for total proteins. Globulins are calculated as (total protein)—(albumin).

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The Cause of Temporary Ventricular Alternation Following a Long Diastolic Pause.*

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A condition of temporary alternation, such as can be induced in dogs' hearts which are normal, according to every criterion we are able to apply, was studied by apparatus recording pressure and volume curves by optical projection.

It was found that temporary alternation induced by the application of premature stimuli to a ventricle or an auricle, by temporary vagus inhibition or by a temporary a-v block, invariably involves both ventricles but does not affect the auricles. Neither the permanence nor duration of the alternation is affected by the volume of venous return or the height of the arterial pressure. Alternation appears to be conditioned entirely by the rate of beat, and cannot be induced in the normal dog's heart when beating at rates below a critical level of about 140 per minute.

The alternating beats resemble those of a more permanent nature induced in other ways. In both cases they differ from the normal, not only in their amplitude of contraction and volume of systolic discharge, but also in the contour of contraction and relaxation and in the duration of their phases. Thus, in the smaller beats the pressure rises more gradually, the isometric contraction phase is prolonged, while systolic ejection and total systole are abbreviated. The isometric relaxation also occurs more slowly so that the interval between the end of systole and the beginning of ventricular inflow is prolonged.

The alternate beats begin with different diastolic volumes and as a

* A detailed report of this research will appear in the Warthin Anniversary Volume, George Wahr, Ann Arbor, 1927.

rule also with corresponding changes in initial tension. Thus, just before the smaller contraction begins the diastolic size is less and the initial tension is lower. The reverse obtains in the case of the larger beat.

A critical study of the possible causes of such variations in filling leads to the conclusion that this is essentially due to the alternating extent of ventricular emptying. Thus, the residual volume retained after a small beat added to the natural inflow results in a larger diastolic volume and higher initial tension, in spite of the slight impediment offered to inflow. Changes in cycle length are obviously not concerned. Alternate changes in filling pressure or concordant variations in amplitude of auricular contraction do not occur. Nor do changes in ventricular relaxation play any rôle. Isometric relaxation, it is true proceeds more slowly after the smaller beats but this is so nicely neutralized by an earlier onset of relaxation that the duration of inflow changes not at all or is affected very slightly. Furthermore, the rate of inflow is slightly impeded after the smaller and not after the larger beat.

Recent observations have shown that such alternate changes in diastolic size and initial tension inaugurated by the longer pause can cause the amplitude, contour and duration of ventricular contractions to change in the same directions as in this form of alternation. It is, therefore, possible to attribute all the effects shown in such temporary alternation to changes in filling. More convincing evidence than we have at present is necessary before we can assume that any specific alteration in the function of ventricular muscle is necessary to produce this type of alternation.

Attention is directed to the fact that this interpretation of the alternation phenomenon may not be applied without further study to those clinical forms which arise under different circumstances.