Oxygen Uptake of the Nonworking Left Ventricle

By W. P. McKeever, M.D., D. E. Gregg, Ph.D., M.D. and P. C. Canney, M.D.

Comparisons have been made in the open chest dog of the oxygen usage of the left ventricle whose external work has been reduced to zero by 4 different procedures. The average oxygen usage of the left myocardium/100 Gm./min. is about 2.0 cc. in complete arrest with vagal stimulation or with intra coronary potassium injection; 3.8 cc. in fibrillation; and 3.4 cc. in the empty but beating heart. The oxygen value in the arrested heart approximates 27 per cent of that in the control state and the uptake during vagal stoppage varies directly with the control metabolic level. In the fibrillating ventricle the higher values are related to the intensity of fibrillatory movements, and in the empty beating heart to the control metabolic rate and to the frequency of the heart beat. The experiments with vagal arrest also permit estimation of the oxygen debt of the myocardium.

In the presence of a normal heart rate, cardiac index, and systemic blood pressure the oxygen uptake/100 Gm. left ventricle is 8 to 10 cc./min. However, the oxygen consumption of the mammalian myocardium performing no external work is not well documented. Initial information was obtained in 1906 by Barcroft and Dixon, who found values ranging from 0.6 to 4.0 cc. and averaging 1.8 cc./100 Gm. heart/min. in isolated puppy hearts with perfused coronary arteries. Subsequent studies have yielded a similar range of values in the Langendorf preparation, but a higher average value of about 4.0 cc./100 Gm. heart/min. in the open chest dog. Average oxygen values of 3.8 to 4.3 cc./100 Gm. heart/min. have been obtained during ventricular fibrillation in the isolated heart and in the open chest dog. Values of 2.1 to 2.7 cc./100 Gm. heart/min. are reported in complete cardiac arrest for the isolated rabbit heart perfused with a calcium-potassium deficient perfusate.

Measurement of the oxygen usage of the nonworking myocardium requires that its work be reduced to and held at zero for a period sufficiently long to permit metabolism to reach a steady state. Furthermore, the oxygen uptake must be measured continuously during these changing states, since it is impossible, employing random samples, to ascertain the attainment of a steady metabolic level. The present study has been concerned with the development of methods to bring cardiac work to zero and back to control levels while measuring oxygen uptake continuously. Comparisons have been made of the oxygen uptake per unit weight of the myocardium whose external work has been lowered to zero by 4 different means.

**METHODS**

Mongrel dogs weighing from 10 to 15 Kg. were used. Anesthesia was induced by intravenous pentobarbital and supplemented thereafter to maintain absence of spontaneous respiratory efforts. Heparin, 10 mg./Kg., was administered intravenously prior to the first blood vessel cannulation. Thereafter 5 mg./Kg. was given at one-half hour intervals. Artificial respiration was maintained through an endotracheal tube with oxygen, using a demand-type valve apparatus (Pneophore) or with room air, using a Phipps and Bird respirator. The vagus nerves were gently isolated in the neck and sectioned. In some cases the peripheral ends were pulled down into the thorax to provide long segments of nerves for...
VENTRICULAR OXYGEN UPTAKE

Electrode placement. Following exposure of the heart through a left thoracotomy in the fourth intercostal space, the left coronary artery was dissected free at its origin from the aorta, and a ligature passed under it in preparation for its cannulation. A cannula introduced into the left common carotid artery in the neck led arterial blood to a rotameter and thence to a specially designed brass cannula whose tip was inserted into the left coronary artery. This was achieved by introducing the cannula into the left subclavian artery and passing it through the ascending aorta so that its tip entered the left coronary artery where it was securely tied in place. By an arrangement of stopcocks the left coronary artery could be perfused either from the carotid artery or from a reservoir containing freshly drawn, heparinized, donor arterial blood.

Perfusion blood was obtained from donor dogs maintained under light anesthesia with pentobarbital, and breathing room air. Heparin, 10 mg./Kg., was given prior to bleeding from an artery. When more than one donor animal was used, their bloods were thoroughly mixed before being placed in the reservoir. Occasionally dextran in small amounts was added to the donor blood to increase the volume and reduce the hematocrit to between 50 and 35. The blood was slowly and constantly stirred and connected to the rotameter through a glass coil contained in a water jacket so that blood was delivered to the coronary cannula at approximately 35 C. During experimental observations the flow from the carotid artery was stopped and the left coronary artery was supplied with arterial blood of known and essentially constant oxygen content from the reservoir. The latter was adjusted to a height above the animal at which the pressure head delivered a coronary blood flow of about the same rate as that which existed when the coronary artery was perfused from the carotid artery. The perfusion pressure generally approximated the prevailing mean aortic pressure. The right coronary artery was not cannulated and consequently was perfused from the carotid artery. The perfusion pressure generally approximated the prevailing mean aortic pressure. The right coronary artery was not cannulated and consequently was perfused with the animal's own blood. The rate of left coronary inflow was measured with a recording rotameter. Systemic pressure was recorded with a Statham strain gage from the left common carotid artery.

A polyvinyl tube with a flanged opening was introduced into the right atrium through the right atrial appendage and passed into the opening of the coronary sinus where it was secured with a ligature. Its peripheral end was connected by a Y tube to the right external jugular vein so that coronary sinus blood could drain either to the exterior or return to the animal through the superior cava.

Fig. 1. Reproduction of an original record showing the effect of asystole for 28 sec. induced by vagal stimulation on left coronary inflow, coronary sinus oxygen content, and systemic arterial blood pressure.

Portions of the flow of blood through the left coronary artery and coronary sinus were drawn through two recording densitometers at the rate of 10 ml./min. each. These instruments respond linearly to changes in oxygen saturation of whole blood within the range of 0 to 100 per cent. They were calibrated at the end of each experiment by drawing through their cuvettes aliquots of coronary sinus blood whose saturation ranged from near zero to full saturation. Subsequently the aliquots were analyzed for their oxygen content. This was determined in a few cases by the colorimetric method employing the Beckman spectrophotometer and in the remainder by the method of Neill and Van Slyke.

Except as indicated, no drugs other than heparin and pentobarbital were administered to the recipient dog. Small infusions of dextran or whole blood were given as required to maintain normal levels of blood pressure. Systemic blood pressure was stabilized at any desired level by connecting both femoral arteries by a Y tube to a liter reservoir bottle filled with blood.

The heart of the experimental animal was carefully inspected after the experiment to assure correct positioning of the coronary artery and sinus cannula. Subsequently the heart was excised, the atria removed, and the weights of both ventricles obtained. The right ventricle was then cut from the left and the combined weight of the left ventricle and interventricular septum obtained.

Four procedures were employed to reduce cardiac work rapidly to zero: (1) Stimulation of the peripheral cut ends of both vagi was carried out with either an electrodynne or Grass stimulator, (2) Injection was made of a solution of potassium chloride or citrate into the coronary artery cannula to produce and maintain systole. The drug was given rapidly manually, or by a syringe connected to a constant speed injection mechanism,
Fig. 2. A. Graph based on record in figure 1 showing the effect of vagal stimulation on heart rate, aortic blood pressure, coronary arteriovenous oxygen difference, left coronary artery flow, and oxygen consumption/100 Gm. left ventricle/min. Control values are at zero time. Values determined from original record at 1 to 2 sec. intervals. B. Graph showing the effect of a constant intracoronary infusion of 10 per cent KCl at a rate of 0.9 ml./min. on same parameters as in A. Just before 1 min. ventricular fibrillation occurred, followed by a supplementary injection of 10 per cent KCl at 2 min., which resulted in asystole. Ventricular fibrillation occurred just before 5 min., and was again converted to asystole by a supplementary injection of 10 per cent KCl at 6 min. Values obtained from original record plotted at 1 to 10 sec. intervals. C. Graph showing the effect of rapid arterial hemorrhage and subsequent reinfusion on the same parameters as in A. Values determined from original record at 1 to 10 sec. intervals. D. Graph showing the effect of electrically induced ventricular fibrillation on same variables as in A. Values determined from original record at 1 to 10 sec. intervals.

(3) Rapid bleeding of the animal was performed by applying suction to the flask connected to the femoral arteries combined with occlusion of venous return through the inferior vena cava. By this means arterial blood pressure and cardiac output (as measured with a rotameter or observed by cutting the ascending aorta) were generally reduced to zero in about 45 sec., and (4) ventricular fibrillation occurred naturally or was produced by stimulating the ventricles with current from an induction coil. In a number of experiments these procedures were performed consecutively in the same dog for better comparison of values.

Oxygen usage of the left ventricle/100 Gm./min. was calculated as the product of left coronary inflow and coronary arteriovenous oxygen difference divided by the combined weight of the left ventricle with the septum. Since the rate of
TABLE 1.—Effect of Prolonged Vagal Stimulation on Oxygen Usage of the Left Ventricle

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<th>Control</th>
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Averages 150  8.1  4.3  2.8  2.4  2.3  2.0  1.9  2.2  3.1  3

*This and other superscripts refer to number of heart beats during time interval.

coronary flow varied and the coronary sinus oxygen saturation was not recorded at the sinus orifice but at the densitometer which was at an additional distance of about 15 cm., some delay in blood transmission occurred. Corresponding values for coronary flow and coronary sinus oxygen were obtained by correcting for the time delay.

RESULTS

Effect on Oxygen Consumption of Procedures That Remove Ventricular Contraction

Vagus Stimulation. The duration and completeness of the asystole produced was unpredictable in the same or different dogs. However, in a number of attempts asystole lasted sufficiently long to permit the attainment of a new equilibrium at what was believed to approximate the level of the resting metabolism. The sequence of events following vagal stimulation lasting for 28 sec. is shown in a reproduction of a segment of an original record in figure 1. The heart rate immediately falls to zero with one heart beat occurring during the fourth second. Within 1 sec. after the onset of asystole, coronary flow increases to a new maximum and then declines gradually so that at the end of vagal stimulation left coronary flow is considerably below the control level. After about a 4 sec. delay the oxygen content in the coronary sinus blood starts to increase, reaching essentially a plateau value within approximately 20 sec.

In figure 2A are plotted at 1 sec. intervals the changes in heart rate, aortic blood pressure, and left coronary flow, together with the changes in coronary arteriovenous oxygen difference and oxygen uptake calculated from the curves in figure 1. The arterial blood pressure falls gradually from 105 mm. Hg to reach a level of 32 mm. Hg in about 16 sec. The coronary arteriovenous oxygen difference initially at 16.5 cc. also declines, reaching a new level of 4.7 cc. in about 18 sec. The left coronary artery flow, after rising quickly from 50 ml./min. to a maximum of 96 ml./min., falls slowly; at 25 sec. its value of 37
ml./min. does not quite approximate a new equilibrium. In the beating heart the oxygen uptake is 10.3 cc./100 Gm. left ventricle/min. With asystole this value increases almost immediately to a peak of 15.4 cc./100 Gm. left ventricle/min. and then declines within 20-22 sec. to a level of 2.1 cc./100 Gm. left ventricle/min., where it is fairly well maintained. During recovery from vagal stimulation the various parameters pass through a supernormal phase before approaching control levels.

This general pattern and timing of response to vagal stimulation is typical of most of our records and calculations. However, the effect of vagal stimulation on the duration and completeness of the asystole produced was erratic. It therefore became necessary to establish criteria as to the duration of asystole necessary to effect a resting level of oxygen uptake. Twenty-one determinations from 12 experiments illustrated in table 1 have been used to determine the level of resting metabolism.

The duration of the approximate asystoles varies from 20 to 48 sec. The total number of heart beats during these periods varies from 0 to 8. Inspection of the data indicates that in almost all instances the resting level of metabolism is reached in 20 to 25 sec. The average values at 20, 25, and 30 sec. are 2.4, 2.3, and 2.0 cc. oxygen/100 Gm. left ventricle/min., respectively. These compare with an average oxygen value of 8.1 cc./100 Gm. left ventricle/min. in the beating heart.

The individual values for oxygen uptake during vagal arrest in the same and different experiments vary considerably, the range being from 0.8 to 3.7 cc. Attempts were made to elucidate the possible effect of certain conditions on these oxygen values. The data plotted in figure 3 indicate that the oxygen uptake in the resting myocardium is related to the preceding level of metabolism in the beating heart. As the metabolism of the beating heart increases from 4 to 20.5 cc. oxygen/100 Gm. left ventricle/min., the oxygen uptake in the vagal stopped heart in-
## Ventricular Oxygen Uptake

### Table 2.—Effect of Intracoronary Potassium Chloride or Citrate Infusion on Oxygen Usage of the Left Ventricle

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<th>C.A.F.</th>
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<th>Cor. A-V.O₂</th>
<th>L.V.</th>
<th>cc.O₂</th>
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*Isolated heart with empty ventricles. Average of 3 separate determinations.
†Ventricles emptied by suction just before potassium infusion.
‡H.R., heart beats/min.; A.B.P., mean arterial blood pressure in mm. Hg; C.A.F., left coronary artery inflow ml./min.; C.S.O₂, coronary sinus oxygen content in cc./100 ml. of blood; Cor. A-V.O₂, arterial coronary sinus oxygen content in cc./100 ml. of blood; cc.O₂ L.V., oxygen usage cc./100 Gm. left ventricle/min.

During prolonged vagal asystole the left ventricle visibly enlarges and the left ventricular end diastolic pressure very often increases significantly. To test the effect of left ventricular diastolic size on the resting metabolism, attempts were made to empty quickly the left ventricle of blood after metabolic equilibrium had been obtained. However, invariably the heart would partially escape from its vagal asystole before a possible new metabolic equilibrium could be established. Accordingly the effect of diastolic size was studied as follows. After the oxygen uptake had been determined in the control and in the asystolic state induced by vagal stimulation, arterial and venous hemorrhage aided by suction was rapidly induced to give essentially zero arterial blood pressure and cardiac output in 40 to 60 sec. The empty heart was again arrested by vagal stimulation and its oxygen uptake calculated. The dog was then re-infused. Typical data from two different experiments are illustrated in figure 4. The value for oxygen usage during vagal asystole in the greatly distended ventricle (V) is essentially the same as that obtained in the ventricle of minimum size following hemorrhage (HV). Similar agreements were obtained in 5 experiments in which the heart rate during hemorrhage was variable.

Potassium Injection. Potassium has been used to produce asystole using an extracorporeal circulation. It is well known that such hearts can be stopped and restarted after a number of minutes. During this period, although there is no coronary inflow, the surface of the heart appears pink and the oxygen usage must be low. Yet as the present results indicate, such hearts extract a considerable amount of oxygen if coronary perfusion is maintained.

The hemodynamic and metabolic responses of the heart to intracoronary artery injection of potassium are generally similar to those obtained with vagal stimulation. The data in figure 2B illustrate the sequence of events that often follows an initial injection of 10 per cent KCl at a constant rate of 0.9 ml./min. into the left coronary artery. The heart beat ceases within about 10 sec.; the coronary arteriovenous oxygen difference decreases within about 20 sec. from a control value of 12.6 to 8 cc. The left coronary artery flow, after an initial rise from 47 to 65 ml./min., decreases below the control level to 29 ml./min. The oxygen uptake initially at 6.7 cc./100 Gm. left ventricle/min. rises to 7.6 cc./100 Gm. left ventricle/min. and then falls...
within the minute to 2.6 cc./100 Gm. left ventricle/min. However, in this experiment the rate of potassium injection was not quite sufficient to maintain asystole. One minute after the start of KCl infusion ventricular fibrillation occurs and the coronary flow, coronary arteriovenous difference, and oxygen uptake increase significantly. At 2 min. a supplementary injection of 3 ml. of 10 per cent KCl was given manually into the left coronary artery. This temporarily caused a complete cessation of coronary inflow following which the oxygen uptake of 2.4 cc./100 Gm. left ventricle/min. again approaches the resting value. However, another episode of ventricular fibrillation raised the oxygen uptake to 3.3 cc./100 Gm. left ventricle/min., which was reduced again by an intracoronary artery injection of KCl. This resulted in a heart maintained in asystole for approximately the last 4 min. of the record. During this period the oxygen values are fairly stable, varying from 1.7 to 2.0 cc./100 Gm. left ventricle/min. These figures are somewhat lower than the values of 2.6 and 2.4 cc./100 Gm. left ventricle/min. obtained earlier in the experiment when there was insufficient time for metabolic equilibrium.

Table 2 summarizes the results of 7 determinations in 7 experiments. The duration of asystole lasts from 1 to 13 min. The range of values for oxygen uptake, 1.0 to 3.3 cc./100 Gm. left ventricle/min. and the average of 2.0 cc./100 Gm. left ventricle/min. are similar to those obtained during vagal stoppage of the heart (table 1).

**Effect on Oxygen Consumption of Procedures that Reduce Cardiac Work to Zero but Retain Myocardial Contraction**

**Hemorrhage.** The effect on myocardial metabolism of rapid hemorrhage to a zero level
VENTRICULAR OXYGEN UPTAKE

Table 4.—Effect of Ventricular Fibrillation on Oxygen Usage of the Left Ventricle

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</tbody>
</table>

*Headings as in table 2.
†Fibrillation induced by potassium infusion.

of arterial blood pressure and cardiac output has been tested successfully in 12 dogs. Except that initially coronary flow and coronary arteriovenous oxygen difference do not rise, the pattern of response is similar to that with vagal stimulation. Figure 2C is a plot of such data from a typical experiment in which essentially zero blood pressure and cardiac output (not shown) are reached in 30 to 40 sec. With the heart rate remaining essentially constant at 70/min., the left coronary flow progressively decreases from 26 to 17 ml./min.; the oxygen uptake decreases from 6.2 cc. in the control state to about 2.8 cc/100 Gm. left ventricle/min. in the bled heart. Following partial reinfusion the different parameters do not quite return to control levels.

Table 3 summarizes the values obtained before and during hemorrhage in 20 determinations in which the ventricles were maintained in essentially an empty state for 1 to 17 min. The arterial blood pressure, coronary artery flow, coronary arteriovenous oxygen, and oxygen uptake of the myocardium all decrease greatly. The directional changes in heart rate are unpredictable. The individual oxygen values in the bled hearts vary from 1.5 to 5.7 cc./100 Gm. left ventricle/min. The average oxygen value during hemorrhage is 3.4 cc./100 Gm. left ventricle/min., as compared with the control of 8.1 cc. This is considerably higher than the average value during vagal asystole.

Figure 5 shows that as with vagal stimulation the end value obtained for oxygen uptake during hemorrhage varies directly with the preceding level of metabolism. This value is not related to the preceding heart rate or arterial pressure (plots not shown).

Ventricular fibrillation. In 8 dogs 8 determinations of left ventricular oxygen uptake were made before and during ventricular fibrillation. The pattern of recorded response is very similar to that with vagal stimulation with a rise in the coronary sinus oxygen content, an initial increase in coronary inflow and with equilibrium being reached in 70 to 80 sec. (fig. 2D). Calculation of oxygen uptake for each second also shows a response curve similar in pattern to that with vagal stimulation. Figure 2D also illustrates that as with vagus asystole a large reduction in the size of the fibrillating left ventricle by massive arterial hemorrhage does not significantly alter the oxygen uptake.

The equilibrium values for oxygen uptake with ventricular fibrillation range from 2 to 5.6 cc./100 Gm. left ventricle/min. and average 3.8 cc./100 Gm. left ventricle/min. (table 4).

Relative Values for Oxygen Usage by Two or More Procedures in the Same Experiment

Complete removal of myocardial activity by vagal stimulation and potassium injection gives essentially similar average values of 2.0 to 2.2 cc. oxygen usage/100 Gm. left ven-
In 5 of 6 experiments the highest values for oxygen usage with any of the 4 procedures was obtained with ventricular fibrillation. These values generally exceed those found with hemorrhage even in the presence of a rapid heart rate (examples: 3.5 vs. 1.5; 3.2 vs. 2.1; 4.2 vs. 3.8). On one occasion in which the metabolism in the beating heart was low the metabolism during fibrillation was about the same.

DISCUSSION

The technic described in this report enabled the oxygen uptake to be monitored continuously. Some objection might be raised on the grounds that the right coronary artery was not perfused and some contamination of the coronary sinus blood with right coronary artery blood might occur. The right coronary artery in the dog contributes a very small fraction to the coronary sinus, and furthermore very little right coronary inflow exists at zero aortic pressure. In addition, the fraction of left coronary flow draining into the coronary sinus was previously found to be fairly constant in the beating heart under a variety of conditions including the administration of drugs. This ratio has also been found to be fairly stable in the presence of asystole from vagal or potassium stoppage, hemorrhage, and fibrillation. Therefore, it is believed that the results truly represent the uptake of oxygen by the left ventricle.

Since immediately following the induction of vagal asystole, ventricular fibrillation, and hemorrhage, left coronary inflow increases in the presence of a maintained coronary perfusion pressure, coronary resistance to flow must initially decrease. However, since with potassium injection coronary inflow decreases immediately and the delayed effect with all 4 procedures is generally a decrease in coronary flow, the sustained effect is an increase in coronary resistance. An explanation for this is not yet available.

The oxygen usage of the left ventricle in the beating but empty heart and during ventricular fibrillation is found to be much lower than that in the working heart. The average
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values of 3.4 and 3.8 cc./100 Gm. left ventricle/min. in these two conditions approximate 40 and 50 per cent of the values in the normal heart. It has been reported that the oxygen usage in a majority of experiments during ventricular fibrillation in the open chest dog may equal or exceed that during the working state. No adequate explanation is at hand for these higher values. However, it has been our experience that such high values can be obtained during ventricular fibrillation if stimulating substances such as adrenaline and noradrenaline are in the coronary perfusing blood in sufficient amounts.

The left ventricle during complete arrest either by potassium injection or by vagal stimulation uses about 2 cc. oxygen/100 Gm./min. The uptake by the left ventricle of oxygen during vagal asystole is thought to approximate a "basal" metabolic value. It seems reasonable that in calculating efficiency of the myocardium this oxygen uptake by the heart performing no work should be subtracted from the oxygen usage of the beating heart. This view is strengthened by the data indicating that the nonworking level of oxygen uptake can be increased by the level of metabolism in the beating state and by nor-epinephrine. However, as yet myocardial efficiency in many instances cannot be thus corrected, since it is difficult to routinely stop the heart by vagal stimulation for a sufficient time to measure its resting metabolism.

In the present experiments the average oxygen values for the 4 procedures tested are in the same over-all range, that is, between 2 and 3.8 cc./100 Gm. left ventricle/min. However, when the values with the different procedures are obtained in the same experiment, the figures for the hemorrhaged heart with rapid heart rate and especially for the fibrillating heart are considerably higher than the oxygen values for the vagus and potassium stopped hearts, which are close to each other.

Presumably most of the difference is attributable to the visibly greater myocardial activity of coordinated heart beats and fibrillatory movements during the latter two conditions. This is borne out by the lower oxygen values obtained in the bled heart with a slower heart rate. In fact, as shown in figure 4, the latter values may approximate those in the ventricle stopped with vagal stimulation. In addition, the oxygen uptake of a fibrillating heart correlates visibly with the intensity of the fibrillatory movements (data not included here). In view of the preceding, together with the additional fact that the actual oxygen usage in the bled empty heart is related to the control metabolic level (fig. 5), it is not surprising that the oxygen uptake in the fibrillating heart may equal, or at times exceed, that in the active but nonworking ventricle.

The experiments with vagal stoppage of the heart have an additional significance in that they lay the groundwork for calculation of the oxygen debt of the heart. Attention is directed to figure 2A, which depicts the course of oxygen uptake during this situation. A constant finding is the apparent increase in calculated usage of oxygen above the control level immediately after induction of vagal asystole and its slow decline to a resting level after cardiac work has ceased. An estimate shows that the excess of oxygen taken up from the onset of asystole to the time of the resting metabolism approximates 1.5 cc. This is approximately the amount of oxygen used during 25 systoles of such a heart before asystole was induced. An adequate explanation on an artefactual or functional basis is not yet available. A portion could arise from an increase in the size of the myocardial vascular bed associated with removal of ventricular contraction. However, it is extremely unlikely that the explanation is mainly upon this artificial basis. If it is assumed that all of the 1.5 cc. oxygen goes to fill the vascular bed, then with the prevailing coronary arte-

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TABLE 5.—Effect of Continuous Intracoronary In-
jection of Levarterenol (dose) on the Oxygen Usage
of the Left Ventricle

<table>
<thead>
<tr>
<th>Date</th>
<th>Oxygen usage (cc./100 Gm. left ventricle/min.)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before levarterenol</td>
</tr>
<tr>
<td></td>
<td>Control</td>
</tr>
<tr>
<td>11-19-56</td>
<td>7.7</td>
</tr>
<tr>
<td>11-26-56</td>
<td>7.2</td>
</tr>
<tr>
<td></td>
<td>9.0</td>
</tr>
<tr>
<td>6-25-56</td>
<td>10.9</td>
</tr>
<tr>
<td></td>
<td>12.6</td>
</tr>
<tr>
<td>5-24-56</td>
<td>9.1</td>
</tr>
<tr>
<td></td>
<td>9.4</td>
</tr>
<tr>
<td>6-4-56*</td>
<td>9.0</td>
</tr>
<tr>
<td>5-24-56</td>
<td>8.7</td>
</tr>
<tr>
<td>Averages</td>
<td>9.3</td>
</tr>
</tbody>
</table>

*Relative values only.

The veno-venous oxygen difference of 4 cc. the increase in intravascular volume would approximate 28 cc., an impossible figure. It is believed that most of the excess oxygen used after cessation of work is caused by the preceding cardiac work and therefore could be called oxygen debt. Since the ventricles were performing at about a normal level of activity before asystole, the oxygen debt of a heart working at capacity could well be considered larger. Unfortunately, thus far it has not been possible to arrest hearts performing at such a metabolic level.

It is of considerable interest that injection of noradrenaline into the left coronary artery significantly increases the oxygen usage of the myocardium whose external work has been reduced to zero by at least two procedures (table 3, 11-19-56). With an arterial blood pressure of 86 mm. Hg, the control oxygen usage is 16.2 cc./100 Gm. left ventricle/min. After hemorrhage, with an arterial pressure of 6 mm. Hg and heart rate of 222, oxygen usage drops only to 13.2 cc./100 Gm. left ventricle/min. With subsequent vagal stimulation the oxygen uptake declines further to 4.8 cc./100 Gm. left ventricle/min. In table 5, in 9 determinations in 5 dogs, the oxygen usage in the beating heart averages 9.3 cc./100 Gm. left ventricle/min., which decreases to 3.3 cc. during vagal stimulation. Infusion of levarterenol after recovery from vagal stimulation raises these values for oxygen uptake to 18.3 and 6.4 cc./100 Gm. left ventricle/min. respectively. The latter value approximates that obtained by Whalen for strips of resting ventricle of the dog to which epinephrine had been added. Since however oxygen uptake in the bled and in the vagal-stopped heart is related to the control metabolic level, it cannot be stated whether levarterenol has a specific effect on the resting metabolism of the myocardium.

**SUMMARY**

Comparisons have been made in the open chest dog of the oxygen usage of the left ventricle whose external work has been reduced to zero by 4 different procedures. The average oxygen usage of the left myocardium/100 Gm./min. is about 2.0 cc. in complete arrest with vagal stimulation or with intracoronary potassium injection; 3.8 cc. in fibrillation; and 3.4 cc. in the empty but beating heart. The oxygen value in the arrested heart approximates 27 per cent of that in the control state and the uptake during vagal stoppage varies directly with the control metabolic level. In the fibrillating ventricle the higher values are related to the intensity of fibrillatory movements, and in the empty beating heart to the control metabolic rate and to the frequency of the heart beat. The experiments with vagal arrest also permit estimation of the oxygen debt of the myocardium.

**SUMMARIO IN INTERLINGUA**

In canes a thorace aperte, comparaciones esseva effectuate inter le quantitates de oxygeno acceptate per le ventriculo in que le travalo externe habeva essite reducete a zero per 4 differente methodos. Le consumption medie de oxygeno in le myocardio sinistre es circa 2,0 cm³ per 100 g per minuta in arresto complete effectuate per stimulation vagal o injection intracoronari de kalium; illo es circa 3,8 cm³ in fibrillation e 3,4 cm³ con le corde vacuate sed pulsante. Le valor de oxygeno in le corde arrestate es approximativamente 27 pro cento de illo in le stato de controlo, e le acceptation durante le stoppage vagal varia...
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direttamente con il livello metabolico di controllo. In le ventricoli in fibrillazione, le
valori (che es plus alto) es relativati al intense
tate del movimento fibrillatori, e in le corde
vacuate sed pulsante illos es relativati al
livello metabolico de controlo e al frequentia
del pulso del corde. Le experimentos con ar
resto vagal etiam permite le estimation del
debita de oxygeno in le myocardio.

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