200 cc. must not be employed.
   (*c*) No change to be made in the schedule unless authorized by Drs. Williams or Stander.

It should be noted that this schedule differs in several particulars from that of Stroganoff, notably in the omission of chloroform, in the use of glucose injections, and in the routine withdrawal of blood. It should be understood that the latter is not for therapeutic purposes, but is solely for obtaining a sufficient quantity for certain routine determinations, as well as for purposes of investigation.

TABLE 2.—*Results in 275 Cases of Eclampsia*
(110 up to Dec. 31, 1911; 165 from 1912 to March 31, 1926.)

	Series 1		Series 2		% Deaths	
	Cases	Deaths	Cases	Deaths	Series 1	Series 2
Antepartum:						
Mild	33	5	30	0	15.1	0.0
Severe	28	10	54	11	35.7	20.4
Intrapartum:						
Mild	16	2	24	1	12.5	4.17
Severe	8	4	21	5	50.0	23.8
Postpartum:						
Mild	15	0	24	0	0.0	0.0
Severe	10	4	12	5	40.0	41.6
Total	110	25	165	22	22.8	13.3

Table 2 shows the results obtained in the two series, and clearly indicates the superiority of conservative over radical treatment. In the table are sixty-one cases of postpartum eclampsia, and, including them, the mortality was 22.8 and 13.3 per cent in the two series, respectively, while the results following postpartum eclampsia were practically identical in the two series.

It will be noted that a distinction is made between mild and severe cases; this was done following the example of Eden, who, analyzing the several reports on the treatment of eclampsia made to the British Congress for Obstetrics and Gynecology in 1922, stated that gross figures alone give very imperfect information as to the value of treatment, as it varies greatly according to the type of the disease. With this in mind, he classified as severe all classes which presented two or more of the following six conditions: (*a*) persistent coma; (*b*) pulse rate more than 120; (*c*) temperature more than 103; (*d*) more than ten convulsions; (*e*) urine containing sufficient albumin to coagulate solidly on boiling, and (*f*) the absence of edema. We have applied the same criteria, except that we have substituted an albumin content of 10 Gm. per liter by Esbach for the boiling test.

It is unnecessary to analyze the table more closely, as a glance will suffice to show that in every rubric the mortality is lower in the second series, with the exception of severe postpartum eclampsia, but in that instance the difference is negligible, as one would expect from the fact that the treatment remained relatively the same in the two series.

A still clearer idea of the improvement in results may be obtained from table 3, in which only the cases of antepartum and intrapartum eclampsia are included. This shows that the gross mortality has fallen from 25.9 to 13.2 per cent. In other words, a reduction of 1.96 times, and this difference is rendered still more striking when the mild and severe cases are considered separately. Thus, in the former, the mortality has fallen from 14.2 to 1.9 per cent, and in the latter from 38.8 to 21.3 per cent; or, to express the difference more graphically, it may be said that in the second series the results in the mild cases were 7.5 times, and in the severe cases 1.8 times better than in the first series.

On considering these figures, it seems fair to conclude that the substitution of conservative for radical treatment has so reduced the mortality of mild eclampsia as to render it an almost negligible condition, whose mortality will scarcely be susceptible of further reduction. On the other hand, while it has served to reduce by one half the mortality of the severe type, a death rate of 21.3 per cent still remains, and urgently demands improvement. In other words, it appears that in mild antepartum and intrapartum eclampsia treatment by narcotics and waiting until after the completion of the first stage of labor before attempting delivery gives almost ideal results, while similar treatment in severe cases is far from satisfactory, but is nevertheless greatly superior to that obtained by radical treatment in the past.

Is it possible to reconcile our results with the mortality of 2.5 per cent reported by Stroganoff? I think so, as it appears from Stander's analysis of his material that the great majority of his cases were mild in type and occurred in the women who were awaiting delivery in his clinic, and who therefore could be treated immediately after the outbreak of the first convulsion. Moreover, as he rarely had to deal with the severe type, his material is not comparable with ours, in which most patients are sent in from the outside. Indeed, our experience goes to show that with increasing efficiency in prenatal care relatively few cases of eclampsia develop in our own material, and they are almost entirely of the mild type, whereas we receive an increasing number of severe cases from the outside.

It must be admitted that in a certain number of the neglected severe cases the patients must inevitably die no matter what may be done for them, but at the same time it is probable that they should show better results than I have as yet been able to obtain, and our problem is as to how it can be brought about. Theoretically, it would appear that fewer deaths might result if delivery could be effected at the opportune

TABLE 3.—*Results in Antepartum and Intrapartum Eclampsia*

	Series 1		Series 2		% Deaths		Reduction, Times
	Cases	Deaths	Cases	Deaths	Series 1	Series 2	
Mild	49	7	54	1	14.2	1.9	7.5
Severe	36	14	75	16	38.8	21.3	1.8
Total	85	21	129	17	25.9	13.2	1.96

time, and the question is how to recognize its necessity and to determine by what means it should be accomplished. Our experience in the first series showed that radical delivery under the usual anesthetics gave much worse results than the conservative treatment now in vogue, and recent studies in our clinic by Stander [1] on the effect of anesthesia on the chemical constitution of the blood may possibly offer a clue toward the solution of the problem. He found that all general anesthetics —chloroform, ether, nitrous oxide and ethylene—after as short a period as fifteen minutes produce changes in the blood suggestive of those noted in eclampsia; namely, a definite increase in uric acid, sugar, lactic acid and inorganic phosphorus, and a decrease in the carbon dioxide combining power of the blood. These changes are shown in table 4, and clearly indicate that

1. Stander, H. J.: Am. J. Obst. & Gynec. **12**: 633 (Nov.) 1926.

the administration of the usual anesthetics even for a short time tends to accentuate the blood changes that characterize eclampsia, and thus superimpose an additional toxemia on that already existing. Such observations render it probable that in the radical treatment of severe cases of eclampsia it is not so much the operation, per se, as the accompanying anesthesia that does harm by converting an already serious condition into a fatal one.

TABLE 4.—*Usual Range of Certain Constituents of Dog's Blood Under Normal Conditions and Under Anesthesia*

	Normal	Anesthesia	Change
Nonprotein nitrogen	40-55	40-55	None
Uric acid	0.0-0.9	0.9-2.5	Increase
Blood urea nitrogen	12-18	12-18	None
Sugar	70-100	150-300	Increase
Lactic acid	12-20	18-35	Increase
Inorganic phosphorus	1.5-3.0	2.0-4.0	Increase
Carbon dioxide	45-60	30-45	Decrease

Too short a time has elapsed since the recognition of these dangers for us to have devised other methods of anesthesia, but it has occurred to us that some form of local or spinal anesthesia may solve the problem, more particularly as Stander has also shown that in dogs the administration of amounts of cocaine which would never be considered in human beings is not accompanied by any recognizable change in the chemical constituents of the blood. Whether improved results will follow the treatment of severe eclampsia along such lines, no one can foretell, nor is it possible to predict what will prove the ideal procedure for terminating the pregnancy. I shall, however, experiment along such lines with my serious cases and hope that in the not too distant future I may be able to report encouraging results.

TABLE 5.—*Fetal Mortality*

	Series 1		Series 2		Percentage	
	Cases	Deaths	Cases	Deaths	Series 1	Series 2
Antepartum	61	40	84	58	65.5	69.0
Intrapartum	24	9	45	15	37.5	33.33
Total	85	49	129	73	57.6	56.6

Thus far I have said nothing of the fetal mortality, but table 5 will make it possible to dispose of the question in a few words. This shows that the mortality was practically identical in the two series, and indicates that conservative treatment at least does no harm. At first glance the results are very discouraging; but when it is recalled that in a considerable proportion of cases the disease occurs before or shortly after the period of viability is reached, it is apparent that a certain mortality is inevitable. Moreover, a great part of the fetal deaths occurs in the severe cases, and in many of them has taken place before the patients come into our hands. The advocates of radical treatment assert that the chances for the child will be greatly increased by cesarean section, but in my opinion this is based on doubtful reasoning as very few children are lost without it in mild cases, while in severe cases experience teaches that under present conditions cesarean section adds materially to the danger to the mother, and I agree with Eden's verdict that after accouchement forcé it is the least appropriate treatment of eclampsia.

One of the important lessons which I have learned from my experience with conservative treatment is that the termination of pregnancy, or at least the death of the fetus, is not essential to the arrest of the disease. Thus, in sixteen of the eighty-four cases of antepartum eclampsia in the second series, I have observed that under the influence of narcotic treatment the convulsions cease, the patients come out of the coma, and all symptoms clear up or at least show marked improvement. The blood pressure may fall to normal, the albumin and casts disappear, and after a variable length of time, in one instance as long as twenty-two days, spontaneous labor occurs without any recurrence of the toxemic symptoms. Unfortunately, this is not the general rule, as in most cases, following a temporary improvement, the symptoms once more increase in severity and become so threatening as to demand the induction of labor. This, however, is not so serious as it sounds, for it enables us to cope with the condition by a relatively simple procedure instead of resorting to radical operative intervention with its associated anesthesia while the patient is in the eclamptic state.

Lichenstein was the first in recent years to direct our attention to this so-called intercurrent eclampsia, and many of his cases, as well as several of those in our series, followed free venesection instead of treatment by narcotics. Such observations are of great importance, as they teach that the eclamptic outbreak is not inevitably due to the existence of a living pregnancy, but may be due to transient conditions, and if they can be temporarily overcome, the normal status becomes reestablished, so that pregnancy may continue without further complication. What it means we have yet no means of knowing, and our ignorance will probably persist until the actual cause of the disease is discovered; but it indicates beyond peradventure that in certain instances at least the termination of pregnancy is not essential to the cure of the disease.

In conclusion, it seems fair to say that in the past not a few of our patients perished who would now recover under conservative treatment, so that pending the discovery of the actual cause of eclampsia, which will inevitably be followed by rational instead of empiric treatment, we should constantly bear in mind the old motto, primum non nocere.

CONCLUSIONS

1. In mild as well as severe cases of eclampsia, the results are better under conservative than under radical treatment.

2. In mild cases, a modified Stroganoff technic gives almost ideal results.

3. In severe cases, such treatment gives twice as good results as more radical treatment, but is still followed by a mortality so high as urgently to demand improvement.

4. It appears that all the generally used anesthetics superimpose an additional toxemia on that associated with the disease.

5. Consequently, the operative treatment of severe eclampsia will probably not show better results until some nontoxic anesthetic is discovered.

6. It appears that, after accouchement forcé, cesarean section performed under the usual general anesthetics is the worst treatment for eclampsia.

7. The necessity for still greater extension of prenatal care is the most efficient means for the prevention of eclampsia.

8. It is necessary to realize that toxemia of pregnancy is a vague general term, and that we have to deal with several types and not with a single one.

9. The treatment of eclampsia must remain empiric and relatively unsatisfactory until the actual cause of the disease is discovered.