of uræmia is the presence in the blood of the ammonium salt of carbamic acid—ammonium carbamate. The recent investigations of Hahn Pawlow, Massen and Nencki render it probable that urea is formed in the liver by the dehydration of ammonium carbonate, and this fact has led some physiologists to suspect that the highly toxic ammonium carbamate may be responsible for uræmic states. This view appears to me to be at variance with the following facts:

1. In watery solution ammonium carbamate is very unstable and is rapidly converted into ammonium carbonate. We know, however, that ammonium carbonate does not occur in uræmic blood in sufficient amount to produce symptoms, and usually cannot be found at all.

2. The toxicity of uræmic blood is not lessened by dialysis, as it would be if the toxicity depended on a diffusible ammonium salt; nor does the diffusate contain ammonium.

3. The urine of uræmic patients does not necessarily contain an increased proportion of N. of ammonia, as it should do if the synthesis of urea were impaired. On the other hand, in liver diseases in which there is extensive parenchymatous destruction, the N. of ammonia may be greatly increased, even in the absence of uræmic symptoms.

Owing to the instability of ammonium carbamate its isolation from the blood is impracticable, and inferences as to its occurrence there depend chiefly on indirect evidence. The conclusion seems justified that such knowledge as we possess does not support the supposition that ammonium carbamate is concerned with the production of uræmic intoxications.

Before passing to the more constructive consideration of the uræmic problem, it is proper to make brief reference to two widely different theories of the nature of uræmia. One of these is the celebrated hypothesis of Traube that renal disease causes thinning of the blood plasma, hypertrophy of the left ventricle and excessive arterial pressure. If the arterial tension is increased beyond a certain point or the plasma of the blood becomes further thinned, œdema and anæmia of the brain are produced and uræmic symptoms result. There are fatal objections to this theory. These are: 1. That there may be marked cerebral symptoms without arterial tension. 2. That the specific gravity of the serum is often normal in typical uræmia. 3. That there are uræmic patients in whom neither cerebral anæmia nor cerebral œdema are found at autopsy. 4. That a marked degree of anæmia of the brain and of œdema is occasionally found in the absence of all symptoms resembling uræmia.

It is, however, clear from clinical study that there is often a close association between certain uræmic symptoms, especially convulsions