cases, it is evident that its constitution plays no role in the diminution of the chlorides in the urine.

Karanyi has proved that the excretion of the chlorides depends on the circulation in the kidneys, and when this is interfered with there is diminished excretion, therefore it is evident that in eclampsia there exists an active contraction of the vessels of the kidneys. Zangemeister proves that a similar condition exists, to a minor degree, in normal labour, and considers that all the phenomena of eclampsia may be due to arterial spasm giving rise to periodic anæmias of kidneys, liver, pancreas or brain. Since eclampsia only occurs during period of active uterine contraction, he suggests that the arterial spasms are brought on with the uterine contractions, or by them.

The view that the toxin develops in the fœtus receives some clinical support from the fact that as a rule the disease comes on in late pregnancy when the fœtus is well developed; and that with the death of the fœtus, in many cases, the symptoms of toxæmia rapidly subside and the patient recovers. Kaltenbach and Fehling are strong supporters of this theory.

Dienst holds that eclampsia results from insufficiency of the maternal excretory organs in the presence of the fœtal waste products, this arising from their previous morbid exudation, or resulting from the deleterious action of the fœtal toxins upon them. He particularly dwells on the harmful action of these toxins on the maternal heart and suggests that the impairment of its function is the most important factor in bringing about the accumulation of toxin in the maternal organism. Dienst has drawn attention to the frequent development of convulsions in the children of eclamptics, and in several such cases has found the characteristic changes, associated with eclampsia, in the fœtal liver and kidneys. He explains this by suggesting that when the maternal blood is saturated with the toxin, a "reinfection" of the fœtus follows, setting up kidney and liver changes in it, which leads to its death, either before or after its birth.

The view of the fœtal origin of the toxin has received a severe setback in a recent paper by Hitschmann, who reports a well marked case of eclampsia in a ii. para at the 18th week of pregnancy. She was delivered of a large hydatid mole and no trace of a fœtus was found. It is thus evident that fœtal toxins cannot be the sole cause of eclampsia.

Recent investigation certainly seems to favour the view that the source of the toxin is in the placenta or the tissues entering into its formation.

Veit and Schmorl think that the deportation of the cells of the fœtal