show absolute agreement between the amount of alkali which was experimentally found to be taken up by the proteins and that which was calculated on the basis of the free dibasic acid groups, the values nevertheless are of the same magnitude and indicate at least from the qualitative standpoint that there is a distinct relationship between the alkali-combining power of these proteins and their content of free dibasic acid groups. The greatest discrepancy is found in gelatin. This, however, is not serious especially since it is very probable that in the estimation of small amounts of aspartic and of glutamic acid considerable experimental error enters.

Our method of estimating the base-combining power of the proteins was carried out according to the procedure which has previously been used by Tague[16] for amino acids and by Loeb and Hitchcock for proteins. On account of the logarithmic increase in pH on addition of alkali the method is not capable of a very high degree of accuracy at high alkalinity.

147 (2379)

Reactions of the urinary bladder in canine anaphylaxis.

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As an index to possible smooth muscle reactions, we have studied the pressure changes in the urinary bladder during canine anaphylactic shock, with parallel tracings of the changes in the arterial blood pressure. The dogs were sensitized to horse serum by Weil's method.[1] They were tested by intravenous injections of 0.5 to 2.0 cc. horse serum per kg. of body weight, about twenty-one days after the final sensitizing dose. The intracystic pressure was recorded by means of a glass catheter (perineal incision, male dogs). The abdomen was opened to avoid errors

[16] Tague, E. L., *J. Am. Chem. Soc.*, 1920, xlii, 173.

[1] Weil, R., *J. Immunol.*, 1923, viii, 233.

from changes in intraabdominal pressure. The following cystic reactions have thus far been recorded:

1. *Typical Anaphylactic Shock.* Arterial blood pressure falls precipitously to about 35 mm. Hg by the end of 45 seconds, gradually decreasing to about 25 mm. Hg by the end of 90 seconds. Recovery usually begins about the twelfth minute, the blood pressure being restored to normal in from 60 to 90 minutes. No recordable change in the intracystic pressure during the first 45 to 75 seconds. The intracystic pressure then gradually increases, usually reaching a maximum of from 25 to 50 mm. Hg by the end of two and a half minutes. Recovery immediately sets in, the intracystic pressure usually being reduced to normal by the seventh minute. No reactions were observed in normal controls.

Assuming that the cystic reactions thus recorded are reliable indices to smooth muscle reactions in other parts of the body, one must conclude that in typical canine anaphylactic shock the smooth muscle structures are not thrown into contraction until the shock is fully established, as determined by the fall in arterial blood pressure. The smooth muscle reactions, therefore, apparently play no rôle in the initiation of the shock. They also apparently have little or no effect on the duration or severity of the shock, as determined by the changes in arterial blood pressure.

2. *Fatal Anaphylaxis.* We have had one example in this series of the rapidly fatal type of canine anaphylaxis previously reported.[2] In this dog, increase in the intracystic pressure began about the 45th second, the pressure reaching a maximum of 30 mm. Hg by the 75th second. The intracystic pressure was maintained at this maximum, practically unchanged till the death of the animal, death taking place in nine and one-half minutes. The thorax of this dog was immediately opened. The lungs remained inflated, were of a rubber-like consistency, and could not be collapsed on moderate pressure. Histological findings will be reported later.

In the infrequent, rapidly fatal type of canine anaphylaxis, therefore, smooth muscle reactions apparently become the dominant factor during the later stages of the shock, and are appar-

[2] Manwaring, W. H., Chilcote, R. C., and Hosepian, V. M., PROC. SOC. EXP. BIOL. AND MED., 1923, XX, 274.

ently the immediate cause of death. Even in this type of shock, however, the smooth muscle reactions apparently play no rôle in initiating the shock, as determined by the fall in arterial blood pressure.

148 (2380)

Convulsions resulting from fluid administration following sucrose injections and water abstinence.

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Convulsions and immediate death were found to result from the administration of fluid to rabbits which had been dehydrated by intravenous injections of sucrose 5 to 10 days previously.

These animals had been used to determine the effect of water abstinence and increased water loss on the excretion of urea and the blood urea concentration. They were kept without food or water and bled 1 to 3 times daily throughout the experiment. Extremely high blood urea concentrations (100 to 450 mg. per 100 cc.) were reached in every animal and fluid ingestion resulted in a very much higher blood urea concentration.

Water to the amount of 200 cc. was administered to a rabbit which had undergone simple water abstinence with negative results. Convulsions were obtained in those rabbits which had received sucrose after the administration of 100 to 200 cc. of distilled water or 0.9 per cent sodium chloride.

The severity of the albuminuria and the height of the blood urea concentration was greatest in the rabbits which had been dehydrated previous to water abstinence by intravenous sucrose injections. Since it was in these only that convulsions were produced it would seem that some damage produced by sucrose was a factor. The cause of these convulsions and their association with kidney damage is being further investigated.