Examples of the experimental data are appended:

Defibrinated Blood		Laked Blood	
Spec. Grav.	Viscosity	Spec. Grav.	Viscosity
1.0566	665.74	1.0563	982.35

61 (204)

The determination of ammonia and urea in blood.

By **W. McKIM MARRIOTT** and **C. G. L. WOLF.**

[*From Cornell University Medical College, New York City.*]

Ammonia is determined by distillation in vacuo. 100 c.c. of blood are treated with 50 c.c. of saturated sodium chlorid solution and 250 c.c. of methyl alcohol are added to the mixture. The precipitate formed is finely granular. The residue is filtered off in a filter press, and the filtrate distilled for 40 minutes, with the temperature of the water bath at 40–50°C. The receivers are charged with $n/50$ sulphuric acid, and the acid titrated with $n/50$ sodium hydroxid free from carbonate. Sodium alizarin sulfonate is used as an indicator. The results are perfectly accurate.

The residue after distillation is made acid with hydrochloric acid, evaporated and hydrolyzed with 10 grams of glacial phosphoric acid at 150°C. The ammonia formed from the urea is then distilled into $n/50$ acid. The duplicates have shown very satisfactory agreement, but it is quite certain that not all the urea which is added to a sample of blood is recovered. It is probable that the carbohydrates in the residue combine with the urea at the temperature of hydrolysis and prevent the formation of ammonia.

62 (205)

The resolution of fibrinous exudates, with exhibition of specimens.

By **EUGENE L. OPIE.**

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The purpose of the experiments which are described has been to determine the part played by enzymes in the resolution of a fibrinous exudate. When turpentine is injected into the subcutaneous tissue of the dog, an abscess results, but when an equal

quantity of turpentine is injected into the pleural cavity, there is abundant exudation of coagulable fluid and the serous surfaces are covered by thick layers of fibrin. Accumulation of fluid which can be followed during life by percussion of the animal's chest reaches a maximum at the end of three days, and then gradually subsides so that at the end of six days, in most instances, the cavity contains no fluid. Fibrin, though diminished in amount, is still present, and gradually disappears, so that at the end of two or three weeks, the cavity has returned to the normal, save for a few organized adhesions.

During the early stage of the inflammation, fibrinous exudate, freed from the serum by washing in salt solution, undergoes digestion when suspended in an alkaline (0.2 per cent. sodium carbonate) or in an acid medium (0.2 per cent. acetic acid). At the end of six days, at a time when fluid has disappeared from the pleural cavity, digestion fails to occur in an alkaline medium, but occurs with great activity in the presence of acid.

During the first stage of the inflammatory reaction, when fluid is abundant and the fibrin which is present digests in the presence of alkali, polynuclear leucocytes are very numerous in the meshes of the fibrin. In the second stage, when fluid has in great part disappeared, and the fibrin contains only one enzyme digesting in the presence of acid, polynuclear leucocytes have disappeared and only mononuclear cells are embedded in the fibrin.

Since the acids, which, in vitro, favor the action of the enzyme present in the second stage of the process, do not occur in the body, the possibility has suggested itself that carbon dioxide brings this enzyme into action. If carbon dioxide is passed through normal salt solution in which strips of such fibrin are suspended, digestion is very greatly hastened. The normal inhibition exerted by blood serum upon the enzyme is overcome by carbon dioxide; in the presence of a small quantity of blood serum, carbon dioxide causes greater enzymotic activity than in the presence of salt solution alone.