Changes in Vascular Resistance During Carotid Occlusion in Normal and Baroreceptor-Denervated Rats

Benedito H. Machado, Leni G.H. Bonagamba, Jaci A. Castania, and José V. Menani

In the present study, we investigated changes in mesenteric, renal, and hindquarter vascular resistance during the pressor response produced by bilateral carotid occlusion (BCO) in conscious, freely moving normal and denervated (aortic, carotid, or both) rats. BCO was performed using special previously implanted cuffs. In control normal rats, the increase in mean arterial pressure (MAP) during early and late responses (37±4 and 21±2 mm Hg, respectively) was related to increased renal (125±12% and 45±10%) and mesenteric (38±13% and 41±5%) but not hindquarter (14±4% and 8±7%) vascular resistance. In aortic-denervated rats, the greater MAP increase in early and late responses (57±4 and 44±4 mm Hg, respectively) compared with normal rats was related to a marked increase in hindquarter (137±26% and 106±26%) and mesenteric (104±14% and 66±9%) vascular resistance. In carotid-denervated rats, MAP increase and change in vascular resistance were similar to those values observed in control rats. Sinoaortic-denervated rats showed a greater MAP increase (34±4 mm Hg) during late response and a reduced increase in renal vascular resistance (46±6%) during early response. The present results show that 1) the pressor response to BCO in normal rats is associated with an increase in renal and mesenteric vascular resistance, 2) the aortic baroreceptors buffer the increase in mesenteric and especially hindquarter vascular resistance during BCO, and 3) the reduced pressor response in late response is probably related to a reduced increase in renal vascular resistance during this component compared with the early response. (Hypertension 1992;19[suppl II]:II-149–II-153)

The pressor response to bilateral common carotid occlusion is generally used to study the role of the baroreceptor reflex in the control of arterial pressure. In dogs, cats, and rabbits, the increase in arterial pressure elicited by bilateral carotid occlusion (BCO) seems to be dependent on the reflex mechanism of carotid baroreceptors.1 In rats, the pressor response to BCO is partially due to a reflex mechanism of carotid baroreceptors, because carotid denervation produces only minor changes in the pressor response to BCO,2 and partially due to cerebral ischemia, because the bilateral occlusion of the internal carotid arteries also induces an increase in arterial pressure.3 The significant pressor response during BCO in sinoaortic-denervated (SAD) rats also confirms the importance of the central ischemic mechanisms in this response.4 The importance of central ischemia in the pressor response during BCO in rats is related to the deficient role played by vertebral arteries in the central nervous system perfusion in this animal.

Through special cuffs previously implanted around the common carotid arteries, it is possible to study the pressor response induced by BCO in conscious, freely moving rats.5 It has been suggested that the pressor response to BCO in unanesthetized rats consists of two components, that is, an early and a late response.24 The early response appears within the first 20 seconds of BCO and is of carotid reflex origin. The late response observed during the last 30 seconds of BCO is smaller than the early response and is probably related to central ischemic mechanisms.

No studies have been performed in conscious, freely moving rats to evaluate the changes in regional vascular bed resistance during BCO, nor have the relative roles of aortic and carotid baroreceptors in the regulation of regional blood flow been investigated. In the present study, we investigated the
contribution of the changes in mesenteric vascular resistance (MVR), renal vascular resistance (RVR), and hindquarter vascular resistance (HVR) to the pressor response to BCO. In addition, we studied the relative roles of the aortic and carotid baroreceptors and cerebral ischemia during BCO using rats with aortic or carotid baroreceptor denervation or both.

**Methods**

Male Wistar rats weighing 250–300 g were used in this study. Direct arterial pressure was measured by means of a cannula (PE-10 connected to PE-50, Clay Adams, Parsippany, N.J.) inserted into the abdominal aorta through the femoral artery. The arterial line was connected to a Statham P23Db pressure transducer and a Hewlett-Packard recorder for measurement of pulsatile pressure and mean arterial pressure (MAP). Another cannula was inserted into the femoral vein of SAD rats for administration of phenylephrine (3–5 μg/kg) to test the efficacy of deafferentation at the end of the experimental protocol.

BCO was performed in conscious, unrestrained rats by means of pneumatic cuffs previously implanted into both carotid arteries with rats under ether anesthesia. The internal balloon of the cuffs was connected to PE-50 tubing that was tunneled to the dorsal region of the neck. A 1-ml syringe connected to both PE-50 catheters by means of a Y-shaped PE-50 tube was used to perform simultaneous reversible BCO.5,6

Blood flow was measured with a Doppler flowmeter (University of Iowa Bioengineering Facility, Iowa City). Through a midline laparotomy, the left renal artery, superior mesenteric artery, and abdominal aorta were isolated along 4 mm of their lengths, and miniaturized pulsed Doppler flow probes were placed around each vessel. The wire leads from the flow probes were tunneled subcutaneously to the head and fixed in a miniature receptacle in the skull. A miniature plug implanted into both carotid arteries with rats under anesthesia.

**Results**

**Mean Arterial Pressure**

Intact control rats presented a MAP of 113±3 mm Hg. Carotid denervation produced no significant change in MAP (117±5 mm Hg) in relation to control rats, whereas sinoaortic denervation and aortic denervation produced a similar and significant increase in MAP (124±3 and 125±3 mm Hg, respectively).

**Changes in Mean Arterial Pressure**

Changes in MAP in early and late responses during BCO are shown in Figure 1. In early response, the increase in MAP of AD rats (57±4 mm Hg) was significantly higher than in control rats (37±4 mm Hg). No difference was observed during early response in the change of MAP in CD and SAD rats when compared with control rats. In late response, the increase in MAP of AD (44±4 mm Hg) and SAD (34±4 mm Hg) rats was greater than in control rats (21±2 mm Hg). No difference was observed between control and CD rats (28±3 mm Hg).

**Changes in Hindquarter Vascular Resistance**

Changes in HVR in early and late responses during BCO are shown in Figure 2. In early response, it is important to note that control rats (14±7%) presented a smaller increase in HVR than AD rats (137±26%) and SAD rats (62±13%). In late response, the increase of HVR in AD rats was greater (106±26%) than in control rats (8±7%).

**Changes in Renal Vascular Resistance**

Changes in RVR in early and late responses during BCO are shown in Figure 3. In early response, the increase in RVR was similar in control (125±12%), AD (121±34%), and CD (123±13%) rats, whereas in SAD rats (46±6%), it was significantly lower than in the other three groups. In late response, no difference was observed among the four groups studied.

**Changes in Mesenteric Vascular Resistance**

Changes in MVR in early and late responses during BCO are shown in Figure 4. In early response, only the increase in MVR of AD rats (104±26%) was greater than in control rats (38±13%). In late response, the increase in MVR remained higher in AD rats (66±9%) than in control rats (41±5%).

**Baseline Vascular Resistance and Blood Flow**

No significant changes in baseline vascular resistance and blood flow were observed comparing normal and denervated rats. For example, the baseline resistances of hindquarter, renal, and mesenteric vascular beds in control rats were 58±6, 110±4, and 69±13 mm Hg/kHz, respectively, whereas in SAD rats, the values were 45±5, 155±42, and 102±28 mm Hg/kHz, respectively. The baseline blood flows in hindquarter, renal, and mesenteric beds in control rats were 2.1±0.2, 1.3±0.4, and 2.1±0.4 kHz, respec-
Discussion

The present results show that the pressor response of normal rats to BCO was associated with an increase in RVR and MVR, whereas small changes were observed in HVR. They also show that the difference between the MAP increases in early and late responses of normal rats was probably related to a small increase in RVR during late response, because no change was observed in the other vascular...
In AD rats, the greater increases in MAP during early and late responses were associated with a marked increase in HVR and MVR. No change in MAP increase was observed during early response in SAD rats compared with normal rats, but the late response of SAD rats was larger. A marked increase in HVR and a smaller increase in RVR was also observed during early response in SAD rats when compared with normal rats.

A greater pressor response in the early and late components of BCO in SAD rats compared with normal rats has been described previously. However,
in the present study, only the late response was significantly greater than in control rats. The extent of the pressor responses in early and late responses in SAD rats indicates the importance of mechanisms not related to baroreceptors for the pressor response during BCO in rats. In SAD rats, the pressor response is attributed to the activation of central chemoreceptors due to cerebral ischemia. The importance of cerebral ischemia does not abolish the involvement of carotid and aortic baroreceptors during the pressor response of rats to BCO. The increase in MAP observed in AD rats or even in SAD rats compared with normal rats shows the powerful inhibitory action of the aortic baroreceptors on the pressor response to BCO. This inhibitory action of the aortic baroreceptors is due to a buffering of the increase in MVR and especially in HVR. Compared with normal rats, no changes in pressor response or vascular resistance were observed during BCO in CD rats. In previous studies,2-4 a reduced pressor response during early response was observed in CD rats compared with normal rats.

Interesting differences were observed when the increases in RVR for the different groups studied were compared. First, it is possible to observe that the increase in RVR was smaller in late than in early response. In the other two vascular beds, these differences were not marked. In this case, the renal autoregulatory mechanisms or local metabolic changes may also play an important role to induce renal vasodilation. Second, the increase in RVR during early response in SAD rats was smaller than in all other groups of animals. This evidence suggests that ischemia or renal autoregulatory mechanisms or both can partially increase RVR, although in the absence of baroreceptor activity, these mechanisms alone are not sufficient to produce a great change in RVR. In this sense, cerebral ischemia alone is not enough to produce a great increase in RVR in the early and late responses. In addition, in all groups studied, except SAD rats, RVR in the late response was smaller than in the early response. As shown in previous studies,2-4 the late response is mainly related to cerebral ischemia. All this information together suggests that cerebral ischemia is not the most powerful stimulus in producing and maintaining the increase in RVR.

In summary, the present results show that 1) the pressor response to BCO in freely moving control rats is associated with an increase in RVR and MVR, 2) the inhibitory action of the aortic baroreceptors on the pressor response to BCO is due to the buffering of the increase in MVR and especially in HVR, and 3) the reduced pressor response in the late response compared with the early response seems to be related to a minor increase in the resistance of the renal vascular bed during the late response. In relation to cerebral ischemia, additional studies are required to evaluate the role of central and peripheral chemoreceptors in the regulation of regional vascular resistance in rats and other species.

References

KEY WORDS • vascular resistance • carotid artery • baroreceptors • ischemia
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