Contrasting Effects of Static and Pulsatile Pressure on Carotid Baroreceptor Activity in Dogs

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The purpose of this study was to contrast the effects of static and pulsatile pressure on carotid baroreceptor activity over a wide range of mean arterial pressure. Static and pulsatile pressure were applied to the isolated carotid sinus of dogs anesthetized with chloralose. Recordings were obtained from single baroreceptor units as well as from the whole sinewave nerve or a large strand of the nerve. Three observations are reported. First, in single units the pulsatile pressure threshold, which averaged 48 ± 8 (SEM) mm Hg, was far below the static pressure threshold, which averaged 79 ± 8 mm Hg. This indicates that pulsatility or dynamic stretch causes more baroreceptor activity than a shift to pulsatile pressure at equivalent pressures. In single units, the decrease approximated 15% (from 32.0 ± 2.1 to 35.5 ± 1.9 spikes/sec, p < 0.05, n = 25). In all units, there was no diastolic nerve activity ("silence") when diastolic pressure was 1 to 10 mm Hg above static pressure threshold; 80% of the units exhibited "diastolic silence" when diastolic pressure was 20-30 mm Hg above threshold and 40% of the units showed silence at diastolic pressures 40-50 mm Hg above threshold. In whole nerve recordings, pulsatility increased activity from 57 ± 15 to 142 ± 29 spikes/sec (p < 0.05) at low mean arterial pressures (50 and 75 mm Hg), as expected from the reduction in pressure threshold noted in single units, and decreased activity by 15% (from 373 ± 69 to 320 ± 55 spikes/sec, p < 0.05, n = 9) at mean arterial pressures of 125 and 150 mm Hg. This decrease in activity with a shift from static to pulsatile pressure at moderate arterial pressures has not been reported previously. Third, the static pressure-activity curve was sigmoid, and its gain peaked sharply at 75-100 mm Hg; in contrast, the pulsatile pressure-activity curve was linear between 25 and 150 mm Hg, and its maximum gain was half the maximum gain during static pressure. These differences between the static pressure-activity curve and the pulsatile pressure-activity curve were noted during both increases and decreases in carotid sinus pressure; both curves exhibited some hysteresis during the decreases in pressure.

Several studies have described the effect of pulsatility or fast and slow pressure ramps on arterial baroreceptors and on the arterial baroreflex. The majority indicate that pulsatility or dynamic stretch augments the baroreflex, suggesting that dynamic stretch causes more baroreceptor activity than static stretch at equivalent pressures.

Although an increase in activity with pulsatile pressure has been reported with multiple unit recordings, the results with single units have not consistently shown an increase. In the present study, we examined systematically single units of carotid baroreceptors as well as multiple units from recordings of sinewave nerve activity at various levels of static and pulsatile pressure. Five levels of arterial pressure were considered. Our rationale for the work was as follows:

First, when static pressure (SP) is below the threshold for activity (SPTH) a shift to pulsatile pressure (PP) may initiate activity even when systolic pressure does not reach the SPTH (Condition I; Figure 1). In 1971, Angell-James showed in the isolated aortic arch preparation that sinewave pulsation initiates aortic baroreceptor activity when systolic pressure is slightly lower than SPTH, usually by 2 to 3 mm Hg and no more than 10 mm Hg. On the other hand, studies on carotid sinus baroreceptors with fast ramps instead of sinewaves showed that a fast ramp initiates activity at much lower pressures, as low as 40-80 mm Hg below SPTH. We elected, therefore, to examine the effect of sinewave pulsation on carotid baroreceptors when systolic pressure was significantly below SPTH.

Second, previous reports indicate that a shift from static pressure to pulsatile pressure when diastolic pressure is above SPTH (Conditions IV and V; Figure 1) does not change single unit activity. On the other hand, two observations suggested that this finding is worthy of further exploration. One was the report by Stegemann et al that in the isolated carotid sinus at relatively high pressure, a shift from static to PP can increase systemic arterial pressure, which would be compatible with a decrease in baroreceptor activity. The other was that of Arndt et al who showed in two
single units a decrease in activity with pulsatility near saturation pressure.11
Our results demonstrate major differences between the static and pulsatile pressure-activity curves. Opposite changes in the magnitude and direction of baroreceptor activity were seen with a shift from SP to PP depending on the overall level of carotid sinus pressure.

Materials and Methods
Mongrel dogs (16–24 kg) were anesthetized with thiopental sodium (30 mg/kg i.v.) and α-chloralose (80 mg/kg i.v.). Supplemental doses of chloralose were administered hourly. The dogs were intubated and mechanically ventilated with room air supplemented with oxygen. Ventilation was adjusted to maintain arterial Pco2 between 30 and 42 mm Hg. Sodium bicarbonate was administered when necessary in order to maintain arterial pH between 7.30 and 7.45. Catheters were placed in a femoral artery and vein for pressure measurements and drug administration, respectively.

Isolated Carotid Sinus Preparation
The left carotid sinus was surgically exposed, and all arteries in the vicinity of the sinus were ligated. Catheters were placed in the common and external carotid arteries. The isolated sinus was flushed and filled with a physiological saline solution equilibrated with 95% O2-5% CO2 and warmed to 37° C. The solution contained the following substances in their respective concentrations (mM): CaCl2-2H2O 2.5, KCl 4.7, MgSO4 1.2, KH2PO4 1.1, CH3COONa 3.0, NaHCO3 24.3, NaCl 98.0, glucose 10.0, and NaHCO3 24.3 was connected to a pressure reservoir via the common carotid artery, and carotid sinus pressure (CSP) was measured through the external carotid-catheter by a Statham (P23AA) transducer. In separate experiments, positive and negative dP/dt were measured by differentiating the amplified output from the pressure channel.

The mean level of CSP was controlled by adjusting a gas regulator valve connected to a pressurized air source. Pulsatile pressure was introduced by two methods. First, during slow ramp increases in pulsatile pressure (Figure 2, right panel) a motor driven piston (Harvard respirator) supplied constant volume sine-wave pulses into the pressure reservoir. Pulse rate was maintained constant within each experiment (90–130 pulses/min) but pulse pressure increased as mean pressure increased, presumably as a result of the

![Figure 1](image1.png)

**Figure 1.** Diagram illustrating the conditions under which the effects of pulsatility on unit baroreceptor activity were examined. For details see text. Interrupted line, static pressure threshold; Syst. P., systolic pressure; Diast. P., diastolic pressure.

![Figure 2](image2.png)

**Figure 2.** Single unit baroreceptor activity during static (left panel) and pulsatile (right panel) pressure ramps. Initiation of unit activity occurred at a much lower pressure during pulsatile pressure (PPth = 45 mm Hg) than during static pressure (SPth = 86 mm Hg). The results from 15 single units are shown in the bar graph. Error bars represent ± SEM. The asterisk indicates that the systolic pressure at which activity was initiated was significantly (p<0.05) less than static pressure threshold.
nonlinear compliance characteristics of the carotid sinus. This technique was only used to determine the pulsatile pressure threshold (PPth) of single units and not to determine the pressure-activity relation of baroreceptors. The pulse pressure was always within a physiologic range (20-40 mm Hg) when threshold was reached.

Second, in the major group of experiments a voltage waveform generator (Millar, Houston, Tx.) fed sine-wave pulses into an electromagnetic pressure converter (Millar) that was connected to the reservoir. The generator enabled instantaneous conversion from SP to PP and vice versa without changing the mean CSP and allowed for control of pulse rate and pulse pressure. In all experiments, the frequency ranged from 90 to 130 pulses/min, and pulse pressure ranged from 30 to 50 mm Hg. Pulse rate did not vary, and pulse pressure and dP/dt remained relatively constant within a given experiment. The characteristics of the pressure generating system along with the manual control enabled us to keep pulse pressure and frequency relatively constant over a wide range of mean pressures. Thus, peak dP/dt was dependent on the rate and amplitude of the pressure pulse but independent of mean pressure; peak positive dP/dt ranged from 200 to 400 mm Hg/sec, and peak negative dP/dt ranged from 175 to 350 mm Hg/sec. Immediately after the introduction of pulsatile pressure, the amplitude of the pulse pressure decreased for 3 to 5 seconds before a stable pulse pressure was obtained (see Figures 3 and 6). Measurements of baroreceptor activity were taken approximately 15 seconds after the initiation of pulsatile pressure, when pulse pressure and nerve activity were stable.

Carotid Sinus Nerve Recordings

The carotid sinus nerve was cut near its junction with the glossopharyngeal nerve, placed on a dissection stage, covered with paraffin oil, and desheathed. Other nerves innervating the sinus region and the vagosympathetic trunk were sectioned. Baroreceptor activity was recorded with a bipolar platinum electrode connected to a Grass high-impedance probe (model HIP 511E, Grass Instrument Co., Quincy, Mass.) and amplified by a Grass (model P511) bandpass amplifier (high-frequency cutoff 3,000-10,000 Hz; low-frequency cutoff 30 Hz). Nerve traffic was visualized on a Tektronix dual-beam storage oscilloscope (model 5113, Beaverton, Ore.) and listened to through a loudspeaker. A nerve traffic analyzer that counts spikes exceeding a selected voltage was used to quantify nerve activity. Carotid sinus pressure, integrated nerve activity, mean activity per unit time, and systemic arterial pressure were continuously monitored on a Beckman dynograph recorder (model R411, Schiller Park, Ill.).

Activity was recorded from either the whole sinus nerve or from large strands of the sinus nerve in 9 dogs. Single unit activity was recorded from 53 units in 45 dogs. Single units were identified by repeatedly splitting the sinus nerve until a fine strand was obtained that exhibited the following characteristics: 1) increased activity of uniform spike height in response to increases in CSP, 2) relatively constant interspike interval during maintained static CSP, and 3) the lack of any interfering spikes above a preselected voltage when CSP was increased to over 200 mm Hg.

The gas tensions and pH of the physiological saline and arterial blood were such that chemoreceptor activity was minimized. When pressure insensitive nerve activity was present the nerve was split to minimize it. Although conduction velocity was not measured, the relatively low thresholds and high discharge rates suggest that most of the single units studied were connected to myelinated afferents.

Protocols and Data Analysis

PRESSURE THRESHOLD DURING STATIC AND PULSATILE PRESSURE (SINGLE UNITS). Static pressure threshold (SPth) and pulsatile pressure threshold (PPth) were determined in 15 single units as described below. In 8 units, both SP and PP were increased from low levels in a slow ramp (<3 mm Hg/sec) until activity occurred (Figure 2). Thus, PPth was defined as the lowest systolic pressure necessary for receptor activation. In the remaining 7 units, both SP and PP were held at a given level near threshold and were either increased or decreased in 1 to 2 mm Hg steps until the minimal pressure necessary for activity was achieved. Comparable results were obtained by both methods.

EFFECT OF A SHIFT FROM STATIC TO PULSATILE PRESSURE AT VARIOUS MEAN PRESSURES ON SINGLE UNIT ACTIVITY. The effect of a shift from SP to PP on unit activity was determined in 38 units. SPth was obtained with a slow ramp increase in SP (<3 mm Hg/sec). SP was then set at either 25 or 50 mm Hg for approximately 10 minutes. In 26 of the single units, the CSP and nerve activity were measured as CSP was converted from SP to PP to SP at various levels of mean CSP from 25 to 200 mm Hg in steps of 25 mm Hg. Pulse frequency remained constant and pulse pressure and dP/dt were relatively unchanged during the changes in mean pressure. At each level of pressure, SP and PP were each held for 15-20 seconds. Nerve activity stabilized within 10-12 seconds after each 25 mm Hg pressure step. If activity was not stable within 15 seconds after the pressure step, the pressure was maintained until the activity stabilized. The effect of PP was reversed promptly with the return to SP.

The nerve activity is reported at five levels of arterial pressure characterized as follows: Condition I, systolic CSP<SPth; Condition II, mean CSP<SPth and systolic CSP>SPth; Condition III, mean CSP>SPth and diastolic CSP<SPth; Condition IV, mean CSP<saturation pressure and diastolic CSP>SPth; and Condition V, CSP at or near saturation pressure (Figures 1 and 3).

In addition, the pressure dependence of "diastolic silence" above SPth was determined from the results obtained during Condition IV. The highest diastolic pressure at which "silence" still occurred was recorded for each single unit (n = 25), and the difference from SPth was calculated.

Because baroreceptors may exhibit hysteresis, i.e.,
their response depends on the direction of the change in pressure,\textsuperscript{5,12,16,17} we contrasted the effect of a shift from SP to PP during step increases and decreases in pressure between 40 and 200 mm Hg in 12 single units from 10 dogs.

**Effect of a Shift from Static to Pulsatile Pressure at Various Mean Pressures on Whole Nerve Activity.**

As with single unit activity the protocol consisted of a sequence of SP, PP, and SP at various levels of mean CSP to examine the effect of pulsatility on multiple unit activity \( (n=9) \). The nerve activity is reported under three pressure conditions: Condition A (at either 50 or 75 mm Hg), which coincides with Conditions I and II in single units; Condition B (at either 125 or 150 mm Hg), which coincides with Conditions III and IV of single units; and Condition C (at 200 mm Hg or saturation pressure), which coincides with Condition V in single units.

**Effect of Pulsatility on Baroreceptor Gain.** Both single and multiple unit activity were analyzed to obtain the slopes of the CSP-activity curve at various mean pressures. The slopes (gains) were determined for each 25-mm-Hg step increase in mean CSP during both SP and PP as follows:

\[
\text{Slope}_{25-50} = \frac{(BA_{50} - BA_{25})}{25} \\
\text{Slope}_{50-75} = \frac{(BA_{75} - BA_{50})}{25}
\]

and so forth where 25, 50, and 75 represent CSP in mm Hg, and BA represents baroreceptor activity. The calculated slopes provide an index of the gain and the change in sensitivity with increasing pressure during SP and PP. Gain as defined here relates the magnitude of the output signal (baroreceptor activity) to the magnitude of the stimulus (carotid sinus pressure).\textsuperscript{18}

**Statistical Analysis.** All results are expressed as the mean ± SEM. Threshold and nerve activity data were analyzed by the paired \( t \) test, and the data on gain were analyzed by analysis of variance (ANOVA). When significance was shown by ANOVA, the paired \( t \) test with the Bonferroni adjustment was used to determine which differences were significant.\textsuperscript{19} Significance was accepted at a \( p<0.05 \).

**Results**

**Pressure Threshold During Static and Pulsatile Pressure (Single Units).**

The record in Figure 2 shows in a single unit that activity was initiated at 86 mm Hg during a SP ramp and at a systolic pressure of 45 mm Hg during a PP ramp.

In 15 single units, activity was initiated during PP at systolic pressures ranging from 13 to 61 mm Hg below SPTH. SPTH averaged 79 ± 8 mm Hg whereas PPTH averaged 48 ± 8 mm Hg (Figure 2).

**Effect of a Shift From Static to Pulsatile Pressure at Various Mean Pressures on Single Unit Activity.**

Figure 3 shows the effect of a shift from SP to PP and back to SP on the neurogram obtained from a single unit at five levels of pressure (Conditions I–V). Figure 4 contrasts the pressure-activity relation in a representative unit during SP and PP (left panel). Activity increased abruptly and markedly from zero when SP reached SPTH. In contrast, during PP, activity was initiated at very low pressure; the activity was modest and increased linearly with pressure. These patterns of responses are also evident in Figure 2. The averaged group data are shown in the bar graphs (Figure 4, right panel) for the five pressure conditions rather than for the various mean pressures because the pressure threshold and consequently the pressure range for each condition varied markedly between units (see Table 1).

The individual data from 26 units are shown in Table 1. The effect of a shift from SP to PP on unit activity was dependent on the mean level of CSP.

a) **Mean PP below SPTH (Conditions I and II).** Pulsatility increased activity from 0 to 10.5 ± 1.5 spikes/sec when systolic pressure was below SPTH and from 0 to 18.5 ± 1.4 spikes/sec when systolic pressure was above SPTH (Figures 1, 3, 4).

b) **Mean PP above SPTH and diastolic pressure below...**
Si Table 1. Single Unit Baroreceptor Activity (spikes/sec) From Individual Experiments During Static and Pulsatile Pressure

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Mean ± SEM 67.1 ± 6.1 10.5* ± 1.5 18.5* ± 1.4 31.4 ± 3.4 26.9* ± 2.2 42.0 ± 2.1 35.5* ± 1.9 55.7 ± 3.2 55.1 ± 3.2

Entries are nerve activities as spikes/sec.

Roman numerals I to V refer to pressure conditions under which the activity was recorded: I, systolic pressure below SPth; II, mean pressure<SPth; III, mean pressure>SPth, diastolic pressure<SPth; IV, diastolic pressure>SPth; V, saturation pressure. All five conditions were not met in all the fibers because the mean pressure was increased in steps of 25 mm Hg from 25 to 200 mm Hg. SP, static pressure; PP, pulsatile pressure; SPth, static pressure threshold.

*S significantly different from SP, p<0.05.

SPth (Condition III). Pulsatility decreased activity from 31.4 ± 3.4 to 26.9 ± 2.2 spikes/sec (Figures 1, 3, 4).

c) Diastolic pressure above SPth (Condition IV). Pulsatility caused diastolic silence and decreased activity from 42.0 ± 2.1 to 35.5 ± 1.9 spikes/sec (Figures 1, 3, 4).

d) Saturation pressure (Condition V). Pulsatility did not cause silence nor did it decrease activity. Activity averaged 55.7 ± 3.2 spikes/sec during SP and 55.1 ± 3.2 spikes/sec during PP (Figures 1, 3, 4).

The PP-induced decrease in activity occurred when diastolic silence was present. Figure 5 illustrates the pressure dependence of diastolic silence. Silence occurred in all units tested (n = 25) when diastolic pressure was 1 to 10 mm Hg above SPth. Silence was maintained in 80% of the units when diastolic pressure was 20-30 mm Hg above SPth, in 64% of the units when diastolic pressure was 30-40 mm Hg above SPth, and in 40% of the units when diastolic pressure was 40-50 mm Hg above SPth. Thus, silence occurred at pressures far above SPth and was responsible for the decrease in activity per unit time that occurred when SP was converted to PP.

Effect of a Shift From Static to Pulsatile Pressure at Various Mean Pressures on Whole Nerve Activity

Figure 6 shows the effect of a shift from SP to PP and back to SP on multiple unit activity at several levels of pressure (Conditions A, B, and C). Figure 7 contrasts the pressure-activity relation in a representative experiment during SP and PP (left panel). A sigmooidal
relation is noted during SP. In contrast, a linear relation up to saturation pressure is observed during PP. The averaged group data are shown in the bar graphs (Figure 7, right panel) for the three pressure conditions.

The individual data from 9 preparations are shown in Table 2. The effect of a shift from SP to PP on multiple unit activity was dependent on the mean level of CSP (Table 2, Figures 6 and 7).

a) At low mean pressures (50–75 mm Hg, Condition A) pulsatility increased activity from $57.2 \pm 15.4$ to $142.2 \pm 28.9$ spikes/sec (Figures 6 and 7), presumably by initiating activity below $P_{th}$ in individual units and by recruiting additional units.

b) At moderate pressures (125–150 mm Hg, Condition B) pulsatility decreased activity from $373.0 \pm 69.3$ to $319.6 \pm 54.7$ spikes/sec (Figures 6 and 7), suggesting that the decrease in single unit activity was greater than the predicted increase in activity due to recruitment.

c) At saturation pressure (200 mm Hg, Condition C) pulsatility did not alter activity which averaged $459.3 \pm 79.8$ spikes/sec during SP and $444.8 \pm 77.2$ spikes/sec during PP (Figures 6 and 7).

**Contrasting Effects of Increases in Static and Pulsatile Pressure on the Baroreceptor Activity Curve**

The pressure-activity relation was markedly different during PP compared to during SP. Static pressure
caused a sigmoid activity curve with the maximal increase occurring around 75–100 mm Hg (Figure 8). In contrast, the increase in PP caused a remarkably linear increase in activity (i.e., constant gain) between 25 and 150 mm Hg and a decline in gain which paralleled that seen with SP near saturation (Figure 8). In single units as well as whole nerve recordings, maximal baroreceptor gain was significantly greater during SP averaging twice that seen during PP: 1.25 ± 0.07 versus 0.59 ± 0.04 spikes/sec/mm Hg in single units (p < 0.05) and 7.04 ± 1.46 versus 3.38 ± 0.53 spikes/sec/mm Hg in multiple units (p < 0.05). The values shown in Figure 8 represent the average gain at the various pressure levels. Since the maximal gain for each nerve preparation did not occur at the same pressure level, the peak gains shown in Figure 8 are lower than the average maximal gain.

**Effect of Hysteresis on Static and Pulsatile Pressure-Activity Curves**

In 12 single units, activity was recorded during step increases and step decreases in SP and PP over a range of pressures from 200 to 40 mm Hg. Figure 9 shows that during the step down phases the activity was somewhat lower than during the step up phases with both SP and PP. However, the characteristic shapes of the curves were retained as well as their relative sensitivities.

**Discussion**

In this study, we characterized the activity of single and multiple units from carotid baroreceptors during static pressure and sinewave pulses at various levels of mean pressure. The following conclusions were reached.

Sinewave pulsation triggers activity of baroreceptors at levels of pressure ranging from 13–61 mm Hg (average of 31 mm Hg) below the pressure threshold determined from a slow nonpulsatile pressure ramp. Sinewave pulsation can cause a decrease in baroreceptor activity of both single units and multiple units even when diastolic pressure is significantly above the pressure threshold. Diastolic silence occurs at levels of diastolic pressure 10–80 mm Hg above the static.


Table 2. Multiple Unit Baroreceptor Activity (spikes/sec)
From Individual Experiments During Static and Pulsatile Pressure

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Entries are nerve activities as spikes/sec.
The letters A, B, and C refer to pressure conditions under which the activity was recorded. A, low carotid sinus pressure (50 or 75 mm Hg); B, moderate sinus pressure (125 or 150 mm Hg); C, high sinus pressure (200 mm Hg). SP, static pressure; PP, pulsatile pressure.

*Significantly different from SP, p < 0.05.

pressure threshold. The decrease in activity per unit time seen in multiple unit recordings when static pressure is shifted to pulsatile pressure at diastolic pressures far above pressure threshold suggests that diastolic silence has a greater effect on activity than the systolic recruitment of fibers or the increase in systolic activity. During static pressure the pressure-activity curve is sigmoid and the gain peaks at 75–100 mm Hg. In contrast, during pulsatile pressure the activity curve is linear over a range from 25–150 mm Hg rather than sigmoid, and the gain is relatively constant from threshold pressure to near saturation pressure. The peak sensitivity during PP is half the peak sensitivity during SP. This contrast between activity with SP and PP is seen also during the hysteresis curve obtained with decreasing steps of SP and PP.

In the "Discussion," we will cover two points. First, we shall compare our findings with information in the literature on dynamic versus static responses. Second, we will attempt to discuss some of the possible explanations for our observations and their physiologic implications.

Initiation of Baroreceptor Activity Below Static Pressure Threshold

The reports which suggest that pulsatility might increase activity at systolic pressures much lower than SPth were done using pressure ramps or step changes in pressure rather than sinewave or natural pulses. In fact, the one study that quantitatively examined the effect of pulsatility below SPth was that of Angell-James in 1971 who showed that systolic pressure was generally within 2 or 3 mm Hg and had to be within 10 mm Hg of SPth for aortic baroreceptor activity to occur. There are no previous studies on single unit carotid baroreceptors which contrast systematically static pressure and sinewave pulsatile pressure. The results from pressure ramps, even when dP/dt is similar to sinewaves or natural pulses, do not necessarily allow prediction of the magnitude of the response to pulsatile pressure because of alternating positive and negative dP/dt. Our results show that, indeed, PPh is much less than SPth during pulsations at physiologic frequencies and dP/dt. The difference

![Figure 8. Baroreceptor gain during static and pulsatile pressure. The results from single units (left, n = 26) and multiple units (right, n = 9) are shown. The average gain peaked during static pressures between 75 and 100 mm Hg. A relatively constant gain was maintained over a very wide pressure range (25 to 150 mm Hg) during pulsatile pressure. Error bars represent ± SEM. The asterisks refer to a significant (p < 0.05) difference from the gain obtained at the lowest level of mean pressure. Since the maximal gain for each nerve preparation did not occur at the same pressure range from 75 to 100 mm Hg, the peak gains shown in this figure are lower than the average maximal gain. During static pressure the maximal gain averaged 1.25 ± 0.07 and 7.04 ± 1.46 spikes/sec/mm Hg in single and multiple units, respectively. Corresponding values with pulsatile pressure were 0.59 ± 0.04 and 3.38 ± 0.53 spikes/sec/mm Hg.](http://circres.ahajournals.org/).
Diastolic Silence When Diastolic Pressure is Above Threshold

Although diastolic silence has been observed, its relationship to diastolic pressure was not quantitated systematically. In 1935, Bronk and Stella first noted that during natural arterial pulses single unit activity often occurs only during systole even when diastolic pressure is at a level sufficient to evoke activity if maintained continuously. Their report, however, was descriptive and there was no indication of the consistency of the finding or its relation to the level of diastolic or threshold pressures. Angell-James, utilizing an isolated rabbit aortic arch preparation and sinewave pulses, observed that diastolic silence above threshold occurs inconsistently. Her data indicate that silence is not present in the majority of fibers when diastolic pressure is above threshold even at relatively high pulse frequency (160 and 210 pulses/min). In 1978, Brown et al noted that “diastolic silence” of rat aortic baroreceptors occurs when diastolic pressure is above threshold during high frequency sinewave stimulation and that the silence is maintained when mean pressure is increased further by 5–15 mm Hg, but no data were presented to assess the consistency of the observation or its relation to the level of diastolic pressure. In 1975, Arndt et al, using dynamic stretch of the aortic arch, concluded that aortic baroreceptors fire continuously as soon as the stretch exceeds threshold during all phases of the cycle.

Thus, although the prior studies described the phenomenon of diastolic silence, it was not observed consistently above the threshold pressure, it was not seen if diastolic stretch exceeded threshold, it was seen only at high frequency of sinewave pulses, and it was not correlated with the magnitude of diastolic pressure. In our study, diastolic silence was seen in carotid baroreceptors above static threshold in all units. Eighty percent of the units showed silence at diastolic pressures 20–30 mm Hg above threshold, 64% showed silence at diastolic pressures 30–40 mm Hg above threshold, and 40% showed silence at diastolic pressures greater than 40 mm Hg above threshold (Figure 5).

Decreased Activity Per Unit Time When Diastolic Pressure is Above Pressure Threshold

Single units. Decreased activity per unit time in Condition III, when diastolic pressure drops below pressure threshold, was described by Angell-James and is not at all surprising. What is new is the decrease in activity per unit time that we observed during the shift from SP to PP, when diastolic pressure is far above pressure threshold (Condition IV). This decrease in activity was always caused by diastolic silence. To our knowledge, this had not been reported previously. Ead et al reported that in 30 preparations of a few or single fibers, a shift from static to pulsatile pressure did not cause a consistent or significant change in activity. Angell-James showed that in 15 single units, a shift from static to pulsatile pressure did not change aortic baroreceptor activity. Arndt et al also described a lack
of a decrease in activity in 3 units with a shift from static to pulsatile pressure, however, at saturation pressures, they described a decline in activity in 2 units. Arndt ascribed this to the fact that the systolic pressure near saturation point does not cause an increase in systolic activity whereas the drop in diastolic pressure will tend to decrease activity resulting in a net decrease in activity of these two units. Brown et al reported that very high pulse rates slightly increased the discharge of nonmyelinated fibers.

On examination of the work of Sleight et al who recorded carotid baroreceptor activity in single units in normotensive and hypertensive dogs during both static pressure and pulsatile pressure, one can calculate from their data that baroreceptor activity was less during pulsatile pressure (21 spikes/sec) that during static pressure (27 spikes/sec). Sleight et al found that the baroreceptor sensitivity was not different in the two groups of animals when static pressure was utilized whereas the sensitivity was less in the hypertensive dogs when pulsatile pressure was utilized. The hypertensive dogs also demonstrated diastolic silence at much higher mean pressure than did normotensive dogs. Their results, along with our findings, suggest that diastolic silence is an important determinant of overall baroreceptor activity and the decreased baroreceptor sensitivity in hypertension may be evident only during pulsatile pressure and not during static pressure.

Whole Nerve Recordings. In agreement with other studies, we found that a shift from static to pulsatile pressure increases sinus nerve activity at low mean pressures and does not alter the activity at very high pressures near saturation. On the other hand, our demonstration that pulsatility decreases baroreceptor activity at moderate pressures has not been described previously. We attribute this effect to diastolic silence in individual units. The PP-induced decrease in nerve activity was usually seen only at pressure levels above 100 mm Hg, probably because at lower pressures the increased activity and "recruitment" of units during systole tended to offset the effect of diastolic silence. Over a moderate range of mean pressure, baroreceptor activity was less during pulsatile pressure than during static pressure in 8 of 9 preparations (Table 2).

Bidirectional Rate Sensitivity During the Pressure Pulse

Our results clearly show that baroreceptors are bidirectionally rate sensitive and refute the concept of Clynes that the receptors are only unidirectionally rate sensitive. Clynes concluded that carotid sinus baroreceptors are only sensitive to the rate of change of pressure when the pressure pulse is increasing and not when it is decreasing. For example, he notes that there is no abrupt diminution in firing rate as blood pressure falls during diastole. Our observation of diastolic silence above threshold and the results of others are inconsistent with his conclusion. Houk and Franz have also concluded that slowly adapting mechanoreceptors including baroreceptors are bidirectionally and not unidirectionally rate sensitive.

It has been stated that baroreceptors are more sensitive to increases in pressure than to decreases in pressure. Our results suggest that this is not always the case. At pressures below threshold the receptors are indeed more sensitive to an increase in pressure (Condition I) but at pressures above threshold the receptors become more sensitive to a decrease in pressure (Condition IV). This is evident when considering that pulsatility decreases diastolic activity more than it increases systolic activity causing a decreased baroreceptor activity per unit time. At saturation pressure (Condition V), the sensitivity to increases and decreases in pressure are equivalent. Our findings are consistent with those of Franz who concludes that the sensitivity to increases and decreases in pressure need not be equivalent.

Baroreceptor Sensitivity During Overall Increases and Decreases in Static and Pulsatile Pressures

The current notion is that the pressure-baroreceptor activity relation is sigmoid. Our results confirm the sigmoid relationship during increases in static pressure but demonstrate an essentially linear relationship during increases in pulsatile pressure from 25 to 150 mm Hg. During PP the increased sensitivity at lower pressures and decreased sensitivity at higher pressures account for the linearity.

Further, during PP, the gain of the response or the maximal sensitivity or slope of the pressure activity curve of baroreceptors is half the maximal sensitivity or gain during SP. At pressures near saturation, baroreceptors are equally sensitive to PP and SP, and the slope flattens (i.e., the gain declines rapidly).

The differences in sensitivity of baroreceptors to changes in PP and SP were not altered significantly by hysteresis. During the step-decreases in pressure from 200 to 40 mm Hg, both PP-activity and SP-activity were shifted to the right compared to the activity during step-increases in pressure, revealing some hysteresis (Figure 9, Panels A and B). The SP curve retained its sigmoid character with an even steeper decline in activity over the range from 175 to 100 mm Hg. The PP curve remained essentially linear over the pressure range of 160 to 40 mm Hg (Figure 9).

The linearity of the PP-activity curve is surprising in view of the known sigmoidal baroreflex response curve when systemic pressure is increased with phenylephrine, for example, and heart rate or sympathetic activity are reflexly decreased. A possible explanation might be that in "closed-loop experiments" changes in heart rate or pulse pressure which accompany changes in arterial pressure may modify the magnitude of afferent activity. Another explanation may be a nonlinearity of the central mediation of the reflex.

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