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CLINICAL TYPES OF PREGNANCY TOXÆMIA[1]

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IN the present paper my attempt is merely to set forth the clinical types of pregnancy toxæmia as I have observed and studied them in my hospital and private practice during the past year or more.

The report is preliminary and imperfect in character, and I am aware that the clinical manifestations in the urine do not always indicate a special toxæmic condition, renal, hepatic, or intestinal. Still, I am of the opinion that all the clinical types of pregnancy toxæmia are closely related, and have possibly a common origin, and the apparently negative urine analyses are due to our present imperfect methods of examination in these cases.

I shall describe seven readily recognizable types, namely:

I. Benign type.
II. Subacute type.
III. Acute type.
IV. Fulminant type.
V. Toxæmic coma without convulsions.
VI. Acid intoxication, simulating hepatic toxæmia of pregnancy.
VII. Cumulative toxæmia.

To these I am adding another type, which I am, at present, unable to classify, namely:

VIII. Persistent hyperemesis without clinical symptoms of toxæmia.

I. BENIGN TOXÆMIA

The benign type comprises the ordinary special petty morbidity of the early months of pregnancy. It is present in many primipara and in a large percentage of all pregnancies. Its most striking feature is its tendency to self-limitation. There is no marked disposition to become worse, and often the reverse is noted up to the time of cessation. Hence it is a slight transitory type of affection.

The symptoms are referred in about equal measure to the gastro-intestinal tract and nervous system. Nausea, vomiting, anorexia, constipation, and perverted tastes appertain to the former, and general nervous irritability, hysterical tendencies, alterations of the mental condition, as exaltation, depression, eccentricity, etc., to the latter. I would enumerate, under benign toxæmia, the chloasma, puritis, etc., which so frequently involves the skin, and which are often in evidence in other toxic states. A study of the urine usually reveals anomalies of metabolism. Various symptoms may be present, which, however disagreeable and atypical they may be, are not necessarily evidences of active toxæmia, as salivation, vertigo, etc.

The chief interest attached to the benign type of toxæmia lies in its possible relationship to the graver forms. Its very great frequency, with its natural tendency to disappear spontaneously before or during the fifth month, causes us to regard it lightly. Even if it does not disappear at this period, it may still be devoid of any bad consequences. Each case, however, must be studied individually. Many factors, insignificant when considered singly, may have a bearing on particular cases. The family and personal history of the woman in regard to biliousness, dysmenorrhœa, and previous pregnancies, the rapidity with which she is bearing her children, her condition after previous deliveries, must all be borne in mind. Even those cases in which a woman feels better in every way when pregnant must not be

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too lightly regarded, for this very state of well-being, of euphoria, has, in itself, something abnormal, as may often be verified.

Generally speaking, however, we cannot look upon mild toxæmia of pregnancy as having a serious prognosis. The spontaneous cessation of its symptoms in such a vast majority of cases argues for the existence of a special defensive mechanism in the internal organism. I cannot yet venture to explain why this provision occasionally fails.

II. SUBACUTE TOXÆMIA

In the present connection I am using the term *subacute toxæmia* for the clinical type, which differs from the acute form only in degree. Thus, even so-called yellow atrophy of the liver, which coincides so frequently with acute toxæmia of pregnancy, is by no means always clinically acute. It may be prolonged for weeks, and exceptionally for months.

The pre-eclamptic state, so-called, is an excellent example of subacute toxæmia. While chiefly known as the precursor of eclampsia, a very similar condition has been described under other circumstances, and as a simple clinical phenomenon. These cases may recover under treatment, and possibly spontaneously. They may disappear after delivery. When death occurs, autopsy may be impossible. There is no doubt that cases coming to autopsy will present structural disease of the liver, perhaps of slight degree. Even marked involvement of the organ does not necessarily exclude the possibility of regeneration.

A few years ago severe toxæmia of pregnancy meant only a few well-marked diseases associated with certain viscera—hepatic eclampsia, renal eclampsia, and, perhaps, pernicious vomiting. Now, we find cases of pregnancy toxæmia which corresponds to none of the above classic types. Roughly speaking, they resemble the pre-eclamptic state, especially as it affects the nerve centers. Vomiting, renal insufficiency, mild jaundice, etc., may or may not be present. In many cases recovery occurs, and we have little means of knowing exactly what took place, or whether the organism has sustained some organic lesion. It is to this type of cases that the term subacute or subacute uncomplicated toxæmia is peculiarly adapted.

CASE I. SUBACUTE TOXÆMIA

Mrs. J. H., seen in consultation with Dr. Charles Henry Moak, of New York, in the night of February 14, 1905.

Age 27; primapara; eighth month of gestation; Patient had enjoyed good health during her pregnancy up to within one week of her eclamptic attack, when œdema appeared, and extended up the lower extremities. Persistent headache and gastric disturbances also occurred at this time.

Prior to this time, Dr. Moak tells me, urine analysis was negative, but with the appearance of the headache and gastric disturbances, albumen was found in the urine, with a few hyaline casts.

Mrs. H. was put upon a milk diet, encouraged to drink freely of water, and free catharsis produced.

An improvement in her condition was observed, but upon the afternoon of February 14, 1905, she had five well-marked eclamptic convulsions. I saw the patient in the evening, and found her with some slight rise in temperature and pulse, the latter with considerable tension, and with the exception of some excitement, as the result of the convulsions, in excellent condition. Mentally, she was perfectly clear.

While I was making my examination, Mrs. H. had a typical eclamptic convulsion. I then recommended that the uterus be immediately emptied, but before the kitchen table could be gotten up, and other preparations made for operation two more convulsions occurred, in spite of the use of chloroform, making eight in all. During one of these last convulsions the patient's tongue was severely bitten.

Preparations being complete, I found no attempt at cervical dilatation, the vaginal portion of the cervix being still present.

By means of Bossi's dilator, followed by bimanual dilation, I obtained a full dilatation of the os, and delivered a living child by podalic version and extraction in forty minutes.

No serious lacerations were produced. Before delivering the placenta, and while the patient was still on the operating table, I irri-

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gated the colon myself with gallons of decinormal saline solution.

The patient was put to bed, thirty grains of compound jalap powder administered as soon as she could swallow, and the free exhibition of veratrum viride ordered.

Mrs. H. suffered from only one convulsion after the emptying of the uterus, that within an hour of the operation. Repeated colonic washings were continued.

Mrs. H.'s mental condition remained dull and apathetic until noon of the following day, February 15, 1905, when her normal mental condition returned, and she made a rapid and uninterrupted recovery.

The urine analysis in this case was particularly interesting to me, as at no time were there symptoms of marked toxæmia, with the exception of the convulsive attacks, nine in all. In spite of these nine eclamptic convulsions, I look upon such cases as subacute in character by reason of the absence of other toxæmic symptoms.

The urine analysis is from a twenty-four-hour specimen, collected February 15th, and with the exception of the low urea percentage does not give evidence of toxæmia, either of the gastro-intestinal, hepatic, or renal origin. Casts were not present in such number as to indicate marked degenerative changes of the renal parenchyma.

February 17, 1905.
Patient, Mrs. J. H. Report No. 6799.

URINE ANALYSIS

Nature of Specimen, Not stated.
Specific Gravity, 1.016.
Volume, 680 cubic centimeters; 23 fluid ounces.
Reaction, Amphoteric.
Albumin, A small amount is present. Quantity stated below.
Sugar, Absent.
Appearance, Turbid and cloudy.
Color, Reddish.
Ehrlich's Diazo Reaction, Negative.
Bile, Absent.
Indican, Trace.

Quantitative Determinations.	Percentage Grammes in 100 c. c.	Grains in a Fluid Ounce	Grammes.	Amount in Specimen Grains.
Urea	1.34	6.12	9.10	140.8
Chlorides	0.83	3.78	5.64	86.9
Albumin, Small amnt..	0.079	0.36	0.54	8.3

Sugar, Absent.
Leucin and *tyrosin*, Absent.

MICROSCOPICAL EXAMINATION OF THE SEDIMENT

Mucus, Small amount.
Blood, Large amount.
Pus, Moderate amount.
Casts, Small number, hyaline.
Phosphates, Few crystals.
Urates, Absent.
Uric acid, Absent.
Calcium oxalate, Absent.
Epithelia, Moderately large number, bladder and vaginal.
Bacteriological examination not made.

III. ACUTE TOXÆMIA

The acute type I have found a fairly constant clinical type in several respects, although subject to much individual variations. It is constant in its pernicious character, when once established, and in the extreme preponderance of cerebral and nervous symptoms. A prodromal stage may be present, resembling the so-called subacute type in its manifestations. This is important to bear in mind, for then if at any time will treatment promise any salutary results. I have observed cases in which, apparently, there was no prodromal stage. The disease proper usually begins with symptoms of cortical excitation—restlessness, agitation, insomnia, confusion, passing into convultions, maniacal agitation, and delirium. This stage leads inevitably to another, characterized by cortical depression—apathy, hebetude, somnolence, stupor, coma, and perhaps death. In exceptional cases the stage of excitation is but slightly, or not at all, in evidence, the patient passing at once into the terminal manifestations.

Acute toxæmia usually ends fatally in two or three weeks. When it continues much longer —it may exceptionally persist for many weeks— it is none the less fatal, although no longer meriting the title acute.

The earliest cases of acute toxæmia are those instances of acute hepatitis and acute yellow atrophy occurring in the gravida. Nearly all have jaundice. We have since learned that the same clinical course may, and usually does, occur without either jaundice or extensive lesions of the liver. In fact, the small area of the latter has led to the belief that the liver, like other parenchymatous organs, may undergo a functional paralysis without necessary gross tissue changes.

The more acute cases supervening in the midst of health, without prodromes, tend to

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show that the liver may suspend its action suddenly. The changes that are present in the liver in such varying degree may only indicate that, after the cessation of its functions, certain areas of the parenchyma are destroyed by autolysis. In any case, the alteration in the liver is not the measure of this disease, and diagnosis and prognosis should not be based upon physical examination of this organ. Normal boundaries, absence of tenderness, etc., should have no weight. The urine is a much more valuable index (presence of leucin), but even this is not infallible. Whenever a woman is taken more or less violently ill without apparent cause, and especially when near the time of her menstrual period, we must always think of the possibility of acute toxæmia of pregnancy. The cortical symptoms, already described, naturally point to this condition. Headache, vomiting, jaundice, general prostration, tenderness over the liver, and certainly the presence of leucin and tyrosin in the urine, strengthen the diagnosis. The patient tends to become worse, yet there is comparatively little tendency to abortion. Cases which linger longer than three weeks evidently represent a milder type of disease.

We must bear in mind that acute toxæmia of pregnancy may be simulated by various affections, as yellow fever, phosphorus poisoning, and acid intoxication of pregnancy (case iv) might lead to confusion.

CASE 2. ACUTE PREGNANCY TOXÆMIA

Mrs. C. G., I saw in consultation with Dr. Q. Feldstein at 8:30 P. M., Christmas eve, December 24, 1904. She was a primipara, twenty-six years of age, and seven and a half months pregnant.

During her pregnancy she had enjoyed good health; and during the afternoon of December 24 had been engaged in her Christmas shopping.

In the evening of this day she suddenly had an eclamptic convulsion, and she had had two when I saw her, and a third occurred during my examination. I advised immediate emptying of the uterus, so a private ambulance was secured, and she was transferred to my service at the Emergency Hospital of Bellevue, and was on the operating table at 9:44 P. M. of the same evening. Mrs. G. here had her fourth convulsion. Four ounces of urine was obtained by catheter for analysis, and under chloroform a rapid manual dilatation of the os was performed, followed by direct podalic version, and the delivery of a living female child, weighing three pounds, six ounces, at 11:05 P. M. The patient was put to bed at 11:20 P. M., and treated with hot packs, colon washing, veratrum viride, large doses of calomel, etc.

Between the hours of one and seven A. M. of Christmas day, this patient had seven convulsions at lengthening intervals, making thus far ten convulsions in twelve hours. After the second convulsion at her home, the intervals were practically conditions of coma, and the patient was apparently blind after the third convulsion. Four more convulsions occurred during Christmas day, making fourteen convulsions in all.

For three days more the patient remained in a comatose condition, and was unable to recognize her husband or family physician until the fifth day post-partum.

After the fifth day, according to her family physician, she returned to her normal condition.

The baby survived for two weeks, and died in convulsions, January 8, 1905.

The first urine analysis, December 25, 1904, showed parenchymatous renal degeneration and renal insufficiency. A trace of acetone was found.

A specimen of urine sent to the Cornell Pathological Laboratory was reported on by Dr. James Ewing, as follows: Quantity, 500 c. c.; bloody; sp. gr., 1.019; reaction, neutral; considerable albumin; indican much increased; many hyaline, epithelial, and granular casts.

Urea (hypobromite method), .642 grms. per 100 c. c. Ammonia, .272 grms. per 100 c. c. In sediment ppt. by lead acetate were many leucin crystals.

Slight jaundice was present in this case, and no apparent physical changes in the liver.

This case I class as one of acute pregnancy toxæmia. The onset was sudden and severe, the pre-eclamptic state being absent, or having escaped the notice of the family physician.

The severity and great number of the con-

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vulsions (namely, 14), the prolonged and profound post-partum coma and stupor, the presence of acetone and leucin in the urine, and the marked parenchymatous renal changes, all attest to the acuteness of the toxæmia present.

The patient made fair recovery, and left the hospital January 17, 1905, in an anæmic condition. I have since heard repeatedly from this patient, and her health has been far from satisfactory for the past four months.

December 25, 1904.
Patient, C. G., at Emergency Hospital. Report No. 6424.

URINE ANALYSIS

Nature of Specimen, During eclampsia.
Specific Gravity, 1 032½.
Reaction, Acid (moderately strong).
Albumin, A very large amount is present. Quantity stated below.
Sugar, Absent.
Appearance, Smoky.
Color, Deep amber.
Ehrlich's Diazo Reaction,
Bile, Absent.
Indican, Absent.

Quantitative Determinations.	Percentage Grammes in 100 c. c.	Grains in a Fluid Ounce.
Urea, Much diminished	1.34	6.12
Chlorides, Normal.....	1.12	5.10
Albumin, Very large amount.....	6.20	28.25

Sugar, Absent
Acetone, Trace only.
Diacetic acid, Absent.
Mucus, Small amount.
Blood, Small number much distorted red corpuscles.
Pus, Absent.
Casts, Numerous granular, some containing fat granules. Moderate number hyaline and epithelial.
Phosphates, Absent.
Urates, Absent.
Uric acid, Absent.
Calcium oxalate, Absent.
Epithelia, Moderate number, bladder and renal.
Leucin and *tyrosin*, Absent.
Bacteriological examinations not made.

IV. FULMINANT TOXÆMIA

The term fulminant may be applied to the most intense type of acute toxæmia of pregnancy, or to sudden death occurring from a masked toxæmia. I am referring here to those cases in which the clinical manifestations resemble closely the fulmiant types of acute infectious diseases, in which death may result before a diagnosis can be made. The symptoms consist essentially of sudden intense prostration, including cardiac failure, anomalies of temperature, and early and progressive implication of the cerebral cortex, which leads to early and fatal coma. As these cases may prove fatal in a day, there are but few—often no opportunities for clinical observation, and the autopsy is of the utmost importance. In uncomplicated toxæmia of pregnancy the liver should be found the seat of alterations, although the extreme rapidity with which death supervenes may be due to cardiac collapse, severe meningitis, or some other factor. Exact diagnosis is important in order to exclude the suspicion of poisoning, suicide, etc. In some instances, sudden death in pregnancy is purely a coincidence—for example, when multiple fat necrosis is found at autopsy. Numerous other coincidences will readily suggest themselves. On the other hand, how often, in cases of sudden death, has the fact of pregnancy, especially early pregnancy, been recorded, without the slightest attempt to connect the lesions found with the pregnant state?

Of sudden death in pregnancy or the puerperium with the patient in apparent health, little is known. Ewing and Norris have attributed as paradoxical cardiac failure to the action of pregnancy toxins upon circulatory organs. Cardiac thrombosis, primary pulmonary embolism, and other affections have been placed in this category.

CASE 3. ACUTE FULMINANT TOXÆMIA

K. M.; para, 1; age twenty-five; no miscarriages or abortions; in the ninth month of pregnancy was received as an antepartum case at the Bellevue Emergency Hospital, May 2, 1905. Nothing unusual was noticed about the patient except that she was slightly jaundiced and that she complained somewhat of nausea and vomiting.

The routine urine analysis of the hospital revealed a moderate amount of albumin.

Specific Gravity, 1.013.
Faintly alkaline.
No sugar.
No casts, leucocytes.
Urea, 4½ grains to Oz. 1.

Upon May 5th the jaundice and vomiting increased, the latter now almost persistent. This went on until May 9th, with increasing amount of albumen and more marked jaundice, when it was decided to empty the uterus. This was done upon May 9th, by manual dilatation of

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the cervix and podalic version. A living child, jaundiced, was delivered.

The patient stood the operation well, but the nausea, vomiting, and jaundice increased. The liver was found to be sensitive, but not noticeably diminished in size.

Upon May 11, two days following operation, the patient had the only convulsion, and so slight that it would probably have escaped observation had not the nurse been standing by the bed. Stupor approaching coma followed the convulsion, which continued until 3:30 P. M. of May 12th, when the patient died in coma, about nine days after the severe vomiting began.

Laboratory examination of the urine before death revealed a small amount of albumen, no casts, moderate amount of indican, leucin and tyrosin, attested to by Dr. James Ewing, of Cornell Medical College.

A full autopsy was performed, a report of which will be subsequently published. The kidneys were normal, the liver weighed [illegible] pounds, and showed acute yellow atrophy.

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V. TOXÆMIC COMA WITHOUT CONVULSIONS

The acute toxæmia of pregnancy may pass directly into coma without any preceding stage of excitement. Under such circumstances we commonly find an acute lesion of the liver, or in very rare instances some other acute organic lesion (kidney, meninges). This type of comatose state belongs in reality under acute toxæmia including its fulminant variety.

Of quite a different type is eclampsia without convulsions. This develops late in pregnancy with the classic picture of the pre-eclamptic state, Convulsions do not occur, or may be very slightly present. Instead, we find somnolence, stupor, or actual coma. Some of these cases may be regarded as abortive (perhaps aborted by prophylactic treatment); others are undoubtedly of unusual gravity.

Transition forms occur between these two types of coma. Thus, in clinical eclampsia without pregnancy-kidney, but with marked lesions of the liver, we see a connecting link between acute toxæmia of pregnancy and eclampsia. If, under such circumstances, a well marked pre-eclamptic stage terminated directly in coma the clinical type would be subacute toxæmia.

CASE 4. TOXÆMIC COMA WITHOUT CONVULSIONS.

Mrs. T. was seen by me, in consultation with Dr. Charles A. Mansen, of New York, upon November 14, 1904.

Mrs. T., at this time, was three and a half months pregnant, and was in a semi-comatose condition, which, at first, bore some resemblance to an hysterical condition.

Her pulse was 120, and hard; temperature, 101 F., and respirations slower than normal. Her pupils were moderately contracted, and responded poorly to light; there was moderate œdema of the ankles, no puffiness about the eyes or face, and the area of hepatic dullness was normal. Not at this time, nor subsequently, was hepatic tenderness observed.

The present pregnancy was the second. The first was three years previous, and at that time the patient suffered from an acute eclamptic attack at the fifth month, and was delivered by rapid dilatation and extracted after several eclamptic convulsions at the Sloane Maternity. During the present pregnancy, Mrs. T. had suffered from persistent nausea and vomiting, and what was described as an aggravated form of "acid indigestion," from which she could obtain no relief. Not being entirely satisfied with the diagnosis, by reason of the apparent hysterical condition, I advised that free catharsis be produced, and the colon irrigated repeatedly until a urine analysis could be obtained. A specimen of the urine was sent to the laboratory of Dr. E. E. Smith, with the request for an immediate report thereon. This was obtained in a few hours, and was rather a surprise:

Specific Gravity, 1.017.
Reaction, Strongly acid.
Sugar, Absent.
Bile, Absent.
Indican and *albumen*, Absent.
Urea, 1.95 grammes in c. c. 8.87 grains in fluid ounce. Small number granular casts.
Phosphates, *urates*, *uric acid*, and *calcium oxalate*, Absent.
Quantitative determinations at this time not made.

I saw the patient again on the evening of the same day, and found the coma more marked and the hysterical manifestations to have entirely disappeared; temperature and pulse the same as in the afternoon.

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In view of Mrs. T.'s previous eclamptic history and the deepening of the coma in spite of the catharsis and colon irrigation, I advised the prompt emptying of the uterus, which was performed by rapid cervical dilatation and curettage. The operation was complete within forty minutes, and the colon irrigations continued, and veratrum viride added to the treatment to reduce the frequency and tension of the pulse.

Under this treatment the stupor gradually lessened, so that there was marked improvement in twenty-four hours, but Mrs. T. did not return to what her family considered to be her normal mental condition for nearly one week.

The total amount of urine obtained in the first twenty-four hours following operation, was sent to the laboratory for a more complete analysis.

The report showed a strongly acid urine, albumen, sugar, bile, and indican absent; small granular casts; no phosphates, urates, uric acid, or calcium oxalate; urea increased in amount over the specimen of the preceding day.

Additional quantitative determinations showed acetone, 0.25 grammes in 100 c. c. 1.28 grains in fluid ounce.

Diacetic acid present with a moderately strong reaction. Ammonium salts present in unusual amount. The laboratory comment upon this specimen was as follows:

"The findings in the urine at this time do not give evidence of a gastro-intestinal toxæmia, and do not give very material evidence of renal changes, a small number of granular casts being the only feature pointing to a renal process. The striking feature of the specimen is the evidence of acid intoxication, a large excess of acetone, diacetic acid and an excess of ammonium salts being present. These findings point to the excessive formation and presence of B-oxybutyric acid, producing an acidæmia of a sufficient degree of severity to explain the acid vomiting and even the existence of coma."

A subsequent twenty-four hour specimen of urine was sent to the Cornell Pathological Laboratory and both Ewing and Beebe attested to the above condition, and also to the presence of leucin.

The urine analysis in this case at first pointed to toxæmic coma, due to acid intoxication. A further analysis of a twenty-four hour specimen, however, revealed the presence of leucin in the urine, thus pointing to hepatic toxæmia as the cause of the coma.

We have thus, in this case, an acid toxæmia complicating an hepatic toxæmia.

VI. ACID INTOXICATION SIMULATING HEPATIC TOXÆMIA OF PREGNANCY

The relationship of acid intoxication, as represented by acetonemia to pregnancy, is obscure. Slight so-called physiological acetonuria is very common in pregnancy, and seems to possess no significance. Acid intoxication proper is said to have been recognized in some cases of fatal toxæmia of pregnancy, and since this condition is manifested by coma, and in some cases by convulsions, it has been accused of causing eclampsia.

As acid intoxication occurs under a great variety of conditions—diabetes, cancer, various gastro-intestinal affections, poisoning, etc. —its presence in pregnancy is doubtless secondary to some other condition rather than to the special or hepatic toxæmia.

Acid intoxication in pregnancy had best be regarded as a condition which may simulate the hepatic toxæmia of pregnancy, especially the comatose forms. Differentiation should be made by urinary tests—the presence of the acetone bodies in unusual amounts with the corresponding absence of the positive findings of hepatic toxæmia.

It is, of course, possible that the two conditions may exist side by side (case 4), and even that acetonemia may be in some way due to the other toxic state (for it has been noted in many kinds of poisoning); but thus far the evidence is very vague. The subject of acid intoxication is one which becomes more complex the more we know of it.

Case 4 at first was considered to be one of coma from acid intoxication. More extended urine analysis, however, revealed the presence of hepatic toxæmia as well.

VII. CUMULATIVE TOXÆMIA

It has long been recognized that rapid childbearing has elements of danger for women, and that even constant breeding with two or

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three year intervals tends in the course of time to react upon the mother, and to increase her exposure to some of the special perils of child-bearing. It is only very recently, however, that the possibility of a cumulative toxæmia of pregnancy has been considered to account for at least a good share of the risks run by women from rapid or excessive child-bearing.

We can hardly speak of a clinical type of cumulative toxæmia since we are dealing not so much with special symptoms as with the history of the patient, and with her condition between pregnancies. If we find that a woman who has passed safely through the puerperal state after having exhibited some form of hepatic toxæmia during pregnancy, continues to show evidences of hepatic insufficiency, or if some local toxic manifestation (pregnancy-kidney, neuritis, etc.) does not undergo the expected resolution, or respond to treatment; and finally if the blood-count shows persistent alterations pointing to anæmia or leukemia, the prognosis of the case forbids gestation until all such symptoms have disappeared. Generally speaking, however, cumulative toxæmia implies such rapid child-bearing that the woman is seen only when actually pregnant. If we find her suffering from any form of toxæmia which while not necessarily severe in itself is graver in character than that presented in the antecedent pregnancy, or if with each succeeding pregnancies the woman exhibits more and more evidence of hepatic insufficiency, we may suspect the presence of cumulative toxæmia. We must not always expect to see these succeeding pregnancies become worse in a logical or orderly sequence; although this tendency is visible in hyperemesis and eclampsia. On the contrary, two or more pregnancies with relatively mild toxæmia may be followed by a third in which the patient perishes without other warning from the fulminant type of toxæmia. If in addition to the evidence of cumulative toxæmia already stated, we find a family history of "biliousness" (cholemia), with tendency of the woman's nearest female relatives to suffer unduly in pregnancy, or a personal history of dysmennorrhœa with mild toxic symptoms, the least we can do with such a woman is to place her in a special class, for which rapid child-bearing is extra hazardous, and pregnancy under any circumstances not to be lightly undertaken.

VIII. PERSISTENT HYPEREMESIS WITHOUT CLINICAL SYMPTOMS OF TOXÆMIA: PSEUDO-TOXÆMIA

I occasionally see cases in which hyperemesis does not cease before the eighth month, but continues despite all treatment until near delivery. In this type of cases the vomiting does not become pernicious in character, but its persistence causes much apprehension in the minds of all concerned. My belief is that these gravidæ have suffered early in pregnancy from slight, transitory, so-called benign toxæmia, but if we examine them carefully in the latter months nothing suggesting toxæmia can be elicited. The urine is free from evidence of disturbed metabolism, and there is nothing which points to renal insufficiency. The mental state is normal, as is also the nervous system, save for the exalted reflex excitability which may be supposed to underlie the tendency to vomit. The fetus does not appear to present any anomalies. We may find that these women have vomited throughout all their pregnancies (as in case 5). It is perhaps hardly just to associate these cases with actual toxæmia of pregnancy, save in connection with differential diagnosis, which is of the utmost importance. But, bearing in mind the rudimentary character of our present knowledge of the toxæmic states of pregnancy, we must watch these cases carefully, even although the clinical picture of a toxæmia is not in evidence. We must watch them not only through labor, but into the puerperium. If a toxic state, latent in its other manifestations, is behind the hyperemesis, it may assert itself in some form after delivery.

As already stated, vomiting in these cases should subside after delivery. It is unnecessary to speculate here on the nature of this clinical type, and wherein it differs from other types. Its chief bearing lies in prognosis. We must not err on either side. The prognosis, once the type is recognized, is good; but not so good as to exclude special watchfulness through the puerperium.

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CASE 5. PERSISTENT HYPEREMESIS WITHOUT CLINICAL SYMPTOMS OF TOXÆMIA

Mrs. L. was referred to me for her confinement by Dr. Charles Gilmore Kerley. Case first seen by me in December, 1904, and was confined at full term of a healthy living child in the latter part of March, 1905. Patient had had four miscarriages, and has seven living children.

In every pregnancy, without exception, beginning in the first month, this patient has persistent daily nausea and vomiting from arising in the morning until noon.

This condition continues until three or four weeks before delivery. At no time has there been any jaundice, and the patient has considered herself always healthy, looking upon the nausea and vomiting as a necessary accompaniment of pregnancy.

In the first pregnancy Mrs. L.'s condition became alarming by reason of the persistent vomiting, and she was kept in bed for the second, third, and fourth months of this pregnancy. She was in another city at this time, and was seen by several consultants with the view to emptying her uterus to stop the vomiting.

This case was of great interest to me and I watched her carefully for clinical symptoms of toxæmia without being able at any time to detect any.

Her mental condition was at all times normal and satisfactory, and repeated unrinary examinations by an expert failed to show evidences of toxæmia, although moderate amounts of indican were at times found. We were never able to detect any leucin and tyrosin in the urine. I append a urine analysis made February 17, 1905, one month before confinement; which is typical of all the examinations of the urine.

This patient was last seen by me May 15, 1905, when her physical and mental condition appeared of the best, she had gained in weight since her confinement, and she stated that she never enjoyed better health.

LABORATORY

February 17, 1905.
Patient, Mrs. L. Report No. 6800.

URINE ANALYSIS

Nature of Specimen, Not stated.
Specific gravity, 1.020.
Reaction, Slightly acid.
Albumin, None found tests for traces obscured by the persistent turbidity
Sugar, Absent.
Apperance, Turbid.
Color, Normal amber.
Ehrlich's Diazo Reaction, Negative
Bile, Absent.
Indican, Moderate amount.

Quantitative Determinations.	Percentage Grammes in 100 c. c.	Grains in a Fluid Ounce
Urea	1.21	5.50
Chlorides	0.89	4.06
Albumin		
Sugar, Absent.		

MICROSCOPICAL EXAMINATION OF THE SEDIMENT

Mucus, Small amount.
Blood, Absent.
Pus, Absent.
Casts, Absent.
Phosphates, Absent.
Urates, Absent.
Uric acid, Absent.
Calcium oxalate, Absent.
Epithelia, Moderate number bladder and vaginal.
Bacteriological examination not made.

50 East 34th Street, New York City.

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