Left Ventricular Energetics

Heat Loss and Temperature Distribution of Canine Myocardium

GABIËL H.M. TEN VELDEN, GIJS ELZINGA, AND NICO WESTERHOF

SUMMARY The sum of total left ventricular heat loss and left ventricular mean total external power was compared with the product of oxygen consumption and its energy equivalent. Myocardial blood flow, measured with 15 ± 3 µm radioactive microspheres, was multiplied by the transcoronary arteriovenous temperature difference and by oxygen content difference to obtain coronary heat loss and oxygen consumption, respectively. Since only part of the heat is carried away by the coronary system, a thermodilution technique was used to obtain the ratio between the heat removed by the coronary system and the external heat loss. A correction was made for the endothermic reactions of hemoglobin deoxygenation and carbon dioxide reactions with blood. Left ventricular oxygen consumption corresponded to 2.26 ± 0.66 W/100 g, and for the sum of total left ventricular heat loss and external power, 2.09 ± 0.51 W/100 g was found (n = 14). In a second series, the measured transmyocardial temperature distribution was compared with the calculated temperature distribution, assuming that heat production in the myocardium is uniform and that heat is lost by coronary flow and diffusion. When thoracic and luminal myocardial surface temperatures were about equal, blood flow was found to be about the same in the various layers of the heart, whereas myocardial temperature was found to be highest near the middle of the wall (0.36 ± 0.07 °C warmer than luminal temperature (n = 6)). When thoracic surface temperature was increased or decreased (by +1.56 ± 0.99°C and −1.10 ± 0.59°C, respectively), consistent changes were seen for the temperature distribution in the myocardium, but not for the local flow (endo/epi ratio: 1.06 ± 0.29 and 0.96 ± 0.21, respectively). These data suggest that myocardial blood flow is independent of tissue temperature. Circ Res 50: 63-73, 1982

HEAT production of the intact working heart has been estimated indirectly through calculation of the difference between oxygen consumption times its energy equivalent, and mechanical work (Neill et al., 1961, 1963; Afonso et al., 1965a, 1965b, 1966). In these studies, heat loss to the coronary blood was obtained from the product of coronary blood flow and the transcoronary temperature difference. It became apparent that not all heat produced was carried away by the coronary blood but that an important fraction was lost through diffusion to ventricular cavities, mediastinum, and lungs. Another rather small fraction of the heat is lost through endothermic reactions in the coronary circulation, i.e., deoxygenation of hemoglobin and reactions of carbon dioxide with blood. The complete equation for the relationship between heat and power produced, and oxygen consumed is therefore:

\[ EE \times \text{MVO}_2 = \dot{H}_{MBF} + \dot{H}_{\text{diff}} - (-\dot{H}_{\text{chem}}) + P_{\text{ext}} \]  

where \( EE \) is the energy equivalent for oxygen, \( \text{MVO}_2 \) is oxygen consumption, \( \dot{H}_{MBF} \) is heat lost through the coronary bed, \( \dot{H}_{\text{diff}} \) is heat lost through diffusion, \( -\dot{H}_{\text{chem}} \) is heat lost through the endothermic reactions, and \( P_{\text{ext}} \) is the external power.

In the present study, the above equation is tested. Therefore we describe a method to measure total left ventricular heat loss. Moreover, we estimate how heat transport through diffusion and by coronary blood flow is related to the myocardial temperature distribution.

Methods

Preparation

Experiments were performed in two groups (A and B) of seven dogs each (24.2 ± 2.4 kg, \( n = 14 \)). After premedication, im (droperidol 0.6 mg/kg, methadone 0.4 mg/kg, atropine 0.01 mg/kg), and induction, iv (pentothal 20 mg/kg, droperidol 0.5 mg/kg, methadone 0.3 mg/kg, atropine 0.006 mg/kg, and xylocaine 2 mg/kg), neuroleptic anaesthesia was maintained by a constant infusion of droperidol, methadone, atropine, and xylocaine (0.5, 0.3, 0.006, and 1.2 mg/kg per hour, respectively). The animals were mechanically ventilated (by a Pulmocat respirator) with a mixture of one-third oxygen and two-thirds nitrous oxide.

In series A, a left thoracotomy was performed through the 4th intercostal space. A pericardial cradle was made and a short silicon catheter was...
tied in the left atrium for thermodilution and injection of radioactive microspheres. Two pacing electrodes were sutured onto the left atrial appendage. An electromagnetic perivascular flowprobe (made in our own workshop) was placed around the ascending aorta and connected to a flowmeter (SE Medic Flowmeter, model 275). Left ventricular blood pressure was measured with a catheter tip manometer (Millar model PC-350) introduced into the left ventricle via the left femoral artery. The chest then was closed in two layers to achieve normal temperature distribution in the thorax.

One thermistor catheter was introduced via the left carotid artery into the ascending aorta just above the valves and another via the left external jugular vein into the coronary sinus. Arterial and venous blood samples were obtained through 6F catheters introduced via the right brachial artery in the thoracic aorta and via the right jugular vein in the coronary sinus, respectively.

The experiments in series B differed from those in series A on the following points. The thorax was now opened through the 5th intercostal space. No electromagnetic flowprobe was used on the ascending aorta. Aortic instead of left ventricular pressure was measured. Use was made of a catheter on a Statham 23 Db pressure transducer. A thermistor injector device (Fig. 1) was tied on the left ventricular anterior wall. The chest was either closed (as in series A) or sealed with a plastic transparent foil (see below).

After these preparations, the animals were anticoagulated with 5000 IU heparine. The following mean and sd values \( n = 14 \) for pH, \( P_{O_2} \), and \( P_{CO_2} \) were obtained using Radiometer apparatus (BMS 3 MK2; PHM 71 MK2): pH = 7.347 ± 0.076, \( P_{O_2} \) = 133.1 ± 35.5 mm Hg, \( P_{CO_2} \) = 36.4 ± 6.7 mm Hg. Oxygen contents were determined with a Lex-O-Ref-TL (Lexington Instruments Corp.). Hematocrit was found to be 36.1 ± 7.2.

**Measurement of Myocardial Blood Flow**

Four differently labeled radioactive microspheres \( ^{125}I \) (3M), \( ^{141}Ce \), \( ^{58}Sr \), and \( ^{41}Sc \) (New England Nuclear), 15 ± 3 μm, suspended in 20% dextran, were injected into the left atrium. Reference samples were taken by a roller pump connected to a Teflon catheter introduced into the right femoral artery. For each flow measurement, \( 2 \times 10^6 \) to \( 15 \times 10^6 \) microspheres were suspended in a vial containing about 1.5 ml saline and a drop of Tween 80. Prior to injection, the vial was agitated mechanically for 15 minutes and ultrasonically for 5 minutes. The spheres were injected and the vial was flushed over a 20-second period with 20 ml of saline at 37°C. After completion of the experiments, the animals were killed with triotal, iv, and the heart was excised. Left ventricular mass (including the septum) was separated from atria and right ventricular mass. By means of a mixer (Moulinex, model 32002), left ventricular mass was homogenized; a sample was taken from the homogenate and weighed (29.2–37.4 g). Radioactivity of reference samples and tissue samples was measured in a four-channel γ counter (Wallac, LKB-1280 Ultrogamma), and isotope sep-
paration was accomplished by standard techniques (Rudolph and Heymann, 1967; Heymann et al., 1977), using a PDP-15 computer.

The reference sample method (Makowski et al., 1968; Domenech et al., 1969; Buckberg et al., 1971) was used for computation of myocardial blood flow (MBF):

\[ \text{MBF} = \text{myocardial activity} \times \frac{\text{flow}}{\text{activity}} \]

and was expressed as ml/min per 100 g.

In series B, prior to homogenization, two additional samples were taken from the left ventricular free anterior wall: one from the location where the needle thermistor was injected and one from an adjacent control location. These samples were divided into three parts for determination of transmural myocardial blood flow. The sample weights ranged from 1.91 to 4.04 g. Thus, in this series, in all, six transmural samples were analyzed, together with one homogenized tissue sample. The reference sample method (Makowski et al., 1968), using a PDP-15 computer.

Oxygen consumption was obtained by multiplying myocardial blood flow (MBF) with transcoronary oxygen content difference. We used an energy equivalent of 20.3 joules per ml oxygen (Geigy, Wissenschaftliche Tabellen, 1968). To obtain the external power, instantaneous aortic flow was multiplied by instantaneous left ventricular pressure. Mean total external power was found by averaging external power over the beat.

Heat generated by the left ventricle is removed in three ways (see Neill et al., 1961):

1. by myocardial blood flow (HMBF),
2. by diffusion into right and left ventricular cavities, mediastinum, and lungs (Hdiff), and
3. by endothermic chemical reactions (-Hchem), consisting of hemoglobin deoxygenation and the reactions of carbon dioxide with blood. From the data of Wyman (1948) it can be calculated that, at physiological pH, deoxygenation heat is 1.29 joules per ml oxygen. For CO₂, a value of 0.39 joule per ml was found (cf. Good and Sellers, 1957). Assuming a respiratory quotient of 0.85, the sum of these two reactions is 1.62 joules per ml oxygen consumed.

We can now rewrite Equation 2 in the form presented in the introduction (Equation 1).

The determination of the sum of HMBF and Hdiff, which equals external left ventricular heat loss, was performed in two steps. First, we measured coronary heat removal (in W/100 g) from the product of myocardial blood flow and transcoronary temperature difference (Tdiff) multiplied by density [ρb = 1.06 g/ml (Schultz et al., 1969) and specific heat of blood [cb = 3.6 J/°C per g (Mendlowitz, 1948)]:

\[ H_{\text{MBF}} = \left( \frac{\text{MBF}}{60} \right) \times \rho_b \times \frac{c_b}{T_{\text{diff}}} \] (3)

Then the ratio of coronary heat loss to external left ventricular heat loss was determined: H_MBF/(H_MBF + H_diff). This ratio R equals the percentage of the heat removed by the coronary system and 100-R the percentage of diffusional heat loss. The ratio R is obtained by dividing the thermoludation area recorded in the coronary sinus by the area recorded in the ascending aorta (i.e., the origin of the coronary system) after injection of a quantity of cold (5 ml saline, room temperature) in the left atrium.

Thermoludation techniques are not usually extremely accurate. However, the determination of the ratio R circumvents errors resulting from deviations in injection volume, temperature, and dura-
tion because the ratio of two areas is taken. We tested the reliability of the ratio R as a measure of diffusional heat loss in a model where the ratio could be measured in two independent ways. The model is shown at the left side of Figure 2. It consists of two compartments thermally isolated from the environment and separated by a membrane over which heat could be exchanged. Water was pumped through both compartments. In the steady state, a certain amount of cold was injected (well mixed) at the entrance of compartment 1. Temperature changes were measured at entrance and exit of compartment 1 and at the exit of compartment 2. The thermal changes are shown at the right side of the figure. The ratio R can be determined in two ways: (1) as the ratio between the thermodilution areas measured at the exit and the entrance of compartment 1, and (2) because the difference in cold at the entrance and at the exit of compartment 1 equals the amount of cold out of compartment 2, R is found also by taking the ratio between the difference of cold introduced into compartment 1 and leaving compartment 2, divided by the cold at the entrance of compartment 1. At a given flow rate in the compartments, the R values measured in this way were 0.49 ± 0.04 and 0.48 ± 0.01 (n = 5), respectively.

In additional experiments, two electrodes were introduced, one at the entrance and one at the exit of compartment 1 to generate heat in the fluid. The compartment (volume = 125 cm³) was filled with small glass beads (o.d. = 1 mm) and perfused with saline. In this way a “homogeneous” heat generation of about 1.5 W was achieved. At a given flow rate (165 ml/min), the ratio R first was determined by the thermodilution technique with and without heating. Second, the R value was measured as the quotient of the amount of heat transported by the flow and the total heat generated. All R values were between 0.64 and 0.66.

For the heart, it has been assumed that the thermal indicator is carried away in a way similar to the heat produced in the heart muscle itself. For each measurement, the values of three subsequent thermodilution ratios (R) were averaged; the areas under the thermodilution curves (Fig. 3) with respect to the baseline were obtained by planimetry. In some experiments, baseline drift complicated the thermodilution area measurement. In these cases, the areas were determined by assuming a linear baseline drift and that the signal had reached its baseline after 85 seconds.

External left ventricular heat loss can now be expressed as $H_{MBF} \times 100/R$, and Equation 1 changes to:

$$EE \times \text{MVO}_2 = H_{MBF} \times 100/R - (-H_{chem}) + P_{ext} \quad (4)$$

**Protocol**

In seven dogs, we compared left ventricular oxygen consumption times its energy equivalent with the sum of total left ventricular heat loss and external power (Eq. 4). Duplicate measurements at two different pacing frequencies were made in the steady state. Time between measurements was at least 15 minutes. For each measurement, we determined transcoronary temperature difference, ratio R by thermodilution (3×), mean total external power, myocardial blood flow, arterial and venous blood gases, and oxygen contents.
**Series B**

**Theory**

In seven dogs, diffusional heat loss and coronary heat loss were determined (at a constant pacing frequency) and compared with the transmyocardial temperature distribution measured with the thermistor injector (Fig. 1). For this comparison, Equation A3 of the Appendix, which holds for a thick-walled cylinder, was used to calculate the theoretical temperature distribution.

**Protocol**

Transmyocardial temperature distribution was measured in each dog at different epicardial temperature levels to alter diffusional heat loss. For that purpose, the thorax was sealed only with plastic transparant foil to prevent exsiccation of the intrathoracic structures. Low thoracic temperatures developed spontaneously, whereas, in order to obtain high intrathoracic temperatures, an infrared lamp (Elstein, 250W) was used, the radiation heat of which easily penetrated the foil. In all situations, measurements were performed after a stationary intrathoracic temperature had been achieved (after about 25 minutes). In all dogs, the temperature distribution was also measured after the chest had been closed in two layers, which resulted in a very small transmyocardial temperature difference. A random order of forced intrathoracic temperature levels was chosen.

At each transmyocardial temperature difference (four per experiment at most), blood samples were taken, ratio R determined by thermodilution (3x), microspheres injected, and transcoronary temperature difference and transmural temperature distribution measured.

**Results**

**Energy Balance (Series A)**

Figure 3 demonstrates how ratio R was determined. It shows that the cold arrives later in the
Coronary sinus than in the aorta. Note also the difference in configuration between the two curves. The oscillation, most clearly visible in the coronary sinus tracing, is synchronous with ventilation. Areas and calibrations are indicated. In one experiment, R ratios obtained with five cold (saline, 23.6°C) and five warm (saline, 50.6°C) left atrial injections yielded a ratio of 65.6 ± 6.1% (mean ± SD) and warm injections gave 65.8 ± 2.2%.

To test the validity of Equation 4, we plotted for each flow and heart rate, three subsequent R determinations (connecting lines) were made. Two heart rates, each with two flow determinations, were studied. The same symbols are used as in Figure 4. The line of regression is significantly different from zero (Student's t-test, P < 0.01).

average of two measurements at the same heart rate. In all experiments, two different pacing frequencies were used. The mean ratio between heat plus external power and left ventricular oxygen consumption of these data points was 0.95 ± 0.20 (mean ± SD, n = 14). Left ventricular oxygen consumption yielded an average value of 2.26 ± 0.66 W/100 g; the sum of total left ventricular heat loss and mean total external power was 2.09 ± 0.51 W/100 g. Higher heart rates were associated with a higher oxygen consumption and with a larger heat loss. Individual data on myocardial blood flow, diffusion and R ratios are given in Table 1. Mechanical efficiency, calculated from mean total external power and left ventricular oxygen consumption, ranged from 10 to 34%. Higher myocardial blood flow.

**Table 1: Flow, Temperature, and Energy Data Per Dog**

<table>
<thead>
<tr>
<th>Experimen-</th>
<th>HR (beats/min)</th>
<th>MBF (ml/min per 100 g)</th>
<th>Tdyr (°C)</th>
<th>R (% avg ±SD)</th>
<th>MVO₂-EE (W/100 g)</th>
<th>H₄₉₉ (W/100 g)</th>
<th>H₄₉₉ (W/100 g)</th>
<th>H₄₉₉ (W/100 g)</th>
<th>Pₑₑₑ (W/100 g)</th>
<th>Eff (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>11010</td>
<td>116</td>
<td>44.6</td>
<td>0.234</td>
<td>45.4 ± 5.1</td>
<td>1.52</td>
<td>0.66</td>
<td>0.80</td>
<td>1.58</td>
<td>0.35</td>
<td>23</td>
</tr>
<tr>
<td>150</td>
<td>49.3</td>
<td>0.262</td>
<td>49.5 ± 5.9</td>
<td>1.85</td>
<td>0.79</td>
<td>0.81</td>
<td>1.75</td>
<td>0.27</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>21010</td>
<td>150</td>
<td>57.5</td>
<td>0.218</td>
<td>53.7 ± 4.6</td>
<td>2.16</td>
<td>0.80</td>
<td>0.69</td>
<td>1.66</td>
<td>0.32</td>
<td>15</td>
</tr>
<tr>
<td>178</td>
<td>69.3</td>
<td>0.266</td>
<td>56.9 ± 2.9</td>
<td>2.61</td>
<td>1.18</td>
<td>0.89</td>
<td>2.28</td>
<td>0.25</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>24010</td>
<td>84</td>
<td>52.4</td>
<td>0.211</td>
<td>44.5 ± 4.4</td>
<td>1.75</td>
<td>0.70</td>
<td>0.88</td>
<td>1.72</td>
<td>0.60</td>
<td>34</td>
</tr>
<tr>
<td>114</td>
<td>62.6</td>
<td>0.226</td>
<td>48.3 ± 4.0</td>
<td>2.43</td>
<td>0.90</td>
<td>0.96</td>
<td>2.06</td>
<td>0.58</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td>31010</td>
<td>106</td>
<td>83.7</td>
<td>0.240</td>
<td>71.4 ± 4.6</td>
<td>2.86</td>
<td>1.28</td>
<td>0.51</td>
<td>2.02</td>
<td>0.45</td>
<td>16</td>
</tr>
<tr>
<td>130</td>
<td>96.1</td>
<td>0.258</td>
<td>69.7 ± 5.2</td>
<td>3.74</td>
<td>1.58</td>
<td>0.69</td>
<td>2.57</td>
<td>0.43</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>01020</td>
<td>98</td>
<td>63.8</td>
<td>0.193</td>
<td>73.2 ± 2.9</td>
<td>2.37</td>
<td>0.78</td>
<td>0.29</td>
<td>1.26</td>
<td>0.42</td>
<td>18</td>
</tr>
<tr>
<td>128</td>
<td>94.4</td>
<td>0.206</td>
<td>74.9 ± 6.3</td>
<td>3.15</td>
<td>1.24</td>
<td>0.42</td>
<td>1.91</td>
<td>0.67</td>
<td>21</td>
<td></td>
</tr>
<tr>
<td>07129</td>
<td>83</td>
<td>77.4</td>
<td>0.102</td>
<td>51.6 ± 13.9</td>
<td>1.79</td>
<td>0.50</td>
<td>0.47</td>
<td>1.11</td>
<td>0.22</td>
<td>18</td>
</tr>
<tr>
<td>108</td>
<td>77.4</td>
<td>0.125</td>
<td>60.9 ± 6.5</td>
<td>2.13</td>
<td>0.62</td>
<td>0.40</td>
<td>1.19</td>
<td>0.33</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>17010</td>
<td>126</td>
<td>42.0</td>
<td>0.231</td>
<td>56.0 ± 4.3</td>
<td>1.84</td>
<td>0.62</td>
<td>0.49</td>
<td>1.26</td>
<td>0.26</td>
<td>14</td>
</tr>
<tr>
<td>150</td>
<td>45.7</td>
<td>0.218</td>
<td>52.7 ± 2.8</td>
<td>1.42</td>
<td>0.63</td>
<td>0.57</td>
<td>1.31</td>
<td>0.25</td>
<td>18</td>
<td></td>
</tr>
</tbody>
</table>

HR = heart rate; MBF = myocardial blood flow; Tdyr = transcoronary temperature difference; R = ratio of coronary heat loss to external ventricular heat loss; MVO₂-EE = left ventricular oxygen consumption times its energy equivalent; H₄₉₉ = coronary heat loss; H₄₉₉ = diffusional heat loss; H₄₉₉ = total left ventricular heat loss; Pₑₑₑ = mean total left ventricular external power; Eff = mechanical efficiency calculated from Pₑₑₑ/(MVO₂-EE).
flow rates were correlated (P < 0.01) with higher R values (Fig. 5). This finding implies that, proportionally, more heat is transported by the coronary system at higher blood flows. In the analysis, all individual R determinations were used, i.e., 3 x 4 per dog.

Transcoronary temperature difference ranged from 0.102 to 0.266°C (see Table 1). Rectal and intrathoracic temperatures were 36.8 ± 1.0°C and 36.5 ± 0.9°C, respectively.

Myocardial Temperature Distribution (Series B)

In one dog, not included in series B, reproducibility of the temperature distribution was measured at stationary intrathoracic temperature (Fig. 6). All three distributions were obtained within half an hour. Each dot represents an individual temperature reading. Luminal temperatures were set equal.

Transmyocardial temperature distributions of six dogs are given in Figure 7; temperature is plotted on the ordinate for the six graphs. The average body temperature of the six dogs was 37.1 ± 1.5°C. Data of the seventh dog were not plotted here because the needle thermistor did not fully penetrate the left ventricular wall. The measured temperature distributions (dots) were compared with the calculated temperature distributions (drawn lines) on basis of Equation A3. The influence of the transmyocardial temperature differences on the measured distribution is shown in Figure 8. Results of two dogs are shown. These measured temperature distributions may be compared with the theoretical distributions presented in Figure A1 of the Appendix.

We investigated whether transmyocardial temperature difference and the associated temperature distribution (Fig. 8) had an effect on coronary blood flow distribution. There was no consistent change of the ratio of endo- to epicardial blood flow with change of temperature difference across the myocardial wall (Mann-Whitney U-test; seven dogs). The mean endo/epi ratio for the seven dogs at an average transmural temperature difference of +1.56 ± 0.99°C (i.e., outside warmer) was 1.06 ± 0.29 for the thermistor location and 1.03 ± 0.23 for the control location; for an average temperature difference of −0.20 ± 0.13°C (about equal luminal and thoracic surface temperatures) the endo/epi ratios for the two locations were 1.11 ± 0.12 and 1.05 ± 0.19. For an average temperature difference of −1.10 ± 0.59°C (i.e., outside colder), these ratios were 0.96 ± 0.21 and 0.98 ± 0.23, respectively. An example of one experiment is shown in Figure 9.

Discussion

Heat produced by cardiac metabolism is removed from the myocardium in different ways. Of the total heat produced, only a small fraction (8%) is consumed (see Neill et al., 1961) by the endothermic biochemical reactions of hemoglobin deoxygenation.
Figure 8 For two dogs, myocardial temperature distributions are shown at three transmyocardial temperature difference levels (★ negative; ■ about zero; ▲ positive), T is thoracic surface, L is luminal surface. This thermal behavior compares well with the computed distributions (Fig. A1).

Figure 9 Endo- to epicardial blood flow ratios at three transmyocardial temperature difference levels (ΔTtrans) and their corresponding temperature distributions (panels). Endo/epi ratios at the thermistor location (●) and at a control location (★) are indicated.

(Wyman, 1948) and carbon dioxide reactions with blood (Good and Sellers, 1957). The remaining heat is lost through the coronary system and by diffusion through the myocardial wall to the epicardial surfaces. The slopes of the temperature distribution at the endo- and epicardial surfaces are related to the amount of heat lost to left ventricular cavity and thorax. The large difference in the capacity of heat removal between the turbulent blood in the ventricular cavity and lung tissue has indirect influence on the temperature distribution; only the temperatures at the two surfaces count (see Equation A3). Determination of the ratio R, relating coronary heat loss to external left ventricular heat loss, forms the basis of the separation between diffusional and convectional heat removal. It is assumed here that metabolic heat is removed in the same way as heat introduced artificially into the myocardium. The fact that we could show that left ventricular energy uptake, measured from the oxy-
gen consumption, did not differ from the sum of total left ventricular heat loss and mean external power (Fig. 4) is in favor of the validity of this assumption.

We found the ratio R to be the same when determined by a small injection of heat or cold. This observation shows that the heat transfer system does not change with temperature. This conclusion is supported by the finding that the distribution of myocardial blood flow, which is part of the heat transfer system, is independent of the temperature distribution over the left ventricular wall (Fig. 9). The heat removed by the biochemical reactions can be considered as a heat production deficit. Therefore, in Equation 4, the ratio R was supposed to apply to convectional and diffusional heat losses only.

The average coronary arteriovenous temperature difference of the experiments presented in Figure 7 was 0.20 ± 0.06°C. When the temperature distributions (Fig. 7) were integrated over the left ventricular wall, an average temperature difference between tissue and cavity of 0.19 ± 0.04°C was found. The fact that these two figures are rather close, combined with the fact that blood temperatures in cavity and aorta are similar, indicates that the temperature of the blood in the small vessels and that of the tissue are in close equilibrium. An increase in the magnitude of myocardial blood flow therefore increases coronary heat loss. This explains the positive relationship between myocardial blood flow and R values shown in Figure 5.

When temperatures inside and outside are kept the same, the highest temperature in the wall, as has been shown previously (Reynolds and Yu, 1964; Bleakley et al., 1965), is found near the middle (Fig. 7). The amount of heat taken up by the coronary blood is proportional to the temperature difference between tissue and blood. Since flow through the various layers of the heart is about the same, and assuming a random organization of the coronary vascular system, one can conclude that at the warmest location heat loss through the coronary system is greatest. Thus, the amount of heat removed from the left ventricular myocardium by the coronary radiator alone, assuming homogeneity of metabolic heat production, is largest in the mid-myocardial layers. Inversely, it implies that diffusional heat loss is greater at epi- and endocardium than in the middle of the wall. This, together with the fact that heat generated by the more central layers and transported by diffusion has to pass through the outer layers, results in the typical temperature distributions across the left ventricular wall (Fig. 7).

The temperature distributions exhibit irregularities (Fig. 7) which may result from the vicinity of large vessels where blood temperature is not equal to tissue temperature. Large deviations from the symmetrical shape or distributions with two maxima may be related to the irregular endocardial surface. Such patterns often were found when the thermistor went through the base of the anterior papillary muscle. These suggested reasons for irregularities in the temperature distribution limit the possibilities to detect differences in local metabolic activity by those measurements.

Because the temperature distribution over the left ventricular wall depends strongly on thorax temperature (Fig. 8), we investigated whether the endo/epi blood flow ratio was related to the endo/epi temperature difference. No consistent effect was found when the temperature in the thorax was made lower or higher than the temperature of the blood in the ventricle (Fig. 9). This finding is in accordance with the small effect on overall coronary blood flow found with alterations in blood temperature (Liedtke et al., 1974).

Appendix
(with E.J.M. Veling*)

Terminology

Total left ventricular heat loss
External left ventricular heat loss
Left ventricular external heat production (in W cm⁻³)
Coronary heat loss
Diffusional heat loss
Heat loss due to endothermic chemical reactions
Energy equivalent of oxygen
Left ventricular oxygen consumption

\[
\text{Mean total external power} = P_{\text{ext}} = \int_0^T P_{LV}(t) F_{ao}(t) \, dt
\]

* Stichting Mathematisch Kruislaan 413, P.O. Box 4079, 1009 AB, Amsterdam.
Left ventricular pressure
Aortic flow
Myocardial blood flow
Heat conductivity of non-perfused heart tissue (Grayson, 1967)
Density of blood
Density of heart tissue, taken the same as blood
Specific heat of blood

Calculations

The differential equation relating left ventricular external heat production ($A$, in watts per cm$^3$) and myocardial temperature ($T$) when heat is lost by diffusion and by the coronary flow, in the steady state (see Pennes, 1948; Hernandez et al., 1979; Eberhart et al., 1980) is given by

$$\frac{d^2T}{dx^2} - \left(\frac{MBF}{6000}\right) \times \rho_b \times c_b \times T/D = -A/D \quad (A1)$$

This equation can be written in cylindrical coordinates assuming cylindrical symmetry:

$$\frac{d^2T}{dr^2} + \frac{1}{r} \frac{dT}{dr} - CT = -A/D \quad (A2)$$

where $C = (MBF/6000) \times \rho_b \times \rho_b \times c_b/D$.

When the luminal temperature is $T_o$ and the thorax temperature $T_i$, we found that:

$$T(r) = T_o + \left[ \Delta - \left( K_0(r_0\sqrt{C}) - K_0(r\sqrt{C}) \right)I_0(r\sqrt{C}) \right.\left. - \left( I_0(r_1\sqrt{C}) - I_0(r_0\sqrt{C}) \right)K_0(r\sqrt{C}) \right] \left[ A/(D \cdot C \cdot \Delta) \right]$$

$$+ \left( K_0(r_0\sqrt{C})I_0(r\sqrt{C}) - I_0(r_0\sqrt{C})K_0(r\sqrt{C}) \right)$$

$$\frac{(T_i - T_o)/\Delta}{A/(D - C - A)}$$

where:

$$\Delta = I_0(r_1\sqrt{C})K_0(r_0\sqrt{C}) - I_0(r_0\sqrt{C})K_0(r_1\sqrt{C})$$

and $r_0$ and $r_1$ are the inner and outer radius, $r$ is the location in the wall, and $K_0$ and $I_0$ are modified Bessel functions (Watson, 1966) of the first kind and order zero. The temperature distribution is calculated for $r_0 = 2$ cm, $r_1 = 4$ cm, $A = 0.02$ W·cm$^{-3}$, $MBF = 75$ cm$^3$/min per 100 g, so that $C = 10.0$ cm$^{-2}$, and is shown in Figure A1 (curve 1). Two other temperature distributions are shown in Figure A1. In one, the outside temperature is increased by 1.2°C (curve 2); in the other, the outside temperature is decreased by 0.6°C (curve 3). All other variables are kept the same.

The six theoretical temperature distributions of Figure 7 are calculated as follows. Left ventricular external heat production ($A$, in W·cm$^{-3}$) was obtained from measurements of coronary blood flow, transcoronary temperature difference ($T_{adiff}$), and the ratio $R$:

$$A = \left( \frac{MBF}{6000} \right) \times \rho_b \times \rho_b \times c_b \times T_{adiff} \times 100/R \quad (A4)$$

The wall thickness and surface temperatures were set as found from the measured temperature distribution. The $D$, $r_0$, and $c$ values were the same as above.
References


Afonso S, McKenna DH, O'Brien GS, Rowe GG, Crumpton CW (1965b) Left ventricular heat production during induced tachycardia in the intact dog. Am J Physiol 208: 31-36

Afonso S, O'Brien GS, Jaramillo CV, McKenna DH, Rowe GG (1966) Left ventricular heat production after lowering left ventricular work. Am J Physiol 210: 553-556


Left ventricular energetics. Heat loss and temperature distribution of canine myocardium.
G H ten Velden, G Elzinga and N Westerhof

*Circ Res.* 1982;50:63-73
doi: 10.1161/01.RES.50.1.63

*Circulation Research* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1982 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/50/1/63

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Circulation Research* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to *Circulation Research* is online at:
http://circres.ahajournals.org/subscriptions/