Comparative Effects of Heart Rate and Aortic Blood Pressure on $M\dot{V}O_2$ in the Anesthetized Open-Chest Dog

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SUMMARY

Comparative effects of heart rate and aortic blood pressure on myocardial oxygen consumption ($M\dot{V}O_2$) were studied in anesthetized open-chest dogs. The left coronary artery was perfused through the external shunt with blood from the left common carotid artery. Heart rate was changed with the left atrial pacing and the constriction of the descending thoracic aorta was utilized to elevate the proximal aortic blood pressure (BP). The left ventricular enddiastolic pressure was insignificantly changed with the atrial pacing and with the aortic constriction. With the constant BP (100 ± 5 mmHg), an unit increase in heart rate per minute augmented $M\dot{V}O_2$ by 0.027 ml/min/100 Gm of left ventricular muscle, while one mmHg elevation of BP caused 0.15 ml increment in $M\dot{V}O_2$ per minute per 100 Gm of left ventricular muscle under the constant heart rate (115 ± 5 beats/min). Therefore an unit change in BP caused five- to six-fold greater increase in $M\dot{V}O_2$ compared with that in heart rate, indicating extremely important roles of BP in $M\dot{V}O_2$.

Additional Indexing Words:

Atrial pacing Cardiac frequency Aortic constriction Left ventricular afterload Myocardial oxygen requirement

MAJOR determinants of myocardial oxygen consumption ($M\dot{V}O_2$) are heart rate, myocardial wall tension and myocardial contractility.[1] Since myocardial wall tension is expressed as the products of the ventricular pressure, ventricular radius, and wall thickness and because of difficulty to measure accurately the ventricular radius and myocardial contractility, heart rate and systemic blood pressure have been used clinically for estimating the left ventricular oxygen consumption.[2),3] There are very few reports available, however, concerning the comparative effects of heart rate and systemic

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blood pressure on $M\dot{V}O_2$.

The purpose of the present paper is to compare the effects of an unit change in heart rate and systemic blood pressure on $M\dot{V}O_2$ in the anesthetized open-chest dog.

Methods

Controlled mongrel dogs were anesthetized with 3 mg/Kg of morphine hydrochloride (subcute) followed 30 min later by the intravenous injection of α-chloralose (110 mg/Kg). The dog was ventilated through an endotracheal tube with gas mixture of 30% O_2 in air by a Harvard respirator pump. The heart was exposed through a left lateral thoracotomy and the left coronary artery was perfused via a Gregg cannula with blood from the left common carotid artery. Coronary blood flow was measured with an electromagnetic flowmeter interposed in the perfusion line and an electromanometer was connected to this line just proximal to the cannula for monitoring perfusion pressure. The left ventricular pressure was measured with an electromanometer through a short polyethylene tube inserted into the left ventricle via the apex. A point-tip side hole catheter was inserted transepicardially into the coronary sinus and sutured to the epicardium served for collection of coronary sinus blood. The dog received a dose of 7,000–10,000 units of heparin (iv) just prior to coronary artery cannulation and thereafter 1,500 units every 15 min for preventing coagulation. A slow intravenous infusion of 1.5 M $NaHCO_3$, adjustments of tidal volume and ventilation maintained arterial blood Po_2, Pco_2, and pH within the physiological ranges. Coronary blood flow, left ventricular pressure, and perfusion pressure were recorded continuously. Control samples of arterial and coronary sinus blood were collected anaerobically and stored in ice. Then, either of following procedures was conducted: 1, the descending thoracic aorta was constricted by an umbilical tape to raise proximal aortic blood pressure (coronary perfusion pressure) by 30–60 mmHg or 2, the left atrial appendage was paced electrically at the rate of 30–90 beats/min above the control. After the new steady state was established, the sampling of arterial and coronary sinus blood was repeated, and then the intervention was stopped allowing the dog to return to the control. After a 20 min recovery period, a different procedure was conducted in the same manner described above. On the average one dog received 3 or 4 levels of the pacing and 1 or 2 levels of the aortic constriction.

Blood Po_2, Pco_2, and pH were measured with a Corning Model 165/II analyser calibrated with a reference gas mixture between each sample. Duplicate estimates of Po_2 and Pco_2 differed by 1 mmHg or less and of pH by less than 0.05 unit. Blood oxygen content was estimated with a Lex-O_2-Con TL fuel cell and duplication agreed within 0.1 ml/100 ml.

After the interventions were over, the dog was killed by a cardiac injection of a saturated KCl solution. The heart was removed and the cannulae of the left coronary artery and coronary sinus were insured to be in proper position, and then the left ventricle was weighed after trimming the heart.

Results

Relationship of heart rate and $M\dot{V}O_2$

Out of 102 runs in 24 dogs paced electrically, 46 in which mean perfusion pressure was in the range of 100±5 mmHg were analyzed. The heart rate and the left ventricular enddiastolic pressure during the control period were 112±18 beats/min and 6±2.0 mmHg (mean±SD), respectively. The left ventricular enddiastolic pressure was insignificantly decreased to 4±

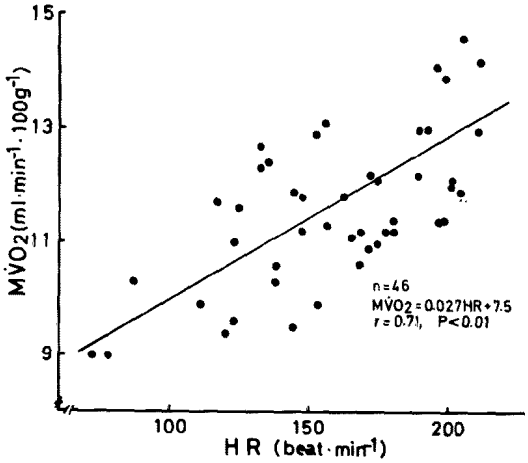

Fig. 1. Relation of $M\dot{V}O_2$ to heart rate (HR). The blood pressure was kept constant (100±5 mmHg). The close relationship was observed between 2 parameters ($r=0.71$, $p<0.01$).

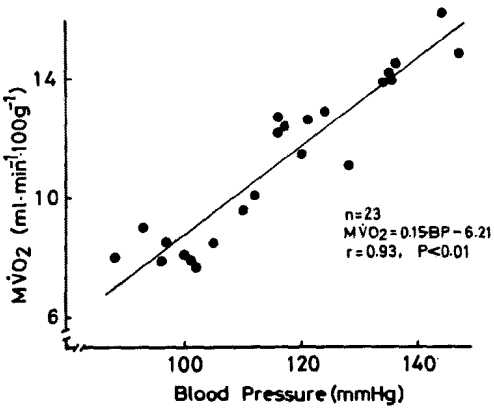

Fig. 2. Relation of $M\dot{V}O_2$ to the proximal aortic blood pressure (blood pressure). The blood pressure was varied with changing the level of constriction of the descending thoracic aorta. The heart rate was kept constant (115±5 beats/min). The close linear relationship was observed between 2 parameters ($r=0.93$, $p<0.01$).

1.8 mmHg by the atrial pacing. With atrial pacing, $M\dot{V}O_2$ varied in close relationship with heart rate in the range of 70 to 220 beats/min, as shown in Fig. 1. The regression equation was as follows: $M\dot{V}O_2$ (ml/min/100 Gm LV)=0.027×(heart rate)+7.5 ($r=0.71$, $p<0.01$).

Relationship of proximal aortic blood pressure and $M\dot{V}O_2$

Aortic constriction decreased heart rate significantly, while the left ventricular enddiastolic pressure did not change significantly: 6±2.5 mmHg during the control period and 7±3.2 mmHg with the aortic constriction. In twenty-three out of 42 runs (17 dogs) heart rate was in the range of 115±5 beats/min and they were used for analyzing the relationship between proximal aortic pressure and $M\dot{V}O_2$. In Fig. 2, $M\dot{V}O_2$ was plotted against proximal aortic pressure (blood pressure). Regression analysis revealed the close linear relation between two parameters: $M\dot{V}O_2$ (ml/min/100 Gm LV)=0.15× blood pressure)–6.21 ($r=0.93$, $p<0.01$).

Discussion

It is well known that heart rate and left ventricular afterload are major determinants of myocardial oxygen requirement. The $M\dot{V}O_2$ varied markedly with the change of heart rate (cardiac frequency) over a wide range.[4),5)] Changing the average heart rate from 100 to 200 per minute caused more than double increase in the $M\dot{V}O_2$.[6)] It is evident therefore that the heart rate is an important determinant of the $M\dot{V}O_2$ primarily through an increased or decreased number of contractions per minute. The present data also indicate a close linear relationship between the heart rate and the $M\dot{V}O_2$ under the approximately constant blood pressure. In the range of the heart rate from 80 to 220 per minute, increasing an unit heart rate (one beat) per minute augmented the $M\dot{V}O_2$ by 0.027 ml/min/100 Gm of left ventricular muscle.

The development of tension by muscle is associated with high energy phosphate utilization and the release of heat.[7)] In the intact heart the development of the left ventricular pressure by active contraction is closely associated with an additional consumption of O_2. McDonald et al[8)] and Monre and French[9)] indicated that the relation between peak wall stress and $M\dot{V}O_2$ appeared almost linear, and five or sixfold increase in $M\dot{V}O_2$ can be produced along the isovolumetric length-tension relationship of the isolated heart. In detailed studies on the isolated supported heart preparation of the dog it was shown that the area beneath the left ventricular pressure pulse per minute, the tension-time index, had a direct correlation with oxygen consumption over a wide range of the heart rate, aortic pressure, and cardiac output.[10)] The product of mean aortic pressure and heart rate, instead of the tension-time index,

is still useful for estimating the directional changes in $M\dot{V}O_2$ under some circumstances.[3] In the present investigation the aortic constriction distal to the left subclavian artery caused a marked increase in the proximal aortic pressure without significant changes in the left ventricular enddiastolic pressure under the constant heart rate. This indicates that under this condition mean aortic pressure reflects well the left ventricular wall tension. Fig. 2 showing the linear relation of aortic pressure and $M\dot{V}O_2$ confirmed this presumption. In the range of blood pressure from 85 to 115 mmHg an unit increase in blood pressure spent additional 0.15 ml/min/100 Gm of left ventricular muscle; five- or six-fold greater change in the $M\dot{V}O_2$ was produced by altering an unit blood pressure in comparison with an unit change in the heart rate. Therefore it should be emphasized that blood pressure, left ventricular afterload, has a greater effect on $M\dot{V}O_2$ than heart rate and is an extremely important determinant of the $M\dot{V}O_2$

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