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Blood Pressure of Sinoaortic-Denervated Dogs Is Not Increased by Cardiac Denervation (42221)

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Abstract. Although blood pressure rises markedly after acute sinoaortic denervation, animals with chronic sinoaortic denervation have normal or only slightly elevated mean arterial pressures. The present study was performed to determine whether reflexes from cardiac receptors exert antihypertensive effects and thereby lower blood pressure in animals with chronic sinoaortic denervation. We made multiple measurements of blood pressures in dogs with chronic sinoaortic denervation before and after their hearts were denervated surgically. Mean arterial pressure after cardiac denervation (100.3 ± 4.2 mm Hg) was not significantly different from the mean pressures recorded before cardiac denervation in these sinoaortic-denervated dogs (104.8 ± 3.1 mm Hg). Also, mean heart rate after cardiac denervation (107.4 ± 5.5 beats/min) did not differ significantly from the mean heart rate recorded before cardiac denervation (107.2 ± 5.9 beats/min). Cardiac denervation did, however, appear to reduce the lability of both blood pressure and heart rate in sinoaortic-denervated dogs. We conclude that cardiac receptors are not responsible for maintaining arterial pressure within essentially normal limits in animals with chronic sinoaortic denervation.

Arterial blood pressure rises acutely to severely hypertensive levels immediately after surgical denervation of the sinoaortic baroreceptors (1, 2). Within 24 hr or less, however, blood pressure decreases toward normal levels. Dogs with chronically denervated sinoaortic baroreceptors have either normal mean arterial pressure (3–5) or only moderately elevated blood pressure (2, 6, 7).

Several investigators (8–10) have suggested that the decrease of blood pressure from severely hypertensive levels toward normal within hours after sinoaortic baroreceptor denervation may be caused, in part, by depressor reflexes elicited by cardiopulmonary receptors. Consistent with this view are data from acute experiments performed under anesthesia. Extensive surgical procedures were used in one study to produce dogs with innervation of only the atria, ventricles, or lungs after acute sinoaortic denervation. Vagal cold block in each of these preparations caused an increase in arterial blood pressure (11). Presumably the rise in blood pressure was caused by the elimination of afferent depressor impulses from the remaining portions of either the heart or the lungs; evidence indicated that receptors in the heart exerted greater reflex effects on blood pressure than receptors in the lungs under these conditions. In another study from the same laboratory, this cardiopulmonary reflex was absent when high pressure was maintained in the innervated carotid sinus, but present when pressure was normal or low in the sinus (12). The abolishment of vasodepressor effects from the heart and lungs when carotid sinus pressure was high could be explained by the fact that afferents from cardiopulmonary and carotid baroreceptors converge on the same neuron pools within the vasomotor center, as has been suggested by Thoren (8). Conversely, the absence of afferent activity from sinoaortic receptors to these neuron pools, as in chronic sinoaortic denervation, conceivably could lead to a potentiation of effects from cardiopulmonary afferents.

In light of the above evidence that low pressure receptors located in the heart and lungs may more effectively buffer blood pressure when impulses from arterial baroreceptors are eliminated, we utilized the technique of surgical denervation of the heart to evaluate whether receptors located specifically in the heart may exert an antihypertensive effect when sinoaortic baroreceptor reflexes are absent. Arterial blood pressure was recorded several times on a number of different days in dogs with chronic sinoaortic denervation to obtain baseline data. Subsequently, cardiac denervation was performed on these dogs, and

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blood pressure was measured again during multiple recording sessions following recovery from this operation. Mean arterial pressure recorded after combined cardiac and sinoaortic denervation was compared with the mean pressure recorded before the cardiac denervation operation.

Materials and Methods. *Sinoaortic denervation.* Selective bilateral sinoaortic denervation was performed aseptically on mongrel dogs (15.6–19.1 kg) under sodium pentobarbital anesthesia (25–30 mg/kg). The trachea was intubated, and an intravenous infusion of isotonic saline containing 250,000 U of penicillin was begun. Aortic arch baroreceptors (and chemoreceptors) were denervated by the method of Edis and Shepherd (13). The aortic nerve was identified as it separated from the vagus below the nodose ganglion. It was identified further by the depressor and pressor responses that could be elicited when the central end of the nerve was stimulated with selected parameters (14). A section of the aortic nerve was then removed between 5-O surgical silk ligatures. This method of aortic denervation destroys selectively only the aortic nerve; care was taken not to damage the vagal or sympathetic trunks during this procedure. Carotid baroreceptors were denervated by removing a section of each sinus nerve between ligatures. In addition, adventitia on the carotid sinus and adjacent vessels was stripped carefully over a length of about 1 cm; 10% phenol was then applied to the area. Catheters were implanted in the descending thoracic aorta and the inferior vena cava via a branch of a femoral artery and a tributary of a femoral vein, respectively, for measurement of arterial and central venous pressures. All dogs exhibited severe hypertension and tachycardia immediately after sinoaortic denervation; they were treated acutely with a single dose of phenoxybenzamine (1.0–2.5 mg/kg) and propranolol (a total of 2–4 mg). Several days postoperatively and periodically thereafter, nitroglycerin (24 μg/kg, iv) and phenylephrine (12 μg/kg, iv) were administered rapidly to confirm the absence of reflex increases or decreases in heart rate. If heart rate changed by more than 3 beats/min during this test (determined by computer as the mean of successive 6-sec intervals), dogs were excluded from further study. Only 5 of the 11 dogs subjected to this operation proved to have acceptable sinoaortic denervation by the above criteria, a ratio comparable to the approximately 50% success rate that we have had with this operation in the past.

Cardiac denervation. Surgical denervation of the heart has been performed in our laboratory over the past 6 years according to the technique developed by Randall and his colleagues (15) as modified in our laboratory by Fater *et al.* (16). Over 90% of the animals denervated by this procedure in our laboratory have proved to be completely denervated when tested after recovery from the operation. The initial test for completeness of cardiac denervation was performed during surgery by electrical stimulation of the ansae subclaviae and the thoracic vagi as described by Randall *et al.* (15). Confirmatory tests for denervation after recovery from the operation included the absence of reflex changes in heart rate after intravenous administration of pressor and depressor agents, the absence of heart rate and blood pressure changes in response to veratridine injection into the left atrium, the absence of heart rate changes after bilateral carotid occlusion just before sacrifice of the animal, and markedly diminished catecholamine levels in all four chambers of the heart; see Fater *et al.* (16) for details. In the present study, cardiac denervation was performed 2–4 weeks after sinoaortic denervation. The effectiveness of cardiac denervation in these animals could not be evaluated completely by our usual means because the sinoaortic reflex had been eliminated previously by sinoaortic denervation. However, the efficacy of cardiac denervation was confirmed during the operation by electrical stimulation of the autonomic nerves to the heart as described above. In addition, injection of veratridine (25 μg) into the left atrium of each of the dogs 3–5 days postoperatively did not decrease heart rate or arterial blood pressure in any of the animals, thus confirming the absence of the Bezold-Jarisch reflex. Catheters were placed in the left atrium and pulmonary artery, and an electromagnetic flow probe was placed around the ascending aorta during this operation. The catheters and flow probe cable were tunneled subcutaneously to the back and drawn through the skin. The chest was closed and the air evacuated by connecting a flexible pleural catheter to a vacuum pump. Following each denervation procedure, penicillin (500,000 U/day, im) was administered for 3 days. The animals were housed in a veterinary hospital where they were fed standard dog chow and had free access to water.

Recording sessions. After allowing at least 1 week for recovery from each surgical procedure, systemic hemodynamics were monitored on alternate days for 30 min. Several sessions were completed for each dog after sinoaortic denervation and again after combined sinoaortic and cardiac denervation. During hemodynamic monitoring sessions, the animals rested quietly in a Pavlov stand in a room to which they had become accustomed. The same person was present during each recording session, and activity and noise were kept to a minimum.

Catheters were connected to pressure transducers (Statham P23Db) positioned at the level of the heart. Aortic and central venous pressures were monitored in the sinoaortic-denervated animals. After cardiac denervation, pressures also were recorded from the pulmonary artery and the left atrium from catheters placed during the second operation. Also, cardiac output was measured by means of an electromagnetic flowmeter (Biotronex Laboratory) and an autozeroing circuit of our own design (17). In all experiments, heart rate was determined from the aortic pressure signal. Blood pressures and aortic flow were monitored continuously on an oscillographic recorder interfaced with a computer (Digital Equipment, Model PDP 11/34). Hemodynamic variables were sampled 100 times per second and averaged each minute by the computer.

Statistical analysis. The data from each dog were averaged to give one representative mean value for each hemodynamic measurement obtained during chronic sinoaortic denervation and a second mean for comparable measurements obtained after combined sinoaortic and cardiac denervation. The differences in corresponding means obtained under the two conditions were evaluated by Student's *t* test for paired samples. Data are expressed as means ± SE.

Results. Immediately after the initial operation for sinoaortic denervation, all animals exhibited severe hypertension and tachycardia (Fig. 1, top panel). Systolic blood pressure typically ranged from 200–230 mm Hg and heart rate was always over 170 beats/min. When measured again 1–3 days postoperatively, however, blood pressure had returned to the normotensive range, and the heart rate had slowed. Subsequently, when the heart was denervated surgically, there was no acute increase in blood pressure or heart rate in any of the animals. Rather, blood pressure was normal immediately after surgery (Fig. 1, bottom panel), and heart rate was in the typical range for animals with surgically denervated hearts (90–130 beats/min).

After recovery from sinoaortic denervation, the dogs displayed considerable variations in blood pressure, (Fig. 2, top panel). Periods of hypertension, as well as periods of hypotension, occurred spontaneously. Hypertensive episodes tended to occur when the dog was excited, and the hypotensive episodes occurred when the dog obviously was relaxed. The extent to which arterial pressure fluctuated varied from one dog to another, but blood pressure lability was present in all. The subsequent denervation of the heart appeared to reduce the blood pressure lability, presumably because an appreciable portion of the blood pressure variability was caused by changes in cardiac output secondary to changes in heart rate. These changes were attenuated following cardiac denervation (Fig. 2, bottom panel).

A total of 36 recording sessions were obtained from the five sinoaortic-denervated dogs, and 67 recording sessions were obtained from these animals after combined sinoaortic and cardiac denervation. Mean arterial pres-

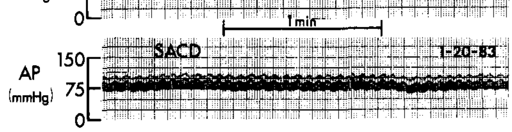

FIG. 1. Arterial pressure (AP) tracing taken immediately after sinoaortic denervation (SAD) and from the same dog 2 weeks later immediately after the heart was denervated to produce combined sinoaortic and cardiac denervation (SACD). The dog was hypertensive immediately after the first operation but normotensive immediately after the second.

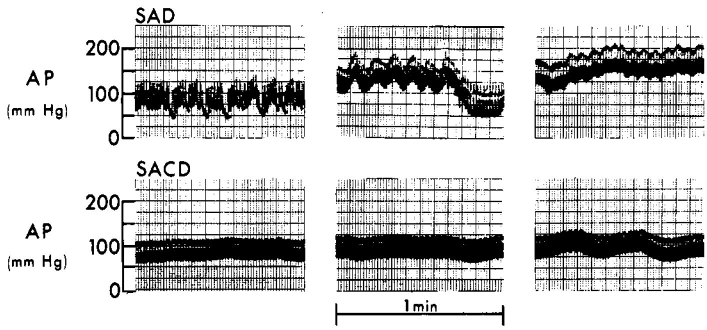

FIG. 2. Arterial pressure (AP) tracings from the same dog after recovery from sinoaortic denervation (SAD) and later after recovery from cardiac denervation to produce combined sinoaortic and cardiac denervation (SACD).

sure (Table I) after sinoaortic denervation averaged 104.8 ± 3.1 and after combined sinoaortic and cardiac denervation averaged 100.3 ± 4.2 mm Hg. Heart rate (Table II) during the same sessions averaged 107.2 ± 5.9 and 107.4 ± 5.5 beats/min, respectively. There were no significant differences in mean arterial pressure or heart rate in the values obtained before and after cardiac denervation. These values are comparable to values obtained from normal instrumented, conscious dogs which have averaged 96.5 mm Hg and 101.3 beats/min in recent studies from our laboratory (16, 18–20) and slightly higher than control data estimated to be 91 mm Hg and 88 beats/min from studies in other laboratories (2, 6, 7). Thus chronic sinoaortic denervation caused, at most, only small increases in mean arterial pressure in these conscious, resting dogs.

A graph depicting a representative 30-min hemodynamic recording from a dog with combined sinoaortic and cardiac denervation appears in Fig. 3. Heart rate, cardiac output, and stroke volume remained stable during the entire recording period. Mean arterial blood pressure ranged from 90 to 110 mm Hg during the recording session. Minor fluctuations occurred in total peripheral resistance and in central venous, left atrial, and pulmonary artery pressures.

Figure 4 compares graphically a typical 30-min recording from a dog after sinoaortic denervation and later, after combined sinoaortic and cardiac denervation. Note that heart rate variability was reduced after the heart was denervated.

Discussion. The major aim of this investigation was to determine whether the elimination of cardiac reflexes produces an increase in arterial blood pressure in dogs with chronic sinoaortic denervation. Results indicated that cardiac denervation did not evoke either an immediate or a delayed increase in blood pressure. These data therefore indicate that reflexes from cardiac receptors are not re-

TABLE I. MEAN ARTERIAL PRESSURE OF CONSCIOUS DOGS WITH SINOAORTIC DENERVATION (SAD) AND WITH COMBINED SINOAORTIC AND CARDIAC DENERVATION (SACD)

SAD	n^a	Mean ± SE	SACD	n	Mean ± SE
1	7	105.9 ± 1.4	1	12	98.7 ± 2.2
2	8	100.8 ± 4.1	2	15	115.3 ± 1.8
3	8	114.9 ± 4.7	3	24	102.3 ± 2.0
4	8	106.1 ± 4.1	4	9	94.0 ± 4.8
5	5	96.4 ± 2.7	5	7	91.4 ± 3.8
Total	36	104.8 ± 3.1	Total	67	100.3 ± 4.2

[a] n = number of 30-min recording sessions.

TABLE II. MEAN HEART RATE OF CONSCIOUS DOGS WITH SINOAORTIC DENERVATION (SAD) AND WITH COMBINED SINOAORTIC AND CARDIAC DENERVATION (SACD)

SAD	n^a	Mean ± SE	SACD	n	Mean ± SE
1	7	115.9 ± 2.0	1	12	97.7 ± 2.0
2	8	91.0 ± 3.0	2	15	103.8 ± 1.3
3	8	98.4 ± 3.8	3	24	101.7 ± 1.2
4	8	124.1 ± 1.8	4	9	128.8 ± 3.5
5	5	106.4 ± 3.0	5	7	105.1 ± 3.0
Total	36	107.2 ± 5.9	Total	67	107.4 ± 5.5

[a] n = number of 30-min recording sessions.

sponsible for maintaining arterial pressure at normotensive or only moderately elevated levels in conscious animals with chronic sinoaortic denervation. This conclusion is consistent with earlier studies on dogs with cardiac denervation alone (16, 21) in which there was no increase in arterial blood pressure after surgical denervation of the heart. The present results provide direct evidence against the suggestion (8–10) that cardiac receptors may be responsible for returning blood pressure toward normal as animals recover from acute sinoaortic denervation. Earlier results from acute experiments utilizing sinoaortic denervation and extensive surgical modifications of the heart and lungs (11) would not seem to apply to the conditions that exist in conscious dogs with chronic denervation of the heart and arterial baroreceptors.

In the present study, we eliminated cardiac afferent fibers, but pulmonary afferent fibers remained essentially intact. It is possible that reflexes from pulmonary receptors may have aided in maintaining blood pressure near normal in the absence of sinoaortic reflexes. However, this appears to be unlikely. Moderate manipulations in pulmonary vascular pressures do not cause any decrease in arterial blood pressure in conscious dogs (19).

We cannot exclude the possibility that some

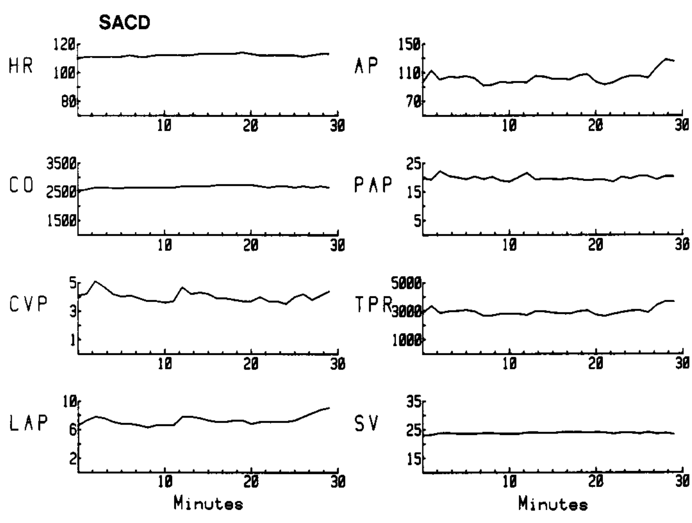

FIG. 3. Hemodynamic variables recorded from a dog with combined sinoaortic and cardiac denervation. Graph was plotted from successive minute mean values calculated by computer. HR, heart rate (beats/min); CO, cardiac output (ml/min); CVP, central venous pressure (mm Hg); LAP, left atrial pressure (mm Hg); AP, arterial pressure (mm Hg); PAP, pulmonary artery pressure (mm Hg); TPR, total peripheral resistance (dynes/sec · cm^{-5}); SV, stroke volume (ml).

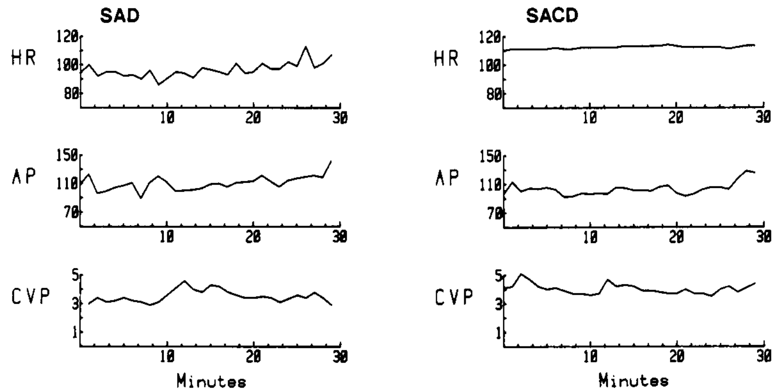

FIG. 4. Comparison of hemodynamic variables from a dog after sinoaortic denervation (SAD) and then after combined sinoaortic and cardiac denervation. Minute mean values are plotted as in Fig. 3.

functional cardiac afferent pathways remained in our cardiac-denervated dogs. We believe, however, that denervation was complete in these animals. Over 90% of our cardiac denervation operations during the past 6 years have yielded animals with complete cardiac denervation as judged by a broad set of criteria (see Materials and Methods). Moreover, animals denervated by this method in an earlier study have been shown to lack the ability to elicit cardiac reflexes; left atrial distension in those animals did not produce reflex tachycardia, natriuresis, diuresis, or inhibition of the secretion of vasopressin or renin (18). All of these responses occur normally in dogs with innervated hearts.

Chemoreceptors, as well as arterial baroreceptors, were destroyed by the sinoaortic denervation in these experiments. Chemoreceptor stimulation can cause an increase in arterial pressure (22). It has been suggested that elimination of chemoreceptors may tend to cause a decrease in arterial blood pressure at the same time that elimination of arterial baroreceptors would tend to cause an increase in arterial pressure (23), but the abrupt, large increase in blood pressure after sinoaortic denervation indicates that the baroreceptors are dominant under these conditions. The decline in blood pressure with time toward normal levels after sinoaortic denervation suggests that neither baroreceptors nor chemoreceptors are responsible for determining the "set point" of prevailing arterial pressure.

Lability of arterial pressure, rather than hypertension, appears to be the most distinctive observation in conscious, sinoaortic-denervated dogs. The extreme lability of blood pressure that may be seen following sinoaortic denervation (2, 3, 5) suggests to us that the role of the arterial baroreflex is to buffer short-term changes in blood pressure and to minimize fluctuations around the prevailing mean. Although it has been suggested that animals do not become hypertensive after sinoaortic denervation because their arterial baroreceptors have been incompletely denervated, there is evidence against this concept. First, although animals with chronic sinoaortic baroreceptor denervation have normal or only mildly elevated levels of mean blood pressure, the denervation procedure routinely produces severe hypertension acutely, thus indicating the effectiveness of the acute denervation. Second, pharmacological testing, as in the present study, indicates that reflex effects on heart rate are absent in animals with chronic sinoaortic denervation; any remaining functional baroreceptor afferents would be expected to reflexly change heart rate during drug-induced hypertensive or hypotensive episodes. Third, periods of severe hypotension, as well as periods of hypertension, are common in animals with chronic sinoaortic denervation (5, 27). The hypotensive periods tend to occur when the animal is very relaxed. We have observed diastolic pressures as low as 12 mm Hg and periods of asystole of over 10 sec in conscious dogs after sinoaortic denervation (27). It is difficult to explain how reduced or absent afferent baroreceptor traffic would cause hypotension and severe bradycardia if one assumes that baroreceptors determine the set point of arterial blood pressure. We believe that similar hypotensive episodes are not observed in normal animals because functioning sinoaortic baroreceptors reflexly increase cardiac output and peripheral vascular resistance during comparable periods of relaxation. Fourth, even when baroreceptors are completely intact, they adapt quite rapidly to changes in the prevailing level of blood pressure (24, 25) and then tend to buffer blood pressure changes from the new prevailing pressure (26). In other words, the baroreceptors adapt to the set point of arterial pressure, but do not appear to be responsible for establishing that set point. For these reasons we do not believe that the absence of hypertension in animals with chronic sinoaortic denervation may be attributed to incomplete sinoaortic denervation.

In summary, the purpose of this study was to determine specifically the effects of cardiac denervation on blood pressure levels in animals with chronic sinoaortic denervation. Since blood pressure did not increase in chronically sinoaortic-denervated dogs after surgical denervation of the heart, we conclude that reflexes from cardiac receptors do not exert appreciable antihypertensive effects in sinoaortic-denervated animals. Other factors must be sought to explain why blood pressure returns toward normal levels in animals with chronic sinoaortic denervation.

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