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Further Contributions to Our Knowledge of the Pernicious Vomiting of Pregnancy.*

By J. WHITRIDGE WILLIAMS, M.D.,

Professor of Obstetrics, Johns Hopkins University, Baltimore; Honorary President of the Glasgow Obstetrical and Gynæcological Society (1910–12).

I CAN perhaps interest the members of this Society by bringing to their attention the results of my investigations concerning the nature and treatment of pernicious vomiting of pregnancy. Some of you may recollect my monograph upon the subject which appeared in 1905, in which I reviewed the literature and reported my observations concerning certain pathological and metabolic changes occurring in the disease. The following year I still further elaborated my views in an article entitled "Toxæmic Vomiting of Pregnancy."

Since that time I have written nothing further upon the subject, but I have had an opportunity to observe a considerable number of cases, which I have studied as thoroughly as possible. This increased experience has served to confirm in great part my original views, but at the same time it has forced me to recognize that some of my conclusions were too far-reaching, and consequently are in need of revision. It is my purpose upon this occasion to discuss with you, frankly and informally, various aspects of the subject, and then to indicate to what extent my original conclusions must be modified.

In my original articles I held that the evidence then available justified the classification of cases of pernicious vomiting into three groups—reflex, neurotic and toxæmic. I made no contribution to our knowledge of the first two varieties, but I pointed out that the toxæmic form should be sharply differentiated from others, as it is associated with profound degenerative lesions in the liver and kidneys, as well as with striking changes in metabolism, which are characterized by a marked distortion in the nitrogenous partition, more

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particularly manifested by an increase in the proportion of the total nitrogen excreted as ammonia. This so-called ammonia co-efficient, which in normal pregnant women varies between 3 and 5 per cent., may become markedly increased, rising in one of my cases to as high as 48 per cent. After discussing the various factors which might be concerned in bringing about such changes, I was obliged to confess my ignorance concerning the mode of production of the high ammonia co-efficient, but I held that it indicated the existence of a toxæmia, and concluded that when it exceeded 10 or 15 per cent. it should be regarded as an indication for the prompt termination of pregnancy.

Accordingly, I recommended the utilization of the ammonia co-efficient as a means of diagnosis, and held, if it were markedly elevated in a given case of pernicious vomiting, that one had to deal with the toxæmic type; while if it were normal the diagnosis lay between the reflex and neurotic variety; the differentiation between the latter being made by the presence or absence of palpable abnormalities of the generative tract. The diagnosis having been established, treatment followed as a matter of course. In the reflex variety, relief followed the correction or removal of the abnormality of the internal genitalia; in the neurotic variety cure should be promptly effected by suggestive treatment, rest in bed, and forced feeding; while in the toxæmic variety prompt interruption of pregnancy was essential in order to check the underlying toxæmia, and, if possible, to give Nature a chance to bring about repair of the organic lesions.

The immediate result of these teachings was a marked reduction in the number of cases requiring the induction of abortion; the discovery that most cases of serious vomiting were neurotic in origin, and therefore amenable to suggestive treatment; and, finally, the recognition that the reflex variety of vomiting occurred but rarely, and even when cure followed the correction or removal of the condition which was supposed to give rise to the reflex irritation, that one remained uncertain whether it was the result of the treatment employed or was merely due to its suggestive influence.

In my original article I was not dogmatic concerning the significance of a high ammonia co-efficient, but, in justice to myself, I feel that I should direct your attention to what I said in that connection in 1905, more particularly because certain of my critics appear to have assumed that I held that the only explanation for such a finding was the existence of a toxæmia complicating the early months of pregnancy.

In view of the fact that in fatal cases the liver presents evidences of extensive fatty degeneration, and occasionally of central necrosis of the individual lobules, quite as extensive as in advanced stages of acute yellow atrophy, I suggested that the high ammonia co-efficient might be due to the inability of the diseased organ to effect complete

oxidation of the nitrogenous material brought to it; but at the same time I was well aware that such an explanation was more than problematical, as we possessed abundant evidence to prove that the preservation of relatively minute quantities of liver tissue may suffice for the ordinary purposes of metabolism. I also pointed out that similar variations in metabolism had been observed in acute yellow atrophy of the liver, as well as in cases of poisoning by phosphorus. Furthermore, I directed attention to the high ammonia co-efficient observed in diabetes and other varieties of acidosis, and also stated that it was observed in certain forms of gastro-enteritis in children, as well as in adults suffering from starvation, and, finally, that it might even follow radical changes in the diet of perfectly normal individuals.

After adducing these facts, I cautiously concluded as follows:— "This being the case, I feel at present that I must remain content with demonstrating that in certain cases of toxæmic vomiting of pregnancy there is a marked disturbance in metabolism, which is manifested by a great increase in the ammonia co-efficient, and that it must be left to future investigations to determine whether the change is directly due to the inability of the diseased liver to effect complete oxidation, or whether it is a manifestation of an acid intoxication, or of some other condition. Moreover, we are absolutely ignorant concerning the nature of the toxic material concerned, and whether it is derived from the mother or fœtus."

Since expressing these views a very considerable literature has accumulated upon the subject, while my own experience has been increased by the careful study of nearly forty patients suffering from serious vomiting of pregnancy. I shall now proceed to consider the criticism to which my views have been subjected, and to indicate to what extent they are justified.

My conclusions have been confirmed in general by Winter, Hofbauer, Czyzewicz, Edgar, Cragin and others; but at the same time they have been subjected to more or less criticism by Leathes, Longridge, Rand and Underhill, Whipple and Sperry, and M'Donald. For the present it is unnecessary to consider critically the views of the former group of writers; but on account of the high standing and recognized merit of those in the latter group, it is essential to give respectful consideration to their views, which I am quite prepared to admit are in part justified, and, together with my own more extended experience, have caused me to modify my own views materially.

Leathes, after directing attention to the fact that a high ammonia co-efficient may be due to various factors, and does not necessarily indicate impairment of the hepatic function, stated that before such a finding could be considered to possess any great diagnostic or prognostic significance in a given case of pernicious vomiting, it would be necessary to take various considerations into account,

particularly the low nitrogen content of the absorbed food, the imperfect nutrition due to incessant vomiting, and the loss of alkali in the vomit, aggravated, possibly, by the requirements of the fœtus. Furthermore, he was inclined to predict that if in the future any prognostic significance could be attached to manifestations of perverted metabolism, it would probably be derived rather from variations in the ratio of the so-called "undetermined nitrogen" than in the ammonia co-efficient.

Longridge, in an excellent article which was based upon theoretical considerations rather than upon personal experience, was inclined to believe that the high ammonia co-efficient accompanying pernicious vomiting should be regarded as a manifestation of an acid intoxication incident to inanition rather than as indicating the existence of a toxæmia. In any event, he held that the limit (10 to 15 per cent.) which I had set as indicating the necessity for the termination of pregnancy was too low, as similar figures may be noted in normal individuals after a radical change in diet. At the same time, he stated that the entire question was still *sub judice*, and that it would be rash to express a definite opinion until additional evidence and experience became available.

Rand and Underhill, in a very painstaking article, which was based upon the accurate metabolic study of four cases of serious vomiting of pregnancy, one of which presented an ammonia co-efficient of 47 per cent., expressed the belief that the urinary changes are identical with those observed in starvation, and are not necessarily indicative of the existence of a toxæmic condition. Furthermore, they hold, in those cases in which inanition is a significant factor, that the exhibition of carbohydrates is of greater importance than proteid material. It should, however, be noted that in one of their cases induction of abortion was necessary in order to save the life of the patient; while in another instance Rand and Underhill did not consider that the patient was suffering from pernicious vomiting, but rather believed that the symptoms were feigned in the hopes of getting rid of an undesired pregnancy.

Following the work of Howland, Whipple and Sperry made an extensive experimental study of the lesions associated with delayed chloroform poisoning, and found that they could produce at will extensive central necrosis of the liver lobules, identical with that observed in acute yellow atrophy of the liver, as well as many of the reported cases of vomiting. Consequently, they felt justified in inquiring whether it was not possible that the hepatic and renal changes observed in patients dying after abortion induced for the relief of vomiting of pregnancy might have resulted from poisoning by the chloroform used for anæsthesia, rather than from a supposed specific toxæmia.

Finally, M'Donald, in a letter to the editor of the *Journal of the*

American Medical Association objecting to an editorial in which the work of Ewing and myself had been favourably mentioned, stated that the differential study of the urinary nitrogen was of no value either from a diagnostic, prognostic or therapeutic point of view, as he held that "such conclusions are unwarranted by facts and unsupported by scientific men."

Although the tendency of most recent writers has been to assume that the underlying factor in the production of serious vomiting is a toxæmia, which is in some way directly connected with the existence of pregnancy, a certain number have advanced other views, which should at least receive cursory mention.

Thus, Chirié and Perrot incline to the belief that the ultimate factor consists in a perverted secretion of the corpus luteum. The evidence adduced in favour of this supposition is not conclusive, and to my mind the contribution is of interest only in that it renews the suggestions of Holladay, Pierrehughes and Turenne which were mentioned in my monograph.

Following the suggestion of Mayer that certain dermatoses of pregnancy may be relieved by the intravenous injection of small quantities of serum obtained from normal pregnant women, Fieux and Dantin employed a similar procedure in a case of serious vomiting. As the patient began to improve immediately after the administration of the serum, they were inclined to attribute the cure to its use, and tentatively suggested that the supposed toxæmia may be due to the lack of some substance which should normally be present in the serum. Their observation is of very considerable interest, but as similar results did not follow the adoption of the procedure in two of my patients, I am inclined to be somewhat sceptical of their interpretation, and would suggest that the improvement in the condition of their patient may have been due to suggestion.

Lequeux considers that all cases of serious vomiting are associated with impaired hepatic function, and holds that an approximate idea of its extent may be gained by determining the glycolytic power of the liver. For this purpose he administers 1 gram. of sugar for each kilo. of body weight of the patient, and if glycosuria ensues he concludes that it affords evidence of such serious insufficiency of the liver as to justify the induction of abortion. The proposal is very interesting, but, unfortunately, my experience would indicate that it is not applicable to the type of cases concerning which information is most desired, as it is apparent that a patient who is vomiting everything she ingests will scarcely be able to retain a solution containing the requisite amount of sugar.

Finally, it may be noted that Rebaudi and Siegmund have reported the recovery of apparently desperate cases following the administration of thyroid extract and adrenalin respectively.

Reverting to my own views upon the subject, I frankly admit that some of my original conclusions are in need of revision.

In the first place, I am inclined to believe that all cases of vomiting of pregnancy, including the ordinary morning sickness, are due to an imperfect reaction on the part of the mother to the growing ovum. Whether this is a manifestation of the inability of her tissues to neutralize or render innocuous distinct chemical poisons originating in the ovum, or is due to a so-called "biological reaction," or to some other mechanism, cannot be determined at this time. That some such connection will be demonstrated in the future I confidently believe, although at present it is impossible to adduce satisfactory evidence in support of such a contention.

Notwithstanding this belief, I still hold that my original classification of reflex, neurotic and toxæmic vomiting should be retained, as in my hands it has demonstrated its value from the standpoint of prognosis as well as of treatment. In the first two varieties I regard the toxæmic element merely as a predisposing factor, and the reflex or neurotic influence, as the case may be, as the determining cause of the condition. Support is lent to such a view by the observation that, if the latter be removed or overcome by appropriate treatment, the underlying disturbance is so slight as to give rise to no symptoms, or to disappear spontaneously, or in response to the employment of simple measures. On the other hand, in the toxæmic variety the toxæmia, with its accompanying organic lesions and perversion of metabolism, is the predominating factor, and must be constantly reckoned with in the treatment of the disease.

In my experience neurotic vomiting is the variety most frequently encountered, and, fortunately, it is ordinarily readily amenable to treatment, although, as will be pointed out later, it may exceptionally terminate in death by starvation if too long neglected. On the other hand, increased experience has led me to conclude that the frequency and importance of the reflex type has been greatly exaggerated. Doubtless vomiting may cease after applications to the cervix, the replacement of a retroflexed uterus, or the removal of an ovarian cyst. Notwithstanding such results, however, I have become more and more sceptical concerning the part played by reflex influences, and now hold that a large part of the curative effect of procedures aiming at overcoming them should be attributed to their suggestive influence. That such a supposition is not entirely theoretical is shown by the fact that in several instances of moderately severe vomiting, complicated by retroflexion of the uterus, I have noted that relief has followed the reposition of the organ and the introduction of a pessary, although a subsequent examination showed that the uterus had returned to its original abnormal position.

The toxæmic type of vomiting occurs much less frequently than

the neurotic, but its incidence is considerably greater than that of the reflex variety. Unfortunately, its diagnosis is not so simple as I had originally believed, but its prognosis is quite as serious; and I still hold that it is practically the only variety of vomiting which necessitates the artificial termination of pregnancy, and even then a successful outcome is not assured.

I admit that increased experience has demonstrated the necessity for a radical revision of my views concerning the significance of a high ammonia co-efficient. In the first place, I have to acknowledge that one of 10 or 15 per cent. does not justify a diagnosis of toxæmic vomiting; and in the second place, that even much higher values do not necessarily indicate the existence of a specific toxæmia.

My experience has taught me that many women suffering from neurotic vomiting gradually pass into a condition of profound inanition, and in such cases examination of the urine may reveal a high ammonia co-efficient, which is probably a manifestation of the existence of a starvation acidosis, as is indicated by the presence of acetone, diacetic and oxibutyric acids. Thus, Charts I, II, III show an ammonia co-efficient of 24, 30 and 47 per cent. respectively, which fell to normal within one week following forced feeding and suggestive treatment, while the patients respectively gained 11, 13 and $9\frac{1}{2}$ lb. in weight during the same period, and were afterwards delivered at full term.

I consider that these women were suffering from neurotic vomiting, with resulting acute starvation, and that the perversion of metabolism was a manifestation of an acid intoxication incident to the latter condition. That such an interpretation is correct is strikingly demonstrated by the history of the patient, whose urinary findings are shown in Chart I. The woman was brought to the hospital by her physician, who stated that she was three months pregnant, had vomited all food for the previous month, and had become markedly emaciated. She weighed $88\frac{1}{2}$ lb., but was not vomiting on admission. She received no food for the first twenty-four hours, during which time she passed 980 cc. of urine containing 7 grains of nitrogen and presenting an ammonia co-efficient of 12 per cent. During the following two days there was no further vomiting, but as she did not care to eat I did not insist upon it, but contented myself with giving saline and nutrient enemata. At the end of that period the patient did not appear to be seriously sick, but as she had lost an additional $3\frac{1}{2}$ lb. in weight, and the ammonia co-efficient had increased to 24 per cent., I concluded that the experiment had been sufficiently prolonged, and talked with her seriously, with the result that she was able to take and retain 200 cc. of milk and 100 cc. of water every two hours. Within a few days the ammonia had fallen to 8 per cent., and the daily excretion of nitrogen had increased to 9 grains. She soon began to ask for solid food, and within a few

days was eating the regular ward diet. She was discharged on the fourteenth day after admission in excellent condition, having gained 11 lb. The vomiting did not return, and she was delivered normally at term.

The interpretation of this case is perfectly clear, as it is apparent that the patient was suffering from starvation dependent upon neurotic vomiting, and was cured by forced feeding and suggestion, while the high ammonia co-efficient was probably a manifestation of an acidosis. I feel confident that Cases ii and iii, as well as several others in my series, may be similarly explained. On the other hand, I am not prepared to admit that this is the only explanation for a high ammonia co-efficient, as will be shown more particularly when we consider in detail our cases of true toxæmic vomiting.

In this connection, however, I wish to refer to the ammonia curve shown in Chart VI. This patient lived for more than a month following the interruption of pregnancy for supposed toxæmic vomiting, and during that period the ammonia co-efficient varied between 20 and 40 per cent. Upon three occasions an intravenous injection of 6 grams of sodium bicarbonate was given, with the result that the acid urine became neutral or alkaline in reaction, and remained so for from six to forty-two hours. Had the high ammonia been a manifestation of an acidosis such an effect could not have been obtained, as the amount of alkali employed lay within the range of normal tolerance, and was insignificant when compared with the quantity necessary to effect a similar change in acidosis complicating diabetes. Furthermore, Dr. Sellard, who was good enough to estimate upon several occasions the amount of acetone bodies contained in the urine, calculated that the increase in the ammonia co-efficient was many times greater than could be explained by their neutralization. Consequently, he concluded that in this case, at least, we had to deal with a primary disturbance in the proteid metabolism, and not with a starvation acidosis. Furthermore, the presence of large amounts of fat in the subcutaneous tissues at the time of autopsy indicated that death must be attributed to some other factor than mere starvation.

In addition to the facts just adduced, certain negative evidence is available as tending to indicate that starvation is not necessarily the sole factor concerned in the production of a high ammonia co-efficient in vomiting of pregnancy. Thus, in Case ii of my monograph the ammonia co-efficient fell from 32 to 12 per cent. in the forty-eight hours following the induction of abortion, although the patient had taken no food. Consequently, it would seem, if inanition were the sole factor concerned, that the ammonia should have risen, but as it fell some other explanation must be invoked, and I am inclined to seek it in the rapid disappearance of the toxæmia following the emptying of the uterus. Furthermore, in

Case v of the present series the patient had ceased vomiting and had been retaining satisfactory quantities of nutriment for several days, yet she died suddenly in coma, with an ammonia co-efficient of 35 per cent. In this instance the starvation had been overcome, and, therefore, could scarcely be invoked as a satisfactory explanation for the high ammonia.

In certain cases of delayed chloroform poisoning a relatively high co-efficient may also be observed, and reached 16 per cent. in one of my patients, who died with the characteristic clinical signs and autopsy findings five days after an operative delivery at full term. Naturally, such an explanation would be inadmissible in patients who are vomiting before operation, but its possibility should always be borne in mind when the ammonia co-efficient rises after the induction of an abortion in which chloroform had been employed as an anæsthetic.

As has already been indicated, Whipple and Sperry have inquired whether it were not possible that the hepatic lesions which are supposed to characterize certain fatal cases of toxæmic vomiting might be the result of poisoning by the chloroform employed at the time the uterus was emptied. As the lesions are identical in the two conditions, there is no doubt that such confusion may occur; and I consider it quite possible that the death of my patient described as Case iv of my monograph may have been due to late chloroform poisoning, particularly as her symptoms were analogous to those observed in the case just mentioned. On the other hand, such an explanation cannot always be invoked, as abortion was not induced in Case v of the present series, while in Case vi the patient died thirty days after the uterus had been emptied by hysterectomy, during which nitrous oxide had been employed as the anæsthetic agent.

We are, however, greatly indebted to Whipple and Sperry for directing attention to this point; but to my mind the great value of their contribution, at least from an obstetrical point of view, consists in having directed our attention to the deleterious effect of chloroform upon the liver, and thereby warning us against its employment in women suffering from toxæmic vomiting, in whom, to say the least, the liver is a *locus minoris resistentiæ*.

Thus far, I have shown that a high ammonia co-efficient may be a manifestation of inanition resulting from neurotic vomiting, but I have also incidentally indicated that such is not its only mode of production. We must now consider the evidence which can be adduced in favour of the existence of the toxæmic type of vomiting, and I may preface the argument by stating that I not only unhesitatingly believe that it occurs, but also that I consider it is the most serious type which we encounter. Unfortunately, it can no longer be contended that its clinical recognition is always easy, as it

is not the only type of vomiting characterized by a high ammonia co-efficient.

Nevertheless, I believe that for clinical purposes we can consider a given case of vomiting as toxæmic in origin whenever a seriously ill patient, presenting a high ammonia co-efficient (20 per cent. or more), fails to improve after a few days' complete rest in bed, combined with suggestive treatment, energetic rectal feeding, and the administration of large quantities of salt solution per rectum and beneath the skin. Furthermore, the appearance of coffee-ground vomit, well-marked icterus, and a semi-comatose condition justifies an almost positive diagnosis, provided chloroform has not been employed as an anæsthetic, and if death ensues characteristic hepatic and renal lesions will be found at autopsy.

Charts IV, V and VI give a clear idea of the variations in the ammonia co-efficient observed in this type of vomiting, and a summary of the clinical histories of the patients concerned cannot fail to impress even the most sceptical with the fact that some other factor than starvation must be concerned.

Thus, in Case iv, the 28-year old patient, who was pregnant for a second time and most anxious for a living child, began to vomit six weeks after the last menstrual period, and soon vomited everything she ingested. Her physician at once put her to bed and fed her by nutrient enemata, which were alternated with rectal injections of glucose solution. As she did not improve, I was asked to see her in consultation ten days after the onset of serious symptoms. At that time she appeared to be well nourished, but was very torpid mentally. The pulse was about 80, and the scanty urine presented an ammonia co-efficient of 16 per cent. Rectal feeding was pushed, and most vigorous suggestion employed, but notwithstanding all we could do her condition gradually became worse. By the end of the fifth day the ammonia had risen to 26 per cent., a purpuric eruption had appeared over the entire body, and she appeared so toxic that interference appeared imperative, although the pulse was still about 80. The uterus was emptied without difficulty under nitrous oxide anæsthesia, and immediate improvement followed, the vomiting ceasing and the ammonia falling to 7 per cent. within the next five days.

The toxæmic nature of the condition was still further confirmed the day after the abortion by the appearance of conjunctival icterus, which soon spread over the entire body, while the purpuric eruption became more pronounced. Furthermore, the ammonia co-efficient immediately began to fall, and reached 20 per cent. before the patient had commenced to take food, and while she was receiving the same quantity of nutriment by the rectum as before the operation.

Still more conclusive evidence was afforded by Case v, which, owing to an unfortunate mistake, acquired almost the exactness of a

carefully planned laboratory experiment. The 32-year old patient, who had had three previous labours, was admitted to the ward when four months pregnant, having vomited incessantly for the previous month, during which she lost 40lb. in weight. At the time of admission she was considerably emaciated and greatly prostrated, with a pulse of 120. During the first twenty-four hours she passed 100 cc. of urine, which contained 9 grains of nitrogen and presented an ammonia co-efficient of 37 per cent. Unfortunately, the assistant who made the determinations, misplaced the decimal point in all his calculations, and did not discover the error until after the death of the patient. He accordingly reported that the first day's urine showed ammonia co-efficient of 3·7 per cent., and that on the subsequent days it remained within normal limits. Consequently, I made a diagnosis of neurotic vomiting, and placed the patient upon rectal feeding and suggestive treatment, which was so efficacious that her condition improved rapidly, and she was able to retain solid food by the end of the sixth day. Improvement continued until the eleventh day, when tonsillitis developed and the vomiting returned. The following morning, however, she ate with relish and retained a fair breakfast. Later in the day she became restless and excited, and passed into a comatose condition in which she died. Autopsy showed marked fatty degeneration of the liver and epithelial necrosis of the kidneys, together with other lesions which will be mentioned in the detailed history at the end of the article.

In this instance, the error in calculating the ammonia co-efficient caused me to overlook the toxæmic condition, and yet the treatment employed enabled the patient to retain the usual ward diet for a week. This being the case, I consider that no one would attempt to attribute the fatal issue to inanition; while the high ammonia co-efficient, which varied between 21·5 and 38·5 per cent., associated with hepatic and renal lesions, indubitably indicated the true nature of the condition.

Case vi, to which cursory reference has already been made, affords even stronger evidence of the existence of toxæmic vomiting, as in it experimental proof was adduced to demonstrate that the high ammonia co-efficient was not due to inanition acidosis.

This patient, in whom a previous pregnancy had been artificially ended on account of supposed toxæmic vomiting (ammonia co-efficient, 13 per cent.), entered the hospital in November 1911. At that time the vomiting was considered as neurotic in origin, and was apparently relieved by rest in bed, rectal feeding, and suggestion. She was discharged in excellent condition, weighing 136 lb. Within a few days after returning home an indiscretion in diet brought about a recurrence of the vomiting, which gradually became so intense that she re-entered the hospital on December 7.

At that time she was able to retain nothing, weighed $124\frac{3}{4}$ lb.,

and presented ammonia co-efficient of 15 per cent. In view of her past history we were still inclined to attribute her condition to inanition incident to neurotic vomiting, and treated her accordingly. She, however, became gradually worse, the ammonia steadily rising until it reached 35 per cent., but there was no loss in weight. In view of the seriousness of her condition, the uterus was emptied by hysterotomy under nitrous oxide anæsthesia ten days after admission. For the next few days there was considerable improvement, which, however, did not continue, and death occurred in coma thirty days after the abortion. During that period the patient retained varying quantities of food, and never became greatly emaciated, losing only 12 lb. in forty days. The ammonia co-efficient varied between 18 and 40 per cent., but did not appear to be due to an acidosis, as was demonstrated by determining the quantity of acetone bodies in the urine, as well as the effect upon its reaction of the intravenous injection of sodium bicarbonate. These data have already received cursory mention, and will be given in detail in the complete history of the case. Autopsy showed that the subcutaneous fat was well preserved, while the liver presented marked fatty degeneration, and the kidneys signs of epithelial necrosis.

As the tests to which reference has been made indicate that the high ammonia was not due to an acidosis, while the preservation of large quantities of adipose tissue as well as the comparatively slight loss of weight speak against acute starvation, it is apparent that the urinary changes could not be regarded as a manifestation of an inanition acidosis. Consequently, we were forced to conclude that, in this instance, we had to deal with a primary perversion of proteid metabolism. Unfortunately, it is impossible to state at present whether this was the primary change, or whether it was dependent upon an underlying toxæmia. In either event, I believe that it cannot be attributed entirely to hepatic insufficiency; for, although the liver was the seat of profound fatty change, sufficient tissue was apparently left to effect the final changes in nitrogenous metabolism.

To my mind the evidence which I have adduced makes it perfectly clear that a high ammonia co-efficient in vomiting of pregnancy is not susceptible of any single explanation, and simply indicates that the patient is seriously ill. On the one hand, it may be a manifestation of a starvation acidosis incident to neurotic vomiting, while on the other hand it may indicate the existence of a profound disturbance of metabolism, associated with serious hepatic and renal lesions, which is most readily explained as being dependent upon the existence of an underlying toxæmia. Unfortunately, it is impossible at this time to make any definite statement concerning the nature of the latter, or even to suggest a satisfactory theoretical explanation. The uniform presence of acetone bodies in the urine in all serious cases would suggest the probability of some interference with fat meta-

bolism; but I believe that such changes are purely incidental, and afford us no information concerning the ultimate origin of the toxæmia.

The fact that serious vomiting frequently occurs in the first weeks of pregnancy renders it unlikely that the toxæmia can be directly due to the entrance of products of fœtal metabolism into the maternal blood, and forces us to consider other possibilities. In this connection, primary disturbances in energy production, the escape of ferments set free by the corrosive action of the rapidly growing trophoblastic cells of the ovum, as well as the possibility of "biological reactions" in the blood, may be thought of; but the information at our disposal is too scanty to permit even the formulation of an hypothesis.

Passing from these theoretical considerations to more practical matters, the time has come to enquire whether the study of the urine, and more especially the determination of the ammonia co-efficient, serves any useful purposes in the treatment of pernicious vomiting of pregnancy. To such a question I would give a most positive affirmative answer.

In the first place, it must be admitted that such studies have materially extended our knowledge of the condition, and it is only by employing all means of research at present at our disposal, or which may become available in the future, that we can expect to solve the problem of the ultimate cause of this malady. In the second place, I know from my own experience that the determination of the ammonia co-efficient is of the greatest practical value in determining the prognosis and treatment of serious vomiting, although I must reluctantly confess that it does not afford quite such precise information as I formerly believed. In my experience, in the absence of palpable lesions of the generative tract a low ammonia co-efficient affords indubitable evidence of the existence of neurotic vomiting, and thereby enables us to say, positively, that the condition is not alarming, is readily amenable to suggestive and dietetic treatment, and will not necessitate ending the pregnancy.

I am frank to confess that in my hands mere clinical observation does not permit such a differentiation, as I frequently see patients who appear so ill that the induction of abortion seems not only indicated but imperative, but in whom the presence of a low ammonia co-efficient gives one such assurance of the amenability of the condition to treatment that I am able to resort to suggestion with such confidence that complete, or at least relative, cure follows within a few days.

Charts VII and VIII illustrate the condition of the urine in two patients who were so desperately ill that at first glance induction of abortion appeared to afford the only prospect of avoiding a fatal issue, yet after the presence of a low ammonia had enabled me to diagnosticate simple neurotic vomiting, I was able to treat them so

confidently by suggestion that the vomiting ceased within a few days without the use of drugs or the induction of abortion. Similar experiences have been so universal in my hands that I now regard with equanimity any case in which the ammonia co-efficient is 10 per cent. or less.

On the other hand, the only positive information afforded by a high ammonia co-efficient is the fact that the patient is seriously ill, but unfortunately it does not enable us to determine whether her condition is due to starvation incident to neurotic vomiting, or to a serious perversion of metabolism dependent upon an underlying toxæmia. In such cases clinical observation alone permits the differentiation. The patient is put to bed, isolated as far as possible from her family, fed energetically by the rectum, given large quantities of salt solution per rectum and beneath the skin, but receives nothing by the mouth until she expresses a desire for it. At the same time she is reassured as much as possible. If improvement occurs within a few days, it is assumed that one has to deal with a starvation acidosis, and cure almost invariably follows suggestive treatment and forced feeding; if, however, the condition grows worse, we assume that the patient is suffering from toxæmic vomiting, and empty the uterus as the only means of averting a fatal issue. Should jaundice develop, or the patient begin to vomit without effort coffee-ground-like material, the diagnosis is assured, and the prognosis becomes even more dubious.

When it is determined to empty the uterus, the operation should be performed by whatever method promises the most rapid and conservative results. In multiparous women, in whom the cervix is soft and patulous, dilatation and curettage is the method of choice; but in all other cases vaginal hysterotomy should be performed, as it enables one to empty the uterus in a very few minutes with a minimum of shock and the least possibility of infection. In view of its deleterious action upon the liver, and the probability of its still further accentuating the lesions associated with toxæmic vomiting, chloroform should be avoided, and ether or preferably nitrous oxide gas employed for anæsthesia.

In connection with the clinical observation of the patient, there are two points upon which I desire to lay especial emphasis. Pinard has stated that a pulse-rate of 100 or more affords a precise indication for terminating pregnancy. This is not my experience, and I wish to issue a note of warning against the adoption of any such arbitrary rule. I have repeatedly observed a pulse-rate of 120 or more in cases of neurotic vomiting, and yet the patients have recovered perfectly. On the other hand, I have already indicated that I emptied the uterus in Case iv when the pulse was only 80, and have recorded in my monograph a fatal case in which it at no time rose above 96.

Likewise, it is generally stated that the presence of albuminuria

is always of most serious prognostic import. In not a few cases a trace of albumen may be without significance, while in fatal cases it may be entirely absent or develop only during the hours immediately preceding death.

Conclusions.

1. The underlying factor in all cases of vomiting of pregnancy is probably an imperfect reaction on the part of the mother to the growing ovum.

2. In most cases this is only a predisposing cause, while a reflex or neurotic influence is the exciting factor, and cure usually follows its removal.

3. I still hold to the classification of reflex, neurotic and toxæmic vomiting. Of these the neurotic is the most and the reflex the least frequent type, while the toxæmic is the most serious.

4. Pronounced toxæmic vomiting is accompanied by characteristic lesions and profound changes in metabolism.

5. The significance of a high ammonia co-efficient is not specific. It may be a manifestation of toxæmic vomiting, of starvation following neurotic vomiting, or of an acidosis due to various causes.

6. It should be regarded merely as a danger signal, while the differentiation between the various types is possible only after careful clinical observation. If improvement does not promptly follow appropriate treatment, the existence of toxæmic vomiting should be assumed and abortion promptly induced.

7. In the absence of genital lesions, a low ammonia co-efficient indicates neurotic vomiting, which can be cured by suggestion and dietetic treatment, no matter how ill the patient may appear.

8. In primiparous women vaginal hysterotomy is the most conservative method of emptying the uterus. Nitrous oxide gas or ether should be used in preference to chloroform for anæsthesia.

Case Histories.

Case I (No. 4492, Morris).—The 29-year old patient was admitted to the Hospital on July 21 1910, and gave the following history:—

One preceding pregnancy, which was ended by forceps six years ago. The present pregnancy followed the menstrual period of April 10 1910, and was uneventful until the latter part of June. Since then she has suffered so intensely from vomiting that she could retain nothing, and has lost greatly in weight.

Examination showed a markedly emaciated woman, weighing 88½ lb., with a scaphoid abdomen. There was no jaundice, and no abnormality could be detected, except the presence of a three months' pregnancy. She was not vomiting at this time, but stated that she did not care to eat. The twenty-four-hour specimen of urine measured 980 c.cm., contained 6 grams of total nitrogen, and presented an ammonia co-efficient of 11·5 per cent. No albumen or casts.

For the first day she received no nutriment of any kind, but on the following day the administration of saline and nutrient enemata was commenced. Within three days she lost an additional 3½ lb., and, as the ammonia co-efficient had risen to 24 per cent., it was felt that the starvation had continued sufficiently long. Accordingly, she was told that she had to eat and retain her food, and to our surprise she took 200 c.cm. of milk and 100 c.cm. of water every two hours, and retained them without effort. She continued to do this for several days, when she began to ask for solid food. This was given in constantly increasing amounts, so that by the tenth day she was able to eat and enjoy the regular ward diet. Coincidently with this the ammonia co-efficient fell, and the amount of total nitrogen and the output of urine increased (Chart I). She gained rapidly in weight, taking on a pound each day she remained in the Hospital after forced feeding had been commenced, and was discharged on the fourteenth day, weighing 96 lb.

Her further history was uneventful, and she was delivered spontaneously at full term.

Case II (No. 4416, Fisher).—The 30-year old patient was admitted to the ward on May 15 1910. She was pregnant for the first time, and nausea and vomiting had appeared shortly after the last menstrual period, which occurred on February 10 1910. She stated that she had eaten no solid food during the month preceding admission, the last week having been spent in bed. She had lost greatly in weight, and suffered from headache, persistent salivation, and a great diminution in the amount of urine.

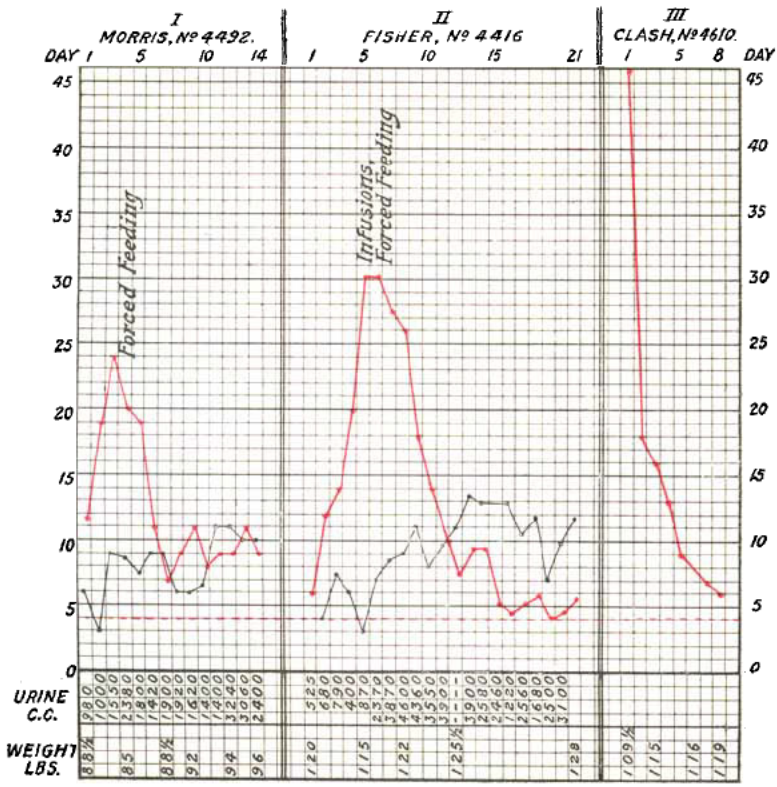
Examination showed a large-framed, emaciated woman, weighing 120 lb., with a distended abdomen. The pulse varied between 90 and 100, while the temperature was normal. She complained of considerable epigastric pain and profuse salivation—330 c.cm. in the first twenty-four hours. There was no jaundice, and a careful physical examination was negative, except for a three months' pregnancy. The patient was very nervous, and there was a distinct odour of acetone to her breath.

As the urine presented an ammonia co-efficient of 6 per cent., a diagnosis of neurotic vomiting was made, and suggestive treatment was instituted. For the first twenty-four hours she was given 1,800 c.cm. of salt solution per rectum, and ice by the mouth. For the next few days she was given nutrient, saline and glucose enemata, but without improvement in her condition. During that time she lost an additional 5 lb., the pulse became more rapid and remained about 120, while the ammonia co-efficient rose rapidly, reaching 30·5 per cent. (Chart II).

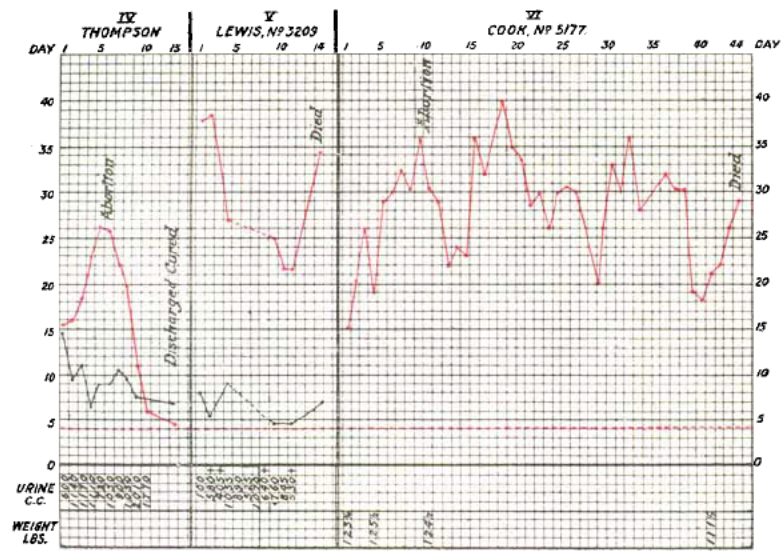
On the following day she received two submammary saline infusions of 1,000 c.cm. each, in addition to the enemata, and was talked to so energetically that she took by mouth 1,270 c.cm. soda water, 520 c.cm. water, the whites of ten eggs, and 1 oz. of sugar. Immediate improvement followed, as was shown by the complete disappearance of nausea and a marked increase in the output of urine.

The same treatment was continued on the seventh day, but was discontinued thereafter, as she began to eat with relish and improved rapidly. The pulse became slower and she gained in weight, while the quantity of urine became still more abundant and the salivation ceased. As shown in Chart II, the ammonia co-efficient fell rapidly, reaching 6 per cent. on the twelfth day and 4 per cent. a few days later, while at the same time the output of total nitrogen increased. The improvement was progressive,

NEUROTIC VOMITING WITH STARVATION.

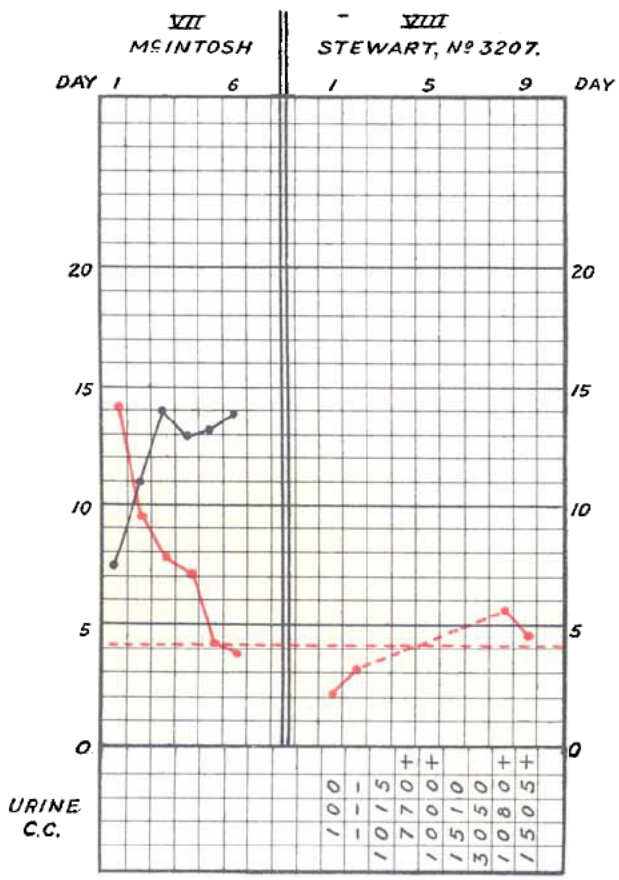


TOXÆMIC VOMITING.



Red lines = Ammonia Co-efficient.
Black lines = Grams Total Nitrogen.

SIMPLE NEUROTIC VOMITING.



Red lines = Ammonia Co-efficient.
Black lines = Grams Total Nitrogen.

so that the patient was discharged on the twenty-first day after admission, having gained 13 lb. since the institution of forced feeding. There was no further recurrence, and she was delivered spontaneously at term.

The urine at no time contained albumen or casts, but the presence of acetone and diacetic acid was repeatedly demonstrated during the first ten days. As the condition improved there was a corresponding diminution in the amount of salivation, which fell from 330 to 35 c.cm. in the course of eleven days.

Case III (No. 4610, Clash).—The 26-year old negress entered the Hospital on October 12 1910. She had aborted spontaneously at eight weeks, two and a half years previously, but had had no other pregnancy. Her general health was not good, having been impaired by several serious illnesses.

Her last menstrual period occurred on July 14, and morning sickness appeared in the latter part of August. This gradually grew worse, so that she vomited almost constantly for the month preceding admission, the last week of which she had spent in bed and had been fed per rectum by her physician.

On admission she was found to be a thin, neurotic woman, weighing 109½ lbs. Pulse, 110; temperature, normal. She did not appear to be very ill, but during the first day in the Hospital vomited everything she ingested. She complained of great tenderness in the epigastrium, but a careful physical examination revealed no abnormality, except a three months' pregnancy.

Notwithstanding an ammonia co-efficient of 46 per cent., a diagnosis of neurotic vomiting was made, and suggestive treatment was instituted. On the second day she was forced to take considerable quantities of milk and water by mouth, and was given subcutaneous saline infusions, as well as rectal saline enemata. This was followed by immediate improvement, and the nausea and vomiting soon ceased. On the second day the ammonia fell to 18 per cent., and thereafter soon approached normal (Chart III).

By the fifth day she was able to retain the ordinary surgical soft diet, and four days later she was eating the ward diet with relish. She was discharged on the tenth day in excellent condition, weighing 119 lb., a gain of 9½ lb., and presenting an ammonia co-efficient of 6 per cent.

As she did not report later, I am unable to give particulars concerning the further course of her pregnancy.

Case IV (A. W. T., private patient).—The 28-year old patient was seen in consultation on account of pernicious vomiting. She had been married seven years, had no children, and one miscarriage at the fourth month six years previously. In the first weeks of that pregnancy she had suffered from persistent nausea.

The present pregnancy followed the menstrual period occurring on August 17 1910. She began to vomit early in October, and had been unable to retain anything after October 5. Her physician put her to bed and placed her upon nutrient and glucose enemata, but as her condition grew worse he asked me to see her, particularly as the urine presented an ammonia co-efficient of 20 per cent.

When I saw the patient on October 12, she appeared to be a well-nourished woman presenting no signs of emaciation. Her lips were parched, her tongue markedly coated, and she presented a torpid expression. Physical examination revealed no abnormality except a two months' pregnant uterus, which was slightly retroverted.

I advised that four nutrient enemata be given each day, as well as an equal number of glucose solution, and talked as hopefully as possible. On the following day the patient was somewhat better, as well as on Oct. 14. On that day 1 oz. of milk diluted with lime water was given and caused immediate vomiting. Following this she was unable to retain anything by the mouth, and was nourished exclusively per rectum. Her condition gradually became worse, and on the seventeenth day she presented a decidedly toxic appearance, was extremely apathetic, and a macular eruption had appeared over the neck and body, while at the same time the ammonia co-efficient had risen to 26·2 per cent. (Chart IV).

In view of these conditions, it was decided that it would be unsafe to allow the pregnancy to continue, and abortion was induced under nitrous oxide anæsthesia, when the cervix was dilated, and the uterus emptied by means of a dull curette ovum forceps.

It is interesting to note that the pulse at no time before or after the operation exceeded 80, while the temperature remained normal. There was no nausea following the anæsthesia, and the day afterwards the patient presented an entirely different appearance, appeared bright and cheerful, and had not vomited. This change occurred without any alteration in the rectal feeding, and while no food was being taken by the mouth.

The following day her condition was still further improved, while the ammonia co-efficient had fallen to 20 per cent., although no change had been made in her diet, but she was ordered to begin taking food that afternoon. At that time there was a slightly yellowish tinge to the conjunctivæ, which had not been present previously. On the third day after the abortion her condition was still further improved, and the enemata were discontinued, as she was taking with relish abundant quantities of liquid food. The icterus, however, had become more pronounced and now involved the entire body, while the macular eruption which had been present before the operation became accentuated. Four days after the abortion the patient was not nauseated at all, and was placed upon solid food. The ammonia co-efficient had fallen to 10·8 per cent. From that time onward improvement was rapid, and one week after the abortion she was out of bed in excellent condition, except for a slight icteric discolouration of the skin which slowly disappeared. Further convalescence was uneventful.

Case V (No. 3209, Lewis).—admitted on October 29, and died on November 9 1907. The 32-year old patient was brought to Hospital on a stretcher by her physician on account of pernicious vomiting, which had resisted all his efforts to cure. She had had three spontaneous labours, and the last menstrual period occurred on June 16 1907. She had vomited more or less since early in September, but for the month prior to admission had been unable to retain anything. She stated that the vomitus was a greenish fluid, whose ejection was accompanied by a marked burning sensation in the throat and epigastrium. She had become greatly emaciated, and weighed 110 lb. instead of 150 lb. as usual.

On admission she was markedly prostrated, and the pulse varied between 110 and 120. Except for emaciation, examination showed no abnormality other than a four months' pregnancy. During the first twenty-four hours in the Hospital she passed less than 100 cc. of urine, and as this apparently showed an ammonia co-efficient of 3·7 per cent. a diagnosis of neurotic vomiting was made. Consequently, the patient was placed upon nutrient and saline enemata and was subjected to suggestive treatment.

Following this there was marked improvement; the pulse became less rapid, the vomiting practically ceased, the urine increased in amount, and the mental condition became much more normal than on admission.

By the sixth day the improvement was so pronounced that she was able to retain fluids, while on the following day she took and retained solid food. The improvement continued until the eleventh day, when a tonsillitis developed and the vomiting returned. The following day she ate and retained a fair breakfast and a light dinner. Later in the day she became restless and excited, and eventually passed into a condition of coma, in which she died. Just before death the temperature was 102°F. and the pulse 126.

Autopsy showed marked fatty degeneration of the liver and kidneys, epithelial necrosis of the kidneys, fatty degeneration of the myocardium, thyroid hypertrophy, aspiration of the stomach contents, hypostatic congestion of the lungs, broncho-pneumonia, accessory pancreas.

On the day after her death the assistant who had made the urinary analyses reported that an error had been made in calculating the ammonia co-efficient, as he had misplaced the decimal point, so that the co-efficient of the first day's urine was 37 per cent. instead of 3·7 per cent., as reported. The corrected figures are given in Chart V.

Case VI (No. 5177, Cook).—28-year old nullipara. Her first pregnancy, in 1908, was ended by the induction of abortion for the relief of supposed toxæmic vomiting. The last menstrual period before the present illness was September 24 1911, and vomiting began on November 5. Within a few days it became so incessant that nothing could be retained, and the patient was put to bed by her physician and placed upon nutrient and saline enemata. As her condition became steadily worse she was admitted to the Hospital on November 14 1911, when physical examination revealed no abnormality, although the urine presented an ammonia co-efficient of 13 per cent. As rapid improvement followed suggestive treatment and forced feeding, a diagnosis of neurotic vomiting was made, and she was discharged two weeks later apparently cured, weighing 136 lb. and presenting an ammonia co-efficient of 3·9 per cent.

Two days after her return home the vomiting reappeared, apparently following an indiscretion in diet. It immediately became uncontrollable, so that during the next eight days she lost 12¼ lb. in weight, and was re-admitted to the Hospital on December 7. At that time she looked very ill, the pulse varied between 110 and 130, and the ammonia co-efficient was 15¼ per cent.

Notwithstanding the employment of nutrient, saline and glucose enemata, as well as of subcutaneous infusions of salt solution, her condition gradually became worse, although there was no further loss of weight. The pulse continued rapid, and the patient appeared torpid and toxic, while the ammonia co-efficient gradually rose to 34·5 per cent. Consequently, ten days after admission (December 16) the induction of abortion seemed indicated, and was readily effected by vaginal hysterotomy, nitrous oxide being used as an anæsthetic.

For a short time her condition seemed to improve, so that three days after the abortion she was able to take and retain surgical soft diet, while the ammonia fell to 22 per cent. Unfortunately, the improvement was short-lived, as the vomiting reappeared on the following day (December 20) and persisted to a varying extent until death occurred on January 16, although at intervals she was able to retain considerable quantities of food.

During this period the ammonia co-efficient varied between 17 and 40 per cent., the pulse remained constantly rapid, and the patient lost gradually in weight, but at no time became greatly emaciated. Just before death her weight was 24 lb. and 12¾ lb. less, respectively, than upon her first and second admissions to the Hospital.

Beginning with January 1, her mental condition became more and more torpid, and for some days before death she would forget to withdraw her tongue after having been told to show it. She became completely unconscious the morning before death, and died in coma. Physical examination gave uniformly negative results. The subcutaneous fat was well preserved until the end. There was no jaundice, and coffee-ground vomit appeared only on the day before death. The temperature was uniformly normal, and reached 100 only upon one occasion eight days after the abortion.

Reference has already been made to the ammonia co-efficient, which is graphically shown in Chart VI. The urine constantly contained traces of albumin, and occasionally a few casts, as well as traces of acetone and diacetic acid. Upon two occasions analysis showed the presence of 0·248 and 0·095 grams of acetone, and of 0·021 and 0·162 grams of oxybutyric acid respectively per litre. Bile pigments were at no time present.

In order to determine whether the high ammonia was due to an acidosis, one grain of sodium bicarbonate was given by mouth for twelve consecutive hours seven days after the abortion, but had no effect upon the reaction of the urine. Furthermore, on December 24, as well as January 6 and 7, 400 c.cm. of a 1·5 per cent. solution of sodium bicarbonate were injected intravenously. In each instance the previously acid urine became alkaline in reaction, and remained so for forty-two, thirteen and thirty-four hours afterwards respectively.

On January 9 10 c.cm., and on January 14 20 c.cm. of blood serum obtained from a normal pregnant woman were injected intravenously, but without apparent effect. On two occasions the fibrinogen content of the blood was determined, and was found to lie within normal limits—0·3850 and 0·3825 grams per 100 c.cm.

At autopsy the anatomical diagnosis was:—Fatty liver and kidneys, œdema and congestion of the lungs, chronic fibrous pleurisy, ulcer of vaginal wall. The subcutaneous fat was well preserved, and the body showed no signs of emaciation. The liver was yellowish in colour, and on section the lobules were sharply marked, presenting a light yellowish periphery and a reddish centre. Fresh microscopic sections stained with Sudan red showed that each lobule was densely infiltrated with fat, but sections through hardened tissue showed that there was no central necrosis, and that the nuclei were well preserved.

Case VII (M., private patient).—I saw the patient in consultation, when I obtained the following history:—25-year old nullipara, in whom two previous pregnancies had been interrupted for the relief of pernicious vomiting—in February and September 1911.

The present pregnancy followed the menstrual period of December 20 1911, and was uneventful until the end of January, when she began to vomit incessantly and could retain nothing. When I saw the patient on February 4 she was in bed and retching at frequent intervals, but bringing up only small quantities of yellowish fluid. She was very thin, and said that she had lost markedly in weight since the onset of the attack. The pulse varied between 80 and 90, and a careful physical examination revealed no abnormality except an early pregnant uterus. In view of their

past experience, the patient and her husband believed that relief could be obtained only by emptying the uterus.

A specimen of urine obtained at this visit showed 15 grams of total nitrogen per litre, and an ammonia co-efficient of 14 per cent., but no other abnormality.

A tentative diagnosis of neurotic vomiting was made, and the patient talked to seriously, placed upon saline enemata, and told that I was confident that I could control the condition without interfering with the pregnancy. Prompt improvement followed. Within forty-eight hours she was able to retain small quantities of fluid nourishment, while the ammonia had fallen to 7·67 per cent. Under the influence of suggestive treatment she grew steadily better, and was able to retain increasing amounts of food, so that by the end of the sixth day she was able to resume her ordinary diet, and to eat and retain "bacon and greens." At the same time the ammonia co-efficient had fallen to 3·56 per cent. (Chart VII). The recovery was permanent, and the patient was uneventfully delivered at full term.

Case VIII (No. 3207, Stewart).—Admitted October 28, and discharged November 10 1907. I saw the patient in consultation with her physician, from whom I obtained the following history:—Age, 17 years; nullipara; last menstrual period, May 6 1907. For the previous three weeks she had suffered from almost constant vomiting, had retained practically no food, and her condition had become so serious that I was asked to see her to consider the propriety of inducing abortion. The emaciated patient was lying in bed and vomiting small quantities of frothy fluid every few minutes. She was greatly prostrated, pulse 136, with the eyes sunken far back into their sockets. Her condition was so serious that I arranged for her to enter the Hospital the next morning, but I advised that she see a priest before doing so.

On admission, the pulse was 130 and the temperature normal. Examination was negative except for a markedly scaphoid abdomen and a six months' pregnancy. The patient complained of constant nausea, associated with burning in the throat and epigastrium; there was no œdema, and the urine was free of albumen or casts. Her condition appeared so serious that the induction of abortion seemed urgently indicated, but it was deferred in order to give an opportunity for the collection of a twenty-four-hour specimen of urine. In the meantime all attempts at feeding by the mouth were discontinued, and she was placed upon nutrient and salt solution enemata.

To our great surprise the ammonia co-efficient was found to be only 2 per cent., so that a diagnosis of neurotic vomiting was made. Consequently, it did not appear necessary to induce abortion, but, instead, the patient was treated by suggestion and the rectal feeding continued. Her general condition improved rapidly, and the pulse fell to 80—100 by the fourth day. The quantity of urine, which on the first day was only 100 cc., increased to 1,015 cc. on the third and to 3,050 cc. on the seventh day. On the fifth day the patient was anxious for food, and retained what she took, so that the enemata was discontinued. She was out of bed on the ninth day with a normal pulse, and was discharged on the fourteenth day in excellent condition. A month later she returned to the Hospital when the change in her appearance was marvellous, and no one could have recognized the red-cheeked girl for the emaciated woman who had been admitted to the ward. There was no recurrence of the vomiting, and a normal labour occurred at full term.