As early as 1915 it was known that an increase in systolic pressure is accompanied by a greater myocardial oxygen cost than a proportionate increase in stroke volume. Recent investigators have related myocardial oxygen consumption to the product of the mean systolic pressure and the heart rate, or to the integral of systolic pressure in time. In the isovolumetrically contracting ventricle, myocardial oxygen consumption could be equally well correlated with peak developed pressure, the integral of systolic pressure in time, or the tension developed by the myocardial fibers, as approximated by the Laplace relationship between wall tension and volume of a sphere.

Inasmuch as intraventricular pressure is the predominant hemodynamic factor in the oxygen cost of the heart's contraction, it was decided to investigate this further by repeatedly releasing the intraventricular pressure at specific points in the pressure cycle and comparing the amount of oxygen used by the ventricle when the pressure cycle was so interrupted to that used by the ventricle with a full pressure cycle. In view of the difficulties in measuring directly the tension developed by the myocardial fibers it was assumed that at a constant end diastolic pressure, volume, and loading, systolic intraventricular pressure would be a reasonable reflection of the tension developed by these fibers.

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through the aortic cannula. Left coronary flow was measured by the rotameter. Blood was withdrawn from the pulmonary cannula under slight negative pressure and returned to the donor dog through a second reservoir and filter.

The left ventricle was electrically stimulated to compress air, as described below and as shown schematically in figure 2. The free end of the mitral cannula was attached to a Krogh spirometer which, in turn, was enclosed in a 100 liter air chamber maintained at the desired diastolic pressure. The heart was immersed to mid position in a saline bath (not shown) which was kept at approximately 37°C. An electronic pulse generator provided the stimulus to initiate contractions. The heart was distended to the desired diastolic pressure through a solenoid valve that was timed to open repeatedly after the stimulus. Both the delay between the stimulus and the opening of the solenoid valve, as well as the length of time the valve remained open, could be adjusted.

With this preparation the left ventricle could be allowed to fill at a desired ventricular end diastolic pressure, and then to develop a full systolic contraction, compressing the air within the ventricular cavity and the tubing below the solenoid valve (Fig. 2). In this circumstance the valve was timed to remain closed in systole and to open, with each stimulus, during a portion of diastole. By advancing the time of opening of the valve into systole the ventricle could be decompressed rapidly, reducing the intraventricular pressure to the diastolic level. By use of the delay circuit this could be made to occur at any time between the start of the development of pressure and the return of the pressure wave to the diastolic level.

Intraventricular pressure was recorded continuously through a pressure transducer* connected to the ventricular cavity by a short piece of rigid tubing. The Krogh spirometer was mechanically connected to a linear differential transducer† so that volume changes could be monitored through a carrier preamplifier. All inputs were recorded on a Sanborn polyviso recorder.

Blood samples from the aorta and coronary sinus were obtained through catheters. These were analyzed for oxygen content and saturation by the method of Van Slyke and Neill. Myocardial oxygen consumption was calculated as the product of coronary flow and the difference in oxygen content of the inflowing and outflowing blood. During metabolic studies, at least two minutes were allowed for equilibrium prior to blood sampling.

Results

With this preparation it was possible to decompress the ventricle rapidly and repeatedly at any point during its pressure cycle and concurrently to determine left ventricular oxygen consumption. Throughout each experiment heart rate and ventricular end diastolic pressure were maintained constant.

In the experiment illustrated by figure 3, left ventricular oxygen consumption is plotted against the integral of pressure in time as determined by the area under the pressure curve above the diastolic level. In this study, left ventricular oxygen consumption was determined four times: first, when the heart contracted isobarically, the solenoid valve remaining open; second, when the ventricle was decompressed during the ascending portion of its pressure wave; third, when the ventricle was decompressed during the descending portion of its pressure wave; and last, when a normal pressure cycle was allowed without decompression. From figure 3, it can be seen that there was only a small difference in left ventricular oxygen consumption between the last two determinations which is within the error of the method.

* Sanborn 267B.
† Sanborn 575DT250.
Figure 3

Plot of left ventricular VO_2 in ml/min against the integral of systolic pressure in time (arbitrary units) in one experiment. Pressure tracings above correspond to four points plotted below.

The left and middle panels of figure 4 are composites of 24 similar experiments performed on nine hearts. In both panels left ventricular oxygen consumption is again plotted on the ordinate while the area under the pressure wave, above the diastolic level, is plotted on the abscissa, in arbitrary units. In both panels, each experiment is represented by a line connecting two or more points. In the left hand panel are shown experiments in which left ventricular oxygen consumption was determined when the ventricle was decompressed during systole before the development of peak systolic pressure. Under these circumstances there appeared to be a good correlation between the oxygen consumed by the ventricle and the area under the pressure wave (P < 0.001). In the middle panel are shown experiments in which left ventricular oxygen consumption was determined when the ventricle was decompressed after the development of peak systolic pressure. In this instance there was no demonstrable correlation between left ventricular oxygen consumption and the integral of pressure in time (0.20 < P < 0.30).

It appeared, therefore, that a consistent relationship between the area under the pressure wave and left ventricular oxygen consumption applied only when the ventricle was decompressed while it was developing pressure. If the ventricle was decompressed during relaxation there appeared to be no appreciable relationship between the area under its pressure wave and the oxygen it consumed. From the right hand panel of figure 4 it can be seen in addition that a good correlation between peak systolic pressure and left ventricular oxygen consumption could be obtained when the ventricle was decompressed while it was developing pressure (P < 0.001).

In all experiments sudden decompression of the ventricle at the peak of the pressure pulse was followed by a slight increase in the
peak pressure of subsequent contractions. This finding, which appeared regardless of the heart rate, is illustrated by the four panels of figure 5 which show diastolic volume (panel 1), intraventricular pressure (panel 2), diastolic volume, continued (panel 3), and intraventricular pressure, continued (panel 4). In the left hand portion of the second panel of figure 5 is seen a pressure tracing in which the ventricle was allowed to develop a full pressure pulse. Then, by advancing the time of the opening of the solenoid valve, the ventricle was decompressed at the peak of the pressure pulse. This is seen in the right hand portion of the second panel in figure 5, where a slight increase in the peak systolic pressure developed by the ventricle can be noted. After readjusting the solenoid valve to its original timing, as seen on the right of the fourth panel of figure 5, which is a continuation of the second panel, the height of the pressure pulse fell to control levels when the ventricle was no longer decompressed at peak pressure.

This finding was noted in all experiments in which the ventricle was decompressed at the peak of its pressure wave. Quantitatively the augmentation was approximately 10%. If, however, the decompression was timed to occur after the peak, the augmentation was less. As can be seen in figure 5, heart rate, ventricular end diastolic pressure, and diastolic volume remained unchanged in these experiments.

By allowing the ventricle to compress air into a slightly larger chamber, while decompressing the ventricle at the peak of its pressure wave, it was possible to achieve the same peak pressure when the ventricle developed a full pressure pulse and also when the ventri-
cle was decompressed at peak pressure, while
ventricular end diastolic pressure, diastolic
volume, and heart rate remained constant.
Under this circumstance, determinations of
left ventricular oxygen consumption were
again made as described under Methods. The
upper half of figure 6 shows the pressure
tracing in one experiment in which the ven-
tricle developed a peak pressure of 158 mm Hg
both when decompressed at its peak and
also when developing a full pressure pulse.
As can be seen from this figure, the amount
of oxygen consumed by the ventricle when
it was decompressed at peak pressure was
10.40 cc/min compared to 11.48 cc/min when
the pulse was full. In 15 additional experi-
ments the oxygen consumed by the ventricle
when it was decompressed at its peak aver-
gaged 91% of that when a full pressure pulse
was allowed.

Discussion
When the ventricle was decompressed at
various points during the development of pres-
sure, left ventricular oxygen consumption
could be correlated with the pressure at which
the ventricle was decompressed and also the
area under the pressure wave, above the dias-
tolic level. This is consistent with the findings
of others who have been able to show a cor-
relation between myocardial oxygen consump-
tion and pressure.

A correlation between left ventricular oxy-
gen consumption and the area under the pres-
sure wave could not be shown if the ven-
tricle was decompressed after the development
of peak pressure. In view of the fact that the
peak pressure developed by the ventricle was
on the average 10% higher when the heart
was decompressed at its peak compared to
that when the ventricle was allowed to de-
develop a full pressure wave, one might argue that any comparison of oxygen consumption under the two circumstances is invalid.

For this reason studies were performed in which the oxygen consumed by the ventricle, when decompressed at its peak, was compared to that when the pressure pulse was full and when, in both situations peak systolic pressure, ventricular end diastolic pressure, as well as heart rate, were comparable. It was then found that there was, on the average, less than a 10% difference in the oxygen consumed by the ventricle in each of the above circumstances. It appears, therefore, that by the time the pressure pulse has reached its peak, myocardial oxygen consumption has been largely determined and the oxygen cost of ventricular relaxation is less than one-tenth of that incurred during the development of pressure.

The cause of the augmentation that accompanies decompression of the ventricle at peak pressure is obscure. A similar phenomenon has been noted by those studying isometric contractions in isolated strips of striated muscle and has been attributed to a small redistribution of the length of the contractile elements in such a manner as to augment the total force of contraction of the muscle strip. Although the studies of striated muscle are far from being completely analogous it would seem possible that a succession of quick releases might predispose a similar small redistribution of length which at a constant loading would manifest itself by a more forceful contraction. It is also tempting to note that both the oxygen consumed during ventricular

![Diagram of pressure tracing and oxygen consumption](image-url)

**FIGURE 6**

Upper portion. Pressure tracing of a ventricle developing a full pressure pulse (left) and the pressure tracing of the same ventricle when decompressed at its peak after peak pressure has been reduced to a comparable level by altered loading (right). Attending left ventricular VO₂ is shown. Lower portion. Schematic representation of the average left ventricular VO₂ in 16 experiments similar to the above.
relaxation and the augmentation that accompanies decompression of the ventricle at its peak were each changes of approximately 10%. One could postulate from this that when the ventricle was suddenly decompressed, its contractile mechanism was left with an excess of chemical energy or activator which was subsequently directed into the development of greater pressure.

**Summary**

Determinations of left ventricular oxygen consumption were made in the isolated heart of a dog while the ventricle performed work by compressing air. In the described preparation the air compressed by the ventricle could be rapidly released and the ventricular pressure pulse repeatedly interrupted at specific points during its cycle. Upon interrupting the pressure pulse at its peak there was a 10% increase in peak pressure that appeared in the subsequent four to five beats. When the intraventricular pressure was released at various points during its ascent to peak pressure, left ventricular oxygen consumption could be correlated with both the pressure, at which it was interrupted, and the area under the pressure pulse. When the oxygen consumed by a ventricle developing a full pressure pulse was compared to that of a ventricle in which the intraventricular pressure was released at its peak—and when the peak systolic pressure of the two compared pulses were equal—then the oxygen consumed by the ventricle when the pulse was interrupted averaged 91% of that when the pulse was full. These studies indicate that by the time the pressure pulse has reached its peak, myocardial oxygen consumption has been largely determined, and that the oxygen cost of the ventricular relaxation is small.

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**References**


