villi, leading to the formation of a syncytiolysin in the maternal circulation, is a probable explanation of the toxæmia; in short, that eclampsia is an auto-intoxication due to a poison of syncytial origin. This view was elaborated before this Society at some length recently, so I will only recall it to your attention on this occasion.

Bandler supports this theory, and in a recent paper advances the view that the placenta is a gland giving off into the maternal circulation an enzyme from the trophoblast and syncytial cells. That the ovary is an organ furnishing an enzyme which has among its attributes the property of resisting or modifying the action of the fœtal enzyme. This he terms the maternal enzyme. Eclampsia, he suggests, is due to a mal-secretion of the fœtal enzyme or a relative mal-secretion or insufficiency due to the action of the maternal enzyme.

The action of the toxin, however it may be produced, upon the maternal tissues must now claim our attention.

The eclamptic attack is simply a symptom of the action of these toxines. The most evident clinical fact, outside of the convulsions, is the irritation of the vaso-constrictors and increase in arterial tension. The whole brunt of the attack is borne by the nervous system.

The oliguria and anuria result from this vascular spasm, which also leads to laceration of the finer blood vessels and results in hæmorrhagic exudations into the tissues. The coagulation necrosis found in various organs may thus be explained, especially when the fibrin of the blood is increased, as is reported to be the case by Dienst, who calls attention to the increased formation of fibrin in this condition. In fatal cases he has found it to be tenfold over the normal. The role of the leucocytes in the formation of fibrin ferment is well known, and Dienst has established the occurrence of well marked leucocytosis in eclampsia; hence it is evident that these poisons have a distinct leucocytic tendency. Their rapid manufacture, due to the toxæmia, results in their lessened resistance, so that they, in breaking down, thus indirectly contribute to the formation of fibrin ferment and pave the way for extensive thrombosis.

I trust that this brief review of the etiology and pathology makes it evident that in eclampsia we have to deal with a toxæmia, which, however produced, is of more or less gradual onset and that the toxin acts by giving rise to arterial spasm and affects certain organs as the kidneys, liver, and heart; and when in sufficient quantity in the maternal organism, precipitates itself on the general nervous system, producing a storm which wrecks the mother and child, or if only the latter tends to subside with or without permanent damage to those structures which bore the brunt of the attack.