sixth month, in 2 cases it began during the fourth, and in 5 cases during the fifth month.

In 6 cases the presence of constipation was noted. In 5 others it was stated that the bowels had been opened regularly.

In 8 cases the trouble was right-sided, in 8 cases the symptoms and signs pointed to an involvement of both kidneys, and in 1 case the left kidney seemed to be affected alone.

One patient gave a history of a similar attack in former pregnancy.

In 2 cases there was no rise of temperature. In 11 the others there was pyrexia, in 14 cases as high as 102° F., and in 6 cases as high as 104°. In cases where there were rigors, which did not occur as frequently as I had expected, the temperature sometimes reached 105° or 106° F.

The urine was acid in 18 cases, alkaline in 1.

In 13 cases bacteriological examination of the urine was made. In 7 cases the *Bacillus coli communis* was found, in 5 cases a coliform bacillus, and in 1 case *Streptococcus albus* and a diphtheroid bacillus.

The symptoms complained of were pain in one or both loins, pain and tenderness along the course of one or both ureters, sometimes pain at the end of micturition, sleeplessness on account of the pain, malaise, etc.

There was 1 case of spontaneous abortion at five months and there was 1 case of spontaneous premature labour. In the latter case the temperature became normal thirty-six hours after labour, and all the symptoms disappeared, although the urine still contained pus. The case in which miscarriage occurred was the only one in which the patient did not make a good recovery.

She was a primigravida, aged 32, five months pregnant. Both kidneys were affected, and the *Bacillus coli communis* was found in the urine. Miscarriage occurred four days after the operation of exploration of the left kidney, and the patient died the next day. At the post-mortem examination pyelonephritis was found on the right side, pyonephrosis on the left side, the kidney being a mere shell filled with pus.

Premature labour was induced only once:

The patient was a primigravida, aged 27, seven and a half months pregnant on admission. She had been ill at home for some weeks, with pain in the loins so severe that she screamed when paroxysms came on, and had been unable to sleep. Raising the foot of the bed gave immediate relief from the pain, she slept well, and her general condition improved a little, but the pain became so acute again that it was thought necessary to empty the uterus. The patient made a good recovery, but the child lived only a few hours.

In none of the other 17 cases was it found necessary to bring the pregnancy to an end or to resort to surgical treatment, medical measures—that is, complete rest in bed, diet consisting chiefly of milk, and administration of urotropin, sometimes potassium citrate, sometimes acid sodium phosphate, with vaccines in a few cases—freeing the patients from pain and pyrexia, though not always from pyuria.

The success of conservative treatment was well shown in the case of a multipara, aged 28. For the first five weeks after admission she had a high degree of pyrexia, the temperature reaching 104° F., and there were several rigors. The question of emptying the uterus was considered, but under medical treatment the temperature eventually came down and stayed down. About fourteen weeks after admission labour came on naturally at term, and both mother and child did well.

Pyelonephritis in the Puerperium.

In cases that occur for the first time during the puerperium the diagnosis may be difficult at first, mainly because pyelonephritis is not thought of. If it is always borne in mind in cases of pyrexia coming on at the end of the first week of the puerperium—that is, at a time when uterine infection is improbable, the correct diagnosis will be made fairly often. This was taught by Olshausen ten years ago. Pyelonephritis occurring during the puerperium is likely to be mistaken for puerperal infection of the uterus, especially in cases in which there is no complaint of pain.

In cases in which there is localized pain a wrong diagnosis of appendicitis or of salpingitis may be made.

The fact that in spite of high temperature with great oscillations for several days the patient's general condition remains fairly good, points to pyelonephritis rather than puerperal infection.

I have seen a case in which the correct diagnosis was not made for many weeks.

Ten days after a normal labour the patient became ill with a fairly high degree of pyrexia. No abnormal physical signs could be found, the uterus was well involuted, the lochia had almost entirely ceased, there was no evidence of any inflamma-

tory trouble in the pelvis, there was no evidence of cystitis, and the urine was apparently normal. After a few days all the untoward symptoms subsided, and the patient got up and about. After a chill contracted when driving the symptoms returned, fever, sweating, and abdominal pain not specially localized. A normal appendix was removed without any benefit to the patient, and then, and not till then, was a deposit of pus found in the urine. With appropriate treatment all the symptoms disappeared and did not return.

A month or two ago I saw a patient who, after induction of premature labour, had no rise of temperature during the first six days of the puerperium. On the seventh day I was called to see her, as she had had a shivering fit and a temperature of 102° F. at 8 a.m. For twelve hours before this she had had great frequency of micturition. There had been nothing else complained of. No catheter had been passed since the confinement. The uterus was rather flabby, and larger than it should have been on the seventh day, but it was not tender, and the lochia were sweet and not unduly profuse. The urine contained pus, and I made a diagnosis of cystitis. In the evening of the same day the patient had a severe rigor, the temperature rising to 105° F. Two hours later, in spite of profuse sweating, the temperature was 106.8° F., pulse 160. The uterus was explored, and nothing was found in its cavity except a small fragment of inoffensive clot. She had had no urinary symptoms whatever during the day, and it seemed unlikely that pyelitis, with no tenderness to be felt in either kidney region, could produce this amount of pyrexia. The temperature next day was normal, and continued to be normal, but the right kidney became tender, and bacteriological examination revealed the presence of the *Bacillus coli communis*. The condition soon cleared up.

The occurrence of pyelonephritis in the puerperium emphasizes the importance of preventing retention during the second stage of labour and the avoidance of catheterization as far as possible after labour.

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THE ETIOLOGY OF THE EXCESSIVE VOMITING OF PREGNANCY.

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By most authorities it is agreed that the cause of the excessive vomiting of pregnancy arises within the uterus and acts through the nervous system, chiefly the sympathetic. Others consider that it is reflex, while some hold that a neurotic temperament is the cause. I have heard it stated that in the early months it is due to a neurosis, and in the later months of pregnancy to toxæmia. The various explanations may therefore be classified under three main groups: (1) Toxæmic; (2) reflex; (3) neurotic.

Toxæmic.

The toxæmic seems to be the most rational explanation of this complication of pregnancy, so that it may be considered first. The questions then arise, Where are these toxins produced, and by what channels do they act?

Dirmosér¹ has given what seems to me the most feasible explanation of the origin and progress of the toxæmic condition. He considers that the excessive vomiting is a reflex irritation of the sympathetic nervous system, commencing in the internal sexual organs, and through the secretory and motor fibres of the sympathetic leading to the production of changes in the biochemical processes, more particularly of the digestive tract, followed by atony of the gut and increased production and absorption of toxins in the gut. In the urine of patients suffering from this complication of pregnancy he found an increased amount of uric acid, aromatic sulphates, indoxyl, phenols,

etc., and also in certain cases urobilin, acetone, diacetic acid, etc. When the intestinal contents of these patients were administered to rabbits and mice the animals soon died, while the contents of normal persons were negative. The urine (catheter specimens) of the cases which have come under my notice was examined; in 11 the reaction for albumen was present, in 4 granular casts were observed, 6 gave the reaction for acetone, and in all cases the urea output was diminished.

Cazeaux² lays stress on the severe constipation associated with excessive vomiting; in my cases I found bad teeth present in all, in addition to constipation.

Williams³ of Baltimore emphasizes the value of the ammonia coefficient of the urine, which is high in the toxæmic type, but if there are no manifest changes in the urine, he holds that the cause of the vomiting is reflex or neurotic.

Behm⁴ refers the autointoxication to syncytiolysin, and thinks this theory proved by the cessation of vomiting with the cessation of pregnancy. He maintains that the toxins are produced in the periphery of the ovum and not by the metabolism of the fetus, thus explaining the excessive vomiting of the early months. He supports his theory by the excellent results obtained clinically by the administration of rectal salines.

Many years ago Smellie⁵ wrote:

Perhaps this complaint is chiefly occasioned by a fullness of the vessels of the uterus, owing to obstructed catamenia, the whole quantity of which cannot as yet be employed in the nutrition of the embryo; over and above this cause it has been supposed that the uterus, being stretched by the increase of the ovum, a tension of that part ensues, affecting the nerves of that viscus, especially those that arise from the sympathetici maximi, and communicate with the plexus at the mouth of the stomach.

Playfair,⁶ about 150 years later, wrote in a similar strain regarding the cause of excessive vomiting. In the Hippocratic writings the cause is attributed to φλέγμα. Matthews Duncan⁷ in 1879 had a fatal case of excessive vomiting in which, *post mortem*, was found acute yellow atrophy of the liver, which condition he suggested was a factor in the production of the vomiting.

Champetier de Ribes and Bouffe de Sainte Blaise⁸ had a case in which *post-mortem* hæmorrhages and areas of cloudiness were found in the liver somewhat similar to those found in eclampsia. They considered that the excessive vomiting was the result of intoxication due to hepatic insufficiency.

The *post-mortem* examination of the fatal case which came under my notice (BRITISH MEDICAL JOURNAL, July 8th, 1911), showed the liver to be enlarged with fatty infiltration, the spleen slightly enlarged, and the kidneys congested. There was a slight congestion of the mucous membrane of the stomach, with a small patch of superficial ulceration near the pylorus. Nothing abnormal was found in the brain.

Freund⁹ thinks the source of the toxins is in the periphery of the ovum or corpus luteum or placenta, but as the toxins in the great majority of cases do not produce excessive vomiting, he advises looking for predisposing causes, such as anaemia, chlorosis, nervous diseases, etc. Giles¹⁰ considers that there are three factors in the etiology: (1) Increased nervous irritability; (2) a local peripheral source of irritation; (3) a ready efferent channel for nervous energy (the vagi).

Reflex.

Displacement of the womb was held by Graily Hewitt¹¹ and others to be the commonest cause of the sickness of pregnancy, but Giles¹⁰ states that out of 300 pregnancies, 200 had vomiting, and he found it difficult to believe that two out of three women had a displaced uterus. McClintock⁵ met with several cases of retroversion of the gravid uterus where vomiting was not a prominent feature, and in some vomiting was entirely absent. Playfair⁶ found that displacement was the exception in cases of excessive vomiting. Williams³ states that displacement may occasionally be the cause, and considers this is proved by the fact that vomiting sometimes ceases immediately after replacement. I have assisted at two Caesarean sections where ventrifixation had been performed on both patients some years previously. In each case the uterus was so held down that at the operation the uterine incision was wholly on the posterior wall, as the anterior wall had taken no

part in the uterine enlargement. In both cases there had been no excessive vomiting, and in each there was considerable uterine displacement, so much so that the children could not be born alive through the natural passages. I have had a patient with excessive vomiting and retroversion. The uterine displacement was left alone and the hyperemesis ceased with treatment.

Hydramnios and twins are also mentioned as causes. Copeman,¹² Cazeaux,² and others considered the cause often lay at the cervix, so that rigidity, deep tears, erosions, etc., should be attended to at once, the first-named by dilatation of the internal os, a procedure which, I think, would involve the risk of miscarriage. Bennet¹³ maintains that where vomiting is excessive and obstinate there is almost always inflammation or disease of the cervix or a diseased uterus, and recommends the local condition to be energetically treated. Freund⁹ records 3 cases in which the turbinates, especially the inferior turbinates, were enlarged and congested; by attending to the nasal condition, he states, he cured the hyperemesis. Giving careful consideration to the above alleged causes, is not one justified in regarding each as a coincidence merely?

Neurotic.

Williams³ has cured several cases by a vigorous lecture on the dangers attending the induction of labour. V. Winckel⁹ holds that hysteria is almost always the cause; but many women are hysterical, while the excessive vomiting of pregnancy is not common. Kaltenbach¹⁴ considers hyperemesis gravidarum to be the exhibition of a latent neurosis, while others have satisfied themselves by the results of treatment that neurosis is a cause in many cases.

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FEVER IN THE NEWBORN.

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It is customary to reserve the term "newborn" for an infant in the first week of life. I propose to use it somewhat less definitely, so as to include all infants in whom the cord is still attached to the navel, for it is in this respect that such infants differ from older ones. As a general rule the cord separates in seven to twelve days, leaving a healed surface. Sometimes the process of separation takes longer, or a raw surface may be left, which perhaps does not heal until two or three weeks after birth. During this period the infant is peculiarly liable to septic and other affections of the cord and navel. In the earlier days it may show abnormal effects of confinement, some of which are prolonged or even permanent—for example, asphyxia, atelectasis, cephal-hæmatoma, sterno-mastoid tumour, various injuries, intracranial hæmorrhage, birth palsies, and ophthalmia. In addition the respiratory tract has to adapt itself to changes in temperature, and the alimentary system to undertake the digestion and absorption of food. Unsuitable diet easily and quickly induces serious illness. Many of the affections mentioned are associated with a rise of body temperature.

"Fever" means a rise of temperature of definite duration. The temperature should be taken in the rectum. In the newborn it is normally the same as, or slightly higher than, that of the mother. It generally oscillates about 99.5° F. (99° to 100° F.). Exposure, bathing, and lack of food quickly reduce it to 96° or 96.5° F., and it may not reach the normal level for three or four days—perhaps in twenty-four hours, perhaps not for ten days or even longer.