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MALARIAL TOXAEMIA AS A CURE OF PUERPERAL CONVULSIONS: WITH REPORT OF TWO CASES.

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To first argue the theory that malarial toxaemia may and does produce the accumulation of symptoms known as eclampsia, it will first be necessary to bring before you the cause of eclampsia. Eclampsia is not really a disease, but an accumulation of symptoms that we recognize as a disease but have certain and well defined causes for their production—they are a collection of symptoms characterized by convulsions of epileptiform seizures that come on prior, during, or after labor, but looking at it from an etiological or pathological standpoint is no more a disease than fever is a disease, but as we never have a variation from the normal standard of health without a cause, so in this disease we have a real etiological factor and to the study of this I now hasten on.

The physician who makes systematic examinatons of the urine of all pregnant women who are placed under his care for the ordeal of delivery, not alone for albumen and casts, but for urea also, and who keeps check on amount of urine passed during the twenty-four hours, such a physician will never be caught napping and he will rarely if ever have a case of puerperal convulsions to treat. So easy to prevent, but when once they come on they are sometimes fearful in their effects, we know with diminished urea that toxaemia is more apt to take place, so you may and do have toxaemia though there has been no suspicion of nephritis throughout the pregnancy, but as the danger of urea

collecting in the system producing toxaemia which we claim is the underlying cause of eclampsia.

The clinical symptoms in cases differ some, but in most cases we have some of the most prominent symptoms of a blood toxaemia and we have some warnings that some impending danger is about to overcome our patients. The first symptoms are those we attribute to the poisoned blood, effects upon the sensitive nerve cell and headache very constant, symptom in all these cases, floating specks before the eyes, very often very sick stomach, difficulty of mind concentration, and in all cases some variation of normal standard. Upon examination of urine you will find diminished excretion so the balance between excretion and secretion are disturbed so you have then the cause that produces the effect and results in blood toxaemia. You may have albumen and casts, it is owing to whether you have nephritis complicating your deficiency of excretion or not, should you have this condition of course much worse for your patient. Now have your most prominent excretory organ diseased and you have quite a much more complicated state to treat. When we accept that the difference that exists between secretion and excretion that we have in disease as the true cause of toxaemia, and accept the theory that toxaemia is the real etiological factor causing this collection of violent symptoms, we call eclampsia. The study of this disease is quite simple for it offers us a reasonable, sensible theory of its causation and does away with the old worn out theory of the pressure of the gravid uterus upon renal veins—a theory that now has no adherents and was never at any time worthy of the sanction of medical men. Of all the ridiculous theories I have ever read, it had less to commend it than any theory I have ever read of upon any disease, and this old worn out theory has done more to make this purely symptomatic disorder difficult and complicated than anything else. We know when the body is in healthy normal condition there is perfect balance between the secretory an excretory organs.

Now let's see what happens when we have this balance disturbed. We have the blood taking up the waste products of cellular action and then we have blood that is pathological and capable of producing disease in our body. During the pregnant state the blood alters in quality, also in quantity, becomes more watery. Albumen and urine falls below the normal and when we remember that all activity is greatly increased during pregnancy excreminitious substances quickly collect in our system, great strain put upon the organs of excretion and at any time this condition of equilibrium between secretion and excretion may be disturbed and we have all the necessary conditions to produce a genuine fit, and it stands very much to reason if this is

apt to occur and does occur where there is no nephritis complicating the toxaemia it will much easier occur where you have a nephritis. When a woman becomes pregnant who has chronic nephritis she is in grave danger of the grave affection of eclampsia. She should be carefully watched after and she can safely be carried through her pregnancy by treating her intelligently along the lines of elimination of waste products and the failure of elimination of the kidneys when diseased is about all the kidneys have to do with eclampsia—that is to say, failure of the function produces or predisposes to blood toxaemia, and should the skin, bowels and lungs act vicariously we may then not have eclampsia, no matter how grave the kidneys are affected. It is very probable that nephritis may be sometimes caused by blood toxaemia, poisoned blood being constantly filtered through the tubules of the kidneys producing irritation and causing them to shed their epithelium and render the tissues good and favorable soil for bacterial development and then we have a true inflammation.

Now if we accept the theory of blood toxaemia as the underlying etiological cause of eclampsia, then we come to our subject proper, malarial toxaemia. We find the same charging of the blood of excrementious substances as we do in any other blood toxaemia; we find the balance existing between secretion and excretion disturbed and find blood charged with malarial parasites as well as other poisonous substances. So I do not claim that malaria itself causes puerperal convulsions proper, but its effect upon the secretory as well as the excretory organs is such as to keep the balance disturbed and blood toxaemia results same as from any other causes, the eclampsia comes on as a consequence of this toxaemia. In malarial toxaemia we find that there is diminished urea, also diminished amount of urine passed during the twenty-four hours. Variations in function of liver and other secretory organs as well as the other excretory organs. Then if some condition that we recognize as eclampsia comes on from this blood toxaemia from the etiological factors, why not from malarial toxaemia. That it does I think my two cases establishes without a shadow of a doubt.

FIRST CASE.

Time, July 20, 1900; Mrs. W. H. H.; first pregnancy, age 21. Stout robust woman, seven months advanced in pregnancy. I will here state I was never asked to wait on her during her confinement. Was sent for on July 20, 1900; patient lived seven miles in country; the messenger said patient was pregnant and had a fit. I suspected trouble and went prepared with the usual remedies. I found her at 2 o'clock in night

having convulsions and unconscious between the attacks, which came on at intervals of about 15 minutes. My first procedure was to perform venesection, which I did successfully, obtaining dark easy co-agulated blood. The convulsions continued on. My next procedure was to give 10 drops Tr. ver. vir. and repeated in one hour. Convulsions continued. During this time I was administering chloroform, but deep chloroform narcosis only controlled the convulsions and did not prevent them returning. Upon examination I found uterus dilated. With my two first fingers I hastened dilatation—head come down, applied instruments and delivered child, which was dead. The convulsions continued. I then gave 60 grs. Bromide Pot. and 30 grs. chloral per rectum, but convulsions still continued. It was now noon, July 21, 1900. I came home, returned at 3 o'clock. She was still having convulsions. Upon examination I found her axillary temperature 104. At 5 in evening she had a free sweating stage. Convulsions continued. I suspected malaria. Gave 10 grs. Bisulp. quinine, hypodermically; repeated in 4 hours. After first dose of quinine she had only three convulsions; when they ceased she became conscious at 4 o'clock on morning of July 22nd. I continued use of quinine; she made a rapid recovery. I will also state no albumen in urine though upon inquiring found she had the premonitory symptoms, though no oedemia of legs.

SECOND CASE.

August 15, 1901. Mrs. E. C. L. Fourth pregnancy. She had never had any complication of pregnancy, but had had phlegmasia. Was at her bedside when first convulsion came on. She lived in an intensely malarial section. Upon examination found uterus dilated. Applied instruments, delivered her of full grown, very well developed child, which lived. At the time of her first convulsion, during the interval, found axilliary temperature 103 1-2 in 2 hours; by wrapping in warm blankets sweating stage came on. She had had up to that time six convulsions. Remembering my experience with the other case I gave her quinine, same method as my other case. One hour after first dose convulsions ceased. She never had any more convulsions and made an uninterrupted recovery. Now if you say that it was my treatment other than quinine in first case, it certainly could not have been that in the second case. I bring the subject before the Society with the report of these two cases, and hope the discussion may prove beneficial to us mutually. I do not know your views upon the subject, but I am convinced that the eclampsia was caused solely from malarial toxaemia,