Selective Contribution of Two Types of Carotid Sinus Baroreceptors to the Control of Blood Pressure

J.L. Seagard, F.A. Hopp, H.A. Drummond, and D.M. Van Wijnsberghe

This study was performed to determine if selective elimination of afferent input from two different types of previously described baroreceptors altered the ability of the dog to regulate blood pressure (BP), examining specifically if there was differential loss of baroreceptor control of tonic levels of baseline pressure versus dynamic changes in pressure. In the first series of experiments in this study, anodal block of the carotid sinus nerve was used to selectively block afferent input in a sequence from large-diameter A-fiber carotid baroreceptors (mostly type I) to smaller A-fiber and nonmyelinated C-fiber baroreceptors (mostly type II). In the second series of experiments, anesthetic block of the carotid sinus nerve with bupivacaine was used to selectively eliminate afferent input in reverse order from anodal block, first blocking input from baroreceptors with small afferent fibers and then additionally eliminating input from the larger-diameter A-fiber baroreceptors. The effects of selective elimination of each baroreceptor type were determined by monitoring baseline BP during constant carotid sinus pressure (CSP) perfusion of a vascularly isolated carotid sinus (tonic control) and obtaining baroreflex sensitivity (slope) during ramp pressure stimulations of the carotid sinus (dynamic control) under various blocking conditions. Low levels of anodal block significantly attenuated baroreflex sensitivity (−0.84±0.11 versus −0.63±0.10 mm Hg BP/mm Hg CSP) at levels of block that had no effect on tonic baseline BP (158.4±16.5 versus 160.7±9.5 mm Hg BP). In contrast, low levels of bupivacaine block produced significant increases in tonic BP (158.8±6.4 versus 169.0±6.5 mm Hg BP), whereas there was no effect on dynamic baroreflex sensitivity (−0.83±0.08 versus −0.73±0.08 mm Hg BP/mm Hg CSP). Thus, blocking large A-fiber baroreceptors resulted in significant decreases in baroreflex sensitivity without changes in baseline levels of BP, indicating primarily an attenuation in dynamic baroreflex regulation. Blocking of smaller A-fiber and unmyelinated C-fiber baroreceptors resulted in smaller decreases in baroreflex sensitivity and significant elevations in baseline BP, indicating a loss of tonic control of pressure. These results suggest that the two types of baroreceptors contribute differently to the regulation of blood pressure. (Circulation Research 1993;72:1011–1022)

Key Words: baroreceptors • baroreceptor reflex • anodal block • local anesthetic block • bupivacaine

Earlier studies in this laboratory identified two types of carotid baroreceptors on the basis of responses to slow increases in ramp pressure stimulations of the carotid sinus. The presence of the two discharge patterns suggested that the two types of baroreceptors contributed differentially to the regulation of blood pressure (BP). The sudden-onset, high-frequency, and more sensitive discharge of the type I baroreceptors allowed these baroreceptors to contribute more to regulation of dynamic pressure changes and were the primary “buffers” to prevent changes in arterial pressure. The continuous firing patterns of the type II baroreceptors, marked by low frequencies and sensitivities and wide operating ranges, suggested that these baroreceptors were better suited to regulate baseline resting levels of BP. The recent finding that type I but not type II baroreceptors acutely reset in response to sustained changes in carotid sinus pressure (CSP) further suggested that the type II baroreceptors provide information that more accurately reflects ongoing tonic levels of BP than the type I baroreceptors.

If the two types of baroreceptors do contribute differently to BP regulation, then selective stimulation or elimination of afferent input of one type of receptor should alter the ability of the animal to regulate pressure. The possibility of differential control of pressure by different types of baroreceptors has been studied by other investigators who examined baroreflex effects of activation of A-fiber versus C-fiber baroreceptors, a classification that closely, but not exactly, follows classification of type I versus type II baroreceptors. Type I baroreceptors have been found to primarily have large myelinated A-fiber afferents, whereas type II baroreceptors primarily have smaller A-fiber and unmyelinated C-fiber afferents. These studies indicated that both A-fiber and C-fiber baroreceptors produced depressor effects...
effects when electrically activated, with some indications that C-fiber baroreceptors appeared to contribute more to the evoked hypotension and were necessary to maintain the depression of BP throughout the stimulation period. One earlier study used anodal block to determine the relative contributions of A- versus C-fiber baroreceptors to the inhibition of sympathetic activity during pressure stimulation. Results from that study suggested that C-fiber baroreceptors contributed to reflex inhibition of sympathetic activity only when arterial pressure was increased well above normal resting levels.

Although our characterization of baroreceptors into types I and II is a functional distinction, the fact that most type I baroreceptors have larger A-fiber axons and most type II baroreceptors have small A- or C-fiber axons is the basis for the blocking techniques used in the current investigation. The experiments in the present study were performed to determine if selective elimination of either larger A-fiber baroreceptors or smaller A- and C-fiber baroreceptors altered the control of BP. The techniques of monopolar anodal blocking and local anesthetic blockade were used to selectively block afferent baroreceptor input during both tonic and dynamic stimulation of the baroreceptors.

Anodal block is the application of low levels of direct positive polarizing current (20–300 μA) to nerve fibers, which produces a localized reversible block of conduction of first larger myelinated (A-fiber) and then smaller myelinated and unmyelinated (C-fiber) axons. Nodal conduction of myelinated fibers makes this type of axon more susceptible to the blocking current. In addition, within each fiber group, the order of blocking is inversely related to membrane resistance, i.e., from largest to smallest. Therefore, careful regulation of blocking conditions can result in a very controllable sequential blockade of nerve fibers from larger A fibers to small A and C fibers. The effects of anodal blocking of baroreceptor afferent information on arterial BP regulation were examined during constant mean pressure and ramp pressure stimulation of carotid sinus baroreceptors to determine the effects of selective elimination of carotid baroreceptor input on tonic and dynamic regulation of arterial pressure, respectively. Results from this first study indicated that elimination of only large A-fiber baroreceptors produced a decrease in baroreflex sensitivity or attenuation of control of dynamic changes in BP, without an associated change in tonic resting levels of BP. Further blocking to eliminate smaller A- and C-fiber baroreceptors produced additional attenuation of baroreflex sensitivity and significant increases in resting levels of baseline pressure.

Application of the local anesthetic bupivacaine (BUP) was also used to produce differential block of carotic baroreceptor input but in the reverse order obtained with anodal block. BUP exposure first blocked small A- and C-fiber baroreceptors and then additionally larger A-fiber baroreceptors at higher anesthetic concentrations. Although the selectivity of the block was not as discrete as that obtained with anodal block, the results of the second study indicate that loss of small A- and C-fiber baroreceptor input preferentially resulted in loss of tonic control of resting baseline pressure, whereas the greatest attenuation of dynamic baroreflex responses to changes in CSP were observed when large A-fiber baroreceptors were blocked. These results were consistent with those obtained in the first study using anodal block, although the blocking order of the baroreceptor types was reversed, and suggested that baroreceptors associated with different afferent fiber types contribute differentially to the regulation of BP.

**Materials and Methods**

**General Methods**

The effects resulting from blockade of carotid baroreceptor inputs were studied using an isolated carotid sinus preparation in anesthetized mongrel dogs (25 mg/kg sodium thiopental bolus +10 mg/kg per hour infusion) as previously described. Briefly, the left carotid sinus was vascularly isolated to permit either a flow-through pulsatile perfusion of the sinus region at constant mean conditioning pressures or a slow ramp increase in CSP (1–2 mm Hg/sec). Buffered lactated Ringer's solution was used as the perfusate and oxygenated with 100% O2 to chemically denervate any chemoreceptors not physically eliminated by the isolation technique. CSP was measured via a catheter in the lingual artery and recorded using a Statham pressure transducer on a polygraph (model 7D, Grass Instrument Co., Quincy, Mass.). Constant CSP was maintained using a servocontroller developed in this laboratory.

The left femoral artery and vein were cannulated to permit measurement of arterial BP and infusion of anesthetic, respectively. Arterial blood gases were measured using a blood gas analyzer (model ABL 30, Radiometer America, Inc., Westlake, Ohio) and kept within normal ranges by adjustment of ventilation and infusion of bicarbonate. Arterial pressure was measured via the catheter in the left femoral artery, which was connected to a Statham pressure transducer and the Grass model 7D polygraph. Arterial BP and CSP were recorded on an FM tape recorder (model D, A.R. Vetter Co., Rebersburg, Pa.) for later data analysis. To limit baroreceptor input to that coming from the isolated sinus, the contralateral sinus was denervated, and both vago-sympathetic trunks were sectioned.

**Anodal Block**

To perform anodal block, direct current was applied through a modified wick-type electrode placed on the isolated desheathed nerve of interest. The monopolar electrode consisted of a solid felt tip, notched for nerve placement, that was epoxied into a hollow plastic tube (diameter, 6.5 mm). An insulated silver wire with a bared end was threaded down the tube and into the felt wick to serve as the electrode lead. The electrode was soaked in saline for several hours before its use to ensure complete conduction of the blocking current. The cathodal electrode consisted of a simple alligator clip, which was placed in muscle tissue lateral to the blocking site to provide multiple current paths from the anode, bidirectionally along the nerve, to the cathode. Current density at the nerve–tissue interface was thus reduced by shunting current through multiple pathways, thereby reducing the excitatory effects of depolarization at the cathode.

Because of the small length of the sinus nerve, the selectivity of anodal block was tested in three dogs on a
small multifiber nerve preparation of the vagus nerve. The vagus was isolated and desheathed, and a small bundle of fibers approximately the diameter of the carotid sinus nerve was dissected from the nerve trunk. The in situ nerve preparation was kept moist to prevent drying of the nerve fibers. Two pairs of stimulating and recording electrodes were placed on the small nerve bundle, with the wick-type electrode used for application of the blocking current placed between the other pair of electrodes. Maximal electrical stimulation of the vagus, determined by monitoring the evoked A- and C-fiber potentials with the recording electrodes, was performed while varying levels of anodal block were applied to the nerve bundle. Currents were applied in random order, from 20 to 300 μA. After each blocking current was applied, the evoked potential was monitored, and no additional current was tested until the evoked potentials returned to control levels.

To perform anodal blocking of baroreceptor afferent fibers, the wick-type electrode was placed on the isolated but intact left carotid sinus nerve. The nerve was carefully desheathed to permit better exposure of the nerve fibers. In two dogs, a very small preparation (few fibers) of the carotid sinus nerve was carefully dissected away from the main trunk of the nerve and sectioned so that afferent traffic in the small bundle could be monitored on a set of recording electrodes. The anodal blocking electrode was then placed distal to the recording electrodes, between these electrodes and the carotid sinus. With this arrangement, the effectiveness of anodal blocking could be monitored by the loss of first large and then smaller action potentials from discharging baroreceptors. Because of the small size of the preparation from which potentials were recorded, spike amplitude and discharge frequency were used as indications of fiber type. In small few-fiber preparations, high-frequency potentials of large amplitude have been ascribed to A fibers, whereas lower frequency potentials with smaller amplitude have been attributed to C-fiber discharge.11-13 In a few cases, as reported by other investigators,4,14,15 when anodal current was applied to the nerve, there was an initial stimulation of the baroreceptor afferent fibers indicated by a reflex decrease in arterial pressure. When this occurred, if the stimulation did not end within 10 seconds, the anodal current was turned off, and the nerve was cleaned of any fluid. The cathodal electrode was also moved to a new location to establish a different current path.

The amount of current necessary to completely block all afferent baroreceptor traffic was different from nerve to nerve, but for cases where few-fiber activity was measured, rarely were C-fiber baroreceptors blocked at currents below 70 μA. Different levels of anodal current were used for each experiment, with each normalized as a percent to 100% maximum blocking current for that animal. Maximum blocking current was defined as the level of current needed to produce maximal elevation of baseline arterial BP during constant CSP perfusion and complete loss of dynamic baroreflex changes in arterial pressure during ramp changes in CSP, indicating complete block of all baroreceptor afferent activity. Maximum blocking current ranged from 80 to 150 μA (mean, 107.3 ± 53 μA).

**Anesthetic Block**

In a manner similar to anodal block, the effectiveness of anesthetic blockade using the local anesthetic BUP was tested in three animals on a small bundle of fibers approximately the size of the carotid sinus nerve dissected from a vagal nerve. An earlier study by Franz and Perry16 had indicated that differential blocking of small versus large fibers using local anesthetics was more selective if only a 2-mm segment of the nerve was exposed to the anesthetic. Since the entire vagal nerve was desheathed to permit dissection of a smaller bundle of fibers, it was difficult to restrict anesthetic exposure to a 2-mm segment of the nerve. Attempts were made to limit the length of the vagal bundle exposed to anesthetic by application of the anesthetic with a wick electrode or a patch of sponge, but some spread of anesthetic was likely. The in situ nerve preparation was kept moist to prevent drying of the nerve fibers. A pair of stimulating and recording electrodes was placed on the small nerve bundle, with the wick electrode or sponge used for application of the anesthetic placed between the other two pairs of electrodes. Maximal electrical stimulation of the vagus, determined by monitoring the evoked A- and C-fiber potentials with the recording electrodes, was performed while varying concentrations of BUP from 3 to 20 mg% were applied to the isolated nerve segment. Anesthetic was applied in doses of increasing concentration, and evoked potentials were tested after 2 and 7 minutes of anesthetic exposure.

To determine effects of selective anesthetic blockade of carotid baroreceptor input, increasing levels of BUP were applied to the isolated carotid sinus nerve. Because selectivity of anesthetic blockade is dependent on length of exposed nerve, an attempt was made to limit the exposure of the carotid nerve by various methods, depending on the available length of the nerve. The carotid nerve was isolated from surrounding tissue and, if possible, placed in a small slotted plastic chamber that could be sealed around the nerve, minimizing anesthetic exposure to a 2-mm segment of nerve. If there was insufficient room, a wick-type electrode was placed around a 2-mm desheathed segment of the nerve and used for anesthetic application. If both methods were impossible, the anesthetic was then directly applied to a 2-mm segment of the nerve that had been desheathed. The remaining sheath on the rest of the nerve served as a diffusion barrier for the anesthetic. All methods appeared to result in differential block, according to the results obtained, but successful block using the last method may have also been dependent on the relatively low doses of anesthetic used, as well as the short exposed segment. Concentrations of BUP used in the study ranged from 5.0 to 20 mg%, which are in the low range of those used by other investigators in earlier studies.17,18

**Experimental Protocol**

The effects of selective blocking of baroreceptor activity on tonic baroreflex control of baseline BP were determined in seven dogs by applications of block during constant mean pressure perfusion of the carotid sinus at a conditioning pressure equal to the resting level of the animal before sinus isolation (105-160
mm Hg; mean, 137.5±17.2 mm Hg). In eight additional dogs, the isolated sinus was perfused at 150 mm Hg, regardless of the resting level of pressure in the intact animal. This CSP was chosen as one that would be above the pressure threshold (Pth) of most baroreceptors, including small A- and C-fiber baroreceptors, and would therefore not limit activation during constant CSP perfusion to primarily larger A-fiber baroreceptors with lower Pths. The effects of selective blocking of dynamic baroreflex control of changes in BP were determined by application of anodal or anesthetic block during the slow ramp increases in sinus pressure. The protocol was as follows. After a 25-minute perfusion of the carotid sinus at the selected conditioning pressure, 30 seconds of resting baseline levels of BP, a measure of tonic baroreflex control, was recorded. To determine dynamic baroreflex responses, pump perfusion of the carotid sinus was then abruptly halted, and the outflow cannula was clamped, temporarily making the sinus a closed pouch. A syringe pump (Harvard Apparatus, South Natick, Mass.), in line with the inflow cannula, was used to infuse lactated Ringer’s solution into the sinus pouch at a constant rate, producing a slow linear increase in CSP at a rate of 1–2 mm Hg/sec from 0 to 250 mm Hg. The response to the pressure ramp was used to construct baroreceptor stimulus–response curves by plotting CSP versus BP, the slope of which was used as a measure of dynamic baroreflex control. After the ramp, the carotid sinus was again perfused at the original conditioning pressure for 5 minutes to prevent any acute resetting effects, and either anodal or anesthetic blockade of the baroreceptor afferents was initiated.

For anodal block, tonic and dynamic baroreflex responses were obtained during application of anodal blocking currents of 20–200 μA to the left sinus nerve in random order, allowing sufficient time for baseline effects of the block to stabilize before initiation of the pressure ramp. After the tonic and dynamic baroreflex responses to each current were determined, the current was turned off, and the sinus was again perfused at constant pressure for 5 minutes. New control baseline and reflex responses were determined after each blocking run to ensure that the effects of the previous block were over. The repeated testing was done until control responses returned to normal. When very high blocking currents were used, the time needed to return to control could be as long as 1 hour. After control was reestablished, a new blocking current was tested. This procedure was repeated until no additional changes in tonic or dynamic baroreflex control of BP were obtained with increasingly higher blocking currents, thus determining the level of 100% maximum current. Once the maximum blocking current was determined, blocking was continued in random order until currents equal to 25%, 50%, and 75% of maximum had been tested.

For anesthetic block, tonic and dynamic baroreflex responses were obtained in six dogs during application of BUP to the left sinus nerve, with responses measured at 2 minutes and then 7 minutes after anesthetic application. In these animals, conditioning pressure of the sinus was set at presilation levels, in a manner similar to the first group of anodal block dogs. Conditioning pressure ranged from 115 to 155 mm Hg, with a mean of 135.2±6.2 mm Hg. BUP was applied in increasing concentrations, starting with 5 mg% and then increasing to 7, 10, and 20 mg% in the following manner. After the tonic and dynamic baroreflex responses at 2- and 7-minute exposures of 5 mg% were obtained, the anesthetic was removed, and the sinus was again perfused at constant pressure for 5 minutes. After this reconditioning period, the next level of anesthetic was applied, and the new 2- and 7-minute responses were measured. This protocol did not allow for full recovery between anesthetic exposures but did allow sufficient time for the major blocking effects of each level of the anesthetic to occur within the experimental period. This procedure was repeated up to 20 mg% BUP, after which the anesthetic was again removed, and recovery was allowed to occur. Repeated ramps in CSP were done at varying intervals until at least 75% of the initial response was obtained at the end of the experiment to ensure that loss of the reflex was not due to time or degradation of the preparation.

Data Analysis

For data analysis, analog-to-digital conversion of recorded parameters was performed using a computer (model 310, Hewlett-Packard Co., Palo Alto, Calif.). Arterial pressure and CSP were sampled at a frequency of 10 Hz for each control and blocking procedure and stored on disk files for quantification and statistical analysis. For anesthetic blockade, both the 2- and 7-minute responses were quantitated, but only the 7-minute responses were used in the final analysis, which represented more complete blocking at each level of anesthetic. Trends in the data at 2 minutes were similar to those obtained at 7 minutes, but the changes were greater at 7 minutes. As a measure of tonic baroreflex control, baseline values of BP were obtained using 30-second averages of this parameter sampled during the period immediately before ramps for control and each blocking condition. To determine dynamic baroreflex sensitivity, mean BP was plotted versus ramp changes in CSP to obtain baroreflex response curves. Nonlinear regression was used to curve-fit the sigmoidal response curves and determine Pth and maximum slope of the linear portion of the curves. Pths, slopes (sensitivities), and baseline BP values for control and each level of anodal or anesthetic block were compared using an analysis of variance. Significantly different means were located using Duncan’s multiple range test. All levels of significance were set at p<0.05 a priori.

Results

Anodal Block

Precise application of anodal blocking current resulted in a selective elimination of A-fiber activity without elimination of C-fiber activity, as shown from the representative small vagal preparation example taken from one dog in Figure 1. Conduction velocities of the fastest evoked A-fiber potentials in this example ranged from 9.8 to 98.9 m/sec, and the large evoked C-fiber potential began with fibers having conduction velocities of 2.8 m/sec. Potentials from smaller A fibers can be seen as smaller deflections in the baseline between the indicated A- and C-fiber potentials. When anodal block was applied to the small bundle of nerve fibers, increasing levels of current up to 60 μA produced
A-decremental decreases in larger A-fiber activity (fast potentials) before significantly altering C-fiber conduction (slow potentials). Some degree of smaller A-fiber activity also remained up to 60 μA of blocking current, demonstrated by retention of the small waves of evoked potentials preceding the C-fiber potentials. At currents above 60 μA, sequential decreases in smaller A- and C-fiber potentials can be seen with increasing levels of anodal block, with complete block of all potentials at 350 μA.

The selectivity of anodal block on carotid sinus baroreceptor activity was examined directly in two dogs by examining loss of pressure-induced baroreceptor potentials during anodal block (Figure 2). In this example from one dog, when anodal block was applied to the sinus nerve during constant pressure perfusion of the sinus, elimination of large pulse-related A-fiber discharges occurred up to 95 μA. Conduction velocity of the largest action potential was 31 m/sec, but conduction velocities of the other potentials could not be determined. At blocking currents of 95 μA and above, only smaller potentials that belonged to small A and C baroreceptor afferent fibers could still be seen riding in the noise of the recording. As shown by the first two figures, the blocking current level at which “complete” elimination of A-fiber activity occurs can vary, depending on nerve size and position of the electrode. However, typically most fibers blocked below 75–90 μA were A fibers. In this animal, 95 μA equaled 63% of maximum blocking current.

Increasing levels of anodal blocking current applied to the carotid sinus nerve produced changes in tonic control of baseline levels of BP and dynamic baroreflex control of changes in pressure (slope). As shown in the representative example from one animal in Figure 3, increasing levels of anodal block from 25% to 100% maximum current (100 μA absolute current for this animal) produced stepwise attenuation of the baroreflex-induced inhibition of arterial pressure during slow ramp increases in CSP. The slope of the reflex response was attenuated at all levels of blocking current starting at 25% maximum current. As seen in Figure 4, this attenuation of dynamic baroreflex control of pressure was accompanied by attenuation of tonic control of arterial baseline BP, but at only the higher levels of blocking currents. The level of baseline BP was the
same at control and during anodal block of the carotid sinus nerve at 25% and 50% maximum current. Thus, the loss of tonic inhibition of arterial pressure, shown by the increases in baseline BP, occurred in anodal block at blocking currents higher than those needed to produce attenuation of dynamic baroreflex control of pressure. Attenuation of the control of dynamic baroreflex changes in arterial pressure occurred at blocking currents that would primarily affect larger A-fiber baroreceptors, whereas attenuation of tonic control of baseline BP occurred at higher blocking currents that would include smaller A- and C-fiber baroreceptors. The results of anodal blocking of baroreceptors from all animals in which CSP was set to preisolation pressure levels are shown in Table 1. As suggested by the single representative example in Figures 3 and 4, significant attenuation of baroreflex sensitivity (slope) was obtained at lower levels of blocking current (25% of maximum current) than was significant attenuation of control of baseline BP (75% of maximum). In addition to attenuation of both tonic and dynamic baroreflex control of BP, anodal blocking also produced a significant elevation in Pth. As shown in Table 1, a significant increase in Pth from control and 25% maximum current occurred at blocking levels of 50% and 75% of maximum current. This effect on Pth preceded any significant changes in control of tonic baseline BP but occurred at a higher blocking current than that which attenuated dynamic baroreflex sensitivity. Since the baroreflex response was blocked at 100% maximum current, Pth could not be determined for this level of anodal block.

The responses obtained from animals in which CSP conditioning pressure was set at 150 mm Hg are shown in Table 2. As seen in this table, the results from this group of animals were similar to those obtained from animals in which CSP was set to preisolation pressure levels, except that no significant attenuation of baroreflex sensitivity was obtained until blocking was performed at 50% maximum current. Significant attenuation of tonic control of baseline BP was not obtained until blocking at 75% maximum current, a response the same as that in the first group of animals. The pattern of changes in Pth was the same in both groups of animals.

**Anesthetic Block**

Application of BUP produced a differential block of carotid baroreceptor afferent fibers, although the selectivity of the block was not absolute. As shown in the example from one vagal nerve preparation in Figure 5, increasing concentrations of BUP produced increasing levels of blockade of both A- and C-fiber conduction in a small multifiber nerve preparation of vagal fibers approximately the diameter of the carotid sinus nerve. In the vagal preparation, increasing the level of BUP to 3.3 mg% resulted primarily in blockade of C fibers (conduction velocities, <1.7 m/sec) and smaller A fibers (conduction velocities, 4.1–8.5 m/sec), shown by the decrease in amplitude of the evoked C-fiber potential and loss of small potentials between the faster A- and C-fiber potentials. Increasing the level of BUP to 5.0 mg% resulted in further loss of C-fiber activity and some of the faster evoked A-fiber potentials (conduction velocities, 8.5–18 m/sec). At 7.0 mg%, C-fiber conduction was almost completely blocked, based on the loss of the evoked C-fiber potential. At this concentration, some conduction of fast A-fiber afferents (conduction velocities, 19–68 m/sec) was still apparent, although greatly reduced. These results suggest that, up

**Figure 3.** Graph showing the effects of anodal block on dynamic baroreflex changes in mean arterial blood pressure in one dog. The typical reflex decrease in arterial pressure was obtained during slow ramp increases in pressure in a vascularly isolated carotid sinus (control). This sensitivity of baroreflex-induced hypotension was attenuated as anodal blocking currents from 25% to 100% maximum blocking current were applied to the carotid sinus nerve from the isolated sinus. Effects of anodal block on control of baseline blood pressure in the same animal are shown in Figure 4.

**Figure 4.** Effects of anodal block in one dog on tonic control of baseline levels of arterial blood pressure (BP) recorded at constant mean pulsatile perfusion of 135 mm Hg of the innervated, isolated carotid sinus. These results were obtained from the same dog whose baroreflex responses are shown in Figure 3. No changes in baseline BP from control were seen when 25% and 50% of maximum blocking current were applied to the sinus nerve. However, increasing the current to 75% and 100% of maximum current produced sequential increases in baseline arterial BP during the constant pressure stimulation of the sinus baroreceptors.
to a given concentration, most of the fibers blocked are small A and C fibers. However, beyond that concentration, there is significant reduction of conduction of all fibers. It is believed that lower concentrations of anesthetic produced C-fiber blockade in this preparation as compared with those used for carotid sinus nerve blockade, as described below, because a longer segment of nerve was exposed to the anesthetic. Attempts were made to limit anesthetic exposure to a short segment of the vagus nerve, but the complete vagal desheathing that was required for isolation of a small multifiber bundle of nerve fibers exposed more of the nerve to the anesthetic. When anesthetic was applied to the sinus nerve, a much smaller segment of nerve was exposed to the anesthetic, and higher concentrations of anesthetics were required.

The effects of anesthetic blockade of carotid baroreceptor afferent fibers on dynamic baroreflex control of BP are shown in Figure 6 and summarized in Table 3. In the example from one animal shown in Figure 6, at 5.0 mg% BUP there was no effect on the slope of the baroreflex-induced decrease in arterial pressure, although there was a slight increase in arterial pressures at all levels of CSP, relative to control values. This suggests that there was some inhibition of baroreceptor activity, although the afferents involved in buffering changes in CSP were not significantly affected. Increasing the concentration of BUP up to 7.0 mg% attenuated baroreflex slope, and higher levels of BUP produced increasing degrees of reflex attenuation until the entire reflex was blocked at 20.0 mg% BUP. This pattern is reflected in the summed data for all animals in Table 3. There was no significant attenuation of baroreflex sensitivity until 7.0 mg% BUP, after which there was an additional decrease in baroreflex slope at 10.0 mg% and 20.0 mg%. In most animals (four of six), the reflex was almost completely eliminated at 20.0 mg% BUP.

The effects of BUP on tonic control of pressure in a representative animal are shown in Figure 7, and data from all animals are summarized in Table 3. Figure 7 shows the effects of increasing blocking concentrations of BUP on the baseline level of BP recorded during constant pressure perfusion of the carotid sinus at 132 mm Hg from the same dog whose reflex responses are shown in Figure 6. As seen in Figure 7, as BUP concentration was increased, there was an elevation in baseline BP at 5.0, 7.0, and 10.0 mg% BUP, indicating inhibition of baroreceptor control of tonic BP. However, there was no additional increase in baseline BP at 20.0 mg% BUP. These results are similar to those for all animals, as shown in Table 3. There was a significant elevation of baseline BP at 5.0 and 7.0 mg% BUP over control BP, and baseline BP was maximally increased at 10.0 mg%. No further significant increases in baseline BP were seen at 20.0 mg% BUP for the summed data for all animals (Table 3), although some individual dogs showed an additional elevation at the highest level of BUP. As seen in Table 3, there were no significant changes in Pth in response to any level of BUP exposure. There was an initial insignificant drop in Pth at 5.0 mg% BUP, but with additional blocking, the Pth actually increased over control Pth. Since the baroreflex response was blocked at 20.0 mg% BUP in most animals, Pth was not determined for this level of anesthetic exposure. These results suggest that anesthetic exposure resulted in a differential pattern of attenuation of baroreceptor control of pressure, with significant attenuation of tonic control of baseline BP observed at the lowest concentration of BUP (5.0 mg%) but no signifi-

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**Table 1. Effects of Anodal Block on Tonic (Blood Pressure) and Dynamic (Slope) Baroreflex Control of Blood Pressure, With Carotid Sinus Perfusion Pressure Set to Preisolation Pressures**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>25%</th>
<th>50%</th>
<th>75%</th>
<th>100%</th>
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<tr>
<td>BP (mm Hg)</td>
<td>158.4±9.5</td>
<td>160.7±9.5</td>
<td>166.4±10.9</td>
<td>181.8±11.1</td>
<td>189.4±13.2</td>
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<td>Slope (mm Hg)</td>
<td>–0.84±0.11</td>
<td>–0.63±0.10</td>
<td>–0.36±0.09</td>
<td>–0.11±0.05</td>
<td>–0.01±0.01</td>
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<tr>
<td>MAP/mm Hg CSP</td>
<td>129.0±9.4</td>
<td>138.4±10.3</td>
<td>160.3±13.7</td>
<td>174.5±7.6</td>
<td>...</td>
</tr>
</tbody>
</table>

BP, arterial pressure during constant pressure perfusion of the carotid sinus; MAP, mean arterial pressure; CSP, carotid sinus pressure; Pth, pressure threshold of the baroreflex. Values are mean±SEM; n=7 dogs.

*Significantly different from control and 25% values. †Significantly different from control, 25%, and 50% values.

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**Table 2. Effects of Anodal Block on Tonic (Blood Pressure) and Dynamic (Slope) Baroreflex Control of Blood Pressure, With Carotid Sinus Perfusion Pressure Set to 150 mm Hg**

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<th>50%</th>
<th>75%</th>
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<tr>
<td>BP (mm Hg)</td>
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<td>Slope (mm Hg)</td>
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<td>–0.53±0.13</td>
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<td>MAP/mm Hg CSP</td>
<td>119.9±7.1</td>
<td>132.7±9.3</td>
<td>154.7±12.9</td>
<td>172.4±6.3</td>
<td>...</td>
</tr>
</tbody>
</table>

BP, arterial pressure during constant pressure perfusion of the carotid sinus; MAP, mean arterial pressure; CSP, carotid sinus pressure; Pth, pressure threshold of the baroreflex. Values are mean±SEM; n=8 dogs.

*Significantly different from control and 25% values. †Significantly different from control, 25%, and 50% values.

†Significantly different from control, 25%, 50%, and 75% values.
c

cant attenuation of dynamic baroreflex control until 7.0 mg% BUP.

Discussion

Possible differential control of BP by different baroreceptor types has been suggested in earlier studies, although classification of baroreceptors in earlier studies was based solely on afferent fiber type.8,19–21 These studies have suggested that, because of the higher pressure thresholds of C-fiber baroreceptors, their primary contribution to BP control was during elevations in pressure from the normal resting level. In addition, because of the lower sensitivity and more irregular firing of C-fiber baroreceptors, it has been suggested that C-fiber baroreceptors may be most important in relaying information about the level of mean arterial pressure.20 Based on the earlier finding that type I baroreceptors generally have larger afferent fibers than type II baroreceptors,1 the present study used two techniques that block fibers according to size to try to establish roles for two different types of functionally characterized baroreceptors. Although both techniques allowed fairly selective blockade of large A fibers versus smaller C fibers, the ability to selectively block small A fibers versus C fibers, especially with local anesthetic blockade, was less discrete. Thus, although some effects can be attributed to loss of only large-diameter afferent fibers, the effects when smaller fibers are blocked must be considered to be the result of blocking both small A and C fibers. The selectivity of blockade for both techniques is presented later in the discussion. However, since the two nerve-blocking techniques reversed the order of blocking of afferent fiber types, their use allowed the different roles of these baroreceptor types to be discerned.

In the present study, large A-fiber baroreceptors (type I) appeared to be required to maintain maximum gain of the baroreflex and produced the greatest changes in arterial pressure when CSP was changing. Small A- and C-fiber baroreceptors (type II) needed to be present to prevent changes in tonic levels of BP, since the greatest changes in baseline “resting” pressure occurred when the afferent fibers from these receptors were blocked. This selective pattern of pressure control is shown graphically in Figure 8. The effects of BUP blockade (top panel) on paired values of baseline BP and baroreflex slope are plotted for different levels of anesthetic. Responses for individual animals are shown by the thinner background lines, and the average curve fit for all animals is shown by the bold line. Slope and baseline BP are presented as percents of maximum: maximum slope was that obtained at control, before initiation of baroreceptor afferent blockade, and maximum baseline BP was that obtained at the higher levels of anesthetic blockade, when no further increase in baseline BP was seen after additional increases in BUP concentration, indicating complete block of all afferent fibers. The starting point for each individual curve reflects the control values for baseline BP and baroreflex slope for each animal (control in Figure 8). The end points of each curve are the points obtained at the higher levels of BUP when all baroreceptor afferents are blocked (maximum block). As seen in this figure, the initial effects of anesthetic blockade were primarily
effects of anesthetic block (bupivacaine) on tonic (blood pressure) and dynamic (slope) baroreflex control of blood pressure, with carotid sinus perfusion pressure set to preisolation pressures

<table>
<thead>
<tr>
<th>BP (mm Hg)</th>
<th>Control 5%</th>
<th>Bupivacaine concentration (mg%)</th>
<th>7%</th>
<th>10%</th>
<th>20%</th>
</tr>
</thead>
<tbody>
<tr>
<td>158.8±6.4</td>
<td>169.0±6.5*</td>
<td>174.3±5.4*</td>
<td>175.7±4.7†</td>
<td>175.2±5.9†</td>
<td></td>
</tr>
<tr>
<td>Slope (mm Hg)</td>
<td>-0.85±0.08</td>
<td>-0.73±0.08</td>
<td>-0.64±0.09†</td>
<td>-0.25±0.08‡</td>
<td>-0.12±0.04‡</td>
</tr>
<tr>
<td>MAP/mm Hg CSP</td>
<td>142.7±8.6</td>
<td>137.4±7.8</td>
<td>145.0±9.1</td>
<td>149.0±7.9</td>
<td>...</td>
</tr>
<tr>
<td>Pth</td>
<td>145.0+9.1</td>
<td>149.0+7.9</td>
<td>...</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

BP, arterial pressure during constant pressure perfusion of the carotid sinus; MAP, mean arterial pressure; CSP, carotid sinus pressure; Pth, pressure threshold of the baroreflex. Values are mean±SEM; n=6 dogs.

*Significantly different from control value. †Significantly different from control and 5 mg% values. ‡Significantly different from control, 5 mg%, and 7 mg% values.

increases in baseline BP, although some reduction in baroreflex slope was also seen. The greatest decrease in slope, however, occurred after the maximal increase in baseline BP was obtained. The initial responses were obtained at lower BUP blocking concentrations, when primarily small baroreceptor afferent fibers were blocked. The later responses occurred at higher BUP concentrations when larger baroreceptor afferents were additionally blocked. This pattern of differential control can be compared with the results obtained with anodal blockade of baroreceptor afferent fibers, also shown in Figure 8 (bottom panel). The effects of anodal block on paired values of baseline BP and slope are plotted for different levels of blocking current. Again, responses for individual animals are shown by the thinner background lines, and the average curve fit for all animals is shown by the bold line. Slope and baseline BP are presented as percent of maximum in a manner similar to that described for anesthetic block, with maximum slope taken as that before any baroreceptor blocking (control in Figure 8) and maximum BP taken as that at 100% maximum blocking current (maximum block), where all baroreceptor afferents are blocked. The order of afferent blocking with anodal block is the reverse of that with anesthetic blockade, with larger baroreceptor afferent fibers blocked before blockade of smaller afferents. The pattern of results obtained with anodal block is the reverse of that seen with anesthetic blockade: There was first a large, significant decrease in slope without much attenuation of control of baseline BP. The greatest increase in baseline BP was seen at the higher levels of blocking current, at which point there was a smaller attenuation of baroreflex slope. Therefore, for both anodal and anesthetic blockade, the greatest attenuation of control of baseline BP occurred during blockade of small A- and C-fiber baroreceptor afferents, and the greatest attenuation of baroreflex slope occurred during blockade of larger A-fiber baroreceptor afferents. Both techniques of blockade indicated that baroreceptors with smaller afferent fibers contributed somewhat to control of dynamic baroreflex sensitivity, whereas baroreceptors with the largest afferent fibers did not contribute to control of tonic levels of blood pressure.

Results from both anodal block studies (i.e., different conditioning pressures) indicated that a significant elevation in Pth was seen after primarily A-fiber blockade with 50% maximum current. Additional anodal blockade to include C fibers did not produce any further significant increase in Pth. This suggests that A-fiber baroreceptors with lower Pths contributed most to determination of Pth of the intact reflex. This result is predictable because of the finding that these baroreceptors contributed most to dynamic baroreflex control. There were no significant changes in Pth during BUP block, suggesting that the overall contribution of C-fiber baroreceptors to Pth for the whole reflex is minimal. This is based on the concept that activation of A-fiber baroreceptors with lower Pths would initiate the reflex decrease in pressure at a point below which most C-fiber and small A-fiber baroreceptors would be active. In addition, it is assumed that the absolute amount of C-fiber activity that contributes to initiating a response to a dynamic change in pressure is less than the amount contributed by A-fiber baroreceptors. This assumption is supported by the results from the anodal block study, which indicated that the relative contributions of C-fiber baroreceptors to dynamic reflex changes in blood pressure were significantly less than the overall contribution...
of A-fiber baroreceptors. It is not known if there would be a significant change in Pth at higher levels of BUP than those used in the present study. However, since the A-fiber baroreceptors with lower Pths would be blocked last, there is a possibility that Pth under these conditions would not change significantly from control.

Activation of arterial baroreceptors, either through pressure stimulation of the receptor directly or by electrical stimulation of afferent fibers, is known to initiate depressor responses, but most studies have focused on the role of larger A-fiber, or type I, baroreceptors. However, the role of C-fiber baroreceptors, which are primarily type II baroreceptors when functionally classified, has been examined in a few studies by other investigators. These studies suggest that, although both A- and C-fiber baroreceptors mediate depressor responses, prolonged control of BP requires C-fiber baroreceptor participation, a finding that agrees with the preferential control of tonic pressure by baroreceptors with smaller afferents observed in the present study. One of the earliest studies, by Douglas et al., showed that the greatest hypotension produced by electrical stimulation of aortic baroreceptor afferent fibers in rabbits required activation of C-fiber baroreceptors. C-fiber baroreceptor activation was also required to maintain the hypotension for the duration of the stimulation, leading the investigators to propose that A-fiber baroreceptors may be involved in rapid adjustment of systemic blood pressure but that the C-fiber baroreceptors may play a longer-lasting role. Similar results were noted by Kendrick and Matson for electrical activation of carotid baroreceptors in dogs. Again, the largest and most-maintained hypotension and bradycardia seen with baroreceptor stimulation required C-fiber activation. Kardon et al. found little effect on BP and heart rate with electrical activation of only large aortic A-fiber baroreceptor afferents in the rabbit. Some depressor responses were observed with smaller A-fiber baroreceptor afferent activation, but the greatest changes accompanied C-fiber baroreceptor stimulation. In a study by Aars et al. that used anodal block to elucidate the role of aortic C-fiber baroreceptors in the rabbit, the investigators found that A-fiber baroreceptors produced a greater reflex decrease in sympathetic activity, especially at low pressures, than did C-fiber baroreceptors in response to a change in aortic pressure. The difference in reflex sensitivity at different pressure ranges is in agreement with the differences in Pths reported for A-fiber versus C-fiber baroreceptors. In the study by Aars et al., tracings indicate no change in baseline levels of pressure or sympathetic activity in response to anodal block of A-fiber baroreceptors at resting aortic pressures, although the reflex effects to increases in aortic pressure were eliminated or greatly attenuated. These data are in agreement with the present study; however, it must be noted that the resting level of arterial pressure was approximately 75 mm Hg, which could have been lower than the Pth for most C-fiber baroreceptors. Finally, an earlier study by Wiemer and Kiwull, who used cold block to attempt a selective block of carotid sinus afferents, suggested that there were different types of baroreceptors—one characterized by larger spikes and a greater sensitivity toward BP changes and a second less-sensitive type with smaller spikes. Figure 3 from the

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**FIGURE 8.** Graphs showing the effects of anesthetic bupivacaine block (top panel) and anodal block (bottom panel) on paired values of baseline blood pressure (BP) and baroreflex slope obtained for different levels of blockade. Slope and baseline BP are presented as percent maximum, with maximum slope taken as that before any baroreceptor blocking (control) and maximum BP taken as that during complete blockade of afferent fibers, when all baroreceptor input is eliminated (maximum block). The fainter tracings in each panel represent the responses of individual dogs, and the bold lines indicate an average curve fit (single exponential) for all tracings in each panel. During anesthetic blockade, the greatest effect on pressure control from control to maximum block is initially an increase in baseline BP, which is maximal after slope is attenuated to approximately 70% of maximum (top panel). The largest response after that point is attenuation of baroreflex slope with little increase in baseline BP until complete inhibition of slope at maximum blockade. The effects on pressure control during anodal blocking are opposite to those during anesthetic block (bottom panel). When blocking is initiated, little effect on baseline BP is seen until baroreflex slope has been attenuated to approximately 25% of maximum. After that point, there is an increase in baseline BP until maximum block is reached. These responses suggest that blockade of large baroreceptor afferent fibers, which occurred first during anodal block and last during anesthetic blockade, resulted primarily in a decrease in baroreflex control of dynamic BP. However, blockade of smaller afferent fibers, which occurred last during anodal block and first during anesthetic blockade, resulted primarily in loss of tonic inhibitory control of baseline BP, accompanied by some attenuation in dynamic baroreflex sensitivity.
study demonstrated that cooling of the carotid sinus nerve to approximately 14°C, which decreased nerve impulse traffic by 10–20%, was required before changes were seen in resting BP, suggesting that elimination of some large A fibers at the higher cold block temperatures did not alter control of tonic levels of BP. However, the results of the study of Wiemer and Kiwull do suggest a greater role for A-fiber baroreceptors in the control of resting pressures than the results of our study. Three possibilities may partially explain this difference. First, the technique of nerve analysis used by these authors did not include “the smallest impulses in the vicinity of the noise level,” so it is difficult to determine the extent of blocking of small A and C fibers in this study. In addition, it is not possible to separate larger A-fiber versus smaller A-fiber axons, since conduction velocities of the potentials were not given. Second, the technique of cold block does not permit as selective a block as anodal block.22 Previous studies have shown that, even at blocking temperatures at which A fibers have been described as completely blocked (7°C), there can be a functional block of C-fiber input.23 This may be due to the slowing effects on conduction of the C fibers that occurs before complete blocking. Finally, the range of blocking temperatures for myelinated fibers (conduction velocities, 20–50 m/sec) versus nonmyelinated fibers (conduction velocities, <2.5 m/sec) has been found to overlap (2.5–8.1°C versus 1.5–7.1°C, respectively), making selective elimination of myelinated fibers in this range difficult.23

Some comments need to be made regarding the techniques of anodal and anesthetic blockade. Anodal block has been successfully used to differentially block A versus C fibers in the aortic4–8 and vagal nerves.24 However, some earlier studies that used the technique found problems with either electrical destruction of the nerve or inadvertent activation of fibers during initial application of the blocking current.14,15,25 Neither was found to be a problem in the present study. The low levels of current needed to block baroreceptor afferent fibers did not produce any damage to the nerve. This was confirmed by repeated testing of control ramps after each level of block. In only a few experiments, some activation of baroreceptor afferent was directly observed, or suggested, by transient decreases in baseline BP as the current was applied. When this excitatory effect was observed, the increase in nerve activity was only transient, and baseline BP returned to control or higher levels as the block was continued. In only one case, BP failed to return to control levels within 10–20 seconds, and this experiment was discarded. It is believed that modifications of the anodal blocking technique used in the present study have minimized the transient activation problem by routing the depolarizing cathodal current through multiple current paths, thus reducing its depolarizing effects.

The use of the local anesthetic BUP to initiate selective blockade was based on the findings of earlier studies. Gissen et al17 found that 250 mg% BUP produced an 80% blockade of C fibers but also a 100% blockade of A fibers in the rabbit vagus nerve in 3 minutes. However, lowering the level of BUP to a range of 1.3–9.3 mg% (0.03–0.40 mM) allowed more selective blockade of C fibers. At these lower concentrations, 79.2 minutes was needed before A-fiber blockade exceeded C-fiber blockade, although the extent of blockade of both fibers was only approximately 20% at that time. This study indicated that two factors were involved in initiating fiber block by BUP— the concentration of the anesthetic used and the time allowed for block to occur. A later study by Palmer et al18 also examined the blocking ability of BUP on the dog vagus nerve. In that study, 20 mg% BUP produced a 78% blockade of C fibers in 3.2 minutes after exposure, whereas 5.4 minutes was required to block 86% of A fibers. Because of the lower lipid solubility characteristics and higher pK of BUP, the selectivity of blocking by BUP was found to be superior to that for lidocaine19 or etidocaine,20 two other local anesthetics. In the two earlier studies cited above, a significant length of nerve was exposed to the local anesthetic, which may have contributed to the decreased selectivity of blockade. As reported by Franz and Perry,16 by limiting anesthetic exposure to 2 mm of nerve or less, at most only three nodes of Ranvier will be exposed for the smaller A fibers and two or less for the larger A-fiber neurons. Since three is the minimum number of nodes that must be blocked to eliminate conduction in the myelinated axons,26 by limiting the exposed length of nerve, a more selective block of small A-fiber and C-fiber afferents can be achieved. Thus, the differential block of small fibers in the study by Franz and Perry was significantly increased when exposure to procaine was limited to 2 mm of cat saphenous nerve and relatively low concentrations of procaine (10–20 mg%) were used. Blockade of conduction in small A fibers was observed at approximately 14 minutes after exposure to procaine, whereas larger A fibers were not blocked at 1 hour after anesthetic exposure. Unmyelinated C fibers were found to block at about the same time as the smaller A fibers.

Thus, four factors must be considered when attempting differential blockade using local anesthetics: 1) the anesthetic used, 2) concentration of the anesthetic, 3) time of exposure, and 4) length of nerve exposed. Also, care must be taken when interpreting the degree of block using amplitude of evoked potentials. For example, if conduction of some C fibers is not completely blocked, but only slowed, the evoked C-fiber potential will spread out, producing an apparent reduced amplitude. Finally, not all A or C fibers will be blocked at the same concentration because of their location within the nerve trunk. However, with these reservations in mind, anesthetic blockade can be used to produce a fairly selective block, evidenced by the separation in responses described in the present study. Our observations were limited to responses observed at 7 minutes after application of low doses of BUP to a limited portion of the nerve trunk. Using these precautions, we were able to differentiate control of two aspects of BP regulation, assessing the contribution of baroreceptor types with different afferent fiber types to tonic versus dynamic pressure control. A similar utilization of local anesthetic blockade was performed in a study to separate the contributions of afferent activity carried by larger myelinated (groups I and II) versus small myelinated (group III) and unmyelinated (group IV) fibers from a muscle to the reflex cardiovascular and respiratory responses initiated by exercise of the muscle.27 Careful application and monitoring of evoked potentials indicated that, although some conduction block of large
A fibers occurred during the more complete blockade of the type III and type IV fibers, reflex attenuation correlated well with loss of conduction in the smaller fibers. Thus, although anesthetic blockade was not completely selective, careful use of the technique permitted discrimination of the reflex pathway to the smaller myelinated and unmyelinated fibers. However, comparison of the two blocking techniques in the present study indicated that anodal block produced a more selective and controllable block than did anesthetic block with BUP.

Results from the present study indicate that selective blocking of baroreceptors unmasks differential roles for two types of baroreceptors in the control of BP. Large A-fiber baroreceptors appear to contribute more to control of dynamic baroreflex regulation of changes in arterial pressure, whereas smaller A- and C-fiber baroreceptors appear to be the primary regulators of tonic baseline levels of BP. This differential contribution to control of two aspects of BP regulation is not absolute, for baroreceptors with smaller afferent fibers were found to contribute in some degree to dynamic pressure control. However, as suggested by the firing characteristics of the baroreceptors, each baroreceptor type appears to preferentially contribute to either dynamic or tonic control of BP. Results from the present study indicate that there can be a functional separation of regulation of these two aspects of pressure control.

Acknowledgment

The authors would like to thank Ms. Claudia Hermes for her technical assistance.

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Selective contribution of two types of carotid sinus baroreceptors to the control of blood pressure.
J L Seagard, F A Hopp, H A Drummond and D M Van Wynsberghe

doi: 10.1161/01.RES.72.5.1011

_Circulation Research_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

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