concludes that both the blood serum and the urine of the normal as well as the eclamptic patient while poisonous are constantly changing, being subject to wide variations in the degree of their toxicity, and that it is by no means certain that the toxic agent, which produces results on experimental animals, is the same which causes eclampsia. The nature of the poison has given rise to much speculation. Early investigators believed it to be ammonium carbonate, the result of a splitting up of urea in the blood. Others have advanced the theory that the poison was a retained constituent of the urine, kreatin or kreatinin. Masin (6), by an elaborate series of experiments, attempted to show that it was carbonic acid, a product of intermediary metabolism, while Poehl attributed the eclamptic seizures to leucomain poisoning, basing his conclusions on the increase of leucomains in the urine of eclamptics.

Recently, Albert (7) has advanced the theory that the poison has a bacterial origin, from a latent infectious endometritis existing during pregnancy. Most of the supporters of the auto-intoxication theory regard the poison as the product of intermediary metabolism of the liver, while others (Fehling) look upon the fetus as being the source of the poison. While each of these theories has arguments in its favour, neither the nature of the poison nor its source are known. The pathologic anatomic findings in patients dead of eclampsia furnish perhaps the strongest proof of the chemotoxic theory. Schmorl (8), who has made an exhaustive examination of the bodies in 73 patients dead of eclampsia found changes in the kidneys in all but one, consisting of cloudy swelling, fatty degeneration and desquamation of the renal epithelium, with frequently but not constantly epithelial necrosis. In addition, thrombi were found in the glomeruli and in the small veins and arteries. The liver also was quite constantly involved, presenting in 71 of 73 cases examined hemorrhagic and anemic necrosis and in the two cases in which these changes were not found, there were present fresh thrombi in the portal vein. Similar changes were found in the brain and lungs, and in the heart fatty and parenchymatous degeneration was common. Schmorl interprets these changes in the different organs as complicated necrotic processes secondary to the thromboses.

The recent experiments of Kohlman (9), and Dienst (10), have shown that there is an increase of fizrin in the blood of an eclamptic.

Volhard, who has confirmed these experiments, attributes this rather to an increase in the fibrin ferment