Right coronary autoregulation in conscious, chronically instrumented dogs

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Bian, Xiaoming, Arthur G. Williams, J r., Patricia A. Gwirtz, and H. Fred Downey Right coronary autoregulation in conscious, chronically instrumented dogs. Am. J. Physiol. 275 (Heart Circ. Physiol. 44): H169–H175, 1998.—Right coronary (RC) autoregulation and right ventricular (RV) function were assessed in conscious dogs, chronically instrumented to measure RC flow and RC pressure (RCP) as a hydraulic occluder on the RC was inflated. Dogs were then anesthetized, and RC autoregulation and RV function were again assessed. In the conscious state, moderate RC autoregulation was present with closed loop gains (Gc) of 0.59–0.27 as RCP was reduced from 100 to 40 mmHg. In the anesthetized state, Gc was not significantly less than in the conscious state at RCP >50 mmHg. The range and potency of RV autoregulation were greater in both groups than for previously reported findings in anesthetized dogs with RC perfused by an extracorporeal system. RV contractile function was well maintained in conscious and anesthetized dogs at RCP >45 mmHg. We conclude the following: 1) modest RC autoregulation is present in the conscious dog, 2) anesthesia limits the range but not the degree of RC autoregulation, 3) extracorporeal perfusion systems appear to depress RC autoregulation, and 4) RV contractile function remains constant in both conscious and anesthetized dogs until RCP falls below 50 mmHg.

right coronary circulation; right ventricular function

We also investigated relationships between RC flow and pressure and right ventricular (RV) contractile function of conscious and anesthetized dogs. Previously, we described well-maintained RV function and myocardial energetics as RC pressure (RCP) was reduced to 60 mmHg and RC flow fell 45% in the open-chest, RC-perfused preparation (17). This contrasts with the view that left ventricular (LV) function is closely coupled to coronary flow (11, 12, 20, 22, 24). Thus it was important to investigate this topic in the conscious state with intact RC.

MATERIALS AND METHODS

Animal Instrumentation

This investigation was approved by the Institutional Animal Care and Use Committee of the University of North Texas Health Science Center at Fort Worth and was conducted in accordance with the Guide for the Care and Use of Laboratory Animals [Department of Health and Human Services Publication No. (NIH) 85–23, Revised 1985]. A total of eight adult mongrel dogs of either sex, weighing 22–29 kg, were studied. Thirty minutes after preanesthesia treatment with acepromazine maleate (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. ID, 0.07 in. OD) was inserted into the aorta through the right internal mammary artery to measure aortic pressure. A similar Tygon catheter was implanted in the right atrium to measure right atrial pressure. A Konigsberg 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 was dissected free for 1–2 cm for attachment of a single crystal Doppler flow probe and a hydraulic occluder. The occluder was positioned distal to the flow probe, so changes in vessel cross-sectional area (CSA) beneath the velocity-sensing Doppler crystal would not occur when the occluder was inflated. A Micro-Renathane catheter (0.014 in. ID, 0.033 in. OD) was inserted into the right coronary artery distal to the occluder through a small side branch to the right atrium. The tip of this catheter was positioned at the origin of the branch. To measure RV segment shortening, a pair of piezoelectric crystals was inserted into the middle wall of the RV. These crystals were positioned 1 cm apart and perpendicular to the main right coronary artery.

At the conclusion of 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. Antibiotics (Clavamox, 6.25 ...
Acute studies were conducted in six of the eight dogs after completion of pressure-flow studies in the conscious state. The dogs were anesthetized with pentobarbital sodium (30 mg/kg iv). In three dogs, data were collected with the chest sutured and opened 10 days after surgery. The RC catheter was flushed with heparinized saline (10 U/ml) and filled with 5,000 units/ml heparin sodium daily. Other catheters were treated similarly.

Data Collection

Beginning the fifth day after instrumentation, measurements were obtained with the animal standing quietly in a sling. Pressure transducers (Narco Telecare model LDI-5) were positioned at mid-heart level. Right coronary flow velocity (CFV) was measured with a Triton Technology model 100 pulsed Doppler flowmeter. Pressure and velocity signals were averaged electronically. Ultrasonic signals from length dimension crystals were processed by a Triton Technology model 120 sonomicrometer and monitored with a Tektronics model 2215A oscilloscope. The hydraulic occluder was inflated if necessary to adjust baseline mean RCP to 100 mmHg, and then further inflated to reduce RCP in 10-mmHg steps from 100 to 20 mmHg. Mean RC flow velocity, mean RCP, mean systemic arterial blood pressure, and RV segment length were recorded on a multichannel Grass model 7D polygraph when RC flow and RCP had stabilized for at least 30 s at each pressure. At least three studies on consecutive days were conducted with at least five successful readings at different RCP in each animal.

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At the end of each experiment, India ink was injected through the RC catheter to identify the perfusion territory of the RC. The heart was removed, and the dyed area was resected and weighed. The internal diameter of the RC under the flow velocity probe was measured, and RC CSA was calculated. RC blood flow was calculated as the product of CFV x CSA/weight of the perfused tissue.

Data Analyses

Lower limits of autoregulation. Data recorded at the same RCP in each animal were averaged. Pressure-flow relationships of both conscious and anesthetized dogs followed initially a logarithmic phase, which was followed by a linear decline at the lower pressures (Fig. 2). These two phases were expressed as logarithmic and linear equations: $y = a + b \log x$ and $y' = a' + b'x$. The pressure at which the flow-pressure relationship changed from logarithmic to linear was considered the lower limit of autoregulation. This point was identified by finding iteratively the intersection of the logarithmic and linear functions previously fit to the data of each dog by regression analyses. In this iterative process, $z = \frac{y - y'}{x - x'}$, and $x$ was varied in 1-mmHg steps to produce a minimal value of $z$, which was taken as the intersection point of the two functions. Logarithmic and linear equation coefficients and points of intersections were averaged for the conscious and anesthetized conditions, respectively.

Potency of autoregulation. The autoregulatory response of the RC circulation at each RCP was expressed as the autoregulatory closed-loop gain ($G_c$) $= 1 - \left[\frac{(\Delta F/\Delta P)}{(F/P)}\right]$, where $F$ is the flow at pressure $P$ and $\Delta F$ is the decrease in flow when pressure $P$ is lowered by $\Delta P$. If $G_c$ equals one, autoregulation is perfect, i.e., flow remains constant despite a change in perfusion pressure; if $G_c$ equals zero, flow changes in proportion to pressure, and autoregulation is absent. $G_c$ values between zero and one reflect the potency of pressure-flow autoregulation. Negative values of $G_c$ reflect flow changes that are proportionally greater than the changes in perfusion pressure.

RV contractile function. RV myocardial segment shortening during systole (SS, %) was calculated as follows: $SS(\%) = \left(\frac{EDL - ESL}{EDL}\right) \times 100$, where EDL is end-diastolic length and ESL is end-systolic length. End diastole and end systole were defined as the onset of positive dP/dt and 20 ms before peak negative dP/dt, respectively.

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Statistical Analyses

All reported values are means ± SE. Linear and logarithmic functions were derived with regression analyses to characterize the RC pressure-flow data. Results of these analyses for each dog in the conscious or anesthetized state were averaged, and the effect of anesthesia on the coefficients of these functions was examined by unpaired, two-tailed t-tests. Gc values for closed-chest and open-chest anesthetized dogs were compared with unpaired, two-tailed t-tests. When no significant differences were detected, data from closed-chest and open-chest anesthetized dogs were pooled for further analyses. Gc values from conscious and anesthetized dogs of this study and those previously published from anesthetized dogs with the RC perfused by an extracorporeal system (26) were tested for difference from zero with one-way ANOVA and the Student-Newman-Keuls tests were used to compare Gc values at corresponding RCP from conscious and anesthetized dogs. Differences described as statistically significant indicate P < 0.05.

RESULTS

RC Autoregulation in the Conscious Dog

At baseline RCP of 100 mmHg, heart rate was 106 ± 4 beats/min, mean systemic arterial pressure was 106 ± 3 mmHg, mean right atrial pressure was 5.5 ± 0.4 mmHg, RV systolic pressure was 22.5 ± 1.1 mmHg, RV dP/dt max was 617 ± 42 mmHg/s, and RV dP/dt min was −558 ± 30 mmHg/s. These variables did not change significantly as RCP was reduced. With RCP of 100 mmHg, mean RC flow averaged 0.51 ± 0.04 ml·min⁻¹·g⁻¹. Constriction of the RC reduced RCP in steps to 20 mmHg, and RC flow fell with pressure (P < 0.001, Fig. 2). Flow values at each perfusion pressure differed significantly. For RCP from 100 to 50 mmHg, the pressure-flow relationship was well fit by a logarithmic function (Table 1) with r² = 0.987 ± 0.006. For RCP from 40 to 20 mmHg, the data were well fit by a linear function with r² = 0.972 ± 0.011. The mean intersection of these two functions (a + b log x = a′ + b′x) was at 42 ± 2 mmHg, which reflects the lower pressure limit of RC autoregulation in the conscious state. At this point, Gc was not significantly different from zero. For changes in RCP above 50 mmHg, Gc values were significantly greater than zero and ranged from 0.39 ± 0.08 to 0.59 ± 0.05 (Table 2). These values of Gc reflect moderately effective RC autoregulation. ANOVA detected no significant differences among Gc values for RCP above 40 mmHg. For this pressure range, linear regression analysis (Fig. 3) showed a poor linear correlation of Gc with RCP (r² = 0.21). Below 40 mmHg, Gc fell significantly to −1.23 ± 0.31.

Comparison of RC Autoregulation in the Conscious and Anesthetized States

At baseline RCP of 100 mmHg in the anesthetized state, heart rate was 148 ± 6 beats/min, mean systemic arterial pressure was 110 ± 4 mmHg, mean right atrial pressure was 4.5 ± 0.7 mmHg, RV systolic pressure was 23.4 ± 2.7 mmHg, RV dP/dt max was 630 ± 49 mmHg/s, and RV dP/dt min was −670 ± 72 mmHg/s. These variables did not change significantly as RCP was reduced. Compared with the conscious state, only heart rate differed significantly. Coronary pressures were reduced to evaluate pressure-flow autoregulation in the same way for both the conscious and anesthe-

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<th>RCP, mmHg</th>
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<td>r²</td>
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<td>-0.34 ± 0.14</td>
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<td>RCP at intersection point, mmHg</td>
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Values are means ± SE of 8 conscious and 6 anesthetized animals. RCP, right coronary pressure; r², coefficient of determination. a and b are coefficients from y = a + b log x; a’ and b’ from y = a’ + b’x. RCP at intersection point is x value for a + b log x = a’ + b’x.
tized states. At a baseline RCP of 100 mmHg, RC flow was 10% less after anesthesia (Fig. 2). RC flow fell with RCP (P < 0.001), and flow values at each pressure differed significantly. The difference in flow between the conscious and anesthetized state was sustained as pressure was reduced to 50 mmHg, such that the coefficients of the logarithmic functions relating flow to RCP were similar for the conscious and anesthetized states (Table 1). However, autoregulation failed at 51 ± 2 in the anesthetized state, whereas the lower limit of RC autoregulation was 42 ± 4 in the conscious state (P < 0.01; Table 1). Gc values are presented in Table 2. For the anesthetized state, opening the chest had no significant effect on Gc. At RCP from 90 to 50 mmHg, mean Gc values were slightly lower in the anesthetized dogs (Table 2), but these differences were not statistically significant. ANOVA detected no significant differences among Gc values of anesthetized dogs for RCP above 50 mmHg. For this pressure range, linear regression analysis (Fig. 3) showed a moderate linear correlation of Gc with RCP (r² = 0.41). Analysis of covariance detected no significant difference in the linear responses of Gc values in the conscious and anesthetized states for RCP >40 mmHg in the conscious state and for RCP >50 mmHg in the anesthetized state (Fig. 3). Reflecting the greater range of autoregulation in the conscious dogs, Gc at RCP between 40 and 50 mmHg was still positive in the conscious state, whereas Gc was negative and significantly less in the anesthetized state. Between 30 and 40 mmHg RCP, Gc values were negative and statistically similar for both states.

Previously reported RC flow data from experiments performed with an extracorporeal perfusion system (26) are illustrated in Fig. 2 for comparison with data from the current investigation. At 100 mmHg RCP, the perfused RC tended to have higher flow than the intact RC of anesthetized dogs in the current investigation. Flow in the perfused RC fell more steeply with pressure than observed in either conscious or anesthetized dogs with intact RC. Figure 4 illustrates values of Gc significantly greater than zero from the current investigation and from the previously reported study of extracorporeally perfused RC (26). At pressures <100 mmHg, the earlier experiments produced a Gc significantly greater than zero only between 100 and 90 mmHg, and this value was significantly lower than that observed in the current investigation. In contrast, Gc values of dogs with intact RC were greater than zero at pressures above 50 mmHg in the conscious state and above 60 mmHg in the anesthetized state. Gc values in Fig. 4 from the conscious and anesthetized states did not differ significantly, although mean Gc values in the anesthetized state were consistently below those in the conscious state.

**RV Contractile Function During Reduced RCP**

Figure 5 illustrates RV SS (expressed as percent of baseline) as a function of RCP. In the conscious state, SS remained similar to baseline as RCP was reduced until a critical pressure of 42 ± 1 mmHg was reached. At this pressure, RC flow was 66 ± 2% of baseline. In the anesthetized state, the critical pressure at which a fall in SS was first detected was 44 ± 3 mmHg, not significantly different from the critical pressure in the conscious state. At the critical pressure in the anesthetized state, RC flow was 44 ± 5% of baseline, which was significantly lower than baseline. The results of linear regression analyses for both states are shown in Fig. 3. Analysis of covariance detected no significant difference in the linear responses of Gc values in the conscious and anesthetized states.
were linearly related to RC pressure. Values are means ± SE. Below these critical pressures, reductions in segment shortening fell abruptly when RC pressure reached 42 ± 1 mmHg in conscious state and when RC pressure reached 44 ± 3 mmHg in anesthetized state. Below these critical pressures, reductions in segment shortening were linearly related to RC pressure. Values are means ± SE.

**DISCUSSION**

This report describes the first investigation of RC pressure-flow autoregulation in the conscious state. Dogs were surgically instrumented for measuring RC flow and RV function as RCP was selectively reduced by an implanted fluid-filled hydraulic occluder. RC autoregulation was also evaluated in the same dogs after anesthesia. The results were further compared with data from a previous study in which the RC autoregulation was evaluated using an extracorporeal perfusion system (26). We found the following: 1) modest RC autoregulation was present in the conscious dog. Although more effective than previously reported for perfused RC circulations of anesthetized dogs, RC autoregulation in the conscious state was less potent than reported for the LC circulation of conscious dogs. 2) Anesthesia did not significantly depress RC autoregulatory gain, although the range of autoregulation was attenuated. 3) RC autoregulation was depressed by extracorporeal perfusion systems used in earlier studies. 4) RV contractile function remained constant in both conscious and anesthetized animals until RCP fell below 50 mmHg. Above this pressure, RV function was not closely coupled to RC blood flow.

**RC Pressure-Flow Relationship**

In a series of investigations, researchers in this laboratory have measured RC blood flow as RCP was varied (14, 19, 26, 27). A consistent finding of these studies was absence of effective pressure-flow autoregulation, i.e., RC flow varied with RCP, although indexes of RV oxygen demand, such as heart rate and RV afterload, were unchanged. Interestingly, RV myocardial oxygen consumption (MV\(\bar{O}_2\)) also varied with flow (14, 26). Metabolic regulation of RC flow effectively compensated for pressure-induced changes in RV MV\(\bar{O}_2\), but this nullified pressure-flow autoregulation. When the change in RV MV\(\bar{O}_2\) was taken into account, a "corrected" RC autoregulatory gain was computed, which agreed well with LC autoregulatory gain (26).

The proclivity for coronary pressure to independently affect ventricular MV\(\bar{O}_2\) was first noted by Gregg (15) and is known as the Gregg phenomenon. The cause of the pronounced Gregg phenomenon in the RV has not been delineated, but in LC experiments we demonstrated that poor autoregulation was a prerequisite for expression of the Gregg phenomenon, and in absence of autoregulation, changes in LC pressure markedly affected LC volume (3). We argued that ventricular systolic stiffness varied with coronary vascular volume and that this affected MV\(\bar{O}_2\) in hearts with poorly autoregulating coronary circulations (18). Because some LC preparations had potent autoregulation and concurrently no Gregg phenomenon (3), we became concerned that the poor autoregulation and pronounced Gregg phenomenon we observed in RC preparations might have resulted from anesthesia, extensive acute surgical procedures, and the extracorporeal perfusion system required for those studies. This concern was heightened by the description by Canty (6) of especially potent autoregulation in LC circulations of conscious dogs. It seemed evident that RC autoregulation should be evaluated in the conscious dog.

We found that RC autoregulation in the conscious dog was, indeed, more potent than we had previously observed in the perfused RC of the anesthetized dog (16, 19, 26, 27). In Fig. 2 we have plotted previously reported RC pressure-flow data (26) for comparison with the findings of the current investigation. RC flow tended to be lower in the conscious dog at 100 mmHg and fell less steeply with reduced pressure. For the conscious dog, G\(_c\) values were positive at pressures of 40–100 mmHg, whereas in the perfused RC, G\(_c\) was significantly greater than zero only between 90 and 100 mmHg. This positive value of G\(_c\) 0.38 ± 0.09, was significantly lower than the G\(_c\) of 0.59 ± 0.05 observed in the conscious dogs of the current study.

The more potent RC autoregulation in the conscious state might have reflected the absence of anesthesia, since cardiovascular function is depressed by general anesthesia (4). However, we found in this study that anesthesia did not significantly depress RC autoregulation at RCP above 50 mmHg (Figs. 2–4). Surgical trauma might also depress autoregulation, but we found that autoregulation was unaffected by opening the chest of anesthetized dogs (Table 2). Thus we conclude that the extracorporeal perfusion system was responsible for attenuating RC autoregulation in our previous studies. However, it should be noted that the current study could only examine RC autoregulation at
RCP less than baseline, whereas studies with RC extracorporeal perfusion systems permitted examination of RC autoregulation at RCP up to 180 mmHg (26, 27).

Contact of heparinized blood with plastic tubing, glass vessels, and rotating stirring bars plus its compression by a roller pump appears to release vasoactive substances (1, 2). Vasoconstrictor substances would reduce the ability of the coronary circulation to dilate as coronary pressure is reduced. On the other hand, vasodilator substances would reduce vasodilatory reserve at normal pressure, thus also limiting the ability of the RC to maintain flow as RCP is reduced. These vasoactive substances have not been completely characterized but include vasoconstrictory thromboxane (7, 25) and vasodilatory prostacyclin (8, 23, 25). Accordingly, some investigators have administered ibuprofen in studies of LC autoregulation (3, 5, 21). However, we (26) and others (9, 16) have observed potent autoregulation in perfused, untreated LC circulations, so the question remains: Is RC autoregulation less potent than LC autoregulation?

This question can be addressed most directly by comparing findings of the current study of RC autoregulation with the findings of Canty (6) on LC autoregulation. Canty (6) observed no significant decrease in LC flow at LC pressures above 49 mmHg. Subendocardial flow began to fall at 37 mmHg, but subepicardial flow was unchanged until pressure fell to 25 mmHg. Thus, above 40 mmHg, LC autoregulation was essentially perfect, i.e., no change in flow in spite of about a 50% decrease in LC pressure. In contrast, we observed a 37% decrease in RC flow over approximately the same pressure range. The lower limit of LC autoregulation cannot be precisely determined from Canty's report, but his Table 1 indicates that subendocardial autoregulation failed between 31 and 28 mmHg, and subepicardial autoregulation failed below 25 mmHg, based on the criterion of $G_c$ equal to 0. We did not measure regional transmural flow in the RV wall, but our measurements of mean RC flow indicate that RC autoregulation in conscious dogs failed at ~42 mmHg, somewhat above the failure point for LC autoregulation in conscious dogs. Canty (6) did not report autoregulatory $G_c$ values for graded reductions of LC pressure, but from 84 to 49 mmHg, he computed mean $G_c$ values of 0.86 for subendocardial flow and 0.80 for subepicardial flow. For approximately the same pressure range, our transmural RC data produced $G_c$ values of 0.56 to 0.39. Thus it appears that RC autoregulation is, indeed, less potent than LC autoregulation. Also, the range of RC autoregulation is less than that of LC autoregulation.

Appreciable collateral blood flow could have provided an alternative blood supply to LV myocardium and resulted in an underestimate of RC autoregulation as RCP was reduced. Collateral flow was not assessed in this investigation, but in an earlier study of RC autoregulation, we found RV collateral flow to be ~5% of RC flow at RCP of 40 mmHg (19). Thus, at RC pressures above the failure point of RC autoregulation, collateral flow should have had no effect on our evaluations of RC autoregulatory function. This view is corroborated by low RCP at near zero RC flow (Fig. 2).

**RC Flow-RV Function Relation**

Tennant and Wiggers first reported in 1935 (22) that myocardial contractile function is closely coupled to coronary flow, although subsequent studies have found considerable variation in the degree of transmural coronary flow reduction required to affect LV contractile function (11, 12, 20, 24). Because the right ventricle differs markedly from the left ventricle in work performed, oxygen extraction and consumption, and transmural pressure, differences in the RC flow-RV function relation might be expected. The canine RC artery does not supply the interventricular septum or at least one-fourth of the RV margin adjacent to posterior longitudinal groove, so it is not surprising that complete occlusion of the RC has been reported to have little effect on RV systolic pressure (13). Clearly, evaluation of the RC flow-RV function relation requires direct assessment of function in the perfusion territory of the RC as was accomplished in the present study.

We found RV function unchanged until RCP was reduced to <50 mmHg in both conscious and anesthetized dogs (Fig. 5). Canty (6) found LV function well maintained over the same pressure range; however, in that study, LC flow was also well maintained. In the present investigation, RC flow fell with RCP (Fig. 2), resulting in a dissociation of the flow-function relation, i.e., flow fell by ~34% in conscious dogs and ~56% in anesthetized dogs before function began to decline. Because the right ventricle has a much greater oxygen extraction reserve compared with the left ventricle (5, 9, 19), it is tempting to attribute the maintenance of RV function with reduced flow to mobilization of this oxygen extraction reserve. However, we previously found that RV oxygen extraction increased only from 44 to 55% as RCP was reduced from 80 to 40 mmHg (19), although RV $M\overline{VO}_2$ fell significantly. Faced with a moderate decrease in RCP, the RV appears able to decrease its oxygen demand with little or no decrement in RV function. This notion is further supported by a recent report from our laboratory that RV function and high-energy phosphates were well maintained as RCP was reduced to 60 mmHg, although RC flow fell 45% (17). We have argued that ventricular systolic stiffness decreases as coronary pressure is reduced and that this reduction in internal cardiac work permits maintenance of normal cardiac external work and cytosolic energetics during moderate coronary hypoperfusion (10, 18). Thus highly effective RC autoregulation is not required to prevent a decline in RV contractile function if RCP is above 50 mmHg.

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