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HEAT GENERATION AND TRANSPORT IN THE HEART

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During contraction of the heart, a large part of the energy in energy metabolism is converted to heat. The article presents the results of measurements of mechanical stresses in the myocardium and blood vessels, temperatures and rate of heat generation. Experimental data correlate well with the numerical solutions of the biothermal problem.

Introduction. During contraction of the heart, a large part of the energy in energy metabolism is converted to heat. The interest in heat generation and transport in the heart arose among others because heat measurements were thought to be useful to assess the local rate of energy metabolism or blood flow. Given the difficulties of local measurements of oxygen consumption in various regions of the heart, for instance to compare energy metabolism in the inner (subendocardial) and outer (subepicardial) muscle layers of the ventricles, investigators tried to deduce local heat generation from the measured local temperatures. This turned out to be very difficult, because heat conduction in cardiac tissue is very fast and myocardial heat transport is more complex than was expected. Below we will see that when the blood flow is stopped for several seconds, at many sites inside the myocardium the temperature rises as one would expect, because convective transport by blood is an important route for heat to be transported out of the heart. One even tried to use the local rate of rise of temperature as a local measure of metabolism. However, at some sites inside the heart muscle the temperature dropped when blood flow was experimentally stopped, despite metabolism and heart generation at such sites, which very likely went on for several seconds after blood flow had stopped. Thus heat transport in the heart turns out to be complex and is incompletely understood.

Besides the use of heat as an indicator of metabolism, there was also interest in heat as an aid in the measurement of local blood flow. Heat has been used very often as a tracer in the thermodilution method to determine cardiac output or coronary blood flow. We will see below in this review that heat injection has also been used to assess the extent of countercurrent exchange between arteries and arterioles entering the heart and veins and venules leaving the heart. The myocardial countercurrent exchange between blood vessels turned out to be large, so that more than half of heat tracer entering the myocardium in the arterial blood does not reach the tissue.

Heat Generation. The time course and amount of heat generated during cardiac contraction can be studied in small isolated pieces of heart muscle, such as isolated papillary muscles, which are connected to the heart valves [1-3]. The temperature of the muscle is measured by placing the tissue on a thermopile [4]. The time course of the temperature is measured after electrical stimulation of the heart muscle, and from the time course of this temperature (with amplitudes of several $m^{\circ}C$) the time course of heat production in the muscle is calculated by deconvolution to correct for the time delay caused by heat conduction. The heat rate is composed of a peak measured during force development of the muscle, which is called the initial heat (see Fig. 1). This peak reflects the enthalpy of the breakdown of the high-energy phosphate metabolite phosphocreatine, which transfers its phosphate group to replace the terminal phosphate group of ATP that was hydrolyzed to directly support the contraction; thus at the end of the contraction the amount of phosphocreatine is decreased. After the contraction a slow tail of heat development is measured, which is called the recovery heat, and which reflects the heat development accompanying resynthesis of ATP in the mitochondria by oxidative phosphorylation. The ATP in turn transfers its terminal phosphate group to creatine and thus phosphocreatine content recovers from the depletion during muscle

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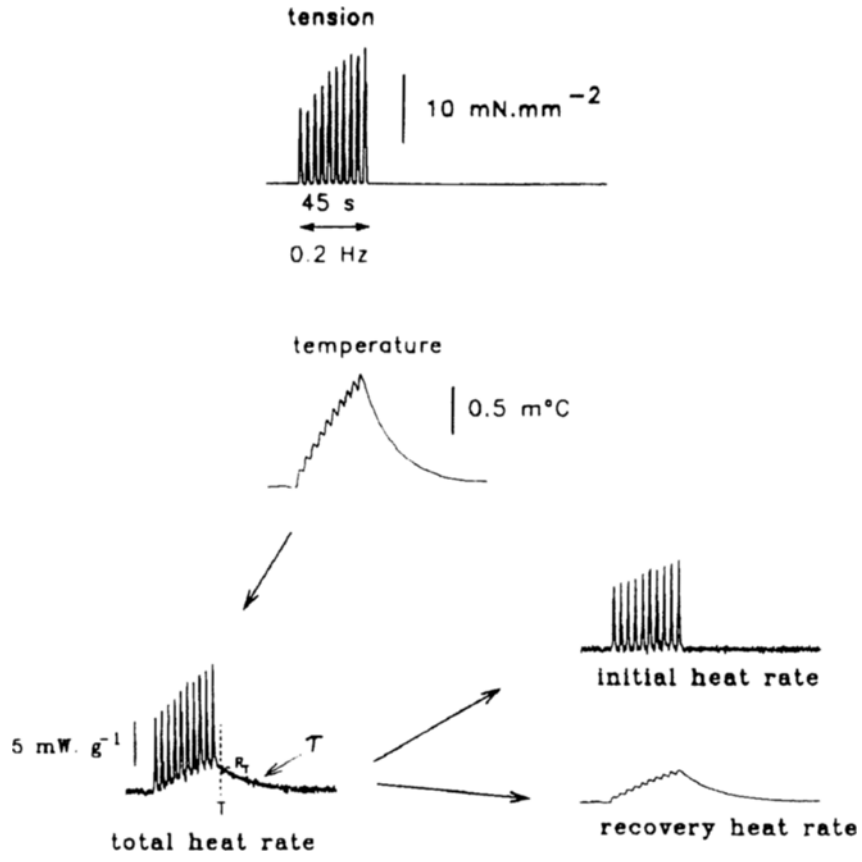

Fig. 1. Recording made by Zuurbier [3] of tension, temperature and rate of heat generation in a papillary muscle that is isolated from the right ventricle of a rabbit heart. The top trace shows the tension (force per unit cross-sectional area) generated after electrical stimulation of the muscle at temperature about 20°C. As often seen in cardiac muscle the force rises gradually when stimulation is started after several minutes without stimulation. In this case ten stimuli at 0.2 Hz are given. The papillary muscle is placed on a metal-film thermopile to measure temperature (middle trace). In all tracings the horizontal direction is the time axis, and the quantity on the *y*-axis is given above or below the trace. By deconvolution of the temperature with the heat conduction response of the measurement system the total rate of heat development is obtained, per gram dry weight. The total heat rate is separated into initial heat, generated during the contraction, and recovery heat, generated during metabolic recovery after the contraction according to previously published methods [1]. The time constant τ is determined starting at T, where the amplitude is R_T.

contraction. The recovery heat decays with a time constant of about 25 seconds at a temperature of 20°C towards a basal heat rate level that is not related to contraction and reflects basal cardiac metabolism [1]. Thus resynthesis of ATP and phosphocreatine is highest in the first seconds after contraction and then gradually becomes lower. The recovery heat constitutes about 52% of the total heat generation caused by isometric contraction of the muscle, and the remaining 48% of the total heat is the initial heat generated directly during the contraction [1].

The heat rate could only be measured reliably at 20 °C because the papillary muscle is not perfused and oxygen diffusion has to take care of all the oxygen supply to the tissue. Although the diffusion coefficient for oxygen increases with temperature, the papillary muscle's core will become anoxic at higher temperatures because oxygen

consumption will increase more strongly. However, we measured the time course of cardiac mitochondrial oxygen consumption after a change in the frequency of contraction in isolated rabbit hearts, which reflects the time course of oxidative phosphorylation just as recovery heat rate does. We found that the time constant of oxygen consumption increases by about 110% per 10°C decrease in temperature [5]. The time constant of the change of oxygen consumption after steps in heart rate is estimated to be around 8 sec at 37°C after correction for oxygen diffusion and vascular transport delay [6]. Consequently, recovery heat generation resulting from one contraction of the heart continues during many following contractions and adds up to an almost constant high level of recovery heat production on which the initial heat generated acutely during each contraction is superimposed. The flashes of heat generation synchronous with contraction have in fact been shown by a group which placed heat sensitive foil, with fast temperature response, on the external surface of the heart and found that the foil changed colour during each beat to indicate temperature increases [7] (and personal communication). The increase in tissue temperature should not be much more than 0.001°C due to the peak of heat generation during systole in the resting heart, as estimated from the amount of heat generated per beat, and as also seen in Fig. 1. However, the temperature variation through the cardiac cycle may be for in large part due to the time-varying coronary perfusion through each cardiac cycle whose effect was estimated to be up to 0.005°C [8].

The total heat rate in a dog heart beating at rest at 80–130 beats/minute is 1.5 ± 0.3 W/100 g wet weight (mean ±SD); the oxygen consumption is 2.7 ± 0.7 μmol/g wet weight/min [9]. The efficiency of energy conversion in the heart showed much interindividual variation among dogs: between 10 and 34% of aerobic chemical energy was converted into work to pump blood. Cardiac oxygen consumptions more than tenfold higher have been found in dogs performing strenuous exercise and heat production may be expected to increase proportionally to oxygen consumption, so that heat production in a dog heart might reach about 15 W/100 g during maximal exercise performance.

Heat Transport by Conduction and Convection. Heat is transported out of the heart muscle convectively by the blood flow, but also by conduction to the adjacent tissue and the blood in the ventricular cavities. Transmural heat conduction plays a substantial role in heat transport, in contrast to the much smaller relative role for diffusion in mass transport, for instance of oxygen. In the isolated heart some diffusion of oxygen across the outer surface of the heart is found and the diffusion front extending from the surface of the heart into tissue is characterized by a space constant of about 120 μm in hearts perfused with saline solutions [10]. However, in the case of the dog heart *in situ* only three quarters of the amount of heat in a bolus of cold saline solution injected into the arterial inflow of a dog heart *in situ* is recovered in the coronary venous outflow and the rest leaves the heart via conduction [9, 11]. Models of heat transport in tissue [12, 13], incorporating not only exchange of heat locally with the blood perfusing the tissue but also transmural heat conduction from the left ventricular free wall to the mediastinum and lung tissue via the epicardial surface and to blood in the ventricular lumen via the endocardial surface, do indeed predict substantial loss of heat via conduction [9].

The heat conduction out of the heart across its external surfaces is driven by temperature gradients across the left ventricular wall. Such gradients were measured directly with a small thermistor mounted in a needle that was driven gradually in small steps through the wall of the heart from epicardium to endocardium [14, 15]. In the center of the left ventricular free wall of the dog, which is about 1.5 cm thick, the temperature usually was about 0.4°C higher than near the surfaces of the left ventricle; see example in Fig. 2. The temperature profile was fitted with a model of heat transport [9] that is based on the bioheat equation, a partial differential equation first proposed by Pennes [16]:

$$\frac{\partial T}{\partial t} = k\frac{\partial^2 T}{\partial x^2} + \frac{A}{\rho c} - \frac{F}{V}(T - T_a)\,. \tag{1}$$

Here T is the local temperature, which depends on position x and time t. The symbol A gives the heat production in W/cm^3, F is the capillary blood flow in ml/sec, V is the volume of the tissue in ml, k is the heat diffusivity which is $1.31 \cdot 10^{-3}$ cm^2/sec, ρ is the density of the tissue, which is 1.06 g/ml in the heart and c the specific heat of the tissue, which is 3.64 J/°C/g. The first term on the right hand side gives the contribution to the local

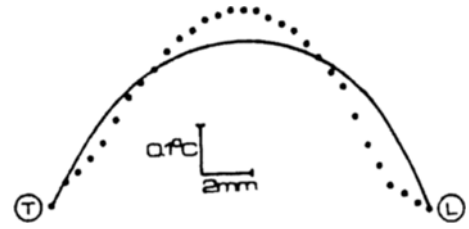

Fig. 2. The steady-state temperature profile across the free wall of the canine left ventricle from epicardium (thoracic side, T) to endocardium (ventricular luminal side, L). Measurements with a thermistor in a needle in an anaesthetized dog are given by the points and the profile calculated from the bioheat equation is given by the continuous line. The blood flow, measured with radioactive microspheres, is about 0.85 ml/g wet weight/min. The temperature of the arterial blood and lumen surface was about 37°C. Modified from [14].

temperature change by heat transport by conduction. The second term gives the contribution by heat that has been metabolically generated. The last term gives the heat exchange between blood and tissue. Here $(T - T_a)$ is the temperature difference between the local tissue temperature and the arterial blood temperature T_a; this difference multiplied by flow gives the amount of heat deposited in tissue or carried out of tissue, assuming that total thermal equilibrium between blood in the smallest blood vessels and tissue has been established, an assumption which holds [17]. Equation (1) is valid if temperature gradients only exist in the x-direction, in our case the transmural direction across the myocardium from outside the heart to the lumen. The temperature profile in the steady state may be calculated by setting $\partial T/\partial t$ equal to zero. Often solutions for various geometrical models may be found from analogous equations given in [18].

However, fitting of the measured profile with the model was not entirely successful, especially in the subendocardial half of the heart muscle. An example of a transmural myocardial temperature profile calculated according to the solution of Eq. (1) for a cylindrical geometry [9] is given in Fig. 2, which can be compared to the measured profile. The measured temperature gradients in the endocardial half of the ventricular wall in this and most other cases become steeper the farther removed from the endocardial surface the measurements were taken; in contrast, the model predicts that the gradients should become smaller toward the middle portion of the heart muscle (see Fig. 2). In the epicardial half of the ventricular wall the gradients do in most cases not become steeper in such a clear way as in the endocardial half. Thus the transport processes considered in Eq. (1), heat exchange between tissue and the local blood flow in the smallest blood vessels and heat conduction through tissue, were insufficient to account for the transmural temperature profile in the left ventricular heart muscle wall. Another remarkable observation that challenges the applicability of Eq. (1) to cardiac heat transport shall now be mentioned.

In an attempt to assess the local rate of metabolism, Ten Velden and the colleagues [19] stopped the coronary arterial blood flow suddenly and measured the resulting increase in local tissue temperature with a small thermistor. They found that in the subepicardial half of the heart muscle wall the local temperature increased after the blood flow was stopped. This was expected because according to Eq. (1) a major part of the heat generated by local metabolism is carried away by the blood flow. Metabolism is not expected to stop immediately when the blood flow is stopped and the generated heat will increase local temperature. However, Ten Velden and colleagues [19] found often in the subendocardial half of the muscle wall that the temperature decreased in the first seconds after they stopped arterial inflow of blood; in the epicardial half the temperature usually increased after flow was stopped.

The finding of decreasing temperature in the first seconds after occlusion of the left coronary main stem Ten Valden [19] suggests that in the subendocardial half of the muscle wall the blood flow does not normally carry heat away but that the blood actually transports heat into the tissue. This phenomenon is incompletely understood and needs to be clarified. Thus the model of heat transport, based on Eq. (1), does not explain two experimental facts: the temperature profile in the subendocardial half of the tissue and the temperature decrease found in the same region when the arterial inflow of blood is stopped.

The decreasing temperature upon occlusion of the left main coronary artery might be explained if the arterial blood flowing through the subepicardial layer is heated up and then heats up the local tissue when it reaches the subendocardial layer. However, by the same reasoning the venous blood, which flows back in general from the inner, cooler layers near the endocardial surface to the outer side of the heart muscle, should then cool the heart tissue it flows through. One would expect that the venous part of the coronary circulation has a higher surface area and slower blood velocities, thus favoring the role of the exchange of heat with the venous blood over the arterial part. This may be offset by the fact that part of the venous blood drains directly into the left ventricular cavity via the Thebesian veins and does not contribute to the transport of heat in the opposite direction, so that the arterial flow, which has a preferential direction from epicardium towards endocardium, is slightly higher. An important role in explaining the decrease of temperature after occluding a coronary artery may be played by the fact that the blood flow in the venous part of the circulation persists for some time after the arterial inflow is topped because of the capacitance (compliance) of the intramyocardial blood vessels. Indeed, Spaan and co-workers [20] have found that the venous outflow decayed with a time constant of several seconds after the arterial inflow was stopped acutely. Thus relatively cool venous blood from near the endocardial surface (see Fig. 2) may cool the tissue toward the middle of the heart muscle layer. The venous blood in the middle of the wall has become warmer and may, together with metabolism, warm up the subepicardial layers, which are not cooled anymore by the arterial blood because of the arterial occlusion.

The models of Hernandez [12] and Ten Velden [9], used for Fig. 2, were based on the bioheat equation of Pennes [16] where heat exchange does take place between the smallest blood vessels and tissue. Other authors, for instance Chen and Holmes [17] and Chato [21], emphasized that exchange of heat between blood and tissue did not occur in the smallest blood vessels but in vessels of intermediate size, with radius in between 10 and 300 μm, which is larger than the capillary radius of about 3 μm, but smaller than the large arteries. Taking this exchange of heat between the intermediate size vessels and tissue into account may explain the measurements of Ten Velden et al. [9] and Duijst et al. [15] of the transmural temperature profile in the heart. However, such an explanation by a full model has yet to be accomplished. In such a model details of the anatomy of the coronary vascular tree may have to be taken into account, and the proximity of arterial and venous vessels probably has to be accounted for. In conclusion, it is clear that the experimental findings on the heat transport in the heart are not yet adequately explained by a comprehensive model of cardiac heat transport.

Heat Transport and Heterogeneity of Blood Flow. The local myocardial perfusion, measured with radioactivelly labelled microspheres, has been found to be very heterogeneous [22]. The heterogeneous flow distribution in the myocardium could be very well described with fractal models because the flow heterogeneity is self-similar. In parts of the whole myocardium, irrespective of size, a similar relative heterogeneity is found as in the whole organ [23]: when one divides a region within the myocardium of arbitrary size in two halves the average flow usually differs by almost 20% between both halves. It is largely unknown whether local blood flow, despite is heterogeneity, is well matched to local metabolism. There is a weak but significant correlation between the local blood flow in the resting heart and the content of the mitochondrial enzyme succinate dehydrogenase [24], but the content of enzyme is not necessarily proportional to local aerobic metabolism.

We developed a model of heat transport in myocardium based on the bioheat equation, Eq. (1), in which the blood flow distribution was assumed to be very heterogeneously distributed according to a fractal model [25, 26]. For lack of knowledge on the distribution of local heat generation with respect to blood flow we assumed that metabolism and heat generation was homogeneous throughout the myocardium; this assumption is very likely not true, but probably represents the worst possible hypothetical case of mismatch of metabolism and blood flow. The flow heterogeneity normally found in myocardium leads only to small changes in the calculated temperature profile when compared with the situation with a hypothetical homogeneous blood flow due to the high conductivity of heat in myocardial tissue; see Fig. 3. In conclusion, the heterogeneity of flow found in the myocardium does not explain the difference in the temperature profile between measurements and model.

Countercurrent Exchange of Heat in the Heart. It is common that small arteries and veins in the muscle wall of the heart lie adjacent to each other, with the blood running in opposite directions. Thus countercurrent heat exchange may be an important feature of heat transport in the heart. Molecules in the arterial blood that exchange

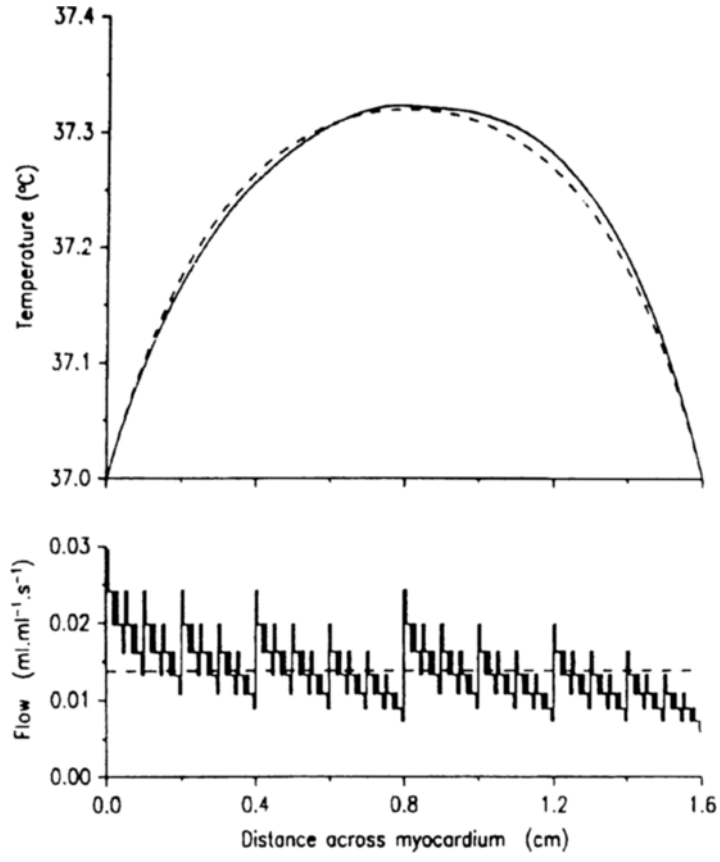

Fig. 3. The steady-state temperature profile across the muscle wall of the left ventricle from epicardium (outer side; $x = 0$) to endocardium (inner side; $x = 1.6$ cm) calculated according to the bioheat equation. The hypothetical blood flow distribution was heterogeneous in this case, and was generated according to a fractal model: the average blood flow in the left half of the myocardium is 110% of the average flow over the whole ventricle, and the blood flow in the right half of the myocardium is 90% of the average flow. Each half of the myocardium itself is divided in turn into two halves, where the average blood flow is heterogeneously distributed between these smaller halves according to the same pattern as found at the larger spatial scale: 110% left, 90% right. This procedure is repeated several times. Thus the pattern of flow distribution is self similar: the relative heterogeneity is similar irrespective of size. Such a self similar pattern is called fractal, and fits the measured blood flow distribution in the myocardium. The temperature profile is calculated on the computer for this heterogeneous blood flow distribution (continuous line) with a finite difference approximation to Eq. (1), and compared with the temperature profile for a homogeneous flow distribution (dashed line).

between the arterial and venous part of the circulation cross over to the venous blood without entering the tissue. Heat, and metabolites such as carbon dioxide that are produced in the tissue, may cross from the venous blood, which leaves the tissue, to the arterial blood and may thus reenter the tissue. Heat or mass is then said to be shunted by countercurrent exchange. Little oxygen [27] and carbon dioxide [28] shunts between arteries and veins. The extent of shunting was even too small to be quantified. However, the diffusivity of heat is much higher than the diffusivity of molecules such as oxygen. We therefore tried to quantify the extent of countercurrent exchange of heat in the myocardium [11, 29].

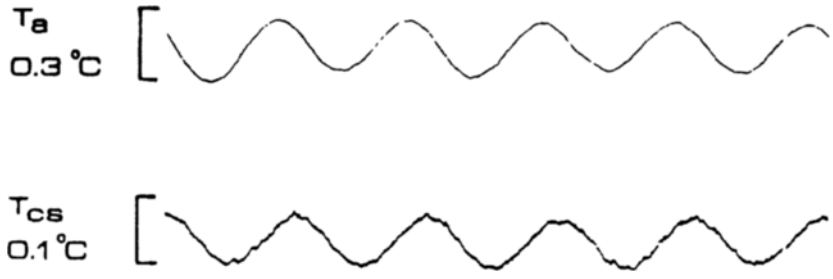

Fig. 4. Example of the venous temperature (T_{cs}) response to sinusoidal variation of the arterial coronary temperature (T_a), measured with a thermistor catheter in the coronary sinus in an anaesthetized dog. The coronary main stem had been cannulated and the coronary circulation was pump-perfused via an extracorporeal circuit fed from a femoral artery. Cold saline solution was infused into the circuit at a sinusoidal rate to obtain the sinusoidal arterial temperature variation.

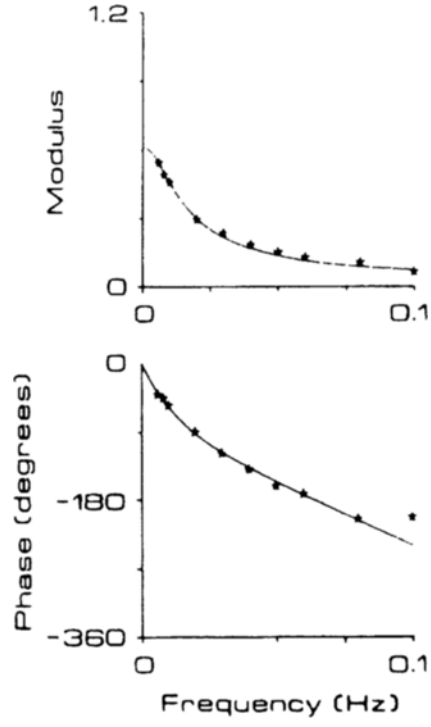

Fig. 5. The response to sinusoidal variation of the arterial temperature in the frequency domain. The modulus (gain) is the amplitude of the venous sinusoidal temperature variation (T_{cs}, see Fig. 4) divided by the amplitude of the arterial variation. The phase shift of the venous sinus relative to the arterial sinus is also shown. The stars give the measured points in one dog; the fit of the model (see text) to the data is given by the continuous line.

We studied the venous emergence of heat infused into a coronary artery of anaesthetized dogs. A cold saline solution was infused at a sinusoidal rate to impose a sinusoidal temperature variation on the coronary arterial blood. The amplitude of the sinus wave of the arterial blood temperature was about 0.2–0.3°C and the frequency was varied from 0.005–0.1 Hz. The resulting sinusoidal venous temperature variation in the coronary sinus was measured with a thermistor catheter. An example of a response is shown in Fig. 4. From such responses at various frequencies, the frequency response was obtained, see Fig. 5.

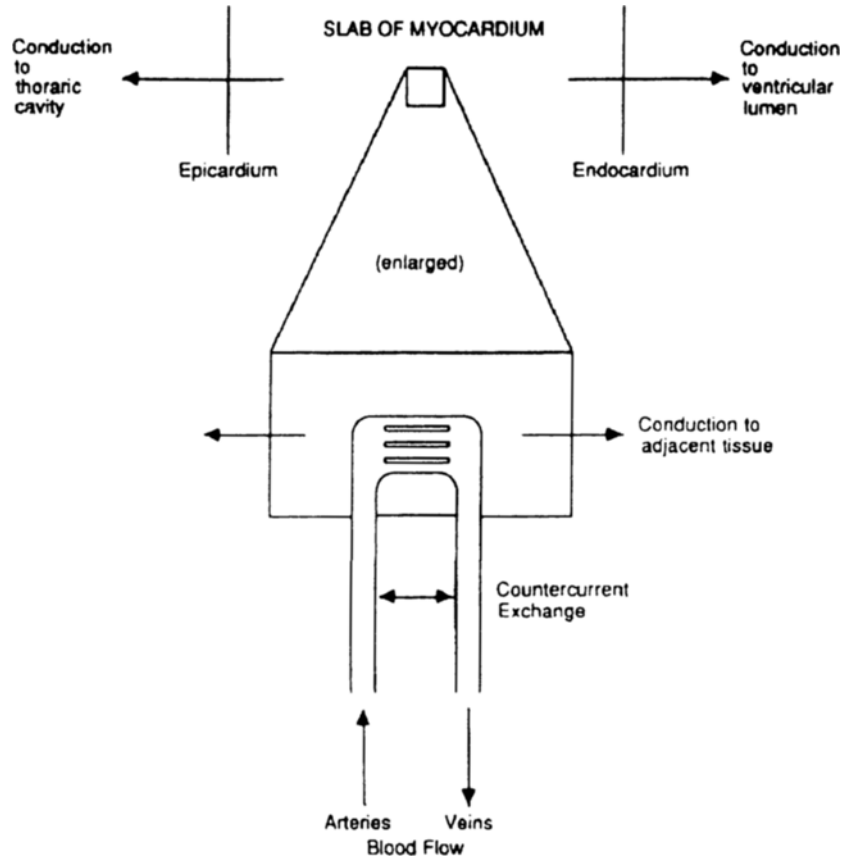

Fig. 6. Schematic representation of model of heat transpor' in the heart. The local tissue is represented by a microvascular exchange region where heat is almost instantaneously distributed. This exchange region is shown in the lower part of the figure as an enlarged view of the left ventricular free wall shown above. Heat is conducted to adjacent microvascular exchange regions, and across the surfaces of the heart to the blood in the left ventricular lumen and to the tissue in the thorax. Arteries and veins or arterioles and venules often lie adjacent to each other so that countercurrent exchange of heat may take place.

The frequency response in the coronary sinus was analyzed with a simplified model of cardiac heat transport, see Fig. 6. The microvascular exchange region was regarded as a mixing chamber in which the heat reaching the tissue was instantaneously distributed due to the high heat diffusivity of the tissue. In the first, simplest form of the model it was assumed for computational ease that the tissue temperature was the same everywhere across the wall of the heart, so that the heart was behaving like a mixing chamber for heat. However, before the heat in the blood was equilibrated with tissue it was subject to countercurrent exchange between the arterial and venous part of the coronary circulation where arterial and venous vessels usually lie adjacent to each other. Heat conduction between heart tissue and the environment across the endocardial and epicardial surfaces of the heart muscle was incorporated in the model. The frequency response, $H(j\omega)$, of coronary venous temperature to infusion of heat indicator into the coronary artery is given by:

$$H(j\omega) = e^{-j\omega t_d}\left[F_s + \frac{f(1-F_s)^2}{(1+j\omega f\tau_m)}\right], \tag{2}$$

j is the complex number $\sqrt{-1}$, ω is the angular frequency: 2π times the frequency of the arterial temperature variation. F_s is the fraction of heat carried by the blood into the countercurrent exchange region that shunts from

artery to vein (or vice versa), t_d is the time taken by transport through the large arteries and veins between the large vessel inlets and outlets of the heart and the countercurrent exchange region. τ_m equals the volume V of the tissue divided by the flow F and is the same as the mean transit time of the tissue for heat (see Eq. (3)) when there is no heat conduction to the environment and no countercurrent exchange. The factor f, which is a well-defined function of the shunt factor F_s and the resistance to heat conduction between tissue and environment, modifies the time constant τ_m which would be found without countercurrent exchange and heat conduction. Thus $f\tau_m$ is the time constant for the transport out of the heart of infused heat that has entered the tissue after bypassing the countercurrent exchange region. The full derivation is given in [11]. In Fig. 5 we see an example of the least squares fit of the model to the data for one dog. Thus the parameters in Eq. (2) were estimated by optimization, and the F_s, the fraction of heat entering the countercurrent exchanger was estimated to be 0.62. Thus 62% of the heat infused in an artery would shunt to the venous blood in the countercurrent exchanger; the same fraction of heat carried out of the heart in the venous blood would be expected to shunt to the arterial blood and be carried back into the tissue.

The conduction of heat over a distance of 1 mm appears to be fast enough with respect to the transit time of blood through the microvascular exchange region for a tissue unit of volume 1 mm^3 to be considered a mixing chamber in the model, but this is not the whole heart muscle wall which is more than 1 cm thick in the dog. Therefore the mixing chamber model above, with uniform transmural temperature, was modified to take transmural heat conduction resulting in heat loss across the surfaces of the heart into account ([25]; Appendix B in [11]). This entailed solving the time dependent form of Eq. (1) for a slab of tissue with temperature not the same across the wall of the heart. However, despite the fact that this slab model was much more complicated than the mixing chamber model, the result for F_s was almost the same.

Analysis of the model behavior showed that countercurrent exchange alone does not diminish the mean transit time of the injected heat. However, loss of heat by conduction to the environment shortens the mean transit time of arterially infused heat. The effect of countercurrent exchange on the mean transit time of heat through the heart may be understood by thinking about a diffusing "quantum" of heat, in analogy with a diffusing molecule: the close mathematical analogy between molecular diffusion and heat conduction allows this way of looking at heat conduction. Consider an organ with a certain distribution of transit times of heat quanta that enter in the arterial blood and can leave only via the venous blood because heat conduction is assumed not to exist. The mean transit time $\bar{t}$ of the heat quanta through the organ is equal to the volume, V, times the partition coefficient devided by the flow F [30]

$$\bar{t} = \frac{\lambda V}{F} . \tag{3}$$

The partition coefficient λ is equal to the specific heat of cardiac tissue divided by the specific heat of blood, and was found to be equal to 1. Now suppose that heat conduction out of the organ is also allowed. Some of the diffusing heat quanta will now leave the organ. The longer a heat quantum is present in the organ performing its random walk, the larger the chance that this heat quantum will leave the organ by diffusion. As a consequence the apparent mean transit time determined from the heat quanta leaving via the blood becomes smaller, and the apparent volume of distribution calculated via Eq. (3) consequently also becomes smaller. However, when many heat quanta leaving the heart via the venous blood are shunted back into arterial blood to reenter the tissue in the regions of countercurrent exchange, these quanta remain in the tissue very long and have a larger chance of leaving the heart by conduction rather than via the blood. Thus the extent of reduction of the mean transit time from the value calculated from Eq. (3) gives information about heat conduction to the environment and about countercurrent exchange.

Experimentally Duijst and co-workers [11] found that 24% of the infused heat was lost by conduction to the environment. The mean transit time was estimated from the frequency response of the phase and amplitude of the coronary venous temperature to the imposed sinusoidal variation in arterial temperature and was about 14 sec. This was only 1.8% of the mean transit time calculated from Eq. (3). The 24% loss of heat by conduction does

explain a reduction of the mean transit time, but the full reduction to 18% is explained if besides the 25% heat loss by conduction 62% of the heat shunts in the countercurrent exchanger.

Injection of cold saline solution in an epicardial vein, running on the surface of the heart, did not result in measurable temperature changes in the adjacent artery, despite the fact that artery and vein lie alongside each other for 2 cm. The peak of the temperature change measured with a thermistor in the myocardial tissue after a bolus injection of cold saline into the left atrium was only one quarter of the peak of the temperature change measured in the venous blood in the coronary sinus where coronary venous blood from the ventricles emerges. This also indicates that a substantial fraction of the injected heat shunts before arrival in the bulk of muscle tissue. It is true that heterogeneity of tissue perfusion cause phenomena that are similar to countercurrent exchange: early emergence of indicator via tissue regions with high blood flow and small temperature changes in regions with poor blood flow. However, the heterogeneity of tissue perfusion would have to be bigger than actually found to explain the measured temperature effects ([25], Appendix C in [11]). Therefore it is likely that shunting of heat by countercurrent exchange is quantitatively considerable in the heart and occurs at the arteriolar level. However, the contribution by flow heterogeneity needs further evaluation.

Because we may assume that heat conduction works both ways, a considerable fraction of the heat generated in the tissue and leaving the heart via the blood is shunted from the venous blood leaving the tissue into the cooler arterial blood back into the tissue. Thus shunting of heat should contribute to higher tissue temperatures. We see that transport of heat by the blood is very important in the heart, but on the other hand heat conduction is responsible for carrying a substantial fraction of the generated heat out of the heart. However, the transport of heat out of the region of vascular countercurrent exchange by the blood stream must be rather effective because more than half of the infused heat seems to shunt in these vascular countercurrent exchange regions and does not reach the bulk of tissue by conduction from the countercurrent exchange regions, but instead is carried out of the organ by the blood.

Summary and Conclusion. Almost half the heat generation in the heart takes place during the contraction. The rest of the heat is generated when the ATP is slowly resynthesized by oxidative phosphorylation. A substantial part of the generated heat leaves the heart by conduction, but most of the heat is transported out of the heart by the blood. Heat transported by the blood is subject to countercurrent exchange between small arteries and veins that lie adjacent to each other. In the countercurrent exchange region convective transport by the blood may compete effectively with heat conduction into the bulk of tissue. Heterogeneity of perfusion might to some extent produce effects that are similar to the effects countercurrent exchange. Despite all this knowledge about myocardial heat transport, it is clear that our model description of heat transport is far from perfect and does not explain all experimental data in a satisfactory way.

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