

Contaminated sera cause depressions.

An antimeningococcus serum, which had produced rashes in humans caused well-marked depressions in  $6\frac{1}{2}$  c.c. doses.

Serum of a diphtheria antitoxin horse, which was recovering from an attack of indigestion gave a slight depression. This serum was secured through the courtesy of Dr. Banzhaf.

These observations have extended over a period of  $2\frac{1}{2}$  years.

*Note.*—We have already reported depressions from the injection of the sera of tuberculous rabbits and from the injection of tuberculins as well as from the injection of the sera of animals inoculated subdurally with normal and hydrophobic brain tissue emulsion.

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**A study by the Meyer method of the effect of blood serum and certain inorganic salts on surviving arteries.**

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The method employed is an adaptation of the Meyer ox carotid method. Instead of strips from the carotid, rings from the mesenteric or hepatic arteries of the ox strung in pairs were used, and from the coronary arteries as controls. Adrenalin, even in every dilute solution, constricts the former, while it causes the coronary to dilate, whether it be added to Ringer-Locke fluid, or to the ox blood serum. This method, then, based on the contrary effects produced by adrenalin on two kinds of arteries, each possessing a different reactive property to adrenalin, should be ideal for the detection of adrenalin and the separation of it from the confusion with other substances in the blood serum exerting a constrictor or dilator action. Ox blood serum as opposed to adrenalin produces a constriction of both coronary and mesenteric or hepatic arteries. Thus it essentially differs in its action from adrenalin. There is, then, so far as surviving arteries are concerned, a vasoconstrictor property of ox blood serum, not to be explained by the presence of adrenalin.

The constriction produced by ox blood serum on ox arteries

occurs abruptly after a latent period of only a few seconds and is comparable in its intensity to that produced by adrenalin on the mesenteric or hepatic arteries. The duration is at least four hours, the limit of our means for recording it. Passing oxygen through it weakens this constricting property, as does time, *i. e.*, allowing it to stand one or two days at room temperature. Adrenalin added to blood serum even at the height of a contraction further increases it in the case of hepatic and mesenteric arteries, but produces an especially marked relaxation in the case of the coronary artery. Adrenalin added to fresh ox blood to make a proportion of one to 800,000, we have identified thirty-six hours later; and when added to make a proportion of one to 100,000 after seven hours' oxygenation under a pressure of more than 100 millimeters mercury at incubator temperature.

Sodium chloride in dilution less than .01 produces a marked constriction of the above-named arteries as compared with Ringer-Locke fluid. The latent period is longer than that of adrenalin or ox blood serum, the ascent more gradual; moreover the height of the curve seems to vary inversely to the sodium chloride water ratio to a point .005, below which we have not investigated. At .013 sodium chloride the strips of artery apparently die. The relations of calcium and potassium to tonus have not been taken up yet. Barium chloride produces a vasoconstriction which exceeds that produced by adrenalin, or, so far as our experience goes, any other substance. The curve produced by it tends to be irregular, frequently assuming a staircase character.

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**The influence of the sugar concentration of the blood on the protein metabolism in phlorhizin diabetes.**

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According to Rubner, the protein metabolism of a normal starving animal is composed of two fractions:

I. Wear and tear quota.