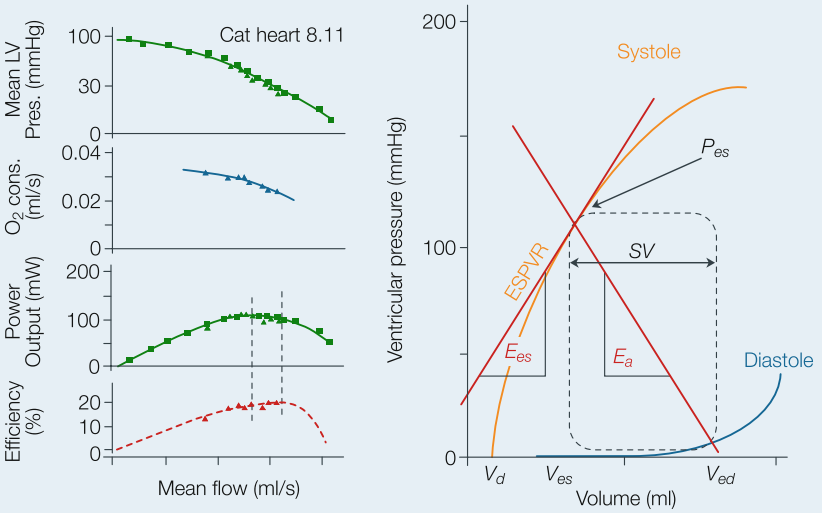
Chapter 17
Cardiac Power and Ventriculo-Arterial Coupling



The heart pumps close to maximal external power and efficiency *(left)*. To test this, ventriculo-arterial coupling analysis is shown on the *right hand side*. On the left relations are shown as a function of *CO*. The *top panel* shows the Pump Function Graph, the *second panel* Oxygen Consumption, and the *third and fourth panels* show Mean Power Output and Cardiac Efficiency. The data were obtained by changing the arterial load while keeping the cardiac filling, contractility and heart rate constant. Both external power and efficiency exhibit a maximum. These maximums are not found at the same flow because O_2 consumption decreases with larger flow, i.e., with lower ventricular pressure (see Chap. 16). Both power and efficiency exhibit shallow relations without sharply defined maximum implying that under normal conditions the heart pumps close to maximal efficiency and maximal power, and efficiency is about 20–25%. The remaining energy is dissipated as heat and carried away by the coronary circulation and by diffusion to the thorax and cardiac lumen. It has been reasoned that the optimum of power results from size constraints of the heart. Whether the heart is pumping at optimal efficiency or optimal power (ventriculo-arterial coupling), or neither, depends on both the cardiac and the arterial state. The *right hand figure* shows how to analyze coupling. *The slope* of the ESPVR (E_{es}, see Chap. 13) characterizes the ventricle and the effective arterial elastance (E_a), which approximately equals total peripheral resistance over heart period, R/T, characterizes the arterial system. When $E_a / E_{es} \approx 1$, external work is maximized, and when $E_a / E_{es} \approx 0.5$ cardiac efficiency is maximal.

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Description

Power and Efficiency

Cardiac efficiency is defined in analogy to that of a hydraulic pump. The external power is calculated from pressure times flow (Chap. 15) and the 'input power' is calculated from cardiac oxygen consumption (Chap. 16). The ratio of external, or produced power, and input power is defined as efficiency. Therefore, both external power and input power need to be expressed in the same units. When glucose or free fatty acids are consumed oxygen consumption can be expressed in Joules and oxygen consumption per time in Watts, through the so-called caloric equivalent. For carbohydrate and fat metabolism it holds that 1 ml $O_2 \approx 20$ J and 1 ml O_2/min ≈ 0.33 W. A review on cardiac energetics has been published by Suga [1].

Maximum Cardiac Efficiency and Maximum Power in the Intact Animal

The pressure and flow generated by the heart and the arterial load can be studied while keeping the Heart Rate, diastolic filling and contractility unaltered [2]. Power can be calculated from the pressure and flow. The Figure in the box, on the left, shows that, when power is plotted as a function of Cardiac Output it exhibits an optimal value. This can be understood with the cardiac pump function graph in mind, (top panel of box Figure, Chap. 14). For a high load (isovolumic contraction) pressure generated is high but flow is zero. Power, the product of pressure and flow, is therefore negligible. Inversely, for a very low load the heart generates a large flow but negligible pressure so that power is negligible as well. Thus, power, at some intermediate value of Cardiac Output, must be maximal. This power maximum was found, in the intact cat [3], to coincide with the working point, i.e., when a physiologic arterial load is present (see Fig. 17.1). It has also been reported that the left ventricle works at maximal efficiency [4].

Local Work and Power

It is of great interest if local power and efficiency could be derived. However, this is difficult since local work, and local oxygen consumption are hard to measure accurately. Local work can be derived by local shortening times local stress. The first, local shortening can be obtained from (surface) markers attached to the muscle or by MRI-tagging (magnetically induced markers in the myocardium). Local forces are only possible to derive indirectly from pressure and anatomy

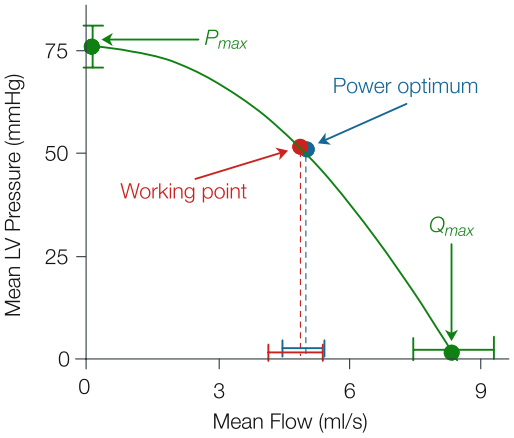

Fig. 17.1 The heart pumps at optimal power output. Power output of the heart studied in the intact animal for different arterial loads. Other determinants of pressure and flow, heart rate, diastolic filling and contractility, are kept constant. When the physiological arterial load is present, shown as the working point, power transfer is maximal. Adapted from ref. [3], used by permission

(sophisticated forms of Laplace's law, Chap. 9). This technique was used by Prinzen et al., to study effect of pacing site on local work [5]. When local oxygen consumption is also determined [6] local efficiency can be derived. However, local stresses derived from pressure are difficult to verify.

Heat Production and Transport

The heart is about 20–25% efficient. This implies that about 75% of the oxygen consumed is converted into heat. The heat is removed by diffusion to lumen and thorax, and convection by coronary flow, in about equal amounts (Fig. 17.2). In the mid-wall of the myocardium the temperature is a few tenths of a degree Celsius higher than in subepicardial and subendocardial layers allowing for diffusion of heat [7].

Assessment of Ventriculo-Arterial Coupling

Optimum power and efficiency are assumed measures of good ventriculo-arterial coupling. Whether the heart functions on optimum power or efficiency can be derived from hemodynamic principles. The two parameters assumed to be the determining ones are the slope of the left ventricular End-Systolic Pressure–Volume Relation, E_{es} or E_{max}, and effective arterial elastance, E_a. The effective arterial elastance is defined as $E_a = P_{es}/SV$, i.e., end-systolic (ventricular or aortic) pressure

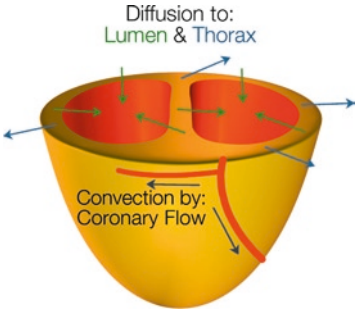

Fig. 17.2 Oxygen consumption produces not only mechanical power but also heat. Convection by coronary flow and diffusion to thorax and cardiac lumen together, each account for about 50% of the heat loss, depending on the magnitude of the coronary flow

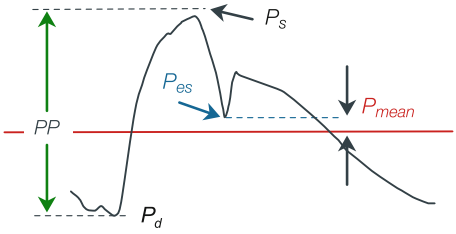

Fig. 17.3 End-systolic pressure is close to mean aortic pressure, allowing for non-invasive determination of P_{es}

over Stroke Volume, (right side of the Figure in the box). The ratio E_a/E_{es} is considered a ventriculo-arterial coupling parameter and when $E_a/E_{es} \approx 1$ external work is maximized, while for $E_a/E_{es} \approx 0.5$ cardiac efficiency is maximal [8].

To determine these two parameters, several simplifications have been used. The E_a can be approximated as follows. End-systolic pressure is close to mean arterial pressure (Fig. 17.3). With Cardiac Output, *CO*, being *SV HR,* and Heart Period *T*, in seconds, the inverse of *HR*, $CO = SV/T$, we find that $P_{es}/SV \approx P_{mean}/CO \cdot T = R_p/T$. Thus the effective arterial elastance, E_a, is primarily a measure of vascular or peripheral resistance, R_p, and hardly reflects the compliant properties of the large conduit arteries. Therefore the term 'elastance' is misleading. Note that E_a depends on vascular resistance, which is a purely arterial variable, and on heart period, *T*, which is a purely cardiac variable. Therefore, E_a is a coupling parameter by itself. The E_a can be derived from noninvasive measurements: mean pressure (by sphygmomanometer) and Cardiac Output (US or MRI), and Heart Rate.

The maximum or End-Systolic elastance, E_{es} is calculated from $E_{es} = P_{es}/(V_{es} - V_d)$. End-Systolic volume can be measured noninvasively, but V_d is hard to estimate. To derive this intercept volume, at least one other point on the ESPVR should be obtained. This would require changes in diastolic filling that are often not feasible in very sick patients and in epidemiological studies. One method to determine the End-Systolic Pressure–Volume Relation is the one suggested by

Sunagawa et al. [8], where an isovolumic left ventricular pressure is predicted from the pressure of an ejecting beat (Single Beat Method). However, none of the so-called single beat methods to determine the ESPVR has been shown to give accurate estimates [9]. Nevertheless, the Single Beat Method is, at present the best approach in practice.

In a number of studies it has simply been assumed that $V_d = 0$ [10, 11]. This assumption leads to a very interesting simplification of the analysis. With the $V_d = 0$, $E_{es} = P_{es}/V_{es} = P_{es}/(V_{ed} - SV)$. The ratio E_a/E_{es} then becomes equal to:

$$E_a / E_{es} = (P_{es} / SV) / [P_{es} / (V_{ed} - SV)] = (V_{ed} - SV) / SV = 1 / \mathrm{EF} - 1$$

with EF, the Ejection Fraction. We see that the P_{es} disappears altogether only leaving the determination of Ejection Fraction. This implies that work is maximal when $E_a/E_{es} = 1$, thus when EF = 0.5. Similarly, cardiac efficiency is maximal when $E_a/E_{es} = 0.5$ or when EF = 0.67.

Thus the assumption of a negligible V_d simplifies matters. However, negligible V_d values are difficult to verify, mostly not correct, and are certainly leading to large errors in case of a dilated heart (Chaps. 13 and 15).

The effective arterial elastance (E_a), has the units of elastance but is not an elastance; it is closely related to total peripheral resistance. It is approximately R_p/T and thus is not related to total arterial compliance. Several authors [12] have interpreted E_a as a measure of total arterial compliance, an interpretation that leads to confusion and is clearly wrong.

Theory of Optimal Heart Size

Why does the left ventricle pump at maximum power, while a feedback control for power is not known to exist? A simple answer to this question can be given based on the following reasoning [13]. Consider the pump function graph (Fig. 17.4). The working point, i.e., the point where maximum power is found, is for a flow which is about 58% maximal flow, Q_{max}. Mean ventricular pressure and Cardiac Output together determine the working point. Pressure is similar in mammals and Cardiac Output is determined by body size (Chap. 30). Several pump function graphs can be drawn through this working point. We begin by assuming that muscle stress is a given quantity, and that the ventricle is a sphere. On the one hand, a larger intercept of the pump function graph with the flow axis, i.e., a larger Q_{max}, implies a larger ventricular lumen requiring a thicker wall (Law of Laplace, Chap. 9), to maintain muscle, or wall stress. On the other hand, with a larger Q_{max} a smaller P_{max} results so that the wall may be less thick. In this way it is possible to calculate ventricular volume for the different pump function graphs through the working point, each with its own Q_{max}. Plotting ventricular volume as a function of Q_{max}, results in the graph given in Fig. 17.4. The minimum volume is found when the working point is at about 60% of Q_{max}, and this is the same value as where maximum power and efficiency

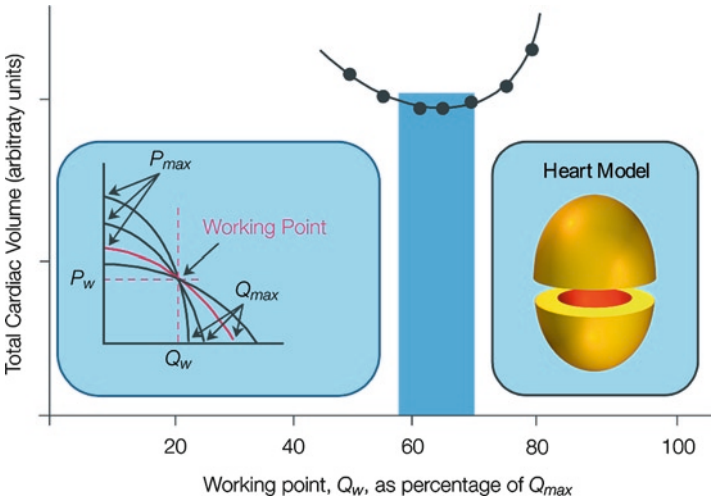

Fig. 17.4 Total ventricular volume, i.e., wall plus lumen volume, can be calculated assuming a spherical shape, right, and a fixed, maximal, wall stress, σ_m, for isovolumic contractions. Thus P_{max} relates to wall volume (Chap. 9), and larger P_{max} implies larger wall volume to keep wall stress the same. Increased Cardiac Output relates to ventricular lumen and wall volume. Many Pump Function Graphs through the working point are possible, left, but the Pump Function Graph where the working point is about 60% of Q_{max}, i.e., where maximal power and efficiency are found, corresponds with the smallest total (wall plus lumen) ventricular volume. Adapted from ref. [13], used by permission

are found. The minimum in heart volume thus corresponds to a working point where power and efficiency are about maximal. In other words, the size of the heart is minimized, and, for this minimal heart volume the heart pumps at maximal power.

Physiological and Clinical Relevance

Cardiac oxygen consumption and efficiency are still difficult to obtain in the patient. Modern techniques, such as Positron Emission Tomography (PET) and Magnetic Resonance Spectroscopy (MRS) may change this. Assessment of glucose metabolism with ^{18}F-fluorodeoxyglucose measures glucose uptake into myocardial cells, but not its conversion by glycolysis. Myocardial oxidative metabolism can be measured by ^{11}C-labeled acetate PET. For lipid metabolism, tracer examples are ^{123}I-beta-methyl-p-iodophenyl pentadecanoic acid and ^{15}-(O-^{123}I-phenyl)-pentadecanoic acid. These tracers can be detected by planar scintigraphy and single-photon emission computed tomography (SPECT), which are more economical and more widely available than PET. With current MRS techniques, ^{31}P-labeled magnetic resonance spectroscopy, Phosphate/Creatine and/or pH can be obtained in humans but this is not common yet. The hemodynamic determinants for oxygen consumption, as discussed in Chaps. 16 and 17,

are only valid in single hearts during acute interventions and cannot be used in comparing different patients.

'Output power' requires the measurement of aortic or ventricular pressure and flow. Thus for the calculation of efficiency, which is the ratio of 'Output power' and 'Input power', many measurements are required and therefore efficiency is not calculated routinely. In Chap. 30 it is shown that in healthy mammals cardiac metabolism is proportional to body mass to the power −1/4, implying that metabolism per gram of heart tissue decreases with body mass.

Related Issues

Contractile efficiency. On the basis of the Pressure Volume Area concept (Chap. 16) the contractile efficiency has been defined as the inverse of the slope of the Pressure Volume Area – VO_2 relation [1]. This definition of efficiency only accounts for the mechanical aspects of oxygen consumption and does not take into account the oxygen expenditure related to activation and basic metabolism. Therefore this contractile efficiency is about twice the actual cardiac efficiency.

Power in cardiogenic shock. Although power is a rather abstract measure, it has been shown that it is the strongest hemodynamic correlate of mortality in cardiogenic shock [14].

Economy. At the extremes of the pump function graph, the heart generates neither pressure nor flow. External power and thus efficiency is zero. In isolated heart studies (Langendorff preparations) where the heart contracts isovolumically or in isolated cardiac muscle studies, when the muscle contracts isometrically, contraction economy can be used instead. Economy of contraction is defined as oxygen consumption used for isovolumic or isometric contractions.

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