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Hypertension; with special regard to pregnancy

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Clarence J. Strand

Senior 1932

HYPERTENSION--With Special Regard to Pregnancy

Hypertension - a combination of words meaning that the fluids of the body are under an increased pressure, that the vessel walls have an increased strain to bear, and that the heart muscle has an increased load to carry. The potential of the blood is increased.

We have after a great many experiments with the accepted instrument, the Sphygmomanometer, established a normal blood pressure, and also realized that there is a physiological increase in blood tension under certain environments or conditions of the organism. For example; exercise, fear, anguish, anger, etc. This increased tension of the blood stream is only temporary and returns to the individual normal, with the restitution of the organism's environment. This is an accepted mechanism which is controlled by a hormonal-nervous balance primarily and amounts to an increased out-put from the

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heart. This action as indicated, is only temporary and rapid, responding in a few minutes or even seconds.

This increased potential, whatever it's cause, physiological or pathological, must be spent on the tissues of the vascular system. This action results in a more rapid flow of blood, or a normal flow through a reduced lumen. These vascular tissues lend all of the resistance to the flow of blood. The capillaries, void of connective tissue or muscular tissue, could not be subjected to this increased tension without the loss of plasma into the tissues, "water-logging", and enhancing the activity of these tissues. Therefore, all of this energy must be expended upon the arterioles, "the sentinels of the capillary beds." To a certain point this tension will cause increased flow and oxygenation. In physiological increased tension, the arterioles seem to guard that upper limit of efficiency at their own expense. That is, the strain and energy absorbed from the blood flow over a number of years

will increase the fibrous content of these vessels, gradually decreasing their lumen and raising the periferal resistance.

The above is an accepted mechanism in the production of arteriosclerosis - a secondary thing to blood pressure. We may look at normal blood pressure as a hypertension, which must be overcome by these small vessels at the expense of their elasticity. This process is a very slow one and over a period of years will act to increase the periferal resistance, which will in turn increase the blood tension, forming a vicious cycle - arteriosclerosis resulting.

There are many factors effecting this action and because of the variability of these factors, we have a variability in the age of the individual manifesting this condition. This is mostly found in mid life. The atherosclerosis of the larger vessels coming on in later life - a culmination of this process beginning early in the small arterioles.

Associated with this process of increased pressure in the arteriole system is increased tonicity of the

ventricle. Herschfelder believes that in the intact animal, any hypertension is associated with increased ventricular tone, with no dilation resulting. This could be readily understood up to a certain point and that point may be in the range of physiological tension variation, but cannot be depended on in pathological exceptional rates. This tonicity is perhaps in addition governed by the condition of the myocardium. In any case, there is increased tonicity of the ventricle. The acute raises are more especially associated with increased tone, which must not be depended on over too long a time. Time is an important factor in the reaction of the individual case.

Pathological hypertension is usually associated with heart and vessel changes of the above nature and exist over a long period of time. No rapid change can be made in the formation or the amelioration. This type is called anatomical or structural hypertension and more or less fixed.

Contrasted with this more or less fixed type,

is the type prone to fluxuation and abrupt changes. The hypertension is characteristic of acute Nephritis, Eclampsia, and Uremia. The tension may raise many millimeters of mercury in a space of a few hours, or even minutes. This, with the fact that the pressure subsides as rapidly with the improvement of the disease, would throw these "hypertensions" into a class due to functional disturbance. These then seem to be the result of a general constriction of the arterioles, whether nervous or toxic in nature.

This paper deals principally with the last type, the "Aggravated" physiological type, so to speak, in which type there is much fluxuation and rapid deviation, as will later be brought out. There is no sharp line of demarkation in these conditions and one will always lead to the other. We must remember that this anatomical type does not occur in a short time, but over a period of years. All of these acute raises, whether physiological or pathological, no matter what their extent or duration, will just to that degree, further the inevitable

anatomical disturbance. With this in mind, we will be less apt to feel that a patient has gone through a toxic state or eclampsia with no damage to her vascular system.

High blood pressure is the most constant sign of a pre-eclamptic state. It must be considered however in light of the individual normal, also the time required for the raise, and not disregarding the diastolic rate.

Hillis reports some cases showing convulsions with a systole of 130 mm., showing all the other signs of an advancing toxemia. Also that the pressure curve showing a rapid rise of 10 or 15 mm. to 140 mm. is much more severe and significant than is the pressure of 150 mm., which has gradually ascended to this point over a long period of time. For this reason, blood pressure readings should start as early in pregnancy as is possible, to establish the individual normal.

Bunzel reports that 6.3 % of all pregnancies are associated with toxemia and .7 % have con-

vulsions. This is no small number of cases. These are classified in regard to their blood pressures as follows:

Mild Toxemia -

Blood pressure below 145 mm. systolic; slight amount of oedema. The albumen of the urine does not exceed 10 % in a 24 hour specimen.

Moderate Toxemia -

Blood pressure 145 - 165 mm. systolic; an albuminuria of 10 - 20 %; pitting oedema.

Severe Toxemia -

Blood pressure above 165 mm. systolic; albumen over 20 %; Oedema massive; with convulsions.

This classification, while simple and useful, does not carry with it a clear picture of the condition, so superimposing the classification of Stouder and Peckham we have:

Low reserve kidney	- - -	A mild Toxemia
Chronic Nephritis	- - -	A moderate Toxemia
Pre-eclampsia	- - -	A severe Toxemia

Eclampsia associated
or not associated with
chronic nephritis - - - A severe Toxemia

While there can be no hard and fast boundaries
set up, this classification will be of great help
in a more or less gross differentiation.

Other symptoms noted will be:

Pre-eclampsia -

The patient is acutely ill;
A very high blood pressure;
Albuminuria;
Amaurosis and epigastric pain;
The blood picture is the same
as eclampsia and is essentially
eclampsia before the convulsions
set in.

The low reserve kidney -

A congenital lack or a patho-
logical loss of glomeruli;
Characterized by a blood pressure
under 150/90 and mild toxemia
symptoms.

Eclampsia superimposed upon Chronic Nephritis -

This is a picture of severe toxemia;
Not essentially different from
eclampsia in management, however,
there is not the rapid drop in the
blood pressure to normal following
parturition; also a retention of
the Non Protein Nitrogen content of
the blood.

The above toxemias are always manifested in the last trimester. We have a toxemia of the first trimester which seems to have no connection with the toxemias of the last trimester. This is Pernicious Vomiting - a truly toxic state and according to Plass oftentimes show a blood pressure raise from 130 - 140 systolic, and may be associated with a polyneuritis.

Another point here in our differentiation is brought out by Draper. The hypertensive toxemias are larger, heavier, and more muscular than the nephritis toxemias. This type is the same as that manifested by Acromegaly.

With these few points in mind, I will present a number of cases with blood pressure charts. These cases will help to correlate and strongly emphasize the symptom of hypertension in pregnancy.

Case No. 1 - Toxemia of Pregnancy - Mrs. H; Primigravida - enters the hospital Sept. 11, 1930. Symptoms: epigastric pain; blood pressure 150/115; amaurosis; oedema of the ankles; in a stuporous condition; decidedly toxic.

The condition was rapid and continued so in the face of treatment for four days. There was then a rapid fall in the blood pressure with amelioration of symptoms. This stayed down only three days and then started rapidly to rise.

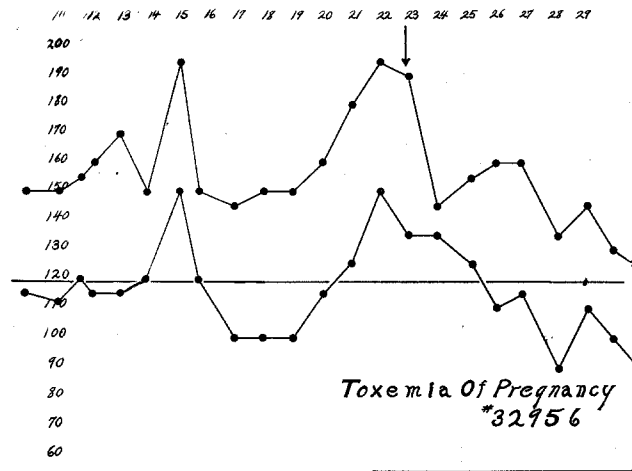


Chart #1

This time it was thought better to interrupt the pregnancy, which was in the seventh month of gestation. Labor was induced with a Vorrhees bag at 10:30 A. M. A four pound baby was spontaneously delivered at 10:00 P. M. Baby lived.

The intensity of the symptoms corresponded exactly with the blood pressure chart. A return to normal in post-partum period..

Treatment used was modified Stroganoff.

Discharged Sept. 29, 1930 in good condition.

Case No. 2 - Toxemia of Pregnancy - Mrs. - enters the hospital on Nov. 20, 1930; Primigravida. Symptoms: Blood pressure 170/120; headache; oedema; albuminuria; eight months gestation.

The course was strong for fifteen days - then rapidly amelioration as the chart indicates - running a normal course through delivery and post-partum.

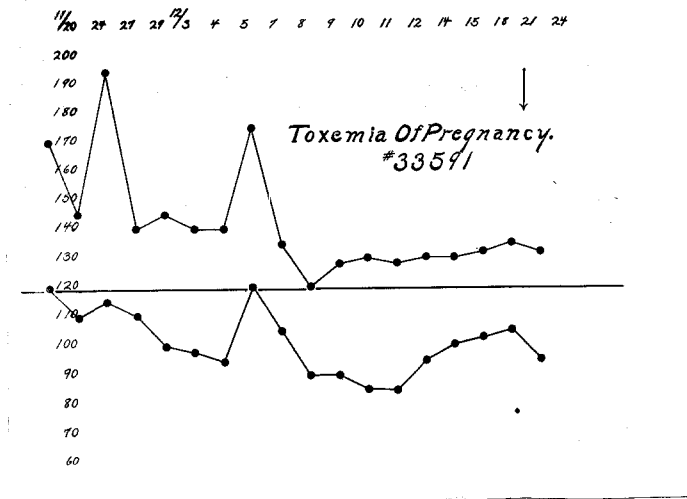


Chart #2

These two cases are very good examples of pre-eclampsia. The symptoms are the same as those of eclampsia, only there are no convulsions.

Case No. 1 would have certainly gone to eclampsia if the gestation had not been terminated. This case did not respond to the Stroganoff treatment, making induction necessary. The blood

pressure rapidly fell to normal with apparently no stigmata.

Case No. 2 is a very comparable case with the exception that it is a month later in the gestation period.

The systole in both cases reached nearly 200 mm., placing them in the classification of severe toxemias. They were both on the verge of eclampsia. They appeared as if they would be seized at any minute by the characteristic epileptiform convulsion of eclampsia. They were both put on a modified medical treatment of Stroganoff - case No. 2 responding and case No. 1 not responding. There is nothing apparent in these two cases that would indicate such action. The diastolic curve as shown in chart No. 2 is immediately effected by treatment which that curve in chart No. 1 seems to be resistant to the treatment. This curve will be noted in the subsequent cases.

Case No. 3 - Toxemia of Pregnancy - Mrs. Rose J. - primipara - white - housewife - entered the hospital June 17, 1930 with the following complaints: Pregnancy; severe headaches; spots before the eyes; blood pressure of 194/106.

Present Illness: last period Dec. 19, 1929 - quickening May 1st, 1930 - due Sept. 26, 1930 - headaches for past two weeks.

Past History: Chronic Nephritis of six years duration.

Laboratory: Albumen 4+; N. P. N. 32.9 mgm., Wass. Neg.

Treatment: Modified Stroganoff, supported by fluids and glucose intra venous.

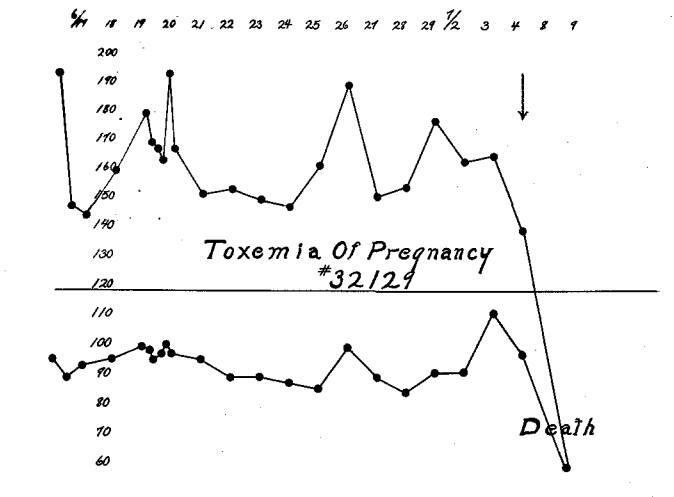


Chart #3

Delivered: July 7, 1930 by Caesarean section - spinal anesthesia.

Death occurred July 9, 1930.

Autopsy findings: No evidence of peritonitis; gall bladder shows one cholesteral stone; liver seems to show a marked palor, which may be fatty degeneration; stomach and small intestines greatly distended.

This case, a severe toxemia, is a very good example of an eclampsia superimposed upon a chronic nephritis. There is already a good deal of kidney destruction. This kidney destruction is an indication of other vascular lesions as well.

Fishberge states that any hypertension first affects the arterioles of the kidney, spleen, and pancreas, with a sclerosing action. This further embarrasses the already inadequate kidney -resulting in a severe process, with a persistent hypertension after delivery, as explained above.

The course is essentially that of a pre-eclampsia as chart #3 will indicate . There is not the same response in the diastolic curve as we noticed in the two previous cases. It remains low and more steady.

There is one finding not shown on this chart. Following the Caesarean, which was carried through with a spinal anesthesia, the blood pressure went

up to 190/100, as high as any time during the course of the disease. Williams brings out the fact that anesthesia is toxic to the eclamptic. This case would seem to carry that fact into the spinal field as well as the general anesthesia.

There was a gradual fall after the final spasm, only however, to 142/100, and never lower than this until the myocardium began to fail.

Case No. 4 - Nephritis and pregnancy -
Mrs. - entered the hospital Feb. 18. -
Complaints: Pregnancy; high blood pressure;
albuminuria.

Laboratory: P.S.P. Feb. 22 - 25%
P.S.P. Mar. 6 - 15%

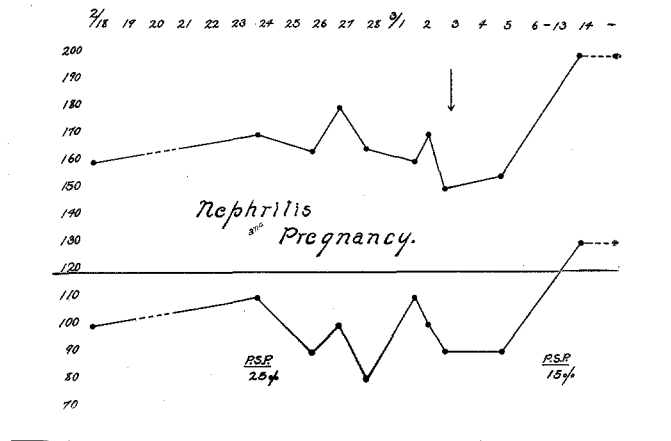


Chart #4

This is a very good case to demonstrate the course of the class labeled "Low reserve kidney". The kidney, as the function tests show, was decidedly insufficient to carry the extra load of a pregnancy. With treatment and diet, the patient was carried with a moderate toxemia. Then following delivery, there is a gradual increase in tension to a very high point. This gives us a fair index of the amount of pathology really taking place in a moderate toxemia. The case starts with practically all of her reserve kidney tissue lost. Then by

comparing the anti-partum normal tension with the post-partum normal tension, we get a measure of the kidney destruction, in terms of blood pressure increase.

Case No. 5 - Chronic Nephrosis - A severe toxemia - Mrs. Celia G. - White - housewife - Age 18 - primipara - entered the hospital June 21, 1930. Complaints: Blood pressure 180/120; Albumin four plus; small quantity of urine passed daily; has been bloated and swollen the past 6-8 weeks.

Present Illness: Pregnancy, last period Dec. 5, 1929; Due Sept. 12, 1930.

Past History: Feet and ankles used to swell years ago; Measles, Mumps, Scarlet Fever, Influenza.

Laboratory: Albumin four plus; N.P.N. 26 mgm. %.

Treatment: Was only supportive.

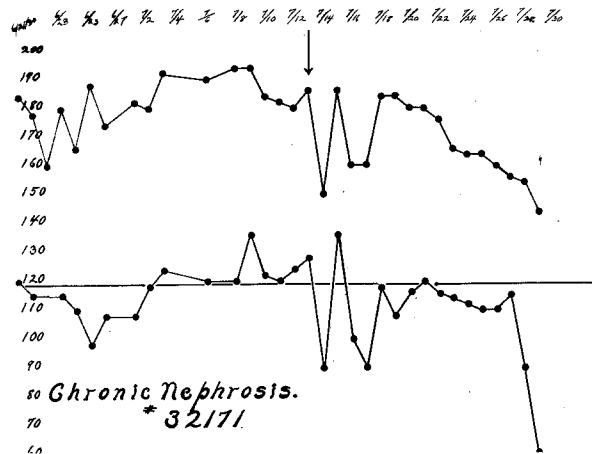


Chart #5

Labor was induced by Vorrhee's
bag 7/29/30 at 9:30 A. M.

Delivery of a seven months' fetus
7/30/30 at 8:55 A. M.

This is the case of a young woman with slightly damaged kidneys entering a pregnancy, only to be subjected to severe toxemia of the pre-eclamptic type. Not being affected by treatment it was found necessary to induce labor in the seventh month to avert the inevitable eclampsia, and further damage. As before, found the diastolic curve to be persistently high. There is a gradual return to the moderate hypertension and does not fall to normal as is the case of an out and out eclampsia. This is a case of carrying the patient at the expense of her arterioles, so to speak, until the fetus is viable, resulting in minimal destruction of maternal tissue and living baby.

ECLAMPSIA

The next three cases will illustrate the curves of a severe toxemia - an uncomplicated eclampsia. These are not essentially different from the records of the foregoing pre-eclamptics.

Case No. 6 - Eclampsia - Mrs. Esther L. - colored - housewife - enters the hospital May 10, 1928 in a stuporous condition.

Complaints: Swelling of feet and ankles. Convulsion at 12:30 P. M. on the day of admittance.

Treatment: Diet of milk only - hot packs - Morphine Sulphate - Glucose and Mag. Sulphate intra venous.

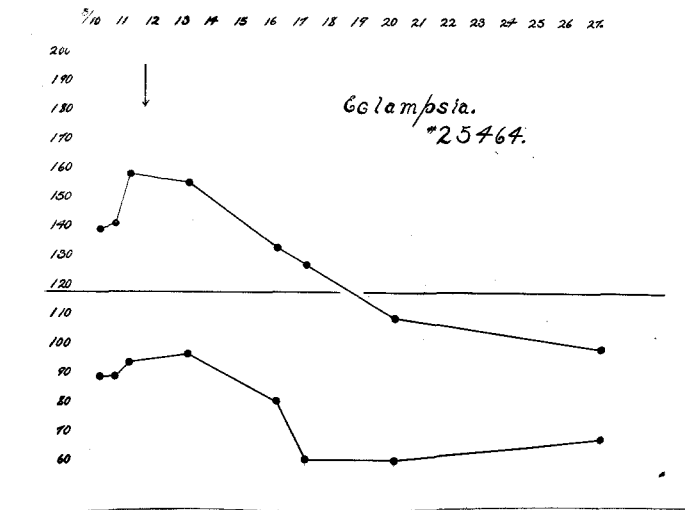


Chart #6

Delivery: May 11, 1928 by forceps and episiotomy. Baby stillborn.

This case shows immediate response to the loss of the fetus. There is a gradual, steady return to normal; thereby, ruling out any chronic nephritis as a complication. As Hillis found, these convulsions appeared at a relatively low blood tension - between 140 and 160 mm., nor was the diastolic curve high. This increase appearing late in the third trimester, was very rapid in it's ascent.

Case No. 7 - Eclampsia - Mrs. Myrtle Mc. - age 18 - primipara - housewife - enters the hospital Sept. 11, 1930.

Complaints: Swelling of ankles - blurring of vision - convulsions - blood pressure 190/120.

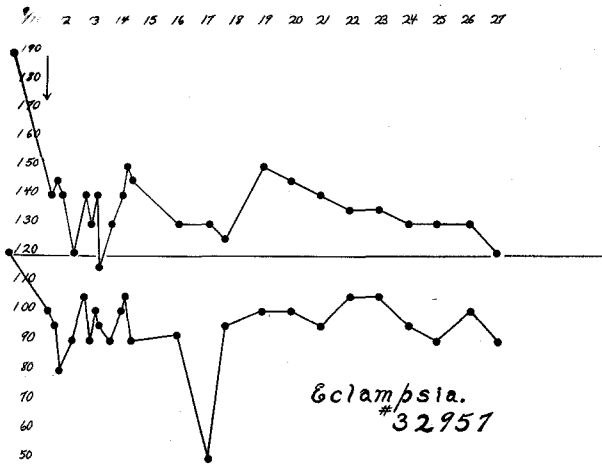


Chart #7

Treatment: Medical - to no avail;
labor induced the following morning.

Case No. 7 is a very good example of the relief afforded the eclamptic by the termination of the pregnancy. This early post-partum period illustrates very well the fluxuation of this type of hypertension, followed by prompt return to normal.

Case No. 8 - Eclampsia - Mrs. Carry J. Colored - housewife - age 28 - gravida 6 - entered the hospital Oct. 11, 1926.

Complaints: Pregnancy; swelling of feet and ankles; headaches; stuporous condition; convulsions four in number.

Past History: Measles - Malaria - rheumatism with another pregnancy - No previous toxemia.

Laboratory: N. P. N. 32 mgm. Wass. four plus.

Treatment: Modified Stroganoff.

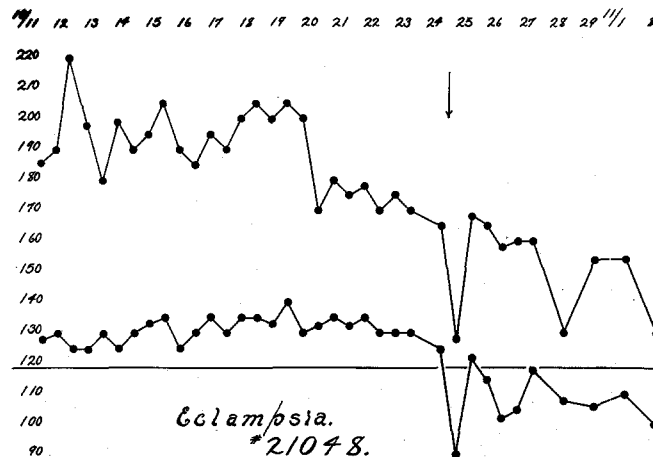


Chart #8

Delivery of a stillborn Oct. 24, 1926.

This case is a very interesting one, and, fortunately we were able to follow this patient to the present day. This pregnancy was the sixth, and in no previous pregnancy was there any toxic signs. The blood pressure curve was very agile. The diastolic curve was unusually high and gradually increased, though the systolic curve showed some improvement with treatment. Following delivery both curves went down to practically normal, 130/100. This diastolic reading is questionable.

Here we have an illustration of the fact brought out by Peckham. It is very fallacious to discharge a patient following the puerperium, showing a normal blood pressure, with the idea that she is in good condition and will not have further trouble. Twenty-two per-cent of Eclamptics will show signs of chronic Nephritis several months after delivery. In this case, a month after leaving the hospital, the blood pressure was 145/96, indicating a chronic nephritis, which was not suspected at discharge. The only indication was the diastolic which was, to say the least, questionable.

Following is a summary of the gestations:

1 - 5;

No toxic symptoms - one miscarriage - rheumatic pains in one, otherwise uneventful.

6th, Oct., 1926;

Described in detail above - severe toxemia - high blood pressure -
Urine analysis: No evidence of sugar,
No red blood cells, Few pus cells
and granular casts.

7th, May, 1927;

Toxic symptoms evidenced here -
blood pressure 128/82.
Urine analysis: no sugar, no red
blood cells or albumin.

8th, Oct., 1927;

Uneventful; blood pressure 115/80;
Urine analysis: no sugar, or red
blood cells.

9th, Aug. 3, 1929;

Entered the hospital in June, 1929
and was diagnosed a mild diabetes
Mellitus. Urine analysis: positive
acetone but no sugar on discharge,
few red blood cells and pus cells.

Entered the hospital in Aug. A
stillborn delivered; Entrance urine
analysis: sugar, 650 mg. per 100 cc.,
red blood cells and casts. Blood
pressure 115/80.

10th, Nov. 21, 1930;

Pregnancy complicated with diabetes.
Precipitate labor Feb. 2, 1931,
Blood pressure 120/80.

11th, Dec. 5, 1931; (last menstrual period)

At the present time this pregnancy is of five months duration. The patient exhibits a blood pressure of 140/90. Vomiting was present the first three months. Urine analysis: no sugar, trace of albumin, red blood cells, pus cells. Patient is on a diabetic diet, and in conjunction, using insulin at the present time.

The most important fact to be drawn from this case is the fact that a severe toxemia may crop out in a multipara after several normal pregnancies. Also, that the subsequent pregnancies need not be effected by this hypertension. It remains to be seen just what will be the outcome of this present pregnancy. Although she exhibits some mild toxic symptoms, the pregnancy will probably follow the course of the last five.

This history has been complicated by Lues all the way through. Following 1926 she took several course of anti-Luetic treatment - resulting in a negative Complement fixation reaction at this time. This disease will account for the miscarriages and stillborn babies.

Another fact brought out by this case is it's relation to diabetes; or as Gessner brings out, a common eteological factor:

"These statistics were taken in Baden. It was found that there were fewer cases of eclampsia during the "World War" than before or since. That this was a result of the scanty diet, poor in proteins and fats; thereby, making the lowest possible oxidation demands upon the organism. At the same time there was noted a 40% decrease in the death rate of Diabetes Mellitus. The lowest incident to eclampsia was shown by the woman forced to do outside work and not the one that remained inside."

Lawrence states that there is no evidence of a relation between the incidence of eclampsia and the incidence of concurrent diseases.

With these findings in mind it would seem plausible that both of these diseases were brought on this woman by environment and diet - bearing no relation to one another.

It was stated that the pancreas is one of the first organs effected by sclerosing due to hypertension. The picture is not that of a persistent hypertension however, and a temporary tension would hardly seem sufficient for the damage unless there was a predilection shown for this tissue. We

must consider however, that this woman has passed through three toxemias of greater or less degree.

These next three cases represent the eclampsia complicated by Chronic Nephritis.

Case No. 9 - Eclampsia - Mrs. Eliza R.- Colored - housewife - gravida 2 - entered the hospital July 10, 1927.

Complaints: pregnancy - headaches.

Previous History: last period Jan. 25, 1927 - Due Nov. 2, 1927. The first pregnancy at seven months was complicated by a toxemia, characterized by dizziness, blurring of vision, swelling of feet and legs.

Treatment: phlebotomy and Caesarean at seven months.

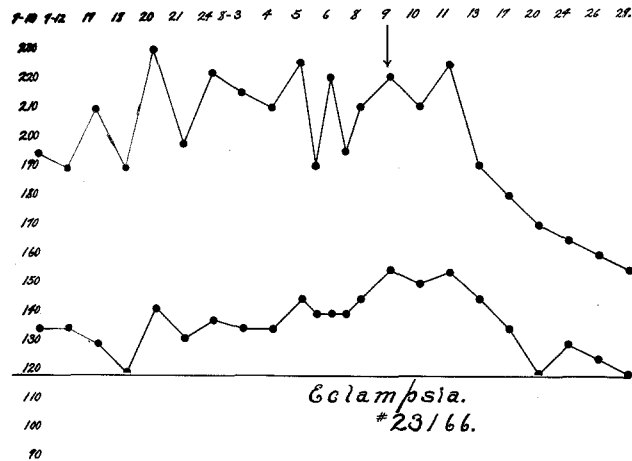


Chart #9

Delivered a stillborn - premature infant of seven months gestation.

This case brings out the rapid fluxuation, characteristic of this type of hypertension. The diastolic curve is very high and gradually ascending as the disease advances. There is prompt amelioration with the termination of the gestation. However, the tension remains high, showing this process to be complicated by a Chronic Nephritis.

Case No. 10 - eclampsia - Mrs. Evelyn A-White - age 17 - Para. 1 - entered the hospital Aug. 10, 1927.

Complaints: severe toxic symptoms - three hours after entrance patient had two convulsions - patient given ether with convulsions.

History: last menstrual period Oct. 18, 1926. Wass. was Neg.

Treatment: Modified Stroganoff.

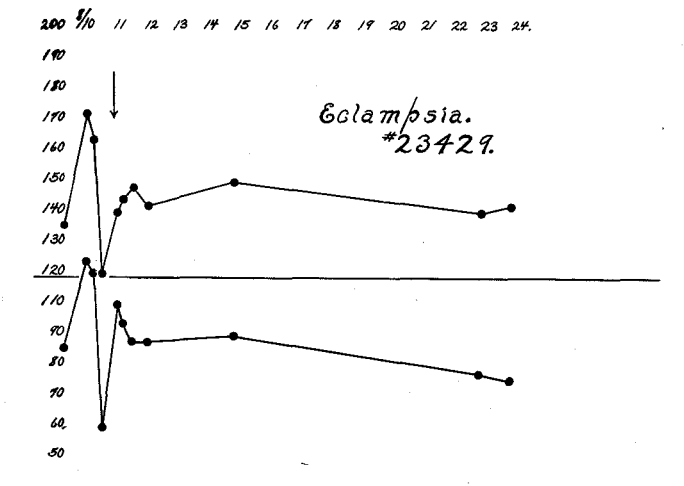


Chart #10

Here is a similar case showing immediate relief after the delivery; the systolic curve remaining high, while the diastolic curve going at once to normal, indicating only a mild Nephritic involvement.

Case No. 11 - eclampsia - Mrs. Lela J. White - housewife - age 19 - para. 1 - enters the hospital June 1, 1927 in a stuporous condition.

Complaints: puffiness of face, legs and abdomen. A convulsion at noon before entrance and two more following entrance. Blood pressure 170/130.

Laboratory: Urine analysis; four plus albumin and many hyaline casts; Blood, N. P. N. 45 mgm.

Treatment: Stroganoff.

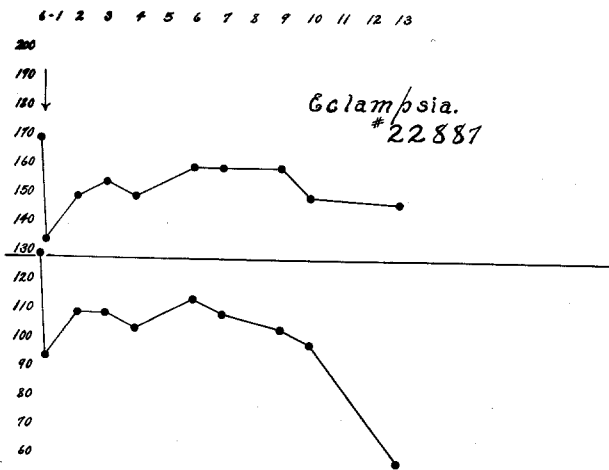


Chart #11

Delivery the following day accomplished by vaginal hysterotomy and forceps.

A similar case showing the immediate response to parturition; and again the systolic curve remains high, indicating a minor kidney involvement. In this case we have a high Non-Protein Nitrogen content of the blood. Fishberge states:

"The Non-Protein Nitrogen content of the blood may be raised by:

1. A coalescence of the arteriosclerotic foci in the kidney.
2. Occurrence of more or less diffuse reaction of glomerular changes.
3. Entrance of cardiac insufficiency."

Plass states that there is no characteristic change in the Non-Protein Nitrogen content of the blood in eclampsia, whether there be convulsions or not, and also is useless as an index of the severity of the condition.

With these facts in mind, this condition would have to be attributed to the kidney, whether arteriolar or glomerular, or both, is not of very much importance, but we then must consider that this woman is a chronic nephritic.

S U M M A R Y

It has been shown that hypertension in pregnancy is a very important finding, and carries a great deal of significance, and may, if properly observed early in pregnancy and at frequent intervals, give us a better knowledge of the individual normal tension, and also a good insight into the condition of the patient.

An understanding of the blood pressure curve will give us a practical classification, which has been shown to be fairly accurate. This would also lead to better handling of the case and a better final prognosis. Case No. 8 especially is very unusual. It gives us an idea of the post-eclamptic behavior and how subsequent pregnancies will be carried out. It is an example of the supreme - of the maximal in gestation following eclampsia; but in using this maximal we can get a better idea of the action of minimal gestation in a post-toxic patient. This case also brings up the possibility that eclampsia and Diabetes Mellitus may have

some relation or common etiological factor. This will bear further study in light of their etiology.

From these numerous charts, we get the idea that this hypertension is very active, rapid, and of a physiological nature; but as brought out, bearing subsequent relation to the structural hypertensions.

The diastolic curve seems to be a better index to the condition of the patient than does the systolic, and carries a great deal of weight prognostically.

While these few cases are very inadequate, they are very good cases to show the behavior of the blood tension in toxemias, the abrupt amelioration with the termination of the gestation, also the toxic response to an anesthesia.

It is here suggested that such a chart might be used in connection with each toxic patient, as the temperature chart is used on the ordinary patient. While the readings should be at shorter intervals and more constant, such a chart, it seems, would be of great value in management of the treatment and eventual prognosis.

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