THEORIES OF ECLAMPSIA.*

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THE gentleman who arranged this symposium on eclampsia asked me to introduce the subject of the causation. I accepted the invitation with a good deal of reluctance, because the time was short, and I wanted to pick out the salient points of the views and theories held by different men in regard to the subject under consideration. In the ten minutes allotted to me I can only briefly present these various views.

You all know the trite expression of Zweifel, that eclampsia is the disease of theories. That I have had more and more proved to me as I waded through the oceans of literature on the causation of eclampsia. As a matter of fact, we know practically nothing of the causation of eclampsia. A theory has only to be set up by one investigator to be knocked down by another, and since there are a large number of theories advanced, we must give both sets of workers credit for the immense amount of labor and time consumed in building up these theories and in knocking them down.

In 1839, Rayer,[1] and in 1843, Lever[2] (England) found albumin in the urine of eclamptics, which was confirmed by Devilliers and Regnault. In 1853 Frerichs declared that eclampsia was a uremia, the result of decomposition of urea in the blood, and that this decomposition resulted in carbonate of ammonia, the convulsions being elicited by this drug. Traube, Munk and Rosenstein[3] believed that eclampsia is due to hydremia and increased blood pressure in the aorta due to cardiac hypertrophy. This theory was disproved by Bidder.[4] Spiegelberg[5] said that sudden cessation of the kidney action was caused by inflammation of the kidneys, or by a reflex similar to the anuria that occurs in cases of catheterization of the urethra and ureters. This theory has not borne the brunt of investigation. Schroeder and Leyden[6] showed that eclampsia is rare in chronic nephritis, but that it is common in acute nephritis. Lantos[7] found albuminuria in 91 per cent. of eclamptics. Ingerslev[8] collected 106 cases without albuminuria or disease of the kidneys. Charpentier[9] collected 141 such cases.

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Prutz[10] found the kidney findings in cases of eclampsia had no relation to the severity of the attacks.

Halbertsma[11] has advanced the theory of eclampsia being due to compression of the ureters, but post-mortem examinations have disproved it. Of 32 post-mortem examinations made by Loehlein, in eight there was dilatation of the ureters. In 37 post-mortem examinations made by Olshausen, there was dilatation of the ureters in seven cases. In 103 non-eclamptic cases, post-mortem examinations showed that in twelve there was unilateral compression of the ureters, and in four bilateral dilatation.[12]

In a case I attended at the Cook County Hospital, eight months pregnant, in which accidental death occurred, post-mortem revealed dilatation of the ureters.

A pregnant woman's nervous system is more sensitive to external irritations, resembling in this respect the condition of children. The exaggerated effects of poisons, of hydremia, etc., may thus be explained. Von Herff[13] believed that a summation of external irritants was necessary to evoke convulsions in women so predisposed.

Nothnagel declared eclampsia an acute epilepsy. Ferè[14] described cases of epilepsy arising as a result of eclampsia. The writer has a case of epilepsy that came on after eclampsia. Epilepsy in some cases is due to toxemia.

Delore,[15] in 1884, suggested that bacteria might be the cause of eclampsia. Doleris, Blanc,[16] Favre,[17] tried to prove it. Gerdes[18] found a bacillus, which he named bacillus eclampsiæ, but Hofmeister[19] and Hägler proved it to be proteus vulgaris. The writer's bacterial studies of eclamptics' placentæ were negative. The tubes remained sterile. Gley found the staphylococcus aureus and albus; and Prutz[20] found a short, thick bacillus. Schäffer found that various germs injected into animals caused convulsions. Schmorl, l.c., Döderlein, Chambrelent,[21] Blanc, Loehlein and others could not prove the microbic origin of the disease. Hofmeister and Hägler proved that several varieties of germs are found in the blood and urine.

Stroganov[22] states that eclampsia is an acute infectious disease introduced through the lungs, and he believes in giving these patients plenty of fresh air and medical treatment.

Albert[23] believes that eclampsia is due to a latent or active microbic decidual endometritis. Mueller[24] agrees with Albert, and says that eclampsia is due to intoxication originating from the parturient canal.

In 1887 the auto-intoxication theory was advanced by Bouchard,[25] which opened up a new field for investigation, and Bouchard tried to prove that eclampsia is an intoxication from impaired action of various excretory organs. Riviere,[26] a pupil of Bouchard, said that (1) pregnancy was attended with an overproduction of toxins; (2) that there was hyperemia of the kidney and liver, with poor elimination; (3) that poisons damaged the kidneys.

In 1890 Laulanié and Chambrelent[27] proved that the blood of pregnant women had in it circulating toxins, and the urine less of them. Ludvig and Savor[28] confirmed the above theories, and sought to prove that the poison is carbamic acid.

Vollard[29] disproved all the above theories. He showed a coagulating ferment in the blood in two cases. Schuhmacher[30] and Stewart[31] also dethroned all these theories, proving that the toxicity of the urines of normal, nephritic, and eclamptic women was the same if the specific gravity was the same.

Zangenmeister[32] found that the excretion of ammonia and urine water were parallel, that the two diminish during labor. Eclamptic urine has a low per cent. of ammonia. From this the deduction is drawn that the eclamptic kidney retains some salts and lets others through.

Pinard, and Bouffe de Saint-Blaise[33] set up the theory of hepato-toxemia, that the toxemia is the result of deficient liver action. This insufficiency of the liver is due to heredity (Pinard), anterior disease of the organ, acute infectious disease, no matter how mild, and intestinal affections.

Turenne[34] believes many of the milder symptoms of toxemia, vomiting, pruritus, etc., of early pregnancy are due to a menorrhemia, that substances which are ordinarily excreted during menstruation now accumulate in the blood. Reasoning from the analogy of the use of ovarin in the treatment of the symptoms of amenorrhea (menopause), Turenne prescribed ovarin for these pregnancy disturbances, and says he always succeeded in curing them.

Planchu[35] reviews the auto-intoxication theory, and says the toxemia is due to the liver, and that the following conditions in pregnancy may be ascribed to it: Nausea, insomnia, somnolence, ocular troubles, neuritis, vertigo, headaches, pruritus, etc. Severer symptoms are vomiting, general puritus, ptyalism, edema without albuminuria, herpes gestationis, puerperal mania, finally pernicious vomiting, albuminuria, and eclampsia.

Juergens,[36] in 1886, found constant changes in the liver and liver cell emboli. Klebs[37] confirmed these observations. Pilliet[38] found necrotic foci in the liver, in addition, and Schmorl[39] found necrotic hemorrhagic and anemic foci in the liver, thrombosis of the portal vein and small veins of the periportal circulation. Schmorl's work, by the way, is classic, and one of the milestones in this history that promises to be permanent. Schmorl found in the kidneys anemic infarcts, in the lungs hemorrhages, and exudations in the alveoli, as well as small hemorrhages in the brain. He also found thromboses and areas of softening in the heart. There were emboli and liver cells, and of giant cells (syncytium) from the placenta in the lungs. Schmorl believed the cause of eclampsia would be found in the placenta, that a substance of the nature of fibrin ferment, the result of degenerative processes or metabolic changes going on in the placenta, circulated in the blood and caused the thrombosis and emboli. Jung,[40] Pels-Leusden,[41] Limfors,[42] Lubarsch[43] support these findings, while Dienst,[44] Fehling,[45] Schuchard say they are not constant. Schmorl at first thought a special pathologic syndrome could be asserted for eclampsia, but his later researches showed the findings in a large majority, but not in all women dying from convulsions.[46]

J. Veit[47] and Opitz[48] are working on a syncitial theory of eclampsia. Schmorl and others have proved the deportation of villi by the blood. Veit and Opitz believe that the syncytium is dissolved in the maternal blood, producing syncytiotoxin, which is held responsible for many of the disorders of pregnancy. Normally, the blood produces anti-bodies, in this case an anti-syncytiotoxin, a precipitine. In eclampsia there is a lack of this anti-syncytiotoxin. By triturating the placenta with sand, Opitz and Weichart obtained a filtrate which they injected in increasing doses into goats. The serum of these immunized goats they tried on themselves, with only local reaction. In eclamptic patients the serum had no effect.

Lange[49] studied the relation of the thyroid to albuminuria and eclampsia. He found that physiologically goitre was present in 60 per cent. of gravidæ, and 100 per cent. of puerperæ. He found that the kidney of pregnancy did not develop if the thyroid gland was enlarged, that if there was albuminuria iodothyrin would usually cause its disappearance. He found that animals from whom he removed part of the thyroid developed tetany, and some of them convulsions.

Nicholson[50] found the thyroid hypertrophied in 81 per cent. of

cases examined, and he also notes the good effects of iodothyrin in the cases of "kidney of pregnancy." No one has yet tried to prove that the para-thyroids have anything to do with toxemia, eclampsia, and allied conditions, nor have the adrenals been incriminated. The high arterial pressure present so often in eclamptics should have led investigators to suspect the adrenal glands.

Cryoscopy is being used to determine the osmotic pressure in the maternal and fetal blood, and in the urine, with a view of discovering the permeability of the kidney. The ultra-microscope has been used for the same purpose by Jankov.[51] Nothing of value has as yet been obtained.

Finally, eclampsia is supposed to take its origin in the child. Ahlfeld,[52] in 1894, expressed the opinion that the toxin was formed in the child or the fetal placenta. Fehling,[53] Kollman,[54] and others have supported this theory. Since the convulsions often cease after the child dies, or is delivered; since they are more common in twin pregnancies; since pathologic changes similar to those of the mother are found in the fetus, some color is given the theory. Baron et Castaigne[55] have proven that substances are absorbed from the child into the mother.

There is much to be said against this theory. Indeed, out of the entire presentation only one point seems to be generally conceded, namely, that eclampsia is due to the action of a toxin in the blood upon the nerve centers. How and where the toxin is formed are unknown.

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