vascular tone of the parts. At the height of the reaction, the perfusion rate is identical with the perfusion rate with amyl nitrite. Marked edema of the hind quarters, especially of the genitalia.

2. *Isolated intestines.* Distinct increase in perfusion resistance (vaso-constriction), decreasing the perfusion rate from 15 per cent. to 50 per cent., depending upon the histamine concentration used. Marked peristaltic movements during the first three minutes of the test, followed by edema, peritoneal transudation, and increased volume of intestinal contents.

3. *Isolated liver.* Marked increase in perfusion resistance (vaso-constriction), decreasing the perfusion rate as much as 90 per cent. with large histamine doses (1:25000). Edema, peritoneal transudation.

4. *Isolated lungs.* Marked increase in perfusion resistance (vaso-constriction), decreasing the perfusion rate from 50 per cent. to 75 per cent. depending upon the histamine concentration used. Marked pulmonary edema.

Histological study of these reactions will be reported later.

88 (2048)

The hepatic mechanical factor in canine anaphylactic shock.

By W. H. MANWARING, R. C. CHILCOTE, and SELLING BRILL.

[*From the Laboratory of Experimental Pathology, Stanford University, California.*]

It has been assumed by certain observers[1] that the sudden pronounced fall in arterial blood pressure, the characteristic feature of acute anaphylactic shock in dogs, is due to a reduction in the available systemic blood volume as a result of splanchnic engorgement. This engorgement they believe is a passie congestion due to hepatic obstruction. We have endeav-

[1] Weil, R., and Eggleston, C., *Jour. Immunol.*, 1916, ii, 525. Simons, J. P., *J. A. M. A.*, 1919, lxxiii, 1437.

ored to test this theory by studying the effects on carotid blood pressure of a mechanical obstruction to hepatic outflow sufficient to produce a combined hepatic and intestinal passive congestion equal to the passive congestion observed during anaphylactic shock. We have taken the increase in portal blood pressure as the measure of this passive congestion.

The normal portal blood pressure averages about 9 mm.Hg. in our series of dogs. This pressure is increased to about 18 mm.Hg. during anaphylactic shock, the maximum being reached by the end of one minute. The portal blood pressure then gradually falls, and is restored to normal in from 8 minutes to 15 minutes.

To prepare animals for the mechanical test, the inferior vena cava was ligated immediately below the liver in a series of dogs. Examination of these dogs six weeks later, at the time of the tests showed an hypertrophied collateral circulation fully compensating for the vena caval ligation.

To make the tests, an unclosed ligature was placed about the vena cava immediately below the diaphragm. By partially closing this ligature, any desired degree of hepatic-intestinal passive congestion could be produced without interfering with the return circulation from the hind quarters. It was found that carefully controlled increased resistance to hepatic outflow, sufficient to raise the portal blood pressure to 20 mm.Hg., which is greater than the maximum portal pressure during anaphylactic shock, was without marked effect on the carotid blood pressure. The carotid pressure usually falls slightly on tightening the ligature, but is restored practically to normal by the end of two minutes. It is only when the vena caval ligature is completely closed so as to produce combined hepatic and intestinal stasis, that a fall in carotid pressure is produced at all comparable with the fall during anaphylactic shock.

We conclude from these tests, that hepatic-intestinal passive congestion, though conceivably a factor of some importance in canine anaphylactic shock, is not the essential or dominant factor in this shock. This finding strengthens our initial theory[2], that the anaphylactic reaction in dogs is essentially an explosive hepatic autointoxication, the formation or liberation of hepatic products having a histamine-like reaction on the extra-hepatic blood vessels.

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[2] Manwaring, W. H., *Zeitschr. f. Immunitätsf.*, 1910, viii, 1; *J. A. M. A.*, 1921, lxxii, 849.

ABSTRACTS OF COMMUNICATIONS

Fourth meeting.

Western New York Branch, Clifton Springs N. Y., December 16, 1922.

89 (2049)

Vital capacity determinations in persons with normal heart and lungs above forty years of age.

By D. C. WILSON, (by invitation).

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The first vital capacity readings were taken by Hutchinson in 1846 on 2,000 persons of all ages by means of a spirometer. His subjects were not given a physical examination. He found the vital capacity to vary with the height and weight. He also stated that the vital capacity increased 1 cu. in. yearly up to the age of 35 and after 35 it decreased 1 cu. in. yearly. Since his time the body surface has been found to measure more closely the vital capacity variations. However, all normal persons studied have been babies, students or young adults. No normal readings have been above forty when the surface area is used as the standard for variation.

The present study is an attempt by accurate physical examination, fluoroscopy of the chest and blood studies to secure patients of all ages who have no cardiac or lung disease. Next to obtain by three separate readings their true vital capacity. Then to compare this by means of a Du Bois chart with the so-called normal for their body surface area. Eighty-five such cases are reported and the results given.

Except for women overweight and between 45 and 55 years of age, the surface area reading is within 500 c.c. of the reading obtained unless there is some cardiac or lung disease. This rule does not hold above the age of 70 when there is great individual variation. There is no such regular decrease in vital capacity after 35 as Hutchinson mentions.