of 10 c.c. of *N* hydrochloric acid.) This is then neutralized with sodium hydroxide and creatinine is determined in the usual manner. When only small amounts of blood are available, 1 c.c. of acid is used and, after evaporation and neutralization, the solution is diluted to 25 c.c. with saturated picric acid and the determination is made by Folin's micro-method. The amount of creatine thus found is about 4 mg. per 100 c.c. of blood. Added creatine is recovered quantitatively, showing that there is no conversion to creatinine in the process of coagulating the protein. No claim is made that the substance reacting with picric acid and sodium hydroxide under these conditions is creatinine derived from the creatine of the blood. However, it is apparently not formed by the condensation of glucose, urea and uric acid. A solution containing 350 mg. glucose, 125 mg. urea and 5 mg. uric acid was evaporated with 10 c.c. of *N* hydrochloric acid. There was an apparent creatine content of 0.1 mg. Smaller amounts of urea and glucose gave no perceptible amounts of chromogenic substance.

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Anaphylaxis in the dog.

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1. Dogs in severe anaphylactic shock have been bled to death, and the blood has been used to transfuse normal dogs. No symptoms of any kind have been produced. Hence the conclusion is drawn that the symptoms of anaphylaxis are not due to the presence of toxic substances in the blood.

2. The liver of sensitized dogs has been perfused with normal blood by means of anastomosis of the portal vein with the carotid of another dog. The blood, as it flows from the inferior cava, clots within a few minutes. If the antigen (horse serum) is injected into the connecting tubing, the blood in the cava soon becomes less coagulable, or quite incoagulable. The conclusion is drawn that the incoagulability of the blood is due, at least in part, to the reaction of the sensitized liver cells to the antigen.

3. Peptone shock has long been known to resemble anaphylactic shock closely, and the inference has generally been drawn that the latter is due to the production and circulation of peptone-like bodies. But the transfusion experiments above described do not bear out this theory. The suggestion is made that these two syndromes coincide for the reason that both alike result from a reaction of the liver. Phosphorus and chloroform, both hepatic poisons, also produce entirely similar changes in the chemistry and coagulability of the blood. Peptone is known to protect sensitized animals against anaphylactic shock. I have found that phosphorus or chloroform poisoning exerts a similar effect. The conclusion is drawn that the liver may be partially exhausted by any of these methods, and will not then react as acutely in anaphylactic shock.

4. Anaphylaxis in dogs is a cellular phenomenon, due chiefly, if not wholly, to the reaction of the sensitized liver cells.

5. It is suggested that the fall of blood pressure in anaphylactic shock may indirectly be due to the liver. That organ is found to be enormously congested, and it is conceivable that the animal is "bled to death" into its own liver. Or, the drop in pressure may be a vaso-motor reflex, comparable to the Goltz phenomenon, initiated by the acute hepatic shock of the reaction.

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The response of the respiratory mechanism to rapid changes in the reaction of the blood.

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For the past year we have been studying the reaction of the blood by two parallel methods: the Fridericia method for alveolar CO_2 and the Van Slyke determination of the carbon dioxide combining power of the blood. We believe that this combination offers a simple method for the study of the state and the reaction of the respiratory mechanism.