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salts, instead of being built up in the body into urea, in order to neutralize the condition of reidosis in the tissues and to save the numeral bases of the body.

It must not, however, be assumed that acid intoxication exists because the percentage of ammonia uitrogen exercted in the urme is very greatly increased. For this proportion may be raised, not because there is any absolute increase in the amount of ammonia introgen, but because there is an absolute decrease in the amount of more exercted. This not infrequently happens in conditions of protein or total starvation. It has been found that when the urea exerction falls below 7 to 8 grin, in the twenty-four hours, the relative percentage of ammonia introgen increases, and that this may occur apart from the presence of organic acids. An absolute as well as a relative increase in the output of ammonia introgen must be looked for, if this is to be used as a basis of prognosis. As much as 12 grin, of ammonia uitrogen have been observed to have been exercted in twenty-four hours, being 49 per cent of the total introgen exercted.

The total quantity of aumonia excreted forms an indication of the degree of acid intoxication, but not necessarily of the amount of organic acid being sumultaneously exercted. For the estimation of this, the test of Hart, mentioned above, forms a very useful guide,

It is of the greatest value to make constant observations on the percentage of the ammonia-nitrogen exerction in any given case, to ascertain the presence of increasing danger; but the figures so obtained are of httle value in comparing one patient with another, for a man may be in fairly good health who is exercting a much greater amount of ammonia introgen than one who is in diabetic coma. There appears to be little doubt that acid intoxication is responsible to the coma and other toxic symptoms in diabetes, and kills the patient; but the exerction of acetone bodies may go on for many months without acid intoxication supervening.

In starvation, the condition is different, I cause, in spite of the fact that the patient suffers from acidosis, he can still assimilate and use carbohydrates if he can get them, and as he still possesses the power of utilizing cleavage carbohydrate derived from his own tissues, he probably dies rather from actual deprivation of food than from acid intoxication, a point which makes the outlook, as far as treatment is conceined, much more hopeful than in the corresponding state of diabetes.

In post-anaesthetic poisoning, it must be remembered that the patients have generally been starved, and that in starvation the total exerction of animonia is to be estimated, not merely the percentage of the animonia uitrozen, as this may appear unduly high, owing to the small amount of urea exercted. The prognosis is much improved if treatment with sodium bicorbonate and glacose is used before the administration of the anæsthetic. Glucose seems to be more effective than the alkali in preventing post-anæsthetic vomiting.