stops the heart beat. With low concentrations of chloroform, a temporary cardiac standstill is produced. 5. Chicken embryos, treated with chloroform in a dosage which causes a temporary cardiac standstill react as normal embryos to the addition of effective serum, *i. e.,* they show typical vascular phenomenon. 6. The phenomenon cannot be produced in embryos whose heart beat is stopped by exposure to ice box temperature.

7836

Pulmonary Blood Velocity in Congestive Heart Failure. Velocity in Pulmonary Venous Circuit.

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Our observations demonstrate that there are changes chiefly, and sometimes solely, in the velocity of the pulmonary venous circulation clearly related to congestive heart failure. This velocity was estimated by utilizing 2 methods for studying pulmonary blood flow (1) the ether time[1] which serves as a measure of the rate of blood flow in the pulmonary arterial system and (2) the taste or saccharine time[2] which serves as a measure of the rate of blood flow through the combined arterial and venous circuit, the so-called "crude pulmonary circulation". By subtracting (1) from (2) we obtained readings of velocity in the pulmonary venous system. The normal average ether time is 5 to 9 seconds, the taste or saccharine time is 14 to 16 seconds, and the average velocity in the venous circuit is 6 to 9 seconds, practically identical with that in the arterial circuit.

Figures within normal limits were observed in 100 hospital individuals from various diseases, the greatest number of whom were free of cardiac disease and the rest recovered from congestive heart failure. On the other hand, in 30 hospital individuals with cardiac disease and in various stages of congestive failure lasting over many months, there were always prolonged saccharine time readings but the ether figures were within normal limits; the arithmetical

[1] Hitzig, W. M., PROC. SOC. EXP. BIOL. AND MED., 1934, **31**, 935; Miller, H. R., *Ibid.*, 1934, **31**, 842.

[2] Fishberg, A. M., Hitzig, W. M., King, F. H., PROC. SOC. EXP. BIOL. AND MED., 1933, **30**, 651

difference between the saccharine and ether readings was always greater than normal, in some instances between 36 and 52 seconds as against normal of 6 to 9 seconds. This evidence of marked retardation in the pulmonary venous channels was noted promptly upon the initiation of congestive failure and the retardation persisted until not only visible external signs but residual central pulmonary congestion disappeared completely. The sluggish blood flow in the venous limb of the pulmonary circulation in a number of instances was accompanied by ether figures which were also increased pointing to some degree of slowing in the arterial limb as well. These figures, however, of the arterial velocity seldom rose (from the normal 6 to 9 seconds) above 11 or 12 seconds and in very exceptional cases to 15 to 19 seconds. Such high ether readings were found in chronic cardiac diseases whether congestive failure was present or absent and led us to believe that disturbances in pulmonary arterial velocity were not the responsible factor. Instead we considered the possibility that alteration in the capillary and alveolar walls led to interference in the diffusion of gases. This problem is receiving further study.

Three further points belong in this report. (1) As a general rule, the extent and degree of congestive failure went hand in hand with the slowing of the pulmonary venous velocity and improvement in both was usually parallel; (2) Although the retardation of blood flow in the arterial channels was absolutely increased in some individuals with congestive failure, from a practical point of view this increase contributed only a minor fractional part to the slowing of the pulmonary circulation as a whole. (3) In the presence of myxedema following thyroidectomy for rheumatic heart disease, blood flow appeared to be markedly held up in the arterial and venous limbs despite marked amelioration from congestive failure.