THE

RELATION OF THE URINARY ORGANS

TO

PUERPERAL DISEASES.

BY

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Modern Renal Pathology may be said to date from the year 1827, when Dr. Richard Bright began to publish his now famous observations and his elegant plates. For several years he was his own commentator, working mostly alone. But in 1839 Christison in Edinburgh and Rayer in Paris published their important treatises confirming and extending the observations of Bright, and directed the attention of students to this interesting field.

From 1840–1850 Kölliker, Rokitansky, Bowman, and Simon elaborated the histology of the kidney; Becquerel, Geo. Johnson, Bence Jones, Berzelius, and Beale the chemistry and micrography of the urine.

From 1850–1860 Frerichs, Virchow, Todd, and Traube classified renal diseases according to the tissues and functions implicated; studied the relations of the liver and the heart to the urinary organs, and formulated theories of albuminuria from retarded circulation; uræmia from restrained excretion;

[1] Read before the Medical Society of the County of New York, February 26, 1877.

ammonæmia from chemical conversion, and eclampsia from hydræmia. In the following decade, from 1860–1870, these theories were made the occasion of many experiments on the human subject and upon animals by Hammond, Harley, Oppler, Parkes, Richardson, Schottin, Treitz, and Zalesky, and many others. An immense amount of careful and well-distributed work was done, which recently has been collated, sifted, and condensed in the works of Rosenstein, Roberts, and Grainger Stewart.

Early in the course of these studies, obstetricians recognized their important bearing upon the convulsive diseases of pregnancy.

The general similarity between puerperal convulsions and those of Bright's disease had been already noticed by Simpson and Rayer, when Lever, of Guy's Hospital, from 1843 to 1847, published a series of cases in which eclampsia and albuminuria were associated. Shortly after followed the observations of Litzman in Germany and Blot in France, agreeing in the statement that twenty per cent. of all pregnant women, and a still larger proportion of primiparæ, showed albuminuria. In 1855, Braun of Vienna made the formal statement that "Eclampsia of parturient women is commonly the result of uræmic intoxication, arising from Bright's disease."

Objections to this statement have been made by many observers; notably by Prof. Barker in papers read to the New York Academy of Medicine, and in his work on Puerperal Diseases, and by Dr. Braxton Hicks of London.

They show that (1) Puerperal convulsions, in many cases, resemble those of reflex irritation quite as much as they do those of Bright's disease; (2) that pregnant women suffering from well-marked Bright's, sometimes go through child-bed without eclampsia; (3) that some women dead of eclampsia, have shown in life no symptoms, and in death no lesions of Bright's disease; (4) that in others the evidences of Bright's follow rather than precede eclampsia.

Hence, these authors, while not excluding the theory of uræmia, incline to that of cerebro-spinal nervous irritation as the most common proximate cause of puerperal convulsions. And they find clinical support for this position in the accepted value of chloroform, chloral, and morphia in these conditions.

It is not the purpose of this paper to consider specially the subject of puerperal convulsions, but we may note in passing these objections, that the absence of convulsions does not speak for the absence of Bright's disease, since, according to Grainger Stewart,[1] three out of four of all cases of positive Bright's also escape convulsions. Neither do the beneficial effects of morphine indicate that Bright's disease may not be present, since, according to Prof. Loomis,[2] morphine is of special use in the convulsions of Bright's. The observation of Braxton Hicks,[3] that uræmia sometimes succeeds rather than precedes eclampsia does not show that the eclampsia is the cause of the uræmia, since it will, we think, appear that various puerperal conditions, not attended with eclampsia, tend to end in uræmia. It is necessary, to the full solution of these questions, (1) that the exact pathological value of albumen in the urine should be determined; (2) that it should be eliminated from consideration when it depends upon extra-renal sources, sc., cystitis, vesical catarrh, hemorrhage, etc.; (3) that the equation between the extrication of albumen and the retention of urea should be fixed; (4) that it should be known whether the liver or the kidneys are most concerned in the production of urea; and (5) whether urea, or some of its derivatives, as carbonate of ammonia, is the real toxic agent.

The observations of Litzman, as to the frequency of albuminuria in pregnancy, were quoted without being accepted, since Abeille,[4] who followed him, reduced the proportion from 20 to 10 per cent., and Elliott and Van Arsdale[5] to 5 per cent. My own observations agree with the latter estimate.

While treating thus fully of the relation borne by the kidneys to eclampsia, authors upon puerperal diseases have not so much considered other modes of morbid action in the urinary organs or so fully developed the circumstances under which they arise, and the influence which they exert. Winkel and Joulin, the later representatives of the French and German Schools, hardly discuss the subject. Dr. Barnes has an interest-

[1] Bright's Diseases, p. 106, Am. Ed.
[2] Med. Record, Vol. 7. p. 160.
[3] Transactions Lond. Obst. Soc., Vol. xii.
[4] Traité des Maladies à Urines Albumineuses set Sucrées.
[5] New York Journal of Medicine, 1856.

ing lecture on Uræmic Excretory Fever, and Dr. Barker frequent casual allusions.

On p. 73 he says: "Clinical evidence has amply demonstrated that convulsions, the various phlegmasiæ incident to the puerperal state, the pyæmic diathesis, septic absorption, and puerperal fever, or any of these causes, may develop an albuminuria not previously existing, and lead to that aggregate of diseases of which albuminous urine is one symptom."

This compact statement might serve as the text for nearly all that I shall have to say this evening, and leads us to regret that the circumstances of publication excluded from a work, so replete in clinical interest, the chapters upon the thoracic, renal, and vesical conditions of child-bed, which, as we understand, had been partially prepared for it.

Hervieux,[1] in his great work published in 1870, has very carefully and fully discussed puerperal nephritis, both inflammatory and metastatic, and puerperal cystitis, but appears to attach little practical importance to them, since he declares them "incapable of diagnosis during life, and minor facts in the great category of puerperal poisoning."

In periodical literature, I have found in the third volume of the Archiv für Gynækologie, an article by Dr. Kaltenbach, of Freyburg, and in the second volume of the Transactions of the Berlin Obstetrical Society, a brief paper by Olshausen, of Halle, to which I am indebted for suggestions, and from which I may cite cases.

My own attention was drawn to this subject by a case in private practice in 1873, and within the last two years, in my service at Charity Hospital, I have seen some cases whose clinical history and necroscopy have enforced the idea *that the urinary tract in its whole length is especially liable to share in any and every morbid process which may follow childbirth, and that the diseases of the urinary tract thus excited have a very marked influence upon the issues of the puerperal state, and sometimes assume the leading rôle among the causes of death.*

It was not until the preparation of this paper was far advanced that I saw for the first time that in two of his Lec-

[1] Traité Clinique et praticale des Maladies puerpérales.

tures upon Puerperal Fever, Dr. Barnes had followed much the same line of thought that I had proposed to myself, and that some matters and forms of statement, which I had supposed somewhat original to myself, had been more fully traversed and better rendered by others.

The compound word, genito-urinary, designed to indicate the intimate association of two sets of organs and functions, is commonly used in reference to the male. But it is even more appropriate in the case of the female, since, morphologically,[1] the vagina and the bladder are both diverticula from the same pouch of the allantois, and the uterus and the ovaries are in much closer contiguity to the ureters and the kidneys than are the testes and the seminal vesicles, which are the corresponding organs of the male.

The proper sexual organs of the male are all extra-abdominal, while those of the female are, for the most part, intra-abdominal, and by a common peritoneal covering, and a common web of connective-tissue, are brought into closer relation to the urinary organs than in the case of the male.

I am not aware that in the male there has been described anything analogous to the nerves connecting the uterus and the kidney, which Frankenhäuser, of Jena, has demonstrated and figured, and which may be supposed to be concerned in the phenomena of hysteric urine almost, if not quite, peculiar to the female.

Now, if in the male and the non-pregnant female diseases and lesions of the superficial genital organs may conduct, either by the continuous mucous surface of their interior, or by the outer investment of connective-tissue, to acute, rapid, and fatal disease of the kidney, we can well understand how the contusions and lacerations, *inevitable in the parturient process*, may more readily and frequently lead to acute disease of the kidney in the system of the puerperal woman, increasingly predisposed, as it is, through the whole term of pregnancy to such a result.

Three such cases I shall cite. The two first are from a report of Sir Charles Murchison to the Clinical Society of London, in Nov., 1875.

[1] Schroeder, Diseases of Female Sexual Organs, p. 568.

CASE I.—A grocer's clerk, aged 28, who had been regularly at his work up to the day before admission to the hospital, and was not known to be ailing in any other way than from a gonorrhœa, which had existed for some time, was received into the wards in a state of profound coma, varied by muttering delirium. There was no eruption on the skin—no sign of thoracic or visceral disease. He died in a few hours. Except some congestion of the base of both lungs and a few small patches of incipient pneumonia, there were no lesions except in the urinary organs.

The entire length of the urinary tract, from the meatus urinarius to the pelves of both kidneys, showed a mucous surface deeply injected and covered with pus. Both ureters, and both renal pelves were filled with pus. Both kidneys were much enlarged, of very dark color, surface smooth and soft. Dark blood dripped from the surface of the section.

CASE II.—A woman of 25, domestic servant, had apparently been quite well on the day previous to her admission. She had made the trip from Paris to London in company with the cook of the same family, and had eaten a fair supper on arrival. Had been somewhat restless during the night; in the morning had a convulsive fit, followed by profound unconsciousness, and was taken to the hospital, where she died on the third day, the symptoms continuing. Post-mortem examination showed the viscera of cranium, thorax, and abdomen in state of moderate hyperæmia; but no structural lesion, except in urinary organs. Both kidneys were in the early stage of acute nephritis, large, smooth, and black from intense congestion. Bladder, ureters, and pelves contained abundant pus. Lining membrane of vagina, urethra, bladder, ureters, and pelves, intensely red.

CASE III.—(From Kaltenbach.) A woman of 35 was operated (colporrhaphy) for prolapse of anterior and posterior vaginal wall. Before operation urine was normal and no bladder symptoms were present. After operation temporary retention, single introduction of entirely new catheter; moderate or partial suppression; the third day after the operation, smarting and burning in urination, increasing pain in bladder, vesical tenesmus after urination. Hypodermic injection of morphine, followed by relief of bladder symptoms, but then followed a dragging pain in the left groin, extending by the seventh day to the neighborhood of the kidney, which was extremely sensitive to pressure. The next day the pain was somewhat less, but the urine was cloudy and acid; specific gravity, 1020, precipitating flocculi of albumen; under the microscope, pus, epithelium of bladder in cohering flakes; small obovate cells, probably from the ureters; in larger quantity elongated and spindle-shaped shrunken cells from the pelvis of the kidney. The patient gradually recovered.

In the two first cases we have acute erysipelatous inflammation travelling rapidly upward along the inner surface of the urinary passages to the kidney, setting up acute nephritis, marked by acute uræmia, producing coma and death, solely as

a consequence of the kidney disease. In the third case we have traumatic inflammation exciting at first moderate cystitis, which was subdued, then travelling along the external surface of the urinary passages to the capsule of the kidney, as shown by the great sensitiveness to pressure, thence through the cortical to the tubular portion, as shown by the late appearance of the urinary disturbance and the proportions of different epithelia.

The *predisposition* of the parturient woman just stated, consists in that circle of changes by which she departs from the non-pregnant state and returns to it in a period occupying about eleven months.

In the following brief statement I shall sometimes use the statements of Dr. Barnes and sometimes those which were prepared before I had seen his language on the same subject. First are to be noted the changes in the blood.

"These probably begin very early. Convulsions with albuminuria have been seen as early as the fifth month, before the uterus is large enough to exert any material pressure. The circulating fluid, the circulatory and the excretory apparatus, have a double work to do. They must suffice for two organisms. The blood must nourish both; it must purify itself of the degraded materials resulting from the molecular changes going on in both.

For two reasons the rapidity of the circulation is increased. First, the quality of the blood being lowered, it must be sent round more quickly in order to compensate for its lesser value. Secondly, there is the increased demand caused by the new organism. There is every reason to suppose that so powerful a local action cannot go on without affecting the chemical properties as well as the volume and the circulation of the blood. The pulse is quickened; the heart has to labor harder to maintain this increased rate of circulation.

There is a physiological hypertrophy of the heart in pregnancy, analogous to the physiological hypertrophy of the gravid womb." (Larcher, Guillot, and others.)

There are two other causes for hypertrophy of the heart; namely, the increased difficulty of circulating an impoverished fluid, which we see illustrated in the palpitations and cardiac tumult of so-called anæmia; and the increased power required

on account of mechanical obstruction from intra-abdominal pressure. These two causes equally, though not in precisely the same form, subsist in chronic Bright's disease, and serve to constitute a more or less remote parallel between the two conditions.

Andral and Gavarret, quoted by Leishman (System of Midwifery, p. 221), state that at term the fibrine of the blood is increased by sixty-two per cent. of its normal amount, the water and phosphorized fat to an undetermined extent. The white corpuscles are also largely increased, while the red corpuscles are diminished about nine per cent., and the albumen to a variable and undetermined extent.

Thus the maternal blood is in a condition of imperfect oxidation. How this may occur, we can see when we consider that, in the first place, the mother's respiratory capacity is decreased by the encroachment of the abdomen on the thorax; and, in the second, that, in the comparative quiet and seclusion which the civilized woman observes in the later months of gestation, the respiratory process is not quickened by exercise; and the air respired, being in-door air, is itself in a condition of suboxygenation. At the same time the red corpuscles give up oxygen in the placenta to supply the aeration of the fœtal blood. Thus, by diminished receipt and increased expenditure, the quota of oxygen is drawn down.

Here also the measure of divergence from the normal state is to be found in the sum of the opposite departures. Thus, if there be an equation between the red and the white corpuscles in the normal state, the inequality of the disturbed state is found by adding together the deficiency of the red and the excess of the white; and the vitality of any particular quantum of the blood will be further lowered by the addition of several equivalents of water. The red corpuscles, or their hæmoglobin, are the only carriers of oxygen, which is the factor of normal metamorphosis by combustion; and in the insufficient supply of it the carbonaceous elements in circulation and in tissue are prone to that rearrangement which we call fatty degeneration, for fat is one form of hydrocarbon.

It is understood that in all zymotic processes there is an indefinite multiplication of parasitic life in all the tissues. And it is the opinion of some that the mischief of the microphytes

or mycrozymes consists in the energy with which these evolving forms, in their nascent stage, abstract oxygen from the weakening vitality of the organism which harbors them.

Have we here a solution of the rapidity with which fatty metamorphosis invades the organs of the parturient woman? It will be seen that the changes of the kidney, of which we are about to speak, fall in this line.

Furthermore, as all retrogressive changes end in solution, so they begin in infiltration[1]—a condition to which the plethora and hydræmia above noted alike conduce. The attenuated quality and the increased quantity alike favor capillary transudation. Again, the increased amount of fibrin not only favors coagulation in the transuded fluid, but within the vessels large and small.

Thus these three states of hyperinosis, hydræmia and oligoerythrohæmia, or deficiency of hæmoglobin, condition a cloud of accidents more or less imminent over the parturient woman.

These are: heart clot; capillary infarction; subserous hæmorrhages; general œdema; specially cerebral œdema, embarrassing the nutrition of the brain by compression of intracranial vessels; and infiltration of those complex excretory organs, the liver and the kidneys.

This hepatic and renal œdema further increases the tendency to fatty metamorphosis, not only by solution, but by extrusion of blood from compression, as in the brain.

It is not in place now to notice how all these blood changes have for a final cause the development of the ovum, nor to remark how seldom the cloud breaks in a rain of accidents. More pertinent is it to note how closely these conditions correspond with those of Bright's disease, and how, in the exceptional puerperal cases, they conduce, as they do in Bright's disease, to headache, amaurosis, eclampsia, pericarditis, pleurisy, and pneumonia.

If, now, to these causes of disease we add another in the shape of direct pressure from the distended uterus upon the emulgent veins of the kidney, we have the cumulative effect on the circulation of the organ in venous hyperæmia of the Malpighian tufts, capillary distention and stasis, effusion of liquor sanguinis and fibrine into the inter-tubular structure, and into the

[1] Rindfleisch, Pathological Histology, §§ 7–16, passim.

tubules themselves, forming within them a fibrinous coagulum, a hyaline cast. Upon its surface the loosened epithelium, detached by a still continuous transudation, may cohere, making it an epithelial cast. If it be awhile retarded in its progress through the convoluted tubule, the devitalized and sodden epithelium breaks up into its germinal granular matter, and we have a granular cast. If it be still longer delayed, the retrograde process in the epithelium goes still further, and the granular matter is resolved into fat globules, giving us a fatty cast. If upon this train, so prepared, there fall a spark of peripheric irritation, of septic or specific poison, we have a fulminant eclampsia or icterus. Otherwise, nature attempts her own relief by setting up premature delivery, and, subsequent to delivery, unloads infarcted liver, kidneys, and intestinal glands by an excretory uræmic fever. Such a case I have fully reported in the sixth volume of the *Medical Record*, p. 265, and will here add other illustrations:

CASE IV.*— Mrs. H., aged 35, was admitted to hospital in the sixth month of her fifth pregnancy, moribund. Was in good health until a fortnight before admission. Thence symptoms developed as follows: Mental depression, gastric oppression, vomiting, progressive jaundice, constipation, hæmatemesis, stupor alternating with violent delirium, premature delivery of twins, coma, death. Autopsy thirty-one hours after. Skin and viscera icterized. Lungs moderately congested and œdematous. Many small ecchymoses beneath pericardium, and capsules of liver and spleen, and through the substance of liver, which was very much reduced in size. On scraping the surface of section a large amount of fatty matter remained on the knife. The cells on the periphery of the lobules were almost completely broken down; towards the interior were enlarged, filled, some with oil globules, some with fatty matter. Kidneys: A very copious exudation occupied the uriniferous tubules, and the epithelium was affected in the same way, as were some of the cells of the liver, *i. e.*, some of the cells were swelled, dense, opaque, granular; some extremely fatty; some had broken down and disappeared.

CASE V.—Mrs. M., æt. 22, near the end of first pregnancy; was always in good health. Became suddenly ill on the 20th of October, with symptoms at first referred to the uterus. Admitted to hospital on the evening of the 22d, premature labor on the morning of the 24th; death on the same evening; illness three and one-half days. At the autopsy: Uterus appendages and peritoneum were found to be all normal for the parturient state, and no morbid anatomy was found anywhere except in the liver and kidneys. In the latter the tubules

* From Grainger Stewart on Bright's Diseases.

were distended, opaque, gorged; their epithelial cells full of granular matter and fatty matter, very easily breaking down; the pressure of a light-covering glass sufficing to reduce the tube casts to a fine débris. The stroma also contained numerous fatty granules, arranged in tubes and lines, as if situated in the connective-tissue corpuscles. Similar changes in the liver were less advanced.

Case VI. (Abridged from report by Dr. Oppenheimer, of Charity Hospital.)—Ellen O'Brien, æt. 29. Delivered December 23d, after natural labor of nine hours. Uterus contracted well. Immediately after delivery lost control over sphincters—an almost continuous flow from the urethra and anus. Urine acid, sp. gr. 1016, abundantly laden with pus, casts and epithelium from bladder, pelves and tubules. Urine after filtration showed one-fourth albumen. Temperature 103½ on the second day, thenceforward 99–101½; in articulo, 102. Diarrhœa incontrollable; tongue moist and slightly furred. Abdomen never tympanitic or painful; lochia normal. Conscious and uncomplaining until three days before death, when she became stupid. Pulse and heart action very feeble. Death, from heart failure, seventeen days after delivery.

By autopsy.—Viscera of thorax all normal; uterus, appendages, vagina, cervix, and peritoneum normal.

Liver large, flabby, pale yellow; section showed several infarcti in yellow stage.

Kidneys large, soft capsule loosened; both pelves showed pyelitis—most marked in right, parenchymatous nephritis; bladder normal; small intestines normal; colon in state of catarrhal inflammation throughout—most marked at the cæcum, where were small superficial ulcerations.

A fourth case speaks for the hyperinosis of the parturient woman.

Case VII.—Lizzie O'Neill, æt. 32, was delivered December 19, 1875, after a short and easy labor of an hour and a half. All the sequelæ were apparently normal, and she was up and dressed on the sixth day for Christmas. On the morning of the 2d of January she went to the water-closet and returned gasping, cyanotic; expiring in a few moments.

The heart was found to be somewhat fatty. In the first divisions of the left pulmonary artery was an old and partially decolorized clot, overlapped by a soft recent clot two and one-half inches long in the main trunk, in diameter as large as a wooden pencil. The liver was soft and friable, weighing 6 lbs. 7 oz. Kidneys somewhat increased in size, cortex swollen, tubules yellowish. Both pelves and ureters contained puriform fluid; left ureter was dilated, and there was marked cystitis. Uterus and appendages all normal.

I regard these cases as not less interesting by their dissimilarity than by their essential identity.

Four women, so far as known, previously healthy, succumbing to the blood changes of pregnancy, without symptom or lesion of the uterine organs; one by acute fatty metamorphosis (I use this word rather than "degeneration," since the latter would seem to imply a slower process) of the liver, plus a minor grade of the same process in the kidneys; another by acute fatty metamorphosis of the kidney, plus a minor grade of the same process in the liver; a third by recent œdematous infiltration of the kidney and fatty metamorphosis of the same, plus similar œdema and catarrh of the overlying intestine; and the fourth by a morbidly coagulable blood, plus a fatty heart, liver and kidneys. Like the aspect of the old Parcæ, "Facies non una, nec diversa tamen, sed quales decet esse sororum."

According to Frerichs and Murchison, more than half of the cases of acute yellow atrophy of the liver occur in women in the later months of pregnancy—almost always inducing miscarriage.

The cases thus far cited were free from every uterine complication; they were in nowise influenced by accidents of the puerperal process, and they therefore show the organic predisposition in the pregnant woman to diseases of the kidney.

This predisposition is farther increased by changes in the tissues corresponding to those in the blood.

The non-pregnant uterus with its appendages weighs perhaps five ounces; the same, containing the matured ovum, not less than fifteen pounds; the same, four weeks later, from seven to ten ounces. To provide for the evolution of the uterus, the extrusion of the ovum and the involution of the uterus, requires a very signal development of the muscular and vascular tissues.

The plates of Cruveilhier and Mascagni demonstrate a mass of venous convolutions enveloping the uterus in such abundance that, when fully injected, they constitute a nearly complete sheath. The same description may apply to the lymphatic vessels in the subserous connective tissue. Clusters of lymphatic ganglia, previously unrecognizable, encircle the cervical zone of the uterus and the inner portion of the Fallopian tubes.

So fully equipped, and so apt for its coming work is the absorbent system that women near term are especially liable to all infectious diseases. Dr. Braxton Hicks, in the 12th volume of the Obstetrical Society's Transactions, publishes 37 cases of

puerperal scarlet fever (27 fatal), commencing either shortly before labor or within five days after, thus showing either extraordinarily shortened incubation or ante-parturient absorption. Also similar observations with regard to diphtheria and erysipelas. Dr. Barnes says it is not uncommon for pregnant women to have a second attack of scarlatina. Dr. Haussmann, whose studies in this direction have for a long time been in high esteem, in the last number of the Berlin Obstetrical Journal says that the vaginal secretions of at least 11 per cent. of women near term show the presence of bacteria.

We may now consider how the process of parturition affects the urinary organs. By this process, in the course of a few hours the woman suffers a loss of substance, and a diversion of the circulation about equal to that involved in the amputation of the thigh at its middle third, often also combined with an equivalent amount of pain and nervous shock, and in addition a degree of muscular effort without a parallel.

The tense contractions of all the abdominal muscles, including the diaphragm, the closed glottis, the compressed but excited heart, and the maximum pressure on the emulgent veins, are all factors in inducing a marked—fortunately, for the most part—a transient congestion of the kidney.

But, at this stage, the lower portion of the urinary organs becomes exposed to great hazard. For days before labor, the low lying head often so flattens the bladder against the pubic arch as to prevent its full evacuation, and the residual urine becoming alkaline, disorganizes the mucous membrane. During labor, the contusion and stretching of the anterior wall of the vagina, determine all grades of accidents from sphacelus to catarrh. Some degree of morbid action from these causes is seen in the majority of cases. A single case may serve as an illustration.

Case VIII.—A lady, the patient of a medical friend, believed to be at the end of her term, and suffering from pains in the hypogastrium, with frequent micturition, summoned her physician. He found the head low down, the cervix obliterated, the os distensible. For a week he waited on a labor which did not begin. Then, being obliged to leave, he passed the case to me. I saw her first when labor was in progress. It was normal and easy. Eight hours after, I found her suffering from retention with much tenesmus. The urine drawn by catheter contained abundantly epithelium of the bladder

and pus. Acute cystitis was developed, but in a few days yielded to washings of warm salt water and anodynes.

This form of cystitis is very common, usually slight in its symptoms and tending to spontaneous recovery. I believe that I have very often overlooked it; confounding its subjective symptoms with after-pains; its tender hypogastric tumor with a sensitive uterus; its troubled urine with urine contaminated by lochial admixture or vaginal secretions. Rarely is there a vesical paralysis permitting a painless hyperdistention of the bladder, to such an extent that we might suppose meteorism was present, did not the fluctuating tumor, soft but dull on percussion, and the stillicidium urinæ, but especially the catheter, readily conduct us to the true diagnosis.

It is the opinion of the Germans, as stated by Kaltenbach and Olshausen, that bladder-catarrh or cystitis is often caused, where it would not otherwise exist, by the introduction of the catheter. They declare that danger from this source is threefold: 1st. The irritation caused by forcing a catheter through the swollen urethra. 2d. The carrying of infectious matter, s.c. lochial discharge, vaginal secretion, pus from vaginal laceration, or unclean matter accidentally remaining in the catheter, into the bladder; and 3d, The introduction of air too often charged with parasitic spores.

To guard against these contingencies, which they declare they have occasionally seen to be very mischievous, they have made the rule, in some of their maternity hospitals, that the catheter shall never be introduced under cover; that the orifice of the urethra shall be fully exposed to view, and previously wiped with a lock of cotton wetted in some mild antiseptic solution; and that only an instrument, new or capable of being disinfected by fire, shall be used. For lack of these precautions they declare that a benign catarrh may become or be made an erysipelatous or a diphtheritic cystitis, occupying the entire bladder, ascending the ureters, and invading the kidney with fatal results. The following case, which fell under my own observation, supports, in part, this declaration.

CASE VIII.—M. M., 28 years of age, an habitué of the Island and a hard character, was delivered by me with forceps of her child, weighing eleven and one-half pounds, naked! It was dead, very much de-

composed, the skin of the arms and back being wiped off by the towel used in drying it. The discharge which followed extrication of placenta was fœtid, no hemorrhage. The perineum was lacerated nearly to the sphincter, not involving it.

The following day—Pulse 120; temp. 101½°–103°. Much tympanitis; no tenderness of abdomen; lochia offensive; water drawn and vagina syringed, ter-in-die, with carbolic solution.

Second day—Temp. 103°–103¼°; pulse 120. Dark slough, forming on the perineal wound. Constant disinfection employed.

Third day, morning—Pulse 94; temp. 101°. Abdomen softer; patient says she is better. Evening, temp. 102°; pulse 130.

Fourth day—Pulse 126; temp. 101°. Patient is much worse. Abdomen very much distended, but there is no pain in it, and it may be freely manipulated without tenderness. Patient grew rapidly worse, and died exactly four days after delivery.

Autopsy, twelve hours after death.—Abdomen, upper portion, tympanitic; dull on percussion over pubis; perineal wound sloughy, covered with a fetid discharge. Thorax: Recent *pleurisy*, with slight amount of fibrinous exudation in lower third of right lung. Also a patch on the outer surface of left lung, about midway. Underlying this pleuritis was a thin stratum of acute pneumonia, averaging six lines in depth. Remainder of lung somewhat congested, with some muco-pus in bronchi. Heart shows a few small superficial hemorrhages; walls contracted, cavities empty, valves and vessels normal. Abdomen: Cavity of pelvis contained a little thin serous and purulent fluid with flocculi of fibrine. Surface of peritoneum: transparency lost; no adhesions; *a yellowish substance may be scraped from the surface.* Intestines distended with gas; uterus reaches fully to umbilicus. Liver: Much enlarged (5 lbs. 10 ounces), flabby, very friable, deeply congested and œdematous; a moderate amount of old cirrhosis; spleen enlarged, diffluent. Kidneys: Much enlarged; one weighing thirteen ounces, the other ten and one-half ounces, or nearly two and one-half times their normal weight; capsule loose and marked with inflammatory mottling. Surface of kidney shows lobulated swellings (Malpighian bodies), sulci depressed and pale yellow (fatty degeneration of intertubular connective tissue); the whole surface shows minute puriform points, scattered miliary hemorrhages, deep redness of the swollen and prominent portions of surface. By section: Tissue soft, cortical substance twice as thick as normal, and, with the pyramids, is the seat of an almost general, acute, suppurative nephritis. The whole mucous membrane of renal pelves shows intense pyelitis with large patches of hemorrhage in substance of membrane. Ureters: Moderately thickened and dilated; the mucous membrane in the same condition as that of pelves. Bladder: Its walls hypertrophied, capacity much increased; mucous membrane much thickened, rough, villous, at fundus filled with miliary hemorrhages; prominences of trabeculæ covered with diphtheritic membrane. Uterus and appendages: Womb, twelve inches long, eight inches across fundus; walls, three-quarters of an inch thick and flabby; in poste-

rior wall several small subperitoneal fibroids; surface of cavity covered with shaggy coagula, at placental site; and throughout with a thick diphtheritic membrane, which superficially is disorganized and gangrenous, and extends into the substance of the uterine wall and sinuses. The cervix is slightly nicked at the right lip of os. Summits of vaginal rugæ, covered with diphtheritic membrane, still intact and firmly united with structure of mucous membrane. Stomach and intestines were normal. (Maxwell.)

This unfortunate woman, containing a putrid fœtus, and therefore septicæmic ante-partum, might have died if all her urinary organs had been sound, but, as I read the autopsy, it seems that the morbid process was farther advanced in the urinary organs than elsewhere, that the thoracic lesions were probably consecutive to the renal trouble, and that death, when it occurred, was determined by the condition of the kidneys. Diphtheritis may have extended to the bladder by contiguity from the anterior wall of the vagina, or it may have been transferred by vaginal secretions adhering to the catheter.

In view of the fact that although the secretions of the diphtheritic fauces are constantly falling through the œsophagus, yet, so far as I know, we rarely have diphtherite of the stomach, it would appear more probable that the extension was by contiguity rather than by transfer.

It will be noted that the diphtherite extended through the entire genital track as far as the oviducts, and through the entire urinary tract as far as the ureters. Beyond these points we have *suppurative* salpingitis and ovaritis; suppurative ureteritis and nephritis. That, in both cases, this was due to the same proximate cause, i.e., swelling of the muscular coat of uterus and bladder, which occluded the distal orifices of these tubes, appears probable from the immense œdema of both kidneys and ovaries, both more than double their normal size, and the dilated ureters and loosened capsule.

At this point the observations of Zalesky become very interesting. He removed the kidneys from a certain number of animals, and in an equal number of the same animals he tied the ureters, at their entrance to the bladder. The latter died promptly from uræmic coma, and from the surface of the peritoneum and pleura he *could "scrape a yellowish substance which proved to be salts of urea;"* an apparently similar deposit was (as above stated) found upon the

peritoneum in this case. On the other hand, the animals, from which the kidneys were removed, lived a much longer time, becoming uræmic very slowly. These experiments having been repeated and confirmed by Oppler, Schottin, and Perls, have been accepted as showing that the urea is not secreted by the kidney from the blood, but formed in the kidney. The observations of Cyon and Meissner show that it is also formed in the liver.

The two cases last cited may be taken respectively for examples, if not types, of inflammation of the mucous and of the muscular coats of the bladder. I have a third observation which I will not cite at length, in which a cellulitis of the right broad ligament, swelling largely, slowly developed albuminuria; cells supposed to represent the ureter being first observed; the hypogastrium then became very sensitive to pressure, and finally vesical epithelium and pus appeared abundantly in the urine. Inferentially, we have in this case, periuteritis, followed by pericystitis striking through all the coats, and becoming a general cystitis, enduring until the cellulitis terminated in recovery.

This matter of the occlusion of the ureters, whether by thickening of the muscularis of the bladder, or by lymphangitic swellings, seems to me of much interest in view of our recent discussion of lacerations of the cervix. By every such laceration, and particularly by such as extend into vaginal tissue, the web of greatly developed lymph spaces and lymph canals is opened, and freer entry of whatever is morbid in vaginal secretions, and of the products of suppuration, is promoted. If now we remember that the cervix is in apposition with the middle portion of the base line of the triangle of Lieutard, connecting the vesical orifices of the two ureters, we can see how readily traumatic swelling, in proportion to its amount, will infringe on the calibre of the ureter.

Having thus considered the conditions subsisting before birth, and the contingencies connected with birth, we may now look at those which arise *after* birth. After the middle of pregnancy the uterus escapes from the true pelvis, and expands under only such moderate pressure as exists in the large cavity of the abdomen. That this expansion sometimes effects a mischievous coarctation of the renal emulgent veins, is on all sides

conceded, but the blood and lymph-moving canals of the uterus itself are in good measure shielded. After birth the conditions are somewhat changed. The uterus descends into the true pelvis as far and as fast as its reduced size will allow. In all the autopsies of women dying in the first week after delivery, the transverse diameter of the fundus is greater than the transverse diameter of the pelvic brim, and the longitudinal diameter is largely in excess of the depth of the true pelvis. Thus the womb remains resting upon the promontory of the sacrum, and the efferent trunks of its blood and lymph vessels occupy the half-enclosed canal between the promontory and the inner border of the psoas muscle (see Hunter's plate). In the condition of tonic contraction, the globo-cylindrical form of the womb will leave this not essentially constricted. But the condition of firm contraction is not persistent, and is interrupted by many physiological causes, and by all morbid processes. When flaccid, the uterus must overlie this canal, and at least, by its weight, oppress the efferent vessels, and the ureters, which lie parallel with them, and in the same web of connective tissue. Thus, the pelvic circulation and urination will be correspondingly impeded. Doubtless, this condition is often very much aggravated by the injudicious employment of compresses and binders, by which, with the purpose of preventing concealed hemorrhage, we crowd the womb hard down on the pelvic brim. Thus, ordinarily, for three days the collapsed rectum cannot deliver the contents of the colon, and the hampered bladder must make partial and unsymmetrical efforts to expel its contents—limited in amount by the coarctation of the ureters. I have repeatedly observed in the reports of autopsies that, accurately at the margin of the brim, the ureter, of normal size below, was upward to the pelvis of the kidney, dilated to twice or three times its normal calibre.

Kaltenbach makes also this observation:

CASE IX.—S. B., twenty years; primipara. Urine, in pregnancy, normal; after delivery, endometritis; diphtheritis of slight lacerations in mucous membrane of vulva, followed by diffuse parametritis and peritonitis; died at the fifteenth day post-partum. In the last three days of life, the urine, which up to that time had been normal, became fetid, turbid; reaction alkaline. It contained albumen in large quantity; and of elementary forms—pus, epithelium of the various passages; various detritus; ammoniaco-magnesian phosphates. Post-

mortem gave extensive peritonitis, diphtheritis of the placental site, patches of diphtheritic membrane as large as a bean upon the mucous membrane; the diphtheritis of external genitals was retiring. In the parametrium, to left of the uterus, cloudy swelling of the connective tissue; the left ureter dilated to double its size; left kidney enlarged; after removal of its capsule, the cortical substance in some places yellow, in some gray, marked by reddened streaks and many superficial hemorrhagic infarctions, also numerous small patches of softening on the periphery, which on section were seen to penetrate like wedges. In the almost normal right kidney only a solitary group of puriform points; mucous membrane of the bladder strongly injected, ecchymosed in several places, and upon it patches of false membrane, which, when removed, showed loss of substance beneath.

Here we have, as a consequence of diphtheritis about the vulva and in the endometrium, the pelvic lymph spaces laid open to the entrance of septic fluid; lymphangitis of the left side causing occlusion of left ureter, which, in turn, produced œdema and rapid fatty metamorphosis of left kidney; the right side remaining exempt.

Case X.—The same observer also reports a very interesting case in which, after difficult labor from contracted pelvic outlet, perimetritis and peritonitis followed, which, by the tenth day post-partum, had set up a secondary nephritis; the causal phlegmasia then seemed to decline and pass away, but the nephritis continued, and in the fifth week post-partum, the patient died from uræmic coma.

Autopsy showed of the early changes in perimetrium and peritoneum, only a slight swelling in the *right* parametrium remaining; abscesses in both ovaries; both kidneys enlarged; cortical substance thickened and discolored, and in the *right* kidney upon surface and through the substance numerous little abscesses, from the size of a pin's head to that of a pea. This case is clearly to be interpreted as metastatic suppurative nephritis—the fatal out-put of a transient peritonitis and cellulitis.

A case from the Hospital Records, in which, unfortunately, no determination of the quantity and quality of the urine seems to have been made for several days after labor, appears nevertheless very fully to illustrate the same process of uræmia from occluded ureters.

Case XI.—D. G., 20 years, primipara; labor normal; thirteen hours; living child; sequelæ normal; forty-eight hours after, pulse 82, temperature, 101; twelve hours later, tenderness in hypogastrium and left iliac fossa; variable fever; pulse, 118. Temperature, 104¼.

4th day.—Same symptoms; weakness; deafness (in this case the first symptoms of uræmia). Details of urine lacking.

5th day.—Epigastric pain; later, general abdominal pain.

6th day.—Vomiting; weakness; epigastric pain.

7th day.—General abdominal pain; great weakness; excitement.

8th day.—Less pain; great weakness.

9th day.—Less pain; great weakness; bowels regular to date; urine not noted.

10th day.—Less pain; great weakness; urination scanty, albuminous.

11th day.—Became suddenly delirious; comatose; suppression; four ounces of densely albuminous urine in bladder; convulsive twitching.

12th day.—Coma. Death.

Autopsy.—Brain, lungs, heart, liver, spleen, nearly normal. Kidneys large; capsule loose; cortex thickened, swollen. Acute parenchymatous and tubular nephritis. Both pelves dilated. Both ureters dilated; right five times normal size. Both contain pus (macroscopic), probably tubular epithelium.

Localized peritonitis, agglutinating the fundus of the uterus, a few loops of intestine, the fundus of the bladder, the broad ligaments, and retro-peritoneal connective tissue, and muscular coat of bladder. The interior of bladder and uterus, the cervix and vagina nearly normal.

This patient clearly died of uræmia. By the great weakness, the disturbance of the senses, the constant nausea, delirium, convulsions and coma, the case is distinguished from simple puerperal peritonitis, and from puerperal septicæmia.

This uræmia was consecutive to a localized inflammation, involving fundus of uterus, bladder, etc., and by swelling occluding the ureters. Hence the kidneys became œdematous, and nephritis with suppression followed.

I am entirely of opinion that the converse relation also exists —that is, that the renal disease sometimes stands as the cause of the peritonitis, for I have a recollection of two young primiparous women, seen for the first time when in labor, both very anasarcous, both in convulsions (which continued with decreasing frequency for some days after labor), and both, when apparently convalescent from the uræmia, developing an unexpected peritonitis.

Compare the facts that peritonitis rather often arises in the course of Bright's disease, and the observation of Zalesky, before quoted, that after tying the ureters, the salts of urea accumulate on the surface of the peritoneum.

I have thus presented cases of simple erysipelatous, diphtheritic, lymphangitic, pyæmic, metastatic cystitis and nephritis.

It may be thought that from a wide search, only such as suited my present purpose have been collated. Such is not the case. I have carefully reviewed the history of obstetrics in Charity Hospital *for several years*, and endeavored to trace the autopsies of women who died within six weeks after delivery, *with an increasing conviction that the urinary organs in fatal puerperal cases are almost uniformly diseased, and in many cases much more significantly than the uterine organs.*

The autopsies of the Hospital are made by the curators appointed for that purpose, and the custom is to summarize the leading facts in the caption or heading of the record. Many of these records in recent years have been made by Dr. Maxwell, whose pathological attainments and discriminate modes of statement are highly appreciated by all who know him. From these records I have obtained a series of cases standing nearly consecutive in the autopsies of puerperal women. These headings of cases, with occasionally a word of explanation from the text, read—

Case I.—Pelvic abscess; acute endometritis; acute suppurative nephritis; thrombosis of uterine veins.

Case II.—Diphtheritic endometritis; dilated ureters and pelves; pyelitis; acute interstitial suppurative nephritis; cystitis.

Case III.—Puerperal septicæmia; advanced parenchymatous degeneration of kidneys.

Case IV.—Uræmia seventh month of pregnancy; parenchymatous degeneration of kidneys.

Case V.—Puerperal convulsions; parenchymatous nephritis.

Case VI.—Diphtheritic cystitis, endometritis; ureteritis and pyelitis; acute suppurative interstitial nephritis; pleuritis; and pleurogenous pneumonia.

Case VII.—Puerperal metritis; gangrene of cervix; kidneys enlarged, pale, fatty.

Case VIII.—Pelvic cellulitis and peritonitis; kidneys normal size, generally congested; pelves and ureters somewhat dilated.

Case IX.—Puerperal pyelo-nephritis; lobular pneumonia; kidneys slightly enlarged; capsule easily stripped over the surface of each; several swollen prominences dotted with yellow points; acute interstitial nephritis; ureters above pelvic brim distended to 2½ times normal size, and are the seat of intense

ureteritis. The tissues surrounding them are œdematous; uterus, ovaries and appendages normal.

CASE X.—Pyelo-nephritis, ureteritis and cystitis; dilated pelves and ureters; cysts of ovary.

CASE XI.—Diphtheritic endometritis; pelvic abscess; hemorrhagic cystitis and pyelitis; suppurative interstitial nephritis of left kidney; acute parenchymatous nephritis of right kidney; entero-colitis; emboli infarcti of spleen and left kidney.

CASE XII.—Puerperal peritonitis; double circumscribed pleuritis. This patient died in uræmic coma, convulsions continuing after delivery; suppression of urine; symptoms of peritonitis developing twenty-four hours before death; no abnormal appearances in any portion of the urinary organs recognized at autopsy.

CASE XIII.—Double pleuritis; double hypostatic pneumonia; perimetritis; localized diphtheritic puerperal endometritis; diphtheritic cystitis; dilated pelvis and ureter of right kidney.

CASE XIV.—Puerperal peritonitis; acute interstitial nephritis; diphtheritic cystitis, ureteritis and pyelitis; double pleuritis.

CASE XV.—Puerperal peritonitis; double pleuritis; parenchymatous degeneration of kidneys; pyelitis; ureters markedly dilated, that of the right side about seven times the normal size.

CASE XVI.—Puerperal endometritis; slight lobular pneumonia; suppurative interstitial nephritis.

CASE XVII.—Puerperal metritis; abscesses in uterine wall; peritonitis; cystitis; hemorrhagic pyelitis; localized acute interstitial nephritis; suppurative arthritis of knee-joint; pyæmia.

CASE XVIII.—Acute parenchymatous nephritis; cystitis; cervical endometritis, puerperal.

CASE XIX.—Localized pelvic peritonitis; pelvic abscess; parenchymatous nephritis.

CASE XX.—Thrombosis and embolism of left pulmonary artery.

CASE XXI.—Puerperal peritonitis; acute interstitial nephritis.

Of these twenty-one cases, *nineteen* showed distinct morbid process in the kidneys, *eight* showed distinct morbid process in the bladder, *eleven* showed distinct morbid process in the interior of uterus. In *nine*, who had distinct lesions of the kidneys, *no distinct* lesion of the uterus was recorded. *In all* who had distinct lesions of the uterus, distinct lesions of the

kidneys were also recorded. Wherever there was diphtheritic inflammation of the bladder or intense lymphangitis there was dilatation of the ureters. The average age of the patients was between twenty-three and twenty-four, and therefore the presumption of Bright's disease preceding pregnancy was small.

During my last term of service, which continued for six months, and comprised about 275 deliveries, I made a diagnosis of greater or less kidney disease in thirteen cases which terminated in recovery. The diagnosis was based upon pain in the lumbar region, increased by pressure, accompanied by pus, epithelium or casts in the urine. In most of these cases there was also a degree of cystitis, sometimes apparently consecutive to the nephritis, descending along the ureters to the bladder, and sometimes primary and followed by nephritis ascendens.

Finally, I venture to formulate the following conclusions:

1. Acute erysipelatous inflammation of the external genitals may ascend to the kidney, sometimes by the inner and sometimes by the outer surface of the urinary tract.

2. The blood of the parturient woman, saturated with fibrine and poor in hæmoglobin, predisposes her to disease of the excretory organs—the kidneys and liver. With a sufficient exciting cause acute fatty metamorphosis takes place. Fatal cases only are demonstrable, but minor grades of the process are probably not unfrequent.

3. Lymphangitis limited (cellulitis), and lymphangitis diffuse may mechanically induce acute œdema of the kidney in the puerperal woman by obstruction of the ureter.

Diphtheritic or other inflammation involving the muscular coat of the bladder produces the same result.

However excited, œdema of the kidney tends to rapid degeneration both of the tubular and inter-tubular structure.

4. Diffuse lymphangitis, commonly attending septic processes, by rapid destruction of the hæmoglobin, tends to the same result, while ulcerative endometritis, suppurative metrophlebitis and cellulitis, tending to pyæmia, not unfrequently are productive of metastatic suppurative nephritis.

5. The condition known as uræmia tends to develop peritonitis in parturient women.

Should the profession confirm and extend these observations,

important inferences as to prognosis, and as to the hygiene of the pregnant, and the management of the parturient woman, may follow from them.

68 W. 40TH STREET, FEB. 20TH, 1877.

[With the consent of the various parties concerned, we append the following remarks, made after the reading of the paper:

The President called upon DR. FORDYCE BARKER, who spoke as follows:

Mr. President and Gentlemen:—I have listened with interest to the paper read by Dr. Chamberlain, and regard it as one characterized by a most thorough and careful research, and also by a very close and extensive observation. I do not propose, in any sense of the word, to criticize the paper; that would be impossible, from merely listening to it, and indeed, I should not wish to, because it states in a full and clear manner most important facts, which should be borne in mind, and in most respects I should agree with the author in his pathological views.

There are, however, some deductions not mentioned in the paper, and it is to those that I shall chiefly allude. In the first place, I will briefly refer to the fact that all these various lesions of the kidneys and liver, to which especial attention has been drawn, are found associated mainly with puerperal diseases, when they exist in an epidemic or endemic form. The tendency of a certain class of observers and writers has been invariably to give prominence and significant importance to local lesions, to regard such manifestations as purely local diseases. For instance, as we all know, in former times, able writers have endeavored to explain puerperal fever by referring it to a purely local inflammation. Now, I am aware that I stand in a very small minority in the professional world, a minority much larger than it was a few years ago, who hold to the view that puerperal fever is an essential, constitutional disease, having its peculiar local manifestations in different types in different epidemics and in different localities; that it is a specific disease, properly speaking, without special characteristic, anatomical lesions. I am well aware that we find in epidemic and endemic puerperal fever, lesions of the kidneys and liver, varying in severity from a mere hyperæmia to extreme inflammation, with its terminal results. I simply wish to call attention, in connection with this point, to this fact, that these various lesions are generally associated with the epidemic and endemic forms of puerperal fever, as I consider it. It is not my intention, however, to discuss the subject of puerperal fever, but simply throw out these suggestions because I feel that the deductions might remain, that such special lesions of the liver and kidney are purely local forms of disease, and are to be studied as such.

I will add, by way of supplement to the paper, not in the light of a criticism, but to make our discussion broader, what my own clinical experience may contribute to the knowledge already obtained upon

the general subject, and I do this simply because it is well to take in the entire scope of the subject, involved in the announcement of the paper.

I will allude to one practical point suggested in the paper, and I mention it because I believe that nothing should be accepted as authority unless it can be demonstrated. The author of the paper referred to Olshausen and other German writers, who object to the use of the catheter for certain reasons which he mentioned. I refer to this because I believe that the catheter is not too frequently used in puerperal cases, but because my experience would lead me to the conclusion that the use of the catheter is too often neglected. I very rarely see cases in which the catheter has produced harm, but I frequently see cases in which its neglect has resulted most hazardously. Not long since I saw for the first time a case of secondary hemorrhage, caused by a distended bladder. The attending physician had been deceived by the statement of both the patient and the nurse that the urine had been frequently evacuated, and he regarded the tumor over the pubis, which was very distinct, to be the large uterus. But on palpating the abdomen, I found the tumor to be more smooth on its surface, more regularly ovoid in its form, and that it was characterized by a more fluctuating kind of elasticity than should belong to the uterus, and I therefore suspected it to be due to a distended bladder. The patient at this time was in a condition of collapse from hemorrhage, which occurred fifty-two hours after labor was completed. A catheter was introduced, and nearly two quarts of urine was drawn off.

The results which follow retention of the urine in puerperal cases, I have repeatedly seen, and one of these is an incomplete evacuation of the bladder, and the effects on the general system of residual urine.

I take this occasion to give a description, which I know to be clinically true, of a condition which I regard as practically important, but which I have been as yet unable to find mentioned in any obstetrical work. It is not unfrequently the case that a woman after her confinement suffers from retention of urine, or, at least, difficulty in evacuating the bladder. This may arise from long pressure upon the urethra, or some portion of the bladder, during the processes of parturition, or from long retention of urine and paralysis or paresis of the urethra or bladder. Subsequently the bladder gets relief to a great extent, but not completely.

Now there is an affection which I am sure results from this condition of affairs, and which has well-marked and characteristic phenomena that I will describe, and which I feel certain many who have been long in the practice of medicine have met. I am certain that the affection exists, and the process by which I reached this conclusion is something as follows:

The first case, in which the symptoms which I am to detail were developed, was that of a woman who had relaxation of the symphysis pubis to such an extent that she fell upon the carpet and was unable to walk afterwards. She was subsequently confined, and had a per-

fectly natural and quick labor. She was unable to walk for a long time after her confinement. For three or four weeks she did well, as regards her general condition, but there had been more or less trouble in passing water, and she began to develop symptoms which were like those seen in the case of a gentleman whom I was attending at the same time. He was paraplegic during a certain period in the progress of his case, which I now know to have been one of locomotor ataxy—a disease which had at that time not been described. He suffered from painful and frequent micturition and the following constitutional symptoms. There were great gastric irritability, nausea, vomiting, and loss of appetite, loaded tongue, general muscular weakness, and great mental depression. Subsequently the man had complete paralysis of the bladder and I was compelled to use the catheter. From the time I began its use there was a rapid subsidence of all these symptoms mentioned. His general condition improved, his appetite was restored, and his countenance looked quite healthy, and the frequent urgent desire to micturate subsided. About five or six weeks after my patient with relaxed symphysis pubis was confined, she began to manifest similar symptoms and suffered from an almost incessant desire to empty her bladder. Finally, a catheter was introduced, just after she had passed all the water she could, and nearly two ounces of exceedingly fœtid urine were withdrawn. That quantity had remained in the bladder after the most complete effort to empty it. The thought at once struck me that it was the decomposition of the urine remaining in the bladder that gave rise to the constitutional symptoms from which my patient was suffering. From that time forward I used the catheter three times every twenty-four hours, and with the most striking and rapid improvement. These cases occurred in my practice more than thirty years ago, and since that time I have seen a considerable number of like cases, although rarely developed, to such a high degree as the two just mentioned. They have, however, manifested the same general constitutional disturbance, and presented the same phenomena. I have often been called in consultation at the expiration of three or four weeks after delivery, and found the condition I have just described.

The patient has gone on very well for three or four weeks, sometimes longer, after her confinement, when it is noticed that she begins to lose her appetite, to complain of nausea, and sometimes vomit, there is increasing muscular weakness, a sunken, haggard countenance, a coated tongue, offensive breath, and great mental depression, accompanied by vesical tenesmus, and a frequent, most troublesome desire to evacuate the bladder. When I have suggested that these symptoms might be due to residual urine in the bladder, it has been objected that she is too frequently called upon to pass water.

Insisting, however, that there might be a residual amount of urine in the bladder, the patient has been directed to void all that she possibly could, and then the catheter has been introduced with the result of drawing off a certain quantity of exceedingly offensive water. The catheter is then used every six or eight hours, and the patients have

been relieved of the constant vesical irritation and the attendant symptoms. Other treatment has been employed, according to the requirements of each case, but no radical measures have been employed, except the use of the catheter.

(Several illustrative cases were related.)

In two clinical lectures, which I have given at Bellevue Hospital, I have ventured to denominate this affection, Puerperal Ammoniæmia.

In connection with the subject of the paper read, there is one other point to which I will call attention, and that is, the occurrence of peri-nephritis as a puerperal disease. An important monograph upon this affection has been written by Bowditch of Boston, and an excellent clinical lecture upon the same disease has been given by Trousseau, but I refer to it as a puerperal affection. It is not of frequent occurrence, but I have met with it several times in connection with epidemics of puerperal fever, and I have seen three sporadic cases.

I will not stop to describe the symptoms or discuss the pathology of this affection, but will briefly relate the three sporadic cases, to which I have alluded.

The first was a woman who entered Bellevue Hospital in my service three weeks after her confinement. As she gave her history, four days after the birth of her child, she was up and engaged in washing clothes, when she was suddenly obliged to cease her work on account of a most severe pain in her side. The next day she had complete retention of urine, and a doctor was called in who gave her medicine which cured this trouble in two or three days. After this she was able to do her house-work for a week or two, but she always suffered from pain in the side. The day before she entered the hospital, she again sent for the doctor, who told her husband that her case was bad and that she must go to the hospital. She had fever, chills of irregular recurrence, but not severe, constant pain over the left kidney, where there was great tenderness, and a circumscribed tumor in which I thought I could detect fluctuation. I called my obstetrical colleagues of the hospital in consultation, but we could not settle upon a diagnosis. Three days afterwards I passed an exploring needle into the tumor and found pus. I then made a free incision over the kidney and gave exit to about six ounces of pus. After its discharge a portion of the kidney could be easily seen. This patient made a good recovery.

The second case was a patient of the late Dr. Stillwell, whom I saw for the first time six weeks after her confinement. A few days afterwards, I evacuated a large amount of pus by an incision, and this lady eventually got well.

The third case was a patient of Dr. Purple, whom I saw, I think, about the fourth week after her confinement, and I then expressed the belief that a peri-nephritic abscess might be forming.

This lady had a peculiar temperament, she was very difficult to manage and she had a great horror of an operation. Some time afterwards, I again saw her with Dr. Purple and Dr. Buck. It was then

evident that the pus had worked its way down to the iliac fossa. Dr. Buck subsequently evacuated the pus by an operation, but I believe that she afterwards died from exhaustion.

DR. MARY PUTNAM-JACOBI remarked that in regard to the alleged anæmia of pregnancy, it was much to be regretted that no other data were appealed to than those established so long ago by Andral and Gavarret. She was not herself acquainted with any more recent investigations than theirs, but the present method of direct numeration of the blood corpuscles was so much more exact than the method of weighing the clot—adopted by Andral and Gavarret—that these early experiments decidedly needed control. Moreover, it seemed to her that there must be a certain illusion on this point, wherever, at least, the number of blood corpuscles for a given volume of blood was calculated; since an increase of plasmatic fluid, relative to the blood corpuscles, would occasion an apparent diminution in the latter, as effectively as if, the plasma remaining the same, the amount of corpuscles were really diminished, we should expect that the plasma would be increased, to meet the demand for plastic material made by the embryo; and the diminution of muscular activity on the part of the mother, must diminish *her* demand for the corpuscles whose oxygen would be required during muscular contractions. Hence again, even if the absolute amount of corpuscles *were* diminished, it should not imply a normal diminution below the real needs of the economy.

We have no assurance to what extent Andral and Gavarret's subjects, hospital patients, were in a normal condition. With regard to the fatty infiltration of the liver found in many of Dr. Chamberlain's cases, it is certain that this is found in a large number of pregnant women, where the pregnancy has been perfectly normal, and death has occurred, sometimes before parturition, from accidental causes, or intercurrent disease. Cornil—to mention no other pathologist—considered such fatty infiltration—to be distinguished of course from fatty *degeneration*—as a normal circumstance of pregnancy—and it is certain that, whenever the oxidation of fats is diminished, these became stored up, first in the adipose tissue, afterwards in the liver. The liver is the internal store-house of fat. Infiltration of the kidneys was less frequent, but even that might occur under the same circumstances. It is known to be normal in dogs.

It was evident from Dr. Chamberlain's histories, that in many cases no symptom of hepatic or renal lesion, no albuminuria existed before parturition, and he himself shows how nephritis has developed as a consequence of the local lesions of metro-peritonitis. In these cases, therefore, at least, of course the kidneys could not be held responsible for the predisposition to the puerperal accidents. Where nephritis did exist, however, during pregnancy, it seemed very plausible to suppose that interference with excretion of nitrogenous substances would favor septicæmic accidents in the same way that, according to Murchison, chronic renal disease extraordinarily aggravates the prognosis in continued fevers.

The very interesting experiments quoted by Dr. Chamberlain upon ligature of the ureters, and the excretion of urea by the peritoneum, have been already invoked to explain the dependence of general peritonitis upon acute nephritis. Bauer admits that the latter disease is sometimes the only cause of the former, and the speaker had recently seen a case where a woman was attacked by general peritonitis, three months after confinement, the health in the interval having been good, and whose urine, at the time, contained forty per cent. of albumen. After death, no uterine or other local disease was found to explain the origin of the peritonitis, but the kidneys showed all the signs of an acute parenchymatous nephritis.

Dr. Chamberlain remarked that these lesions of the urinary system in general were very largely the effects of the general processes of puerperal fever, and that it was not to represent them as exclusively local lesions that he brought them forward in his paper. It was rather to show that as we reckoned metritis, phlebitis, and cellulitis, so we must also consider nephritis and fatty metamorphosis of the kidneys as varieties of the morbid puerperium, more frequent and more grave than commonly represented.

In a certain number of cases the patient recovered entirely from the metritis, peritonitis, lymphangitis, cellulitis, etc., yet had more permanent renal lesions, as evidenced by careful examination of the urine.

Regarding the catheter, he wished to be understood as maintaining that it was not necessarily its use that was detrimental, but that the use of an impure instrument, or the blind and rude use of a clean one, might be a frequent cause of puerperal difficulties. The use of the catheter for the purpose of removing decomposing urine from the bladder, was not only justifiable, but imperative.

With reference to hydræmia, the points made were, that the woman was shown to be in that condition, because, by absolute measure, the water of the blood was increased, and by absolute count the red corpuscles were diminished; the albumen was diminished, and the fibrin and white corpuscles were increased. It was the appreciation of the total change, as indicated by actual measure, that gave rise to the condition.

With regard to the hæmoglobin, the doctor regarded it as the oxygen carrier, and as such, it was the agent of normal combustion, and that normal combustion was the natural method of consuming the residual excretory elements in the blood, and that in the absence or diminution of that element in the blood, there was an accumulation of carbonaceous matter, which required only a slight change to take the form of fatty degeneration, both in the blood and the tissues. The statements of Andral and Gavarret were old, it is true, but he did not understand Dr. Jacobi to say that they had been invalidated. If not, age argued authenticity.—Ed.]