CORRESPONDENCE

Etiology of eclampsia

To the editors

It is amazing how obstetricians cling to the idea that eclampsia (convulsive metabolic toxemia of late pregnancy) is caused by the fact that the expanding pregnant uterus interferes in some manner with the physiologic function of abdominal and pelvic blood vessels. Abitbol and colleagues raised the question again in the March 1, 1976, issue of the JOURNAL, on page 460, in the article "Production of experimental toxemia in the pregnant rabbit." They credit Young, in 1927, with the idea that primary, etiologic "uteroplacental ischemia" plays a fundamental role in the pathogenesis of eclampsia.

However, Denman,[1] in 1829, a century earlier, wrote:

> It has been presumed, that the pressure made by the expanding uterus upon the descending blood vessels, causing a regurgitation of the blood to the superior parts of the body, to the head in particular, by overloading the vessels of the brain, produced convulsions.

Page simply added to this speculation with the idea of the "Goldblatt kidney"; thus, myth has built upon myth and our obstetric-gynecologic academicians remain today, in 1976, as much in the dark about the etiology and pathophysiology of eclampsia as Denman was 147 years ago!

In my book, *Metabolic Toxemia of Late Pregnancy: A Disease of Malnutrition*,[2] in commenting on the theory and experiments devoted to trying to prove the theory of primary "uteroplacental ischemia" in the etiology of eclampsia, I stated:

> All of these experiments have one vital weakness: We never find in Nature, in toxemic women such clamps on the abdominal aortas nor ligatures on the ovarian and uterine arteries.

Denman[1] also clearly stated one of the most perplexing observations about this "ancient enigma of obstetrics," eclampsia:

> It is said that women are far more liable to convulsions, in first than in subsequent labors, which is true. . . .

It seemed logical that the primigravid woman might have a "tighter" abdominal wall or even a "tighter" uterus which impeded blood flow, and this assumption fit well with another clinical observation that women with twins had a higher incidence of metabolic toxemia of late pregnancy. However, we know now from the work of many people that *the well-nourished primigravid woman escapes eclampsia* while the grand multiparous woman in many nations has been shown to be at "high risk" for the disease.

It is compelling to try to clarify the nutritional etiolgy of eclampsia; prevention and therapy will never be rational or scientific on the clinical level until the correct ideas are taught in obstetrics. As long as the etiology of this problem remained "unknown" to Denman, he was justified in "trying" various methods of prevention and therapy.

> At the time of labour it is a rule generally observed that their minds should be kept composed, their apprehensions quieted, their present sufferings soothed by the tenderness of their friends and attendants; that they should be encouraged with the hope of a happy event. But when any symptoms of disease appear . . . no symptoms can require more attention than those which have been recited as threatening convulsions. Bleeding is known to lessen, in a very effectual manner, all the complaints in pregnancy which arise from uterine irritation, and to a certain degree, in pregnant women, from most other causes. It is therefore universally recommended in all cases, when convulsions exist, or are to be apprehended . . . in some very urgent cases, besides the blood taken from the arm, it will also be found necessary . . . to use local bleedings, by scarification and cupping at the nape of the neck, or by opening the jugular vein, or sometimes by cutting the temporal artery; a thing so easily done as not to deter us from the practice, and often so efficacious as to invite our doing it on many other occasions.

Denman described taking 40 ounces of blood "with the happiest effect" and ". . . in a labour of long duration, when the convulsions have continued and been severe, not less than sixty or seventy ounces" of blood were removed from the eclamptic woman.

Do modern physicians take a more scientific position here? There is little evidence to indicate that they do in 1976! We have identified hypovolemia, especially a contracted plasma volume, as a central problem in metabolic toxemia of late pregnancy.[3, 4] It was observed in the 1920's when blood-letting was being abandoned that the eclamptic woman seemed to need all the blood she had and more, too! But our erudite academicians with the help of the private drug industry found a less obvious method to attack the blood volume of the eclamptic patient: low sodium diets and sodium diuretics! The use of diuretics became universal in obstetrics in the United States in the late 1950's among physicians who would be the first to condemn "blood-letting" by slashing the jugular vein or the temporal artery!

At the Food and Drug Administration hearing, "Certain thiazides: their use in pregnancy," on July 17, 1975, in Rockville, Maryland, the members of the obstetric-gynecologic Advisory Committee, Bureau of Drugs, were amazed to hear Professor Leon Chesley join our attacks on the diuretic hoax! You would have thought we all had just descended from outer space with such a "radical" idea! The testimony evidently left the committee in a state of shock because no word of the revelation has been issued by the Food and Drug Administration in more than nine months! We remain in the Dark Ages here, side by side with Dr. Denman in 1829!

Tom H. Brewer, M.D.

President
Society for the Protection of the Unborn Through Nutrition
17 N. Wabash
Suite 603
Chicago, Illinois 60602

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