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

colored than white individuals, as indicated by our first results, was confirmed by examination on a larger scale; among 902 white individuals 165 (18.3 per cent), and among 338 colored 95 (28.1 per cent) whose blood reacted negatively. A differentiation of bloods (in the same group) lacking or possessing the factor M, was possible also with dried specimens.

The heredity of the property M was studied in more than 100 families. The results are in keeping with the assumption that M is inherited as a mendelian dominant. In the following table the families are arranged in 3 classes according to the presence or absence of the factor M in the parents, and its incidence among the offspring is given.

TABLE I.

Number of families	65		39		3	
Parents	+	+	+	—	—	—
Number of children	252+	25—	114+	52—	0+	11—

It may be mentioned incidentally that the character[2] A^1 present in most bloods of group A, seems to be an inheritable quality. For, in our tests, if the property was not or slightly developed in the blood of both parents, this, as a rule, was also the case for the children.

With a method similar to that employed for the detection of M, namely, suitable absorption of certain rabbit immune sera for human blood, two other agglutinable qualities were found which may be denoted as N and P. These reactions, further distinguishing individual human bloods, vary in intensity from very strong to weak or negative.

The reactions for N seem to be strong in most cases where M is absent. The property M could be demonstrated also in the blood of eight chimpanzees examined, but was not found in the blood of five gibbons.

Strong reactions for P were considerably more frequent with the blood of colored than white individuals; similar results were also obtained with suitably absorbed normal rabbit and horse sera.

[1] Landsteiner, K., and Levine, P., PROC. SOC. EXP. BIOL. AND MED., 1927, xxiv, 600.

[2] For the terminology see Landsteiner, K., and Witt, D. H., *J. of Immunol.*, 1926, xi, 221.