pellicle enclosing several highly refractive globules. Both the pellicle and the globules are insoluble in water and in cold 95 per cent alcohol.

Chloroform similarly but more rapidly dissolves the plasmalemma. A small drop on touching the plasmalemma quickly passes through it and the endoplasm in its vicinity becomes coagulated.

An interesting phenomenon which possibly is related to the problem of pseudopodial formation occurs when chloroform is allowed to diffuse against the ameba from the tip of the micropipette. A local elevation of the pellicle takes place, into which the granular endoplasm flows. As the pipette is gradually withdrawn the incipient pseudopodium enlarges and extends in the direction of the moving pipette. In this way the entire ameba can be made to move to all appearances in a normal fashion along any path taken by the tip of the pipette.

I wish to thank Dr. Robert Chambers most sincerely for his kind help in this work.

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Influence of Vegetative Nerves Upon Distribution of Arsenic After Salvarsan Injections. II. Pilocarpinized Animals.

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A previous publication[1] gave a comparison between the distribution of arsenic after intravenous injection of arsenicals in normal animals[2] and in animals under the influence of epinephrin. When the involuntary nervous system is under the influence of epinephrin, almost 5 times as much arsenic is retained in the blood stream and only a small part of the arsenic normally distributed to the brain and liver reaches those organs. The general aspect of these tests led to the assumption that epinephrin inhibits the normal distribution of arsenic into the body organs.

In a second series of tests, 2 groups of rats were given subcutaneous injections of pilocarpin previous to the intravenous injections of salvarsan, neosalvarsan and silversalvarsan. 160 mg. of salvarsan per kg. body weight was the dosage used for the normal rats and the pilocarpinized rats were given 80 mg. per kg. body weight. The figures for neosalvarsan and silversalvarsan are similar to those

reported for salvarsan and will be given in detail in a later paper. The rats receiving pilocarpin were divided into two groups. One group received one injection of 5 mg. of pilocarpin 1 hour before the injection of the arsenical while, in the other group, four doses of 5 mg. each of pilocarpin were given 72, 48, 24 and 1 hour before the intravenous injections of the same arsenical preparations. The results for the two groups, as shown in the chart below do not differ greatly. Minor differences will be discussed in a later publication.

TABLE I.

*Average arsenic content of organs after intravenous injection of arsenicals preceded by subcutaneous injection of pilocarpin.**

Interval	Salvarsan alone	Salvarsan preceded by 1 previous pilocarpin	Salvarsan preceded by 4 previous pilocarpins
LIVER.			
Immediate	9.33	70.87	77.82
5 minutes	33.84	87.27	76.89
20 minutes	96.02	86.79	68.11
40 minutes	39.52	83.26	102.73
BLOOD.			
Immediate	14.75	204.19	147.25
5 minutes	42.21	157.72	119.29
20 minutes	13.72	80.18	80.27
40 minutes	13.69	106.93	47.17
BRAIN.			
Immediate	39.12	1.126	0.878
5 minutes	5.51	1.236	1.129
20 minutes	1.90	0.298	0.593
40 minutes	2.66	2.148	1.439

*Arsenic figures are given in mg. per 100 gm. dry specimen. Each figure represents an average of five determinations.

Since the pharmaco-dynamic action of pilocarpin is the reverse of that of epinephrin, one would expect to have found an even greater distribution of arsenic into the organs of pilocarpinized animals than in normal rats. Actually there is a greater retention of arsenic in the blood and the character of the curve for the arsenic content of the blood differs considerably from the normal. Usually, the arsenic content of the blood first decreases due to the rapid distribution and deposition of arsenic into the body organs, and then increases between the fifth and tenth minutes after the injection of salvarsan as the metal is released from the organs where it was first deposited. The arsenic then gradually passes from the blood into

the liver where it is accumulated. These findings indicate a distinct participation of the liver in the process of arsenic distribution.

This mechanism is entirely changed in the pilocarpin tests. The arsenic figures for the liver are higher than normal, but the concentration in the liver is much less than that in the blood. The arsenic content of the liver remains practically constant throughout the experimental period. The tests show that much smaller quantities of arsenic are deposited in other organs such as brain and spleen. Therefore, starting immediately after the injection, comparatively large quantities of arsenic reach the liver and remain deposited in that organ. No increase in the concentration takes place later because no arsenic is released from other organs.

Summary. Studies on arsenic localization and distribution in rats under the influence of pilocarpin show, as do previous tests with epinephrin, how essential a normal state and a normal, undisturbed activity of the vegetative nerves are for the distribution of arsenic therapeutically administered in a mammalian body. They demonstrate that one has to deal, not with a particular state of vascular or capillary permeability, that regulates and controls arsenic distribution, but with a rather complicated mechanism. Every disturbance of the vegetative nerves alters the character of arsenic distribution, regardless of the type of change actually made in the vascular or capillary permeability. This gives evidence of the important part which has to be attributed to the vegetative nervous system for the cooperative synergistic action of the body organs participating in, and responsible for, the distribution of arsenic after the injections of salvarsan.

This is a preliminary report.

[1] Müller, E. F., Myers, C. N., and Marples, E., PROC. SOC. EXP. BIOL. AND MED., 1927, xxiv, 689.

[2] Fordyce, J. A., Rosen, I., and Myers, C. N., *Am. J. Syph.*, 1924, viii, 377.

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Further Observations on Individual Differences of Human Blood.

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In a previous communication[1] we described an agglutinable factor (M), independent of the blood groups and present in many but not in all human bloods. A somewhat higher incidence of M among