of the virus in the host, or to the combination of both factors. When a positive fixation reaction occurred, it has persisted to the time limit of experimentation, in one instance to 21 months after onset of illness.

The animal test for protective antibodies as a means of diagnosis is expensive and not without danger to the worker. If the complement-fixation reaction is developed so as to give a high proportion of reliable results without false positives, it appears essential to apply it not before several weeks after the suspected illness.

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The Cardiac Output in Hyperventilation.*

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Henderson[1] described the depression of blood pressure in acapnia, advanced the hypothesis of a venopressor mechanism, and still believes that the decrease of the venous return to the right heart is a principal factor in the production of this effect.[2] Dale and Evans[3] showed by cardiometer experiments that the cardiac output is not significantly altered in hyperventilation, and thought the effect due to depression of the vasomotor centres of the bulb and spinal cord. Voluntary over-breathing in man results inconstantly in a reduction of the blood pressure; and McDowell[4] reported that in some dogs anesthetized with chloralose no fall, or even a rise, of blood pressure occurred with acapnia, while in all animals anesthetized with ether a fall occurred. These observations were explained by the hypothesis that acapnia has a dual effect on the circulation, (a) vasodilatation due to a central action, and (b) constriction of smaller and more peripheral vessels due to a local action.

In studying the effects of acapnia, it was desired to measure by the Fick method the cardiac output before and during hyperventila-

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[1] Henderson, Y., *Proc. Am. Physiol. Soc.*, Dec., 1905.

[2] Henderson, Y., *J. Am. Med. Assn.*, 1930, **95**, 572.

[3] Dale, H. H., and Evans, C. L., *J. Physiol.*, 1922, **56**, 125.

[4] McDowell, R. J. S., *J. Physiol.*, 1930, **70**, 301.

tion, and to compare the effect on the output with the effect of partial obstruction of the venous return to the heart.

Dogs of 5 to 8 kgm. body weight, anesthetized with sodium-barbital, were used in all experiments. Ventilation of increased rate and volume was obtained by alternate positive and negative air pressure applied to the entire body except the head, in the manner of the Drinker respirator. A cylindrical metal chamber, 35 cm. x 15 cm. in diam., was used; its open end was closed by a thick rubber cuff, sealed about the dog's neck by several layers of adhesive tape. The chamber was connected by a side-arm to 2 Palmer respiration pumps, operated synchronously by an electric motor, at 115 or 160 r.p.m. Oxygen consumption was measured by a Benedict type spirometer. The spirometer could not freely follow rapid respiratory movements; hence a breathing chamber with one wall a rubber diaphragm was devised to take up the oscillatory movement, while the position of the float continued to indicate the oxygen consumption. The chamber used was a metal cylinder, 19 x 22.5 cm. diam., connected directly to the tracheal cannula, and containing soda-lime on its floor and in a gauze bag suspended over the tracheal connection. Air was continuously circulated through the whole apparatus by a motor blower. Samples of the mixed venous blood were obtained from the right heart by means of a metal cannula 20 cm. long, passed down the right jugular vein; samples of arterial blood were collected through a T-cannula in the right carotid artery. The arterial blood pressure was recorded by a manometer connected to a cannula in the left carotid artery. Sufficient heparin (Howell) was given intravenously to render the blood incoagulable. Calculations of the cardiac minute volume were by the Fick formula, the arterio-venous oxygen difference being estimated by means of a Van Slyke manometric blood-gas machine.

It was found that in hypocapnia the blood pressure fell proportionately more than the cardiac output. In 3 dogs, the cardiac minute volume remained at 90 to 110%, while the blood pressure fell to levels 72.5 to 83% of the normal. This contrasts with the effect of obstruction to the venous return by ligation of various large veins, when the output of the heart is diminished proportionately more than the blood pressure; in one dog the cardiac output fell 78.5% while the blood pressure remained at the normal level, and was 41% at a blood pressure level 67% of the normal.

These findings confirm the opinion that the fall in blood pressure in hyperventilation occurs without much depression of the circulation, and is probably due to an arteriomotor relaxation.