Traube advances the theory that the basis of uraemic symptoms is œdema of the brain which, from its increased volume, induces anaemia. Two conditions predispose to œdema, marked hydraemia and increased arterial tension, which, as we know, are so frequently found in disease of the kidneys. Traube considers that the special symptoms depend upon the degree and localization of the œdema, namely, convulsions occur when the middle lobe is affected, coma when the œdema extends to the whole cerebrum.

This theory is supported by: (1) The frequency of cardiac hypertrophy and hydraemia in cases of uraemia; (2) The production of coma and convulsions in dogs by the injection of water into the carotid, after ligature of the ureters and one of the jugular veins.

The objections to this theory may be stated briefly thus: That these symptoms in dogs are only produced experimentally by the injection of enormous quantities of water, and that even then the brain may present no indications of œdema. Further, hypertrophy of the heart and hydraemia are not always found in cases of uraemia, while post mortems frequently fail to demonstrate œdema of the brain. In cases where cerebral œdema has been found, it has been thought by some to be the effect rather than the cause. Notwithstanding these objections, Traube's theory has, in a modified form, several adherents.

Chemical Theories.

(a) Retention of urea.—This theory is favored by the following:—(1) The appearance of marked symptoms when the secretion of urine and elimination of urea are much reduced. (2) The detection of urea in the blood under such conditions. (3) The production of drowsiness, convulsions and vomiting after ligature of the renal arteries, or ureters, or removal of the kidneys.

Against this theory it must be admitted: (1) That all symptoms of acute uraemia may be absent, when complete suppression has lasted for many hours or even days, without vomiting or diarrhœa, which might produce vicarious elimination. (2) That the symptoms, in their frequency and severity, do not bear any relation to the quantity of urea excreted. (3) That, occasionally, no symptoms of uraemia may be present even though large quantities of urea are found in the blood. (4) That when urea is given to animals with their food, no symptoms are produced, so long as it can be freely excreted. (5) That frequently in dogs no symptoms follow the injection of large quantities of urea into the circulation.

In any case that urea cannot be the sole determining factor in uraemia, is indicated by the fact that death, with uraemic symptoms, occurs much more quickly when the ureters are ligatured than when the kidneys have been extirpated.