Right ventricular oxygen supply/demand balance in exercising dogs

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Hart, Bradley J., Xiaoming Bian, Patricia A. Gwirtz, Srinath Setty, and H. Fred Downey. Right ventricular oxygen supply/demand balance in exercising dogs. Am J Physiol Heart Circ Physiol 281: H823–H830, 2001.—This is the first investigation of right ventricular (RV) myocardial oxygen supply/demand balance in a conscious animal. A novel technique developed in our laboratory was used to collect right coronary (RC) venous blood samples from seven instrumented, conscious dogs at rest and during graded treadmill exercise. Contributions of the RV oxygen extraction reserve and the RC flow reserve to exercise-induced increases in RV oxygen demand were measured. Strenuous exercise caused a 269% increase in RV oxygen consumption. Expanded arteriovenous oxygen content difference (A-VΔO₂) provided 58% of this increase in oxygen demand, and increased RC blood flow (RCBF) provided 42%. At less strenuous exercise, expanded A-VΔO₂ provided 60–80% of the required oxygen, and increases in RCBF were small and driven by increased aortic pressure. RC resistance fell only at strenuous exercise after the extraction reserve had been mobilized. Thus RC resistance was unaffected by large decreases in RC venous PO₂ until an apparent threshold at 20 mmHg was reached. Comparisons of RV findings with published left ventricular data from exercising dogs demonstrated that increased O₂ demand of the left ventricle is met primarily by increasing coronary flow, whereas increased O₂ extraction makes a greater contribution to RV O₂ supply.

Myocardial oxygen consumption (MV\textsubscript{O₂}) reflects the oxygen demand of the myocardium when oxygen supply is not limited; coronary blood flow and oxygen extraction reflect the coronary vascular responses required to meet the myocardial oxygen demand. This study was designed to define contributions of RC blood flow (RCBF) and oxygen extraction reserves, which might be mobilized to increase myocardial oxygen supply when RV oxygen demand is increased by graded treadmill exercise.

Numerous investigations have focused on cardiac hemodynamic and metabolic responses to dynamic exercise. LV oxygen extraction is high (~75%) at rest, therefore, the LV oxygen extraction reserve available for increasing LV oxygen supply during exercise is small (14, 40, 41, 43). Thus increases in LV MV\textsubscript{O₂} during exercise always produce concomitant increases in coronary blood flow (1, 2, 12, 14, 16, 18, 39, 40, 41, 43). On the other hand, the RV has a large extraction reserve at rest (5, 17, 22, 26, 28), and this reserve might be mobilized to contribute significantly to RV oxygen supply during increases in RV oxygen demand. The RV also has a large RC flow reserve (8, 21, 27, 33, 41), and several studies have reported that RC flow increases during exercise (2, 23, 25, 30, 31). Interplay between the RV flow and oxygen extraction reserves has not been described in the conscious animal.

In this investigation, we observed that the RV oxygen extraction reserve was mobilized as exercise progressed, and this reserve contributed importantly to RV oxygen supply during exercise. RC flow initially increased along with an increase in aortic blood pressure, but only during strenuous exercise did RC vascular resistance fall. In addition to defining contributions of RC flow and RV oxygen extraction reserves to RV oxygen supply during exercise, these findings demonstrate that RC resistance is insensitive to large changes in venous oxygen tension, at least above a critical threshold. Results are compared with published LV data, and important differences in mechanisms of ventricular oxygen balance are apparent.

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MATERIALS AND METHODS

Animal instrumentation. This investigation was approved by the Institutional Animal Care and Use Committee and was conducted in accordance with the Guide for the Care and Use of Laboratory Animals (NIH Publication 85-23, Revised 1996). Seven adult mongrel dogs of either sex, weighing 24–32 kg, were studied.

Thirty minutes after preanesthesia treatment with PromAce (0.03 mg/kg im), anesthesia was induced by thiopental sodium (5 mg/kg iv). After endotracheal intubation, a surgical plane of anesthesia was maintained by mechanical ventilation with isoflurane gas (1–3%) with equal offset of oxygen (1 liter). Under sterile conditions, a thoracotomy was performed in the fourth right intercostal space, and the dog was instrumented as illustrated in Fig. 1. A Tygon catheter (0.04 in. inner diameter, 0.07 in. outer diameter) was inserted into the aorta through the right internal mammary artery to measure aortic blood pressure. A Königsberg model P6.5 pressure transducer was inserted through a stab wound in the RV infundibulum and secured with a purse-string suture. A nonbranching section of the right coronary artery (RCA) was dissected free for 1–2 cm to affix a 2-mm diameter Transonics flow transducer. A coronary venous catheter prepared from Micro-Renathane tubing (type MRE-025, 0.012 in. inner diameter, 0.025 in. outer diameter) was inserted into a superficial vein draining the RV myocardium, as described previously (6). The venous catheterization site was in the central region of the RV free wall, well within the perfusion territory of the RCA (20).

At the conclusion of the instrumentation, catheters and wires were brought out of the thorax through the third and fifth right intercostal spaces, tunneled under the skin, and exteriorized between the shoulders through individual puncture wounds. The chest was closed, and the pneumothorax was evacuated through a chest tube. Antibiotic (Clavamox, 6.25 mg/lb., bid, po) and aspirin (162–300 mg po) were given for 10 days after surgery. The RC venous catheter was attached to an Access Technologies “C” Series Balloon Pump (60-ml volume elastomeric pump, 2.0 ml/h flow rate) for continuous infusion of heparinized saline (10 U/ml) for the duration of experimentation. The RC catheter was flushed with heparinized saline (10 U/ml) and filled with heparin (5,000 U/ml) daily. Other catheters were treated similarly at 3-day intervals.

Data collection. After the animals recovered from the surgical procedures, resting measurements were obtained with the animal standing quietly on a treadmill. RC flow was measured with a Transonic T106 series flowmeter. A disposable Isotect pressure transducer (Quest Medical) was positioned at midheart level and connected to the mammary artery catheter to measure aortic blood pressure (AoP). AoP, RCBF, and RV pressure (RVP) were recorded on a multichannel Coulburn chart recorder.

Blood sample collection and analyses. Arterial and RC venous blood samples were collected to determine oxygen content, Po2, and glucose and lactate concentrations. All samples were collected anaerobically and chilled on ice until analyses. Oxygen content was measured with an Instrumentation Laboratory model 682 Co-Oximeter, and Po2 was measured with an Instrumentation Laboratory Synthesis 30 blood gas analyzer. MVo2 was determined by multiplying the RC arteriovenous difference of oxygen content (A-V O2) by RCBF, normalized per gram tissue mass. Oxygen extraction was computed as A-V O2 divided by the arterial oxygen content multiplied by 100. Blood glucose and lactate concentrations were determined by a Radiometer model EML105 Metabolite Analyzer. Glucose and lactate uptakes were calculated by multiplying the RC arteriovenous substrate difference by RCBF (39).

Exercise protocol. A standardized submaximal exercise protocol was used (38). After baseline measurements were taken with the animal resting quietly on the treadmill, exercise was begun with a 3-mile/h warm-up period for 3 min. The speed of the treadmill was then increased to 4 miles/h for the first level of exercise (exercise 1). This treadmill speed was continued for the remainder of the experiment. For the second level of exercise (exercise 2), the incline of the treadmill was elevated to 4%. Further elevations of the treadmill incline to 8 and 16% were defined as exercise 3 and exercise 4, respectively. The animal was exercised for 3 min at each level. Blood samples were taken and measurements recorded during the last minute at each level of exercise.

Measurement of RCA perfusion territory. After termination of the experiments, the animal was euthanized with pentobarbital sodium (30 mg/kg iv) followed by potassium chloride sufficient to cause ventricular fibrillation. After the chest was opened, the proximal RC artery was clamped and −15 ml of 2.5% Evans Blue dye was injected into the RC arterial catheter to delineate the perfused territory. In all cases, the RC venous catheter was found to be positioned within the dyed RV wall. This dyed territory was then carefully excised and weighed to normalize RCBF per gram of tissue mass.

Statistical analyses. All values are expressed as means ± SE. Results were analyzed with one-way repeated measures (within subject design) analyses of variance (ANOVA). When significance was found (P < 0.05), a Student-Newman-Keuls multiple comparison test was performed. Statistical procedures were performed with Sigma Stat statistical software, version 2.0, and interpreted according to Keppel (21).

RESULTS

Table 1 presents hemodynamic and metabolic data collected at rest and during the exercise protocol. AoP increased 19% to 118 ± 4 mmHg during exercise 1. AoP
Arterial oxygen content was 16.8 then increased further to 34 increased 38% to 29
increased 230 increased further during
P 66% to 174
remained elevated during the exercise protocol, but did not increase further (P > 0.05). Heart rate increased 66% to 174 ± 9 beats/min during exercise 1 and increased further during exercise 3 and exercise 4, reaching 230 ± 6 beats/min at exercise 4. Peak systolic RVP increased 38% to 29 ± 2 mmHg during exercise 1 and then increased further to 34 ± 3 mmHg at exercise 4. Arterial oxygen content was 16.8 ± 0.9 ml O₂/100 ml blood at rest and was 17.5 ± 0.9, 18.2 ± 0.8, 18.8 ± 0.8, and 19.3 ± 0.7 ml O₂/100 ml blood at exercise 1, exercise 2, exercise 3, and exercise 4, respectively. Compared with rest, exercise stimulated glucose uptake during exercise 1, exercise 2, exercise 3, and exercise 4, respectively. Combined with exercise 1, exercise 2, exercise 3, and exercise 4 uptake was further enhanced during exercise 4. Lactate uptake was unaffected by exercise until the highest level, where lactate uptake significantly diminished.

RCBF was 0.59 ± 0.02 ml·min⁻¹·g⁻¹ at rest and tended to rise with each exercise step (Fig. 2); exercise 2 and exercise 3 produced significant but modest increases in RCBF compared with rest. RCBF at exercise 1, exercise 2, and exercise 3 did not differ significantly. When compared with exercise 3, exercise 4 produced a 49% increase in RCBF. Right coronary venous Po₂ (PvO₂), oxygen extraction, GU, glucose uptake; LU, lactate uptake; *P < 0.05 vs. rest; †P < 0.05 vs. exercise 1; ‡P < 0.05 vs. exercise 2; §P < 0.05 vs. exercise 3.

Values are means ± SE; n = 7 dogs. AoP, mean aortic pressure; HR, heart rate; RVPmax, right ventricular peak systolic pressure; PVO₂, right coronary venous Po₂; O₂E, oxygen extraction; GU, glucose uptake; LU, lactate uptake; *P < 0.05 vs. rest; †P < 0.05 vs. exercise 1; ‡P < 0.05 vs. exercise 2; §P < 0.05 vs. exercise 3.

Exercise data are shown by shaded rectangles, and resting data are shown by white area within the exercise 1 rectangle. Different from resting baseline condition, P < 0.05; †different from prior, less strenuous condition, P < 0.05.
state of the myocardium. Also plotted on Figs. 3–6 are published data from comparable LV studies (14, 40, 41, 43). Figure 3 confirms that resting MV\(\dot{O}_2\) is less in RV than in LV, although blood flows were similar. This is consistent with low resting RV oxygen extraction. From rest to exercise 3, RCBF increased only moderately, although RV MV\(\dot{O}_2\) more than doubled. In contrast, left coronary (LC) flow appeared to increase linearly from rest to the highest level of exercise. From moderate to strenuous exercise, blood flow increased similarly in both ventricles. Therefore, as the RV oxygen extraction reserve is exhausted, the RV mobilizes its RC flow reserve, as does the LV, to supply further increases in myocardial oxygen demand.

Coronary P\(v_O2\) is plotted against MV\(\dot{O}_2\) in Fig. 4. Changes in coronary P\(v_O2\) reflect changes in myocardial P\(o_2\) and are a sensitive index of changes in myocardial oxygen supply/demand balance. RV venous P\(v_O2\) decreased steeply from 29.9 to 19.3 mmHg as RV MV\(\dot{O}_2\) increased from rest to exercise 3, but then fell more gradually from exercise 3 to exercise 4. Clearly, P\(v_O2\) is higher in RV than in LV at rest and during mild exercise. In contrast to the steep fall in RV P\(v_O2\), exercise-induced increases in LV MV\(\dot{O}_2\) caused only small decreases in LC P\(v_O2\). At the highest level of exercise, RV P\(v_O2\) was similar to that of the LV at comparable MV\(\dot{O}_2\).

Coronary resistance was estimated by dividing the driving pressure (AoP) by coronary flow (36), and this variable is plotted against MV\(\dot{O}_2\) in Fig. 5. RC resistance did not vary significantly from rest through exercise 3, although RV MV\(\dot{O}_2\) more than doubled. At exercise 4, RC resistance fell significantly. In contrast, LC resistance fell abruptly and continuously with exercise-induced increases in LV MV\(\dot{O}_2\).

RC and LC resistances are plotted against their respective coronary venous P\(o_2\) values in Fig. 6. Above 20 mmHg, RV vascular resistance was not significantly affected by large decreases in P\(v_O2\) caused by exercise-induced increases in oxygen extraction. Below 20 mmHg, RC resistance fell significantly. With resting P\(v_O2\) values near 20 mmHg, any decline in LC P\(v_O2\) was associated with concomitant decreases in LC resistance.

**DISCUSSION**

This report describes the first investigation of RV oxygen supply/demand balance during exercise. Although LV oxygen supply mechanisms have been investigated extensively in exercising animal models (1, 13–16, 18, 24, 40, 41), RV mechanisms have not been investigated, apparently due to the difficulty of collecting RC venous blood samples from conscious animals. This difficulty is due primarily to the small size and fragility of the superficial veins draining the RV and to the absence of a common drainage path, such as the
coronary sinus. We recently developed a procedure for collecting RC venous blood samples from conscious dogs (6). Utilizing this procedure in the current investigation, we found the following results. First, the RV has a large oxygen extraction reserve under resting conditions, and this reserve contributes preferentially to supply oxygen when RV oxygen demand increases during light and moderate exercise. Only at more intensive exercise is the substantial RC flow reserve mobilized. Second, RC vascular resistance is not affected by large decreases in RC PvO2 until the RV oxygen extraction reserve has been mobilized. When compared with the LV, the RV relies more on mobilization of a large oxygen extraction reserve to meet increased myocardial oxygen demands of exercise.

RV oxygen extraction reserve. Myocardial oxygen supply is a function of coronary blood flow and oxygen extraction. The LV extracts much of the coronary arterial oxygen (16, 22, 34, 37, 39–41, 43), with resting values reported as high as 82% (39). LV oxygen extraction does increase with exercise (Fig. 7), and at very strenuous exercise, LV oxygen extraction values as high as 97% have been reported (43). Although increased oxygen extraction contributes to LV oxygen supply during exercise, the oxygen extraction reserve is relatively small, therefore changes in LV oxygen demand must be met primarily by altering LC flow (Fig. 3). LV experiments consistently demonstrate decreased left coronary resistance during increased LV oxygen demand, even at mild exercise (Fig. 5). In contrast, RV resting oxygen extraction is only 40–50% (7, 22, 28, and present findings), therefore the RV has a large oxygen extraction reserve as well as a substantial flow reserve (8, 22, 23, 28). Both of these reserves are potentially available to supply significant amounts of oxygen when RV oxygen demand increases, as during exercise.

Studies in anesthetized dogs (22, 32–34) have demonstrated that the RV oxygen extraction reserve can be mobilized to meet increased RV oxygen demand produced by pacing, isoproterenol infusion, or pulmonary artery constriction. However, anesthesia, open-chest surgery, and perfusion systems may have blunted RC vasoconstrictor tone in these experiments (7, 9, 12, 35), and, thus, yielded inappropriately high RC flow and low RV oxygen extraction values. Experiments in conscious dogs were required to ascertain that resting RV oxygen extraction reserve is, indeed, greater than that of the LV, and then to define the extent to which this reserve contributes to RV oxygen supply during exercise. Figure 7 clearly demonstrates that initial increases in RV oxygen demand produce large increases in RV oxygen extraction that approach resting LV oxygen extraction. The enhanced RV oxygen extraction occurred even in the presence of moderate exercise-induced increases in arterial oxygen content. More strenuous exercise is required to determine whether RV oxygen extraction will parallel LV oxygen extraction at higher oxygen demand.

Taking values observed in this investigation for resting RCBF and RV oxygen extraction, arterial O2 content at rest and during exercise, and assuming a potential increase in oxygen extraction to 97%, as observed in the LV of exercising dogs (43), such an increase in oxygen extraction, could have contributed an additional 6.4 ml O2·min⁻¹·100 g⁻¹ with no increase in flow, or about 53% of the overall increase in RV MV˙O2 we observed. However, as shown, in Fig. 2, the effects of increases in RV A-VO2 are amplified by increases in RCBF, especially during strenuous exercise. Taking into account the higher RCBF that experienced greater oxygen extraction, 58% of the increased RV oxygen demand during strenuous exercise was provided by the increase in oxygen extraction. Furthermore, exercise-induced release of red blood cells increases the arterial oxygen content and enhances the RV oxygen extraction reserve. At the less intensive exercise levels of this study, the oxygen extraction reserve could have supplied the entire increase in RV MV˙O2, and, in fact, did supply more than 80% of the

**Fig. 7. Ventricular oxygen extraction expressed as a percentage of arterial oxygen content is plotted as a function of as a function of myocardial oxygen consumption.**

Coronary resistance is plotted as a function of coronary venous PO2. Filled circles show RV data from this investigation. Open symbols show comparable, published LV data.
required oxygen. Clearly, the RV oxygen extraction reserve is an important factor in RV oxygen supply/demand balance.

Although basal LV oxygen extraction reserve is small (16, 22, 34, 37, 39–41, 43), Dole and Nuno (11) observed a marked expansion of this reserve when heart rate was decreased from 120 to 40 beats/min in atrioventricular-blocked dogs. When LC perfusion pressure was then reduced from 120 to 80 mmHg, autoregulation, i.e., vasodilation to maintain constant flow, was ineffective. As LC perfusion pressure and flow fell, the LV utilized its oxygen extraction reserve rather than mobilizing its flow reserve. Although these circumstances clearly differ from exercise-induced changes in oxygen demand, it should be appreciated that the LV and RV preferentially use oxygen extraction reserves, if available, before decreasing coronary vascular resistance. Similar observations have been made in skeletal muscle, which, like RV, has a relatively high oxygen extraction reserve at rest (3, 15).

Right coronary flow reserve. RCBF, the other determinant of RV oxygen supply, has been measured in exercising dogs (2, 30), horses (25), and ponies (23, 31). These studies all reported increases in RC flow, therefore, no differences in RC and LC flow responses to exercise were noted. In contrast, we detected very modest increases in RCBF during the first three stages of exercise (Fig. 2), and these increases in flow were due to elevated AoP because RC resistance was not significantly reduced (Fig. 5). Only when a significant portion of the oxygen extraction reserve had been mobilized during exercise 4 (Table 1) was there a pronounced increase in RCBF (Fig. 2). Because RCBF can increase to about 4 ml·min⁻¹·g⁻¹ (28), a large RCBF reserve was still available at the most strenuous exercise employed in this study.

It is difficult to compare results of earlier RC flow studies with our findings because RV oxygen extraction and MV\(\dot{O}_2\) was not measured and, thus the degree of oxygen demand during exercise cannot be readily equated. However, in one earlier canine study, Ball et al. (2) measured RC flow with radioactive microspheres during graded treadmill exercise. Their resting flows greatly exceeded those we observed at comparable heart rates. For example, the RCBF reported by Ball et al. for moderate exercise (heart rate = 185 ± 2 beats/min) was 2.8 times our measured flow at exercise 3 (heart rate = 191 ± 5 beats/min). Ball et al. also measured LC flows and concluded that RC and LC flows increase at similar rates during exercise. Our findings of little change in RC flow during mobilization of the oxygen extraction reserve (Fig. 2) clearly differ from the conclusions of Ball et al. The discrepancy is unlikely to be due to different measurement techniques because resting RC flows are similar in both studies. One difference is the time of measurement. In our protocol, data were collected at 3 min of exercise at each level, whereas Ball et al. injected microspheres at 45 s after initiating exercise, a time when heart rate had stabilized, but perhaps not RC flow. In fact, we have observed transient increases in RC flow during protocol steps that required 60–84 s to subside. It is also possible that the dogs of Ball et al. had a lesser oxygen extraction reserve, and, therefore, had to mobilize their flow reserve to a greater degree. Once the oxygen extraction reserve is exhausted, our data (Fig. 2) do agree with Ball et al.’s suggestion that RC flow increases in parallel with LC flow.

Initiation of exercise caused a 19-mmHg increase in mean aortic blood pressure (Table 1). Because RV MV\(\dot{O}_2\) is affected by changes in RC perfusion pressure (5), a small portion of the observed increase in RV MV\(\dot{O}_2\) was due directly to increased AoP. However, the focus of this investigation was to delineate mechanisms of RV oxygen supply during exercise irrespective of specific factors responsible for increased RV oxygen demand. It should also be recognized that changes in coronary perfusion pressure produce changes in coronary blood flow independent of pressure-induced changes in oxygen demand, as demonstrated in the perfused LV by Vergroesen et al. (42). In the current investigation, the initial rise in RC flow paralleled the rise in AoP; RC resistance was not decreased (Fig. 5), although RV MV\(\dot{O}_2\) was significantly elevated. In contrast, the large increase in RC flow at the most intensive exercise was associated with a marked decrease in RC vascular resistance.

We did not determine the mechanism responsible for the decrease in RC resistance during the most strenuous exercise. Indeed, the mechanism responsible for left coronary vasodilation during exercise remains elusive (41). Our data do impact on understanding the role myocardial oxygen tension might play in regulating coronary arteriolar tone. Assuming RC Pv\(\dot{O}_2\) is a valid index of RV PO\(\dot{O}_2\), it is apparent that values greater than ~20 mmHg are not associated with changes in RC resistance. Interestingly, resting left coronary venous PO\(\dot{O}_2\) is about 20 mmHg (40, 43), and increases in LV oxygen extraction are associated with left coronary dilation. Whether these decreases in tissue PO\(\dot{O}_2\) directly or indirectly cause coronary vasodilation remains to be determined.

Substrate selection. In this investigation, RV glucose uptake was enhanced during exercise, in agreement with findings of LV investigations (4, 27). As arterial lactate concentrations rise during dynamic exercise, it is generally accepted that lactate uptake increases, as long as oxygen is available (29). In our investigation, however, arterial concentrations were not significantly elevated during the 12-min exercise protocol, and RV lactate uptake did not increase (Table 1). During the highest exercise level, glucose uptake was further enhanced and lactate uptake decreased, suggesting an increased preference for glucose as a metabolic fuel.

Validation that RC venous samples reflect RCA drainage. For our conclusions to be valid, RC venous blood samples must contain only blood draining RV myocardium supplied by the RCA. Two possible sources of contamination are blood drawn retrogradely...
from the right atrium and blood originating from vessels other than the RCA. To investigate whether there was contamination from right atrial blood, radioactive microspheres were infused simultaneously for 5 min into the superior and inferior vena cavae of four dogs. Three dogs were instrumented and studied in the conscious state, and one was studied in an acute experiment. During infusion of radioactive microspheres, blood samples were collected from the right atrium and the RC vein and later analyzed for radioactivity. Because circulating microspheres were trapped in the pulmonary circulation, any radioactivity within the RC venous samples would have come from right atrial contamination. Mean radioactivity counts emitted by the blood samples were 2,636 ± 702 for right atrial blood and 2 ± 1 for RC venous blood samples. These data demonstrate that there was no right atrial blood withdrawn into the venous samples using our technique.

It is also possible, given the vascular anatomy of the right heart, that blood from the LV circulation may have contributed significantly to RV venous samples. This possibility of venous contamination was explored in an earlier canine study in our laboratory by Murakami et al. (28). They infused Evans blue dye systemically while perfusing the RCA from an uncontaminated blood supply. With RC perfusion pressure reduced to 80 mmHg and with normal systemic arterial pressure, the LC contribution to RC venous drainage was 1.2 ± 1.0%. In the present study, there was no disparity between RC and LC perfusion pressures, therefore RC venous contamination from other coronary sources should have been negligible.

In summary, this report presents the first data describing RV oxygen supply/demand balance during graded exercise. The results documented a substantial RV oxygen extraction reserve at rest that is utilized preferentially during exercise-induced increases in RV MVO₂. Small increases in RC flow during mild exercise were the result of elevated AoP; RC resistance did not fall until the RV oxygen extraction reserve had been mobilized. In this process, RC PVO₂ decreased to ~20 mmHg without concomitant RC vasodilation. Strenuous exercise caused a 269% increase in RV oxygen consumption. Expanded A-VΔO₂ supplied 58% of this increase in oxygen demand, and increased RCBF supplied 42%. Considerable differences exist between the ventricles as to the relative contributions of the coronary flow and oxygen extraction reserves mobilized to increase myocardial oxygen supply as oxygen demand is elevated.

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REFERENCES


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