ENERGY METABOLISM OF THE FAILING HEART

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Starling (1915) showed that the normal heart dilates only when increased work is thrown upon it. In disease the heart dilates without any increase in work imposed upon it. It is obviously of importance to know in what particulars the failing heart differs from the normal. The experiments recorded here amplify the results of Starling and Visscher (1927) in elucidating this problem. In their experiments it was found that the energy liberation in the normal heart is fixed by the size of the heart at the beginning of contraction; i.e., at the end of diastole. The size of the heart is a measure of the length of the muscle fibers; consequently their results may be expressed by saying that the quantity of energy liberated in contraction is determined by the length of the fiber at the time of contraction.

As a matter of fact, we are more fundamentally interested in the amount of work the heart can do than we are in the total energy it can liberate. Therefore the proportion of the total energy which can be put to useful work, which is the efficiency, becomes a matter of first importance. In cardiac decompensation in man, as well as under certain conditions in experiments on hearts of lower animals, the heart dilates progressively in spite of a uniform or even decreasing load. One ordinarily speaks of such a heart as having poor tone. The heart of a decompensated patient does little work in spite of its enormous size and its apparent effort. The question as to whether the heart under these circumstances is defective in liberating less energy than a normal heart would, or is defective in being unable to use the energy for work in ejecting blood, was investigated by Starling and Visscher (1927). They found that when the mammalian heart lost its normal tonus, and dilated enormously in order to do amounts of work that when fresh it could do at small volume, the total energy
liberation was not at all diminished; only the proportion of the total energy which could be converted into work was diminished. That is to say, the efficiency of the heart muscle as a machine was found to fall as the condition of the heart deteriorated. There was no lessening in the quantity of energy made available.

It has been necessary to reinvestigate these problems because Stella (1931) questioned the reliability of the earlier results. Decherd and Visscher (1933), and Moldavsky and Visscher (1933, 1934), have indicated the errors which led Stella to fallacious conclusions. Certain observations upon failing hearts are described here because of their importance to an understanding of the physiology of decompensation.

In these experiments the total energy liberated in contraction was calculated from measurements of the oxygen consumption. The latter was measured by a modified Barcroft differential manometer method, observing the decrease in volume of oxygen in the vessels containing the turtle heart and the perfusion fluid, while the carbon dioxide was being removed from the air by potassium hydroxide present in a porous plate suspended in the vessel. A compensating vessel of equal air volume automatically corrected for temperature changes, which were within 0.005°C., since the bath itself was controlled within that limit. The relatively large volume of oxygen in the system made such accuracy in temperature control necessary. The energy converted to work was calculated from the pressure and the output. The pressure was measured with a membrane manometer and the output was observed by employing the closed chamber around the heart as a cardiometer, recording changes in the volume of air within it by means of a properly calibrated tambour. A more detailed description of the apparatus and methods used has been published by the authors (1933) in a previous paper.

A heart perfused with Ringer's solution for a long time spontaneously dilates and fails to eject as much blood as enters between contractions. It can be therefore said that when the heart begins to fail the condition becomes evident in an inability to keep up its output per beat at a given diastolic volume. In Fig. 1 are shown the results of a typical experiment in which the volume in diastole was kept constant while the heart was failing. More than twenty-five
concordant experiments have been performed. It can be seen that
the work the heart was able to do falls off very rapidly. It is equally
obvious that the energy liberated per beat does not diminish at all
during the time of observation. This has been the invariable result
of many observations. It appears, therefore, that in the decompensa-
ting heart the defect is not in a failure of the heart to liberate enough
ergy to do its work. It can still liberate as much energy as it ever
could. Its defect lies rather in an inability to employ the energy for
useful work. This is entirely aside from any valvular defects which,

![Graph](image)

**Fig. 1.** In this experiment the ventricle was held at constant diastolic volume
over several hours. The energy liberation remains constant, but the work done
falls off markedly as the condition of the isolated heart deteriorates.

of course, also diminish the efficiency of the heart. The machine
which does mechanical work with the energy of contraction is out of
order. The extent of this defect is measured by the efficiency of the
heart. In Fig. 2 are plotted the results of two experiments in which
the efficiency of the failing heart was measured for several hours during
which its condition was becoming steadily worse. When this im-
pairment occurs the ventricles are obliged to dilate to a greater diast-
tolic fiber length in order to do the work imposed upon them. Thus it
is apparent that the dilated heart, which one describes as having poor
tone, is in reality simply one with low mechanical efficiency.
These observations have many implications for clinical medicine. It is at once apparent that the failing heart is not in need of fuel, but rather of materials for repairs. Visscher and Müller (1927) and Bodo (1928) investigated certain agents which improved the condition of the isolated heart and found that insulin was conspicuous among all substances employed in restoring the tone or, in other words, efficiency, of isolated working hearts. Further experimentation along this line is in progress in this laboratory.

![Graph showing mechanical efficiency over time.](https://jem.rupress.org/)

**FIG. 2.** The results of calculations of efficiency in two experiments are plotted in this figure. It will be noted that the mechanical efficiency gradually falls off over the period of several hours' observations. The diastolic volume was held constant throughout.

There are also agents which diminish the efficiency of heart muscle. Starling and Visscher found that adrenalin, although it caused a great increase in total energy liberation, produced a marked lowering of efficiency, and therefore left the heart in worse condition than it had been before. Although temporarily stimulating to energy liberation by the heart, adrenalin eventually leaves the heart muscle less efficient and must therefore frequently be very harmful to a weakened myocardium.
SUMMARY

The failing heart is found to liberate energy in contraction to the same extent at any fiber length as the normal heart muscle, but it is unable to convert as much of the energy into useful work. Its efficiency as a machine is impaired. In the light of these observations the word “tonus” as applied to heart muscle becomes synonymous with “efficiency.” The implications of these findings in applied cardiology are discussed.

BIBLIOGRAPHY

Bodo, R., J. Physiol., 1928, 64, 365.
Stella, G., J. Physiol., 1931, 72, 247.