

5-1-1932

## Low reserve kidney of pregnancy

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THE LOW RESERVE KIDNEY

OF

PREGNANCY

Senior Thesis

April 14, 1932

John T. McGreer

THE LOW RESERVE KIDNEY  
OF  
PREGNANCY.

There are two schools of thought regarding the reaction of the maternal organism to pregnancy; one considers it as physiological, the other pathological in which there is developed defensive mechanism against an invading organism, the developing ovum. Williamson (41) states that, "the distinction between the physiological and the pathological is ill-defined- there is a boundary country but no border-line." Nevertheless normal gestation is always accompanied by profound anatomic and metabolic alterations. (36,40)

The renal load, as well as that of the heart, is increased during pregnancy, and any impairment of efficiency becomes inadequate more easily during such a time. The carbohydrate, protein and fat metabolism is changed; the neuro-vascular system shows evidence of increased instability; and the entire endocrine system is altered. (36,37). A mild acidosis occurs in normal pregnancy, particularly during during the last weeks. (37,41). Anemia frequently occurs and occasionally may be severe. (37). All of these alterations adversely affect the physiological and chemical processes of the kidney during pregnancy and no doubt contribute indirectly to renal affections.

The term "low reserve kidney" may be insufficient and not exact, however, it characterizes a certain group of pregnancy toxemias probably as well as any other term thus far suggested. This so-called mild type of toxemia manifests

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itself in the second half of pregnancy, usually in the eighth or ninth month, and consists of a slight elevation of blood pressure, a slight amount of albumin in the urine and moderate swelling of the ankles. (27,36). Upon delivery of the patient the symptoms disappear entirely, and in a future pregnancy the condition may recur or may be absent. In such a condition it is impossible to demonstrate any signs, symptoms or laboratory findings suggesting nephritis. (36). Perhaps the "low reserve kidney" falls into the "boundary country" between the physiological and the pathological processes of pregnancy previously mentioned.

Because of its indefiniteness, particularly as to pathology, this condition was and still is to a slight extent undifferentiated. Leyden (29) was among the first to recognize the condition and the very first to designate it by a specific term, "the pregnancy kidney." In 1881, he reviewed the literature pertaining to the subject, adding his contribution to it. Lever in Guys Hospital Report of 1843 discussed edema with albumin in the urine during pregnancy. Frericks thought the condition was due to an alteration of the blood and to the mechanics of blood lakes in the abdomen, and not a chronic affair. Rosenstein had the idea that the venous stasis was linked up with the pregnancy kidney in the same manner as in passive congestion. He saw the weight of the pregnant uterus on the veins of the abdomen as the causative factor. Bartels believed that the mechanism of the stasis was a basis for the kidney affection. Hofmeier and Morike found that this condi-

tion was not always followed by eclampsia. Leyden stated that the affection occurred in the last half of pregnancy, most often in "first born", and is relieved immediately after birth, the pathology not being deep seated and healing readily after delivery. He associated the condition with puerperal fever. Leyden recognized that "pregnancy kidney" may simulate chronic nephritis and that a differential diagnosis is difficult, requiring the utmost care. In conclusion he states, "when a chronic nephritis stands latent and when the pregnancy case has a kidney affection with hydrops we do not make a diagnosis of a typical pregnancy kidney. Study the case which during pregnancy gives a history of nephritis and you will not put them in the typical case."

There has been comparatively little added to the statements and observations recorded by Leyden, and with but one exception, the relation of pregnancy kidney and puerperal fever, his work has been corroborated by further investigations. The "pregnancy kidney" was more or less forgotten, apparently, for there were no very extensive investigations carried out on the subject until recent years. (18).

The etiology of this intoxication of pregnancy as well as that of the others is still grossly obscure. The theories which have been advanced and the experimental procedures may be divided roughly into two groups. One group is based on the production of toxins due to alterations of the maternal metabolism, fetal elements and placental products; and the other is founded on mechanical changes. The mechanistic theory was the

first one to be advanced. Frerichs and Rosenstein believed that stasis in the abdominal and pelvic veins, "blood lakes", was the etiological factor. (29). Johnson (23) concluded that the mechanical interference caused by the gravid uterus hinders the normal respiratory function. As a result there is an imperfect combustion of nitrogenous materials and therefore the blood plasma is congested, the systolic force is increased, arterial tension is raised, osmosis is disturbed, nutrition is impaired, and functions suffer according to the disturbance of the organs to which they belong. Intra-abdominal pressure arising from hydramnios, twins and ovarian tumors crowding the ovum and producing a placental stasis or toxicosis has been ascribed as the cause. (3,5,8). Other observers suggest that the increased pressure resulting in abdominal stasis and hyperemia of the kidneys induces albuminuria and accompanying symptoms. (10,29,38).

A very thorough and illuminating investigation on the causation of albuminuria during pregnancy was quite recently undertaken by Theobald. (38). By producing lordosis in pregnant and non-pregnant dogs and later in men and women he was able to cause transient albuminuria with no accompanying symptoms. Also he was able to produce an albuminuria by lowering the respiratory rates. He suggests that the albuminuria accompanying pregnancy can be accounted for by mechanical means, namely, lordosis (the position assumed by pregnant women, particularly in the latter part of pregnancy in order to maintain their balance), the diminished thoracic capacity associ-

ated with the latter months of pregnancy, and the weight of the gravid uterus. He suggests that this type of albuminuria, in itself, has no relationship to the toxemias of pregnancy, except that disturbances of the metabolism and diet are factors in the causation of both. "Albuminuria is an important symptom, but it is only of serious concern when associated with a high or increasing blood pressure." (38).

The normal kidney possesses a considerable reserve, approximately 50 per cent, which may be called upon in emergencies. (36). Thus it seems reasonable to assume that certain individuals' kidney reserve may be greatly decreased by having congenitally small or congenitally defective kidneys. (36,20). Kellogg (24) expresses this as a faulty kidney balance and terms it "recurrent toxemia of pregnancy." In such a kidney there would be very little reserve left for an emergency. When pregnancy occurs the kidney reserve is too low to respond normally to the extra demand. As a result there is a passage of a certain amount of albumin through the glomerular epithelium and a moderate rise of blood pressure. (36,20). These manifestations usually disappear within a few weeks after delivery, the extraload having been removed and the kidney substance not being permanently damaged. Because of this deduction Goodall (20) believes that the term, "low reserve kidney", is confusing, and every kidney that is damaged has a reduced reserve. He suggests the term "congenital renal deficiencies", it being more descriptive of the true condition.

French observers, notably Chambrelent and Tarnier, found

that during pregnancy the blood serum was more toxic, while the urine was less so than normally. (30). With few exceptions, it is believed that there is an unknown toxin circulating within the maternal blood. (1). Mörliche noted albuminuria accompanying hydremia and chlorosis in pregnant women. (30). The fetus has been accused of introducing toxic substances into the maternal circulation. (8,27). Trantenroth demonstrated the disappearance of albuminuria upon the death of the fetus. (30). Kolischer (27) has observed that during the last half of pregnancy, the efficiency of the reticulo-endothelial system is impaired by the toxin, which weakens the body defense. The deportation of placental villi within the maternal circulation, a physiological process, which with the maternal erythrocytes form a cytotoxin, producing a toxemia, has been accorded the etiologic factor by Peterson. (32).

Not a few investigators are of the opinion that the "low reserve kidney" is a mild type of nephritis. Baer (3) thinks that the nephropathy occupies a place between nephritis and nephrosis and that the initial changes probably occur in the renal vascular system. It has been classed by Mussey and Norman (31) as a "nephrosis." Gibson (18) agrees with them that the "low reserve kidney" is a degenerative lesion of the kidney and not inflammatory.

Experimental and clinical studies have shown that a mild chronic nephritis may exist in an individual and yet give no clinical indications of its presence. However, upon the impregnation of this individual, the added strain of pregnancy



causes an exacerbation of this low grade chronic nephritis. (7,14) Browne and Dodds (7) believe that the "recurrent toxemia" of pregnancy is really a mild degree of chronic renal damage, and that perhaps the "low reserve kidney" may be in the same category. Gibberd (16) thinks that recurrent albuminuria of pregnancy is the result of a "subliminal" pregnancy toxemia acting in conjunction with a very mild chronic nephritis, the lesion being hidden except when the most delicate test of renal function is applied—the test of pregnancy. To this picture he gives the name "occult nephritis."

For a long time it has been stated that affections of the placenta may be associated with albuminuria and toxemia. (35). Young and Miller (42) believe that toxemia is produced by placental disease in the nature of infarction, ablation placenta, separation of the placenta, placenta previa and thrombosis, the underlying factor being an interference with the maternal blood supply. They found albuminuria in accidental hemorrhage in nineteen out of twenty-one cases. Also they report the presence of albuminuria in five of ten consecutive cases of placenta previa and there were other symptoms of toxemia in three of the remaining five. Danby (12) suggests that the extensive extravasation of blood into the pelvic tissues may be a manifestation of toxemia and that this effusion is due to changes within the vessel walls, dependent upon a toxic substance.

Stieglitz (37) states that the term "low reserve kidney" as used is grossly perverted, for an actual reduced renal reserve resulting from previous renal disease manifests itself

far earlier in pregnancy and is associated with persistent and often excessive hypertension which is aggravated by the intoxication and the increased burden of pregnancy. He believes that the tissues of the whole body are involved in this intoxication and designates it the "syndrome of tissue and renal fatigue in pregnancy." He suggests the possibility that these changes are protective mechanisms. He notes that "we" think of edema with nephritis rather than nephritis causing edema. Also that albuminuria may be an active protective mechanism as glycosuria in diabetes mellitus. This group, he believes, represents approximately forty per cent of the total number of the nephritides in pregnancy.

Though there be no abrupt line between physiological and pathological pregnancy, as alluded to before, it will be advantageous to review rather briefly the changes noted in normal pregnancy, which are relevant to the subject. This will enable a more acute recognition of the toxemia designated as the "low reserve kidney" of pregnancy. In a normal pregnancy there is a low total of non-protein nitrogen low urea nitrogen and a very low ratio of urea nitrogen to the total protein nitrogen in the blood. (9,36). Normal obstetrical patients eliminate phenolsulphonephthalein more slowly than normal non-pregnant individuals. (19). It is generally accepted that albuminuria is not present in the normal pregnant woman, although some believe that it may be found in such individuals living in unhygienic surroundings. (30,37). The basal metabolic rate is increased more than thirty per cent above the normal, parti-

cularly during the latter part of pregnancy. (2,35). A slight elevation of blood pressure is found in a small percentage of all normal pregnancies. (36). Donaldson (13) concludes that during normal pregnancy there is no increase in blood pressures. Schulze (33) states that the normal range of the systolic blood pressure is between 100 and 130, with an average of 114 to 118 (pressure in mm. of Hg.). Irving (22), in 5000 consecutive cases, found that in four-fifths of them the systolic blood pressure ranged between 100 mm. and 130 mm.

Formerly a considerable proportion of the patients with "low reserve kidney" were grouped as pre-eclamptic, and a smaller proportion as nephritic. Now that the "low reserve kidney" is recognized it constitutes the largest percentage of pregnancy toxemias. Stieglitz (37) believes that this nephropathy accounts for forty per cent of the total number of nephritides in pregnancy. In 293 repeat cases, Kellogg (25) diagnosed 26 per cent of them as "low reserve kidney". A review of the histories of all toxemic patients entered in the Johns Hopkins Hospital since 1897 indicates that about one-third of them suffered from this mild type of toxemia. (36). Gibbered (16) declares that all normal pregnant women have a slight toxemia. Stander (36) claims that the number of pregnancies has nothing to do with the development of this entity. Primiparae compose from 5 to 6 per cent of the patients according to Allbutt. (1). This nephropathy of pregnancy is thought by some to be essentially a disease of primiparae. (2,5,21). The highest percentage of toxemia occurs in the British, North

American negroes and Irish nationalities, while it is uncommon among the Russians (practically all Jews) and the Slavs (Australians and Poles). (22). Albuminuria and toxemia occurs most frequently in gravida under twenty years. (22). In a series of twenty-one cases Stieglitz (37) determined that the average age was twenty-six and four-tenths, the average number of pregnancies one and eight-tenths, the maximum number of pregnancies five, and the number of primipara eleven or 52 per cent. However, it would seem that such a series of cases would be far too small to reach any accurate deductions. Stander (36) believes that "low reserve kidney" is present in about 5 per cent of all full term pregnancies.

The "low reserve kidney" manifests itself in the latter half of pregnancy, usually during the eighth or ninth month. (3,22,28,36,37,40). Perhaps the only symptoms which may be noted by the patient is a moderate and, usually, a slight amount of edema of the ankles, legs, hands, or arms, or it may be present in all four members. (3,10,25,28,36,37). There may be some edema of the lower abdomen. (28,3). Rarely a slight headache is present. (28,36). Because of these very mild symptoms, unless a patient is under a doctor's care and receiving periodic examinations, the condition goes unrecognized or, as Gibbered and some others believe, a more serious toxemia develops.

During one of the periodic examinations, usually in the eighth or ninth month, the attending physician may discover the patient has a slight rise in the systolic blood pressure,

above 135 mm., or a small quantity of albumin in the urine, or both-edema as described may or may not be present. Such findings are characteristic of the "low reserve kidney." According to Young and Miller (42) albuminuria is the earliest definite sign of toxemia. Crook (11) disagrees for, in his experience, the amount of albuminuria has been of little or no value, either for the prognosis or the diagnosis of the toxemia. The blood pressure is considered the best criteria on which to establish a diagnosis and to determine an index of a toxemia. (2,21,22,34,36,37). Never-the-less, both are of great importance. In most instances the blood pressure elevation is not very marked, rarely exceeding 150 mm. systolic and 90 mm. diastolic. (36). Schulze (33) regards a systolic pressure of 150 mm. or over as a definite sign of toxemia. A systolic pressure of 140 mm. or over is taken by Harris (21) and Kellogg (25) as a fair index of toxemia. In a series of twenty-one patients Stieglitz (37) found that the blood pressures averaged 153/97. The quantity of albumin in the urine is never very large, varying before delivery between a fraction of a gram and two grams, although the lower figures are the most prevalent. (28,36). A few of the investigators claim that there is a slight failure on the part of the kidney to concentrate the urine, a tendency to retain sodium chloride and a moderate amount of retention. (3,28,37). A few writers, notably Kosmak (28), report that occasional hyaline and granular casts and red blood cells are found on microscopical examination, perhaps the reason being that they consider this

toxemia a nephrosis or a so-called "occult nephritis." Cook (10) writes, "in the average case of albuminuria or pregnancy there is remarkably little clinical or biochemical deviation from the physiological standard with the exception of edema." "The blood chemistry as well as the urinary analysis reveals nothing abnormal," is the conclusion drawn by Stander. (36). Baer (3) claims that he is able to distinguish this nephropathy of pregnancy from the others by capillary microscopy. He says that the capillary changes are transient and are characterized by slight deformities of the capillary loops as bulging at the convexity, hair-pin shapes and elongation. Basal metabolic determinations have not proved of any real help in differentiating between the various toxemias in late pregnancy. (35).

Several observers believe that the "low reserve kidney" cannot be definitely diagnosed until a few weeks post partum, or is diagnosed with considerable difficulty. Goodall (20) states that the condition is difficult to diagnose and is arrived at only after a process of exclusion. In no case did Harris think it was possible to make a conclusive diagnosis until three weeks after delivery. (21). Gibson (18) finds it difficult to distinguish degenerative changes in the kidney from inflammatory, as does Stander. (36). "A sharp line of distinction between pregnancy kidney and chronic nephritis does not exist, there is every grade between them." (14). Occasionally there are patients having an elevated blood pressure without any other signs of toxemia. If the patient had

an elevated blood pressure without demonstrable disease before she became pregnant, she is probably suffering from essential hypertension. (36). Stander (36) notes that Pinard and Varnier believe albuminuria is sometimes blamed on the state of pregnancy when syphilis is really the cause. A patient may have albuminuria in the urine without any rise in blood pressure or other evidence of toxemia, and on careful examination a pyelitis or, more rarely, a nephrosis of pregnancy is diagnosed. (21).

It is the opinion of the majority of observers that the prognosis in "low reserve kidney" is excellent. In this nephropathy the elevation of blood pressure and the albuminuria usually disappear completely during the two weeks after delivery, but there is a small fraction of cases in which four to six weeks pass before the blood pressure becomes normal and the urine albumin free. (3,9,36,37). Most characteristic of the group is that in subsequent pregnancies, the patient's condition does not become aggravated, and she is as well as or better than she was in the preceding pregnancy. (4,9,36,37). Of the twenty-one patients whom Stieglitz (37) was able to follow for a considerable period of time, 94 per cent were in good condition, 6 per cent in fair health, and no known instances of poor or bad health were observed. Patients in this group deliver at or very close to full term. (36,37). The twenty-one patients Stieglitz followed delivered on an average of eight and six-tenths months of gestation. (42). If the albuminuria is reduced to a faint trace in the course of one week

there is no permanent kidney defect, and the arterial tension drops more promptly in these cases than in those in which the kidney is primarily defective. (34). Banister states that the "low reserve kidney" does not tend to recur. (4). He reports the finding of case histories of albuminuria in succeeding pregnancies up to the seventh pregnancy, and in the eighth and ninth pregnancies the urinary examination is recorded as being normal. Allbutt (1) and Baer (3) make the dogmatic statement that immunity is conferred in most of these nephropathies, while Slemons (34) says that autointoxication does not recur in 80 per cent of the cases. Kellogg (24), who terms this nephropathy the "recurrent toxemia of pregnancy", subdivides it into two groups: (1) in which, under the strictest possible prenatal care, the prognosis for both mother and child is good and, (2) in which the prognosis for the child is bad, no matter what the prenatal care may be. Following the subsequent histories of twenty-seven women with toxemia of pregnancy, Von Gelden (39) found that thirteen of them had one or more normal pregnancies following the toxemia; while fourteen had a recurrence. He finds that the onset in subsequent pregnancies usually occurs early. Also he believes that with ordinary clinical means it is difficult and often impossible to determine which patient can be safely carried through her pregnancy without endangering her life.

Gibbered (14) states that chronic nephritis may be a sequel to "low reserve kidney." He has found that about 40 per cent of this group recover their kidney function after delivery and



have normal pregnancies subsequently, while 50 per cent are free from signs only in intervals between pregnancies. (15). Further, he thinks that women who were previously healthy and who have albuminuria during a pregnancy run a risk of eclampsia, patent chronic nephritis, 10 percent; occult nephritis, 50 per cent; death of the foetus in utero, 9 per cent; and death of the infant due to prematurity, 3 per cent. (17).

Several investigators believe that the most delicate method for determining kidney damage following such a nephropathy is another pregnancy. (6,7,17).

The treatment, employed by the various writers in "low reserve kidney", is, with few exceptions, fundamentally the same. Immediately upon the diagnosis of "low reserve kidney" the patient should be put at rest in bed with a low protein diet. Edema of the legs and ankles will occasionally disappear more rapidly upon the restriction of salt from the diet. Saline cathartics and diuretics are often given. Improvement generally follows this type of treatment.

Rest, mentally as well as physically, is very important. (17,20,22,28,36). Rest is of value in that it reduces the patient's endogenous protein metabolism to a minimum, thereby reducing the work of the kidneys. (17,20). The patient should remain in bed until she is free from all signs and symptoms. Gibbered (17) states that in spite of active treatment it is uncommon for the signs and symptoms to entirely clear up before delivery.

A diet which entails the least amount of work for the

kidneys is very desirable. (17,20,22,28). The diet should contain no more protein than is necessary to replace the results of katabolism. (17,20). The basal caloric requirements of the body must be supplied, preferably in a form of food which will not add to the load of the kidney. (17,20). Gibbered (17) chooses sugar in the form of glucose-common liquid glucose containing about 60 per cent of carbohydrate. He states that it avoids completely the onset of ketosis and, being less sweet than cane sugar, it may be given by mouth. Gibbered has four diets which he gives the patients, depending on their condition. When first admitted to the hospital he places them on diet number one for the first twenty-four hours, in order to obtain an estimate of the blood pressure and albumin content of the urine under standard conditions. The subsequent diet depends upon the severity of the signs and symptoms which the patients present. Kosmak (28) places the patient on a diet of milk, cereals, toast and as much water as possible. He prescribes what he calls the "imperial drink": cream of tartar one teaspoonful, juice of half a lemon, and water one pint with sugar to flavor. It is slightly diuretic.

The body elimination is of importance for an attempt at ridding it of toxins. It is desirable to secure one or two loose stools daily, preferably with a saline cathartic as magnesium sulphate. (17,20,22,28). It is usually given in one or two dram doses once a day. Stieglitz (37) believes the best diuretic is water and, if necessary, acid salts such as calcium chloride, calcium nitrate or ammonium chloride may be

administered. The skin should be kept functioning with daily warm baths. (28). There are no drugs of particular value except alkalies and their indiscriminate administration is to be deplored. (37). Edema is not necessarily renal in origin, but a protective mechanism in intoxicated tissues and deprivation of water is illogical therapy. (37). Baer (3) believes that pursuit of treatment depends upon the relative predominance of nephritic or nephrotic symptoms, restricting fluids in the former and forcing them in the latter. It is suggested by Kolischer (27) that the efficiency of the reticulo-endothelial system be increased with aseptic protein shocks by injecting 8 to 10 cubic centimeters of the patient's own blood into the gluteal muscle. The administration of 10 grains of bismuth subnitrate three times a day orally has proven to be useful in hypertension. (36,37). However, under this therapy, the blood pressure becomes inaccurate as a guide to the course of the intoxication. (37).

If, in spite of the treatment as outlined, the condition of the patient remains stationary for a period of from two to three weeks, or it becomes worse, termination of the pregnancy may be necessary. (17,22,36,). If such be the case the patient is probably suffering from a chronic nephritis. (17,36). Gibbered (17) claims that the longer the first toxemia is allowed to continue the greater danger there is of recurrence in subsequent pregnancies. He observed a 36 per cent recurrence in those patients whose first toxemia lasted less than three weeks, and a recurrence of 76 per cent in those patients whose

pregnancies were permitted to continue over three weeks. No set rule can be made governing the termination of pregnancy in this condition, for it depends upon the individual patient and the discretion of the attendant. (17).

To summarize, "low reserve kidney" is manifest in approximately 5 per cent of all full term pregnancies. It is the mildest type of toxemia, but there is some doubt as to whether it is, in itself, an entity. Observers disagree as to whether there is any injury to the kidney, but the majority believe that if there be any, it is not permanent. This toxemia may or may not recur in subsequent pregnancies. If recurrence should occur it is usually not more severe. The diagnosis is made on a small amount of albumin in the urine, ranging from a fraction of a gram to one gram per liter of urine, and a moderate rise of blood pressure, usually about 150 mm. systolic and 90 mm. diastolic. There may also be some edema, and very rarely a complaint of headache. These signs and symptoms of toxemia appear during the last few months of pregnancy. Of much importance in arriving at a diagnosis is the fact that by three weeks post partum the blood pressure has resumed the normal level, the urine is albumin free, and any edema which may have been present has disappeared. There is no abnormality in the blood constituents at any time, and the nitrogen ratio in the urine is normal. In subsequent pregnancies the patient may present a similar picture or be entirely free of any signs. The etiology of this mild toxemia, as in all of the toxemias of pregnancy, is not definitely known.

The treatment consists of rest in bed, the reduction of protein and the quantity of food just sufficient to supply the body demands, and a careful maintenance of normal body elimination. This course of treatment usually brings about improvement, and interruption of pregnancy is not necessary. Should the condition become worse, in spite of the measures just mentioned, it is probably a chronic nephritis and not a "low reserve kidney", in which case more radical treatment may be necessary.

"Low reserve kidney is probably identical with 'kidney of pregnancy', with certain types of 'recurrent pregnancy toxemia', with the simple 'albuminuria of pregnancy' as well as with certain of the 'nephroses' of pregnancy." (36). By not a few, the term "low reserve kidney" is thought to be inadequate and inaccurate. However, since so little is known about this condition, except that the kidneys are slightly below par and are accompanied by the characteristic signs, it seems incorrect to affix a more definite title. It is illogical and misleading to designate any one type of toxemia as "albuminuric." The term "albuminuria" is not a disease entity and should be used only to denote a laboratory finding. Gibbered (17) states that "functional albuminuria of pregnancy, or 'simple albuminuria of pregnancy' are misleading terms, implying that the condition is without danger to the mother or foetus—an implication that is entirely untrue.....'Simple albuminuria' cannot be justified on pathological grounds, and since it is dangerously misleading on clinical grounds it

should be abolished." The objection to the title "recurrent toxemia" is that it does not tell whether the process is benign or becomes progressively worse in subsequent pregnancies.

Stander (36) says, "it is a matter of great importance for the physician to know whether the patient is suffering from a mild and benign toxemia or from a kidney condition which, if treated inadequately, may prove fatal in the near future. Furthermore, even eclampsia may be recurrent."

Excluding the nephritics, it does not seem unreasonable to consider the "low reserve kidney" as the first phase or stage of the group known as the "toxemias of pregnancy." Also that the stage to which the toxemia develops is dependent upon the intensity of the toxin and the susceptibility of the individual.

In a warning to the physician, Kellogg (25) writes the following, "While we admit the academic existence of the 'low reserve kidney' group, we feel strongly that it should not be preached for public consumption until more facile methods are put forth for its ready and certain identification."

## CASE HISTORIES.

## Case I.

White; American; Female; Age 32; Gravida iii. Her past pregnancies were essentially normal. She had some edema of the ankles, and slight nausea for one and one-half months before delivery. She entered the hospital on 12-17-31. At that time her blood pressure was 130/80. Her Wassermann was negative. The only treatment was magnesium sulphate once a day. She had an uneventful delivery at term of a normal infant on 12-23-31. Her urine and blood findings were negative. She was dismissed perfectly well on 1-7-32. She was examined one month later in the dispensary and was found to be normal.

Diagnosis: Low reserve kidney.

## Case II.

White; American; Female; Age 36; Gravida vi. All of her pregnancies but the last one were normal. The last pregnancy was interrupted at four and one-half months because of bleeding. A transfusion was necessary. She was in the hospital for seven days during the eighth month because of edema of her ankles, legs, hands and arms and occasional slight vomiting. Wassermann-no report. The blood pressure varied from 110/78 to 124/82. The blood count was normal. Blood chemistry: NPN, 22.3 mg.%; CO<sub>2</sub> combining power, 34.3 Vol.%.  
 The urine contained a slight trace of albumin continuously, and there were occasional white blood cells and epithelial cells on examination. On two examinations the specific gravity was 1.014 and 1.003.

Water tolerance test:

<u>Time</u>	<u>Quantity Urine Passed</u>	<u>Specific Gravity</u>
7:30	180 cc.	1.004
8:30	190 cc.	1.006
9:30	115 cc.	1.006
10:30	130 cc.	1.005
11:30	44 cc.	Difference 1.008
Total	659 cc.	of 0.004.

The patient was put on a low salt and protein diet. The elimination was maintained with magnesium sulphate.

The patient reentered the hospital on 11-3-32 with no edema or complaints. The blood pressure was 135/95 and 135/85 on two occasions. The blood findings were essentially negative. The urine contained albumin, 14, and had a considerable number of white blood cells and occasional epithelial cells. The



Case II.

patient had a normal delivery at full term. No further laboratory examinations were made and the patient was dismissed on 11-15-31. No follow up examination.

Diagnosis: Low reserve kidney.

## Case III.

White; American; Female; Age 34; Gravida ii. In the patient's first pregnancy her feet swelled at night for the last two months. The baby was small. No history of nephritis. Entered the hospital on 11-13-31 with large varicosities of both legs and thighs, and a bluish coloration of right leg. No edema. Wassermann negative. There was slight arteriosclerosis of the retinal vessels. The blood pressure was 155/90, and gradually rose to 150/110 up to the time of delivery. The blood findings were normal. The urine showed a trace of albumin, 6-8 white blood cells, 3-12 epithelial cells and occasional hyaline and granular casts per high power field, up to the time of delivery. The patient felt dizzy and nauseated and labor was induced. The patient had a normal full term delivery on the following day, 11-25-31. The blood pressure was 146/100 the next day. No laboratory work was reported following the delivery. The patient was on a low protein diet up to the time of delivery. Dismissed 12-24-31. The patient has not been examined since.

Diagnosis: Low reserve kidney.

Note: It is doubtful if this case was a true "low reserve kidney." The hypertension plus the white blood cells, epithelial cells and hyaline and granular casts is very suggestive of a nephritis.

## Case IV.

White; American; Female; Age 20; Gravida i. Entered the hospital 1-1-32. The patient had slight swelling of feet, ankles and hands for the last two months. Wassermann negative. The blood pressure ranged from 135/90 to 116/64. The urine contained albumin, 4  $\mu$ , on two occasions, and varied between 1  $\mu$  and 2  $\mu$  the rest of the time. Red blood cells varied from 4-5, white blood cells from 4-6, and occasional epithelial cells per high power field. The water diuretic test only showed a small lowering of the water output. The blood findings were negative. Eye grounds showed diminishing of size and paling of the arterioles. These findings were thought to be due to ischemia of glomeruli. Delivery induced by rupture of membranes on 1-7-32. On dismissal the eye grounds were normal, blood pressure 116/64, albumin negative with 4 red blood cells, 4 white blood cells and a few epithelial cells per high power field in the urine. No edema present. Dismissed 1-18-32. There was no follow-up examination.

Diagnosis: Toxemia of pregnancy.

## Case V.

White; American; Female; Age 28; Gravida i. Entered the hospital on 6-27-31 with slight edema of the lower extremities and some abdominal discomfort which had been present for the past three months. The blood pressure ranged from 110/70 to 160/100. Albuminuria ranged from 3  $\mu$  to 1  $\mu$ . White blood cells and epithelial cells were present in the urine from many to a few. The water diuretic test showed considerable inability to concentrate the urine. The phenolsulphonephthalein test showed moderate retention of the dye. There was quite severe secondary anemia. The blood chemistry was essentially normal except for an NPN of 15 mg% on one occasion. The patient was on a low protein and low salt diet. The patient had a normal full term delivery on 7-5-31. At dismissal, 7-19-31, her condition was much improved, and she was free of all toxic signs and symptoms. No follow up records were available.

Diagnosis: Low reserve kidney.

## Case VI.

White; American; Female; Age 18; Gravida i. Entered the hospital and delivered on 1-14-32. There had been swelling of the feet and hands for the past week. The blood pressure was 152/96. There was albumin, 24, and 6 red blood cells, 8 white blood cells and a few epithelial cells per high power field in the urine. The blood examination and chemistry was negative. On 1-15-32 there was 3 grams of albumin per liter of urine. On 1-22-32 there was a trace of albumin in the urine and the blood pressure was 144/80. No further laboratory work was done and the patient was dismissed on 1-24-32. There was no edema present on dismissal. An examination two months later found the patient to be normal.

Diagnosis: Low reserve kidney.

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