69 (1651)

The influence of venous return and arterial resistance on the pressures within the right and left ventricles.

By CARL J. WIGGERS.

[*From the Physiological Laboratory, Western Reserve University Medical School, Cleveland, Ohio.*]

I. ARGUMENT.

The question, is the response of the ventricle, under conditions of equal irritability, fundamentally determined by the initial length of its muscle fibers or by the initial tension exerted upon them, is of general physiological importance. The related question, can variations in initial volume (*i.e.*, initial length) occur independently of changes in initial tension, in the mammalian heart, is of far-reaching clinical interest as well. As regards the second question, the experimental results of Frank[1] and those of Straub[2] supply an answer which is contradictory to that of Patterson, Piper and Starling[3]. The latter investigators believe to have demonstrated that initial length alone determines the magnitude of the cardiac response, irrespective of whether initial tension is simultaneously altered in the same or reverse direction. Gesell[4] holds that both factors may be concerned but seems inclined to believe that changes in initial length play their important rôle when ventricular filling is relatively small.

While the fact can not be denied that the bulk of evidence apparently points to the conclusion that initial length fundamentally determines the magnitude of contraction in skeletal muscles, it is not so clear how such changes can *promptly* adjust the work of the heart to sudden changes in venous inflow or arterial resistance—except, in so far as these length changes are primarily due to changes in initial tension. This thought is suggested by the following premises: The diastolic volumes of the ventricles

[1] Frank, *Ztschr. f. Biol.*, 1895, xxxii, 370.

[2] Straub, *Deut. Arch. f. klin. Med.*, 1914, cxv, 531; 1914, cxvi, 409.

[3] Patterson, Piper and Starling, *Jour. Physiol.*, 1914, xlviii, 465.

[4] Gesell, *Am. Jour. Physiol.*, 1916, xxxix, 239; 1916, xl, 267.

can be increased beyond their normal capacity either (*a*) by an increased initial pressure overcoming the inherent tendency of the ventricles to resist stretching, or (*b*) by a reduction of this inherent power of the ventricle to resist stretching, *i.e.*, by a reduction of tonus.[1]

According to the hypothesis of Patterson, Piper and Starling it would be necessary to assume that an augmented venous return, for example, causes a prompt reduction of tonus. Our entire experience teaches us, however, that the degree of tonus in a muscle of any type is not capable of being rapidly changed; on the contrary, such changes occur very slowly. The results of Patterson, Piper and Starling, corroborated in my own work, indicate, however, so prompt a response on the part of the ventricle, as to make this almost presumptive evidence against the view that tonus changes are primarily or chiefly concerned. To supply quite certain proof that such changes are, on the other hand, associated with and probably due to simultaneous changes in initial tension requires a careful study of the pressure changes in the right and left ventricles during the early response of the right heart to changes in venous inflow and arterial resistance.

II. EXPERIMENTAL RESULTS.

The venous inflow into the right heart of intact animals was increased by allowing a graded inflow of normal saline into the jugular vein. Increased arterial resistance was produced, in experiments here reported, by partial compression of the thoracic aorta. Records of right and left intraventricular pressures were synchronously recorded by optical manometers. The results of 17 such experiments may be briefly summarized:

1. *Effects of Saline Infusion.*—Beginning with the very first beat when the initial (diastolic) volume of the heart increases, after such saline infusion has started, the initial tension in the

[1] To prevent misunderstanding, it should be noted that I define the term "tonus" as that partial state of contraction which persists during diastolic rest, and by virtue of which muscle resists stretching. The statement of Patterson, Piper and Starling, that tonus is "synonymous with the physiological condition or fitness of the muscle and its measure is the energy set free per unit length of muscle fiber at each contraction of the heart," I believe, expresses the end effects of tonus changes but does not describe the nature of the tonus phenomenon itself.

right ventricle is at once elevated. As the heart continues to dilate during diastole, this elevation of pressure increases more and more. After the second beat the pressure-maximum is also increased in the right ventricle. Two to three beats are usually required before the initial and maximum pressures in the left ventricle are similarly altered. Systolic discharge and the intraventricular pressure-maximum continue to increase only so long as initial tension also continues to increase. In these cases, increased diastolic distention is, therefore, never dissociated from increased initial tension. Tonus changes may simultaneously operate to lengthen the muscle fibers independent of initial tension but, if so, their effects are entirely obscured. If this condition of increased inflow persists in a stationary manner, for a matter of 15 to 20 minutes, however, it may happen that then the heart dilates further, even while the initial pressures in the right and left ventricles decline. Such a dilatation, evidently due to a decrease in tonus, is always accompanied by a reduction in systolic discharge and in the pressure-maximum in both ventricles.

2. If arterial resistance is suddenly elevated during partial compression of the thoracic aorta, the systolic discharge is decreased for a few beats (usually 2–3) resulting, as also shown in Patterson, Piper and Starling's results, in a diastolic distention and increased initial length. Systolic discharge returns to normal about the fourth or fifth beat. Careful study shows that the pressure-maximum is elevated at once in the left ventricle, and by the third beat the initial pressure is also measurably although but slightly increased. At the fourth or fifth beat where the systolic discharge returns to normal there is a significant increase in the initial pressure in the left ventricle also. Then, for the first time, initial tension and pressure-maximum in the right ventricle also increase.

These results favor the conclusions, (1) that initial tension changes are apparently always associated with changes in initial length resulting from alterations in venous inflow or arterial resistance; (2) that in the intact animal, changes in initial tension play the predominant rôle in determining the response of the mammalian ventricle.