High-Salt Diet Elevates Baroreceptor Pressure Thresholds in Normal and Dahl Rats

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Dahl Salt Sensitive (DS) rats rapidly develop high blood pressure when exposed to a high-salt diet. Recent studies suggest that DS rats have poorly functioning baroreceptor afferents and baroreflexes even when salt intake is restricted. This study examines baroreceptor pressure- and mechano-transduction in DS, Dahl Resistant (DR), and Sprague-Dawley (SD) rats during low-and high-salt conditions. Single unit, regularly discharging baroreceptors were studied using an in vitro aortic arch-aortic nerve preparation. Pressure thresholds and suprathreshold pressure sensitivities were determined from responses to slow ramps of pressure. Pressure-diameter relations measured in each rat were used to transform pressure threshold and pressure sensitivity values to their mechanical equivalents in terms of aortic wall strain. A total of 407 unit baroreceptors were studied from 49 rats. Tail systolic blood pressures were significantly higher only in DS during high salt. Pressure threshold was similar for all groups on low salt. Exposure to a high-salt diet increased the mean pressure threshold for all three groups. Pressure threshold for high-salt diet was highest in DS and lowest in DR. Pressure sensitivities were lowest in DS and highest in DR on low salt. High salt had no significant effect on pressure sensitivity. The differences in threshold apparent when expressed in terms of pressure were eliminated by conversion to their mechanical equivalents (strain threshold and strain sensitivity). The results suggest that baroreceptors in the two Dahl rat strains represent two extremes from normal baroreceptor function. DS tend to be less pressure responsive than normal (SD), and DR tend to be somewhat more responsive to pressure. High salt altered baroreceptor properties in all three rat strains. The elevation of pressure threshold in DR and SD occurred without increases in systolic blood pressure suggesting that high dietary salt can alter baroreceptor function independent of blood pressure effects. The mechanism of this effect appears to be related to the local mechanical properties of the vessel wall or the way in which the receptor is coupled to the vessel wall. (Circulation Research 1989;64:695–702)

Epidemiological and laboratory studies suggest that an elevation in dietary salt may increase the risk of developing hypertension. Little is known about the precise mechanisms by which increased salt intake might produce elevated blood pressures. One animal model of salt-dependent hypertension was developed by Dahl et al. The two strains of Dahl rats were selected for inbreeding on the basis of the change in their blood pressure in response to a diet high in salt (generally 4–8% NaCl). The Dahl Salt Resistant (DR) rats remained normotensive on high-salt diet, while the Dahl Salt Sensitive (DS) rats rapidly became hypertensive. Both DS and DR were normotensive on a low-salt diet. Several genetic loci are believed to be involved, and mechanisms involving the kidney, adrenal cortex, nervous system, and unidentified humoral factors have been implicated in the salt-dependent hypertension of DS.3

The baroreflex control of blood pressure is abnormal in DS.4,5 The major efferent mechanisms of baroreflexes are very similar in DS and DR on low salt. Recordings of whole aortic nerve activity suggest that the function of arterial baroreceptor afferents is impaired in prehypertensive DS on a low-salt diet, and this afferent defect may account for the low sensitivity of baroreflex control mechanisms in DS during low-salt treatment.6 Similar experiments in DR exposed to high salt suggest that DR aortic baroreceptors become "sensitized" or show enhanced pressure-discharge characteristics during elevation of dietary salt.7 In Sprague-Dawley rats, genetically unselected for blood pressure responses to dietary salt, multifiber discharge from aortic nerves decreased during high salt.8 The mechanism or cellular basis for the differences in baroreceptor responses in these two Dahl...
strains on low salt and the effects of high salt on baroreceptor function are not known.

To determine which baroreceptor properties are altered in Dahl rats and which properties are affected by excess dietary salt, the discharge characteristics of single-fiber baroreceptors from the aortic arch were determined using an in vitro preparation. Baroreceptors were sampled for quantitative, direct comparison of three different rat types including both Dahl strains and the Sprague-Dawley (SD) strain. Groups of each rat strain were raised from weaning on either low-salt or high-salt diet. The mechanical properties of each aortic arch in the region of baroreceptor innervation were determined so that discharge properties could be expressed in terms of vessel distortion, the direct stimulus for these mechanoreceptors. The results suggest that the two Dahl strains represent opposite extremes of the normal range of baroreceptor function and that high dietary salt can significantly alter baroreceptor function even in normal animals genetically unselected for salt sensitivity of blood pressure. The local vessel wall plays an important role in the differences in baroreceptor responses between these rat strains.

**Materials and Methods**

Male SD, DS, and DR rats were obtained at weaning (3–4 weeks of age) from Harlan Sprague Dawley, Indianapolis, Indiana, and Brookhaven National Laboratories, Upton, New York, respectively, and placed on either a low-salt (0.15% NaCl) or a high-salt (8.0% NaCl) chow diet (ICN Nutritional Biochemicals) and distilled water ad libitum. The birthdate of each rat was supplied by the vendor and used to calculate rat age on the day of the experiment. The rats were housed in the same room and maintained on a fixed light-dark cycle (12:12). Tail systolic blood pressures were measured by the indirect method9 (IUTC, Inc.).

Baroreceptor experiments were performed on rats between 8 and 12 weeks of age. To maintain this limited age range, it was necessary to conduct experiments in two separate series of approximately equal numbers of rats. In total, experiments in 10 rats in each of the six experimental groups were completed: three groups of rats on low-salt diets and three groups of rats on high-salt diets. Data for a given rat was excluded from subsequent analysis for cases in which fewer than two single-fiber baroreceptors were successfully isolated and tested. Each 4-week series of experiments included five rats for each experimental rat group and were delivered in two separate shipments from the above vendors.

The properties of single aortic baroreceptors and the vessel wall mechanical properties of each aortic arch associated with those baroreceptors were studied using an in vitro aortic arch-aortic nerve preparation. All experiments were conducted in conformity with the "Guiding Principles for Research Involving Animal and Human Beings" of the American Physiological Society. Methods for testing baroreceptors have been described in detail previously.9 Briefly, under pentobarbital anesthesia (30–50 mg/kg), the aortic arch and aortic nerve were exposed. Metal cannulas were placed in the innominate artery and the descending aorta, and ligatures were placed on the ascending aorta, left common carotid, and left subclavian arteries. The aortic arch and nerve were then removed and transferred to a temperature-regulated perfusion bath, where the vessel was fixed to approximate its in situ length and shape. The lumen of the aortic arch was perfused with Krebs-Henseleit solution equilibrated with 95% O2-5% CO2 gas mixture, and the preparation was covered with warm mineral oil.10

**Measurement of Baroreceptor Discharge Characteristics**

The baroreceptor pressure threshold, the minimum pressure at which the receptor discharges, depends on the most recent level of the conditioning mean arterial pressure to which it is exposed.11 After mounting in the perfusion apparatus, all aortic arch preparations were perfused at a fixed control conditioning mean arterial pressure of 80 mm Hg for at least 1 hour before isolation of the first single-fiber baroreceptor. Only regularly discharging receptors were tested, and these were presumed to be connected to myelinated axons.12 When a single active baroreceptor was isolated, perfusion was halted and mean arterial pressure was reduced to 20 mm Hg. After 30 seconds at 20 mm Hg, pressure was increased in a slow ramp using a shaker driver-bellows system (Ling Dynamic Systems 411). The ramp rate never exceeded 2 mm Hg/sec and generally included the range of pressures from 20 to 220 mm Hg. Baroreceptor responses measured with these slow ramp inputs are very similar to the responses measured after 30 seconds of adaptation to various levels of step inputs.10 After completion of tests for a given baroreceptor, fiber splitting continued until isolation of another unitary baroreceptor. This process of splitting and testing was continued until the nerve trunk became too short to record responses to the entire pressure ramp without collision of the expanding aortic wall with the recording electrodes.

Experiments were recorded on analog FM magnetic tape and later played back for digitization by a microcomputer (PDP 11/23, MDB, Inc, Orange, California). Action potentials were 1) detected directly with a simple Schmitt-trigger voltage level detector for unit baroreceptor recordings or 2) sorted from small multifiber recordings (less than four unit baroreceptors) using a pair of cascaded, time-voltage amplitude window discriminators (DIS 1, BAK, Inc, Rockville, Maryland) to detect (sort) unitary action potentials. Discharge rate was calculated as the reciprocal of the interspike interval, that is, the instantaneous frequency. From the ramp responses, a pressure-discharge relation was constructed for each baroreceptor. Typically, these relations have a
distinct minimum pressure at which discharge begins and a suprathereshold region in which increases in discharge are quite linearly related to increases in pressure in the range of 50–60 mm Hg above threshold or up to frequencies equal to one half the difference between the threshold and maximal discharge rates. In these regularly discharging baroreceptors, discharge at threshold typically begins by a jump from zero spikes to a rate of between 15 and 30 spikes/sec as pressure exceeds the threshold level. Thus, the pressure values for discharge from the first 10 action potentials were averaged to represent the pressure threshold. The range of pressures for this averaging was generally less than 1 mm Hg, but use of this procedure avoids basing this critical measure on the location of a single action potential (the first one). The average discharge frequency of these first 10 action potentials at threshold was also calculated for comparison. The slope of the linear suprathereshold region was used as an index of receptor gain or sensitivity to pressure. Pressure threshold and pressure sensitivity threshold were used as the basic parameters for comparison of baroreceptors.

For each experiment, the pressure-discharge curves of all unit baroreceptors were compared for possible duplicate tests of the same baroreceptor. Superimposable matches of the pressure-discharge curves between two baroreceptors recorded at different times during the experiment were considered duplicate tests of the same baroreceptor. Identical baroreceptor curves were found for about 15% of all fibers split. Only one baroreceptor from these replicate fibers was retained for subsequent analysis. This procedure ensured that the analysis would not be biased by inclusion of multiple recordings of the same unit baroreceptor repeatedly isolated at several points along the length of the aortic nerve.

**Vessel Wall Mechanics**

At the end of each experiment, the pressure-diameter relation of the aortic arch preparation was measured between the left common carotid and left subclavian arteries, the region of baroreceptor innervation. The ocular micrometer method of diameter measurement has been previously described. This method has a resolution of 1% for diameter measurements. In some experiments of the second series of replicates, a custom-made, high-resolution (0.01% or 1 μm) photoelectronic caliper (Iwazumi, Calgary, Alberta, Canada) was also used to measure vessel diameter. Results were equivalent with either method. Briefly, the diameter was measured 30 seconds after a step change in pressure. Pressure was randomly varied from 0 to 200 mm Hg at intervals of 20 mm Hg. The length of the aortic segment at the inside border of the ligatures was measured. Finally, the aortic segment was trimmed to include only the vessel region between the ligatures, and the trimmed aortic arch was patted dry and weighed. Thus, mechanical data were obtained directly from each aorta for which baroreceptor pressure characteristics had been measured.

Several vessel wall parameters were calculated from the aortic diameter, length, and segment weight measurements to quantify the static vessel wall mechanics. Based on an assumption of constant vessel wall volume, the inner radius of the vessel wall (Ri) was calculated from the following expression:

\[ Ri = \frac{Re^2 - (w/d \cdot \pi \cdot L)}{12} \]

where Re is the measured external aortic radius, w is the vessel segment weight, d is the tissue density of 1.06, and L is the length of the vessel segment. The wall thickness, h, could then be calculated as the difference between Re and Ri. Circumferential wall strain (ε) is considered to be a good index of vessel mechanical properties and appears to be linearly related to baroreceptor discharge rate. ε was used to quantify vessel distorsion and calculated as

\[ \epsilon = \frac{(R_p - Ro)}{Ro} \]

where Rp is the midwall radius at a given pressure and Ro is the unstressed, usually minimum midwall radius. In addition, to express distensibility, the incremental elastic (Einc) modulus was calculated as

\[ E_{inc} = \frac{\Delta P}{\Delta Re} \left(1 - \theta^2\right) Re \cdot R_i^2/(Re^2 - R_i^2) \]

where θ, the Poisson ratio, was assumed to be 0.5. Three measures were calculated at a common pressure, 100 mm Hg, to facilitate comparison of the average vessel wall properties as used previously. These were ε100, h100, and Einc100; the wall strain, wall thickness, and incremental elastic modulus, respectively, at 100 mm Hg.

The mechanical equivalents of baroreceptor pressure threshold and sensitivity were determined in units of wall strain by linear interpolation of the vessel wall measurements (which were made each 20 mm Hg). The error in the equivalent baroreceptor strain parameters introduced by interpolation was generally less than 1% in experiments where both stepwise measurements and continuous measurements (in response to slow pressure ramps) were compared using the electronic caliper. For pressure threshold, the procedure was a direct interpolation. For suprathereshold pressure sensitivity, the value was calculated beginning at the wall strain equivalent to the pressure threshold. This is the functional setpoint for that particular baroreceptor and the point in the pressure-strain relation at which action potential encoding begins.

**Statistical Treatments**

Two types of measurements were made in this study: 1) rat parameters, those related to each rat and thus shared in common by all baroreceptors from that rat, and 2) baroreceptor parameters, those unique to each individual baroreceptor. Descriptive statistics were calculated for rat measures (tail systolic blood pressure, age, weight, ε100, h100, and Einc100) and for the five baroreceptor properties
(pressure threshold, pressure sensitivity, threshold frequency, strain threshold, and suprathreshold strain sensitivity). Analysis of variance (ANOVA) was performed and, where significant overall treatment effects were identified, comparisons were made with Scheffe's procedure. With the exception of blood pressure, no significant two-way interactions were detected between rat type and diet for any rat or baroreceptor measures. No differences in within-rat or rat-to-rat variances for baroreceptor properties were found. Reported probability values are truncated at $p=0.0001$. Values of $p<0.05$ are considered significant.

**Results**

Baroreceptor experiments were successful in a total of 49 rats in the six experimental groups (Table 1). Tail systolic blood pressure (Figure 1) was similar in all groups on low-salt diet ($p=0.072$), but the blood pressure response to high-salt diet was clearly different across rat types (a significant two-way interaction, $p<0.0001$). Systolic blood pressure of DS on high-salt diet increased to clearly hypertensive levels ($p=0.0001$), but the same high-salt diet slightly lowered SD blood pressure ($p=0.012$). Body weights of SD were lower than DR and DS on both low-salt and high-salt diets ($p<0.03$). Animals on high-salt diets had lower body weights than their counterparts on low-salt diets for all three rat types ($p>0.03$). No other differences were detected in age or measures of mechanical properties of the vessel wall (Table 1). Thus, high-salt diet was associated with an increase in systolic blood pressure only in the DS rat, and blood pressure decreased somewhat in SD and nonsignificantly in DR. No effects of high salt or the hypertensive phenotype of DS on high-salt diet were apparent in global measures of vessel wall function (Table 1).

**Baroreceptor Pressure Properties**

A total of 407 aortic arch baroreceptors were successfully recorded in the six groups of rats (three groups on low-salt diet: DR, 90; SD, 53; DS, 69; and three groups on high-salt diet: DR, 75; SD, 60; DS, 60). Pressure thresholds were similar across all groups on low-salt diet based on the value of $p=0.05$ (Figure 2). High-salt treatment substantially increased pressure threshold in all groups ($p=0.001$). The magnitude of this increase in pressure threshold during high-salt treatment was not different among the three rat strains ($p=0.179$). The rank order of mean pressure threshold for the three rat strains was the same on both low-salt and high-salt diets. The DR group had the lowest mean Pth of the three rat strains on both diets, and the DS group had the highest Pths of the three strains on both diets.

Marked differences in mean suprathreshold pressure sensitivity were found across the different rat strains ($p<0.0001$), but these differences were unaltered by diet ($p=0.32$). On low-salt diet, DS baroreceptors had significantly lower pressure sensitivity values ($p=0.012$) than DR baroreceptors (Figure 3).

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Baroreceptor pressure sensitivities in DS remained lower than in DR on high-salt diet ($p=0.032$). Note that the rank order of the mean pressure sensitivity values placed DS baroreceptors as the least sensitive of the three rat strains and DR baroreceptors as the most sensitive. SD baroreceptors were intermediate in pressure sensitivity, and this relative rank order did not change during high salt treatment or significantly change the average absolute level of baroreceptor gain (pressure sensitivity) within each rat strain. In addition, the frequency of barorecep-
FIGURE 1. Tail systolic blood pressures in Dahl Salt Sensitive (DS), Dahl Salt Resistant (DR), and Sprague-Dawley (SD) rats. Bars represent the means of each group with plus one standard deviation indicated by the narrow bracket. There were no differences in blood pressure among the low-salt groups. High salt increased blood pressure substantially in DS and decreased blood pressure slightly in SD.

FIGURE 2. Baroreceptor threshold pressure values were similar in the low-salt groups. High salt significantly increased pressure threshold in all groups, and Dahl Salt Sensitive rats (DS) on high salt values were greater than Dahl Salt Resistant rats (DR) on high salt. Bars represent the means of each group with plus one standard error of the mean indicated by the narrow bracket. SD, Sprague-Dawley rats.

FIGURE 3. Baroreceptor suprathreshold pressure sensitivities were significantly lower in Dahl Salt Sensitive (DS) than in Dahl Salt Resistant (DR) rats. High salt did not alter suprathreshold pressure sensitivity significantly. Pressure sensitivity was lower in DS than in DR on both diets. Bars represent the means of each group with plus one standard error of the mean indicated by the narrow bracket. SD, Sprague-Dawley rats.

FIGURE 4. Baroreceptor threshold frequencies were similar across all groups on low-salt and high-salt diets. Bars represent the means of each group with plus one standard error of the mean indicated by the narrow bracket. DS, Dahl Salt Sensitive rats; SD, Sprague-Dawley rats; DR, Dahl Salt Resistant rats.

Baroreceptor discharge at threshold was not different across rat types \( (p=0.065) \) or salt treatments \( (p=0.258) \). Thus, in terms of pressure properties, DR baroreceptors had the lowest mean pressure thresholds and the highest mean pressure sensitivities throughout these experiments, suggesting a greater pressure responsiveness than the other rat strains. DS baroreceptors had the highest mean pressure threshold and the lowest mean pressure sensitivities, suggesting a generally lower pressure responsiveness. In all cases, the SD rats were situated between these two extremes with what might be termed moderate pressure responsiveness. The largest changes in pressure threshold with high-salt treatment occurred in DS and the smallest in DR. Note that blood pressure did not change or was somewhat lower in DR and SD and, thus, the high-salt treatment effects in these two strains cannot be attributed to blood pressure changes. The studies of these six groups of rats were run in two separate halves with different shipments of approximately equal numbers of animals (see "Materials and Methods"). All of the basic statistical findings (rankings of means and significance) were similar whether each half of the study was analyzed separately or together.

Baroreceptor Distortion-Sensing Properties

When threshold was expressed as the circumferential wall strain at the pressure threshold and means calculated, there were no differences in strain threshold between DR and DS on low-salt diet (Figure 5). High-salt treatment did not alter strain threshold in the Dahl rats \( (p=0.07) \). SD tended to have a somewhat higher strain threshold than DR on both diets \( (p<0.045) \). The suprathreshold strain sensitivity was not different across rat types \( (p=0.346, \text{ Figure 6}) \) and was not altered by high-salt treatment \( (p=0.614) \). Thus, expression of baroreceptor discharge properties in terms of circumferential wall strain eliminated the differences between DS and DR baroreceptors that were so apparent in terms of pressure.
Baroreceptor Properties During Low- and High-Salt Diets

Baroreceptor pressure thresholds were similar in DS, DR, and SD rats on the low-salt diet. There was a tendency for baroreceptors from DS on low salt to have somewhat higher pressure thresholds than baroreceptors from DR on low salt, but this difference ($p<0.05$) was just below the general significance level ($p<0.05$). The absolute magnitude of the difference in pressure thresholds between DR and DS on low salt was less than 5 mm Hg. This small difference on low salt plus the marginal probability value leads me to conclude that DR and DS baroreceptor pressure thresholds are similar on low salt and unlikely to be functionally significant at a population level. Suprathreshold pressure sensitivities of DS on low-salt diet were substantially lower than those of DR on low-salt diet. Thus, these results suggest that lower pressure sensitivities are probably responsible for the lower slopes of pressure-discharge relations for whole aortic nerve activity found in vivo in DS on low-salt diet compared with DR on low-salt diet.6-23 Differences in baroreceptor recruitment patterns which depend on the distribution of pressure threshold values probably make only a minor contribution to the differences in whole nerve activity under these conditions. The lack of significant differences in systolic blood pressure in these young rats on low-salt diet suggests that the baroreceptor differences are not secondary to chronic resetting.9

Conflicting results have been reported on the effects of high-salt diet on whole aortic nerve discharge in Dahl rats. Aortic baroreceptors and the baroreflexes arising from both arterial and cardiopulmonary mechanoreceptors are reported to undergo a "sensitization" in female DR during high-salt treatment.7,27 Mark and coworkers found that relative to low-salt conditions, pressure-discharge relations for whole aortic nerves had greater slopes during high-salt treatment. These investigators also found that the slopes for high-salt treated DS, aortic nerves were nonsignificantly decreased compared with DS on low salt. Among several mechanisms discussed was the possibility that a humoral factor might be responsible for the
“sensitization” of DR cardiovascular afferents during high-salt treatment. A role for unidentified circulating factors has previously been suggested for renal abnormalities in the Dahl model. Contrary results of the effects of high salt on DR baroreceptors were reported by Miyajima and Buñag. Using similar in vivo techniques to measure activity in whole aortic nerves, except in male rats, Miyajima and Buñag reported decreased slopes for the phenylephrine portions of their response curves during high-salt treatment for both DR and DS compared with low-salt treatment. The source of the difference in the observations of these two studies is unclear, but it seems unlikely that the gender of the rats is involved. In a separate, earlier study in SD rats, Miyajima and Buñag found that a similar high-salt diet also reduced discharge slopes in whole aortic nerve.

In the present study, addition of excess salt to the diets of the three rat strains resulted uniformly in a selective increase in baroreceptor pressure threshold with no change in suprathreshold pressure sensitivity. Pressure threshold increased significantly in all high-salt groups over their low-salt values. Blood pressure was elevated by high salt only in DS and actually declined slightly in SD. Thus, the increases in pressure threshold observed in both DR and SD on high-salt diets cannot be attributed to chronic hypertensive baroreceptor resetting. Chronic baroreceptor resetting in response to hypertension generally results in an increase in pressure threshold and a decrease in pressure sensitivity. Blood pressure was substantially elevated in DS on high-salt diets at the time of the experiments and had been hypertensive for 4 weeks in most animals (author’s unpublished results). At least four observations suggest that the major portion of the changes in DS baroreceptor function during elevated dietary salt is most likely the result of high-salt effects independent of blood pressure changes: 1) pressure threshold was affected selectively with no change in pressure sensitivity. 2) No measurable changes in global vessel wall distensibility were detected in DS on high-salt diets. 3) None of the compensatory changes in strain transduction normally observed during chronic resetting were evident in DS on high salt. 4) Despite the potential additional influence of the elevated systolic blood pressure in DS on high-salt diet, there was not a significant difference in the magnitude of the effect of high salt on pressure threshold among the three rat strains. Based on my own work and a survey of chronic and rapid resetting across a number of laboratories (see Table 1 in Reference 11), the blood pressure change alone would be expected to increase pressure threshold by 10 to 15 mm Hg. Thus, the duration of elevated systolic blood pressure may have been insufficient to induce measurable chronic resetting through vessel wall restructuring. By 50 weeks of age, adult DS on a diet of restricted salt have significantly greater systolic blood pressures than DR maintained on low salt and show the hallmarks of chronic hypertensive baroreceptor resetting: elevated pressure threshold coupled with decreased pressure sensitivity and decreased strain threshold with elevated strain sensitivity.

The results reported here do not support a “sensitization” of baroreceptors during exposure to a high-salt diet. On the contrary, a selective increase in pressure threshold was found for all rat strains studied, which is consistent with the findings of Miyajima and Buñag. Thus, local baroreceptor or vessel wall properties alone cannot be responsible for the reported baroreflex “sensitization” with high salt. Our results cannot rule out the possible presence of a “sensitizing” modulator in the blood of DR on high-salt diet that was eliminated in our saline perfused in vitro preparation. The direct effects of high salt on baroreceptors are to selectively increase pressure threshold. Thus, it appears that changes in the pattern of recruitment of newly active baroreceptors produce the decreased discharge rates (slope) in whole aortic nerves in vivo.

Splinting Hypothesis

When information concerning the mechanical properties of the vessel wall from each preparation is paired with pressure threshold and pressure sensitivity values for baroreceptors from the same rat, the resulting baroreceptor strain characteristics fell into a surprising pattern. All differences in threshold and sensitivity, which were apparent when expressed in terms of pressure, disappeared with strain equivalents (Figures 5 and 6, Table 2). This result is unlike any previous baroreceptor study, yet fulfills the prediction of the so-called “splinting hypothesis” of baroreceptor resetting. This hypothesis suggests that baroreceptor resetting might result from mechanical restraint of the stretch sensitive endings by the vessel wall. Thus baroreceptor responsiveness to distortion (strain threshold, strain sensitivity) would not be altered, but increased increments in pressure would be required to overcome local decreases in wall distensibility to stimulate the baroreceptor in an equivalent manner. This hypothesis is not consistent with the baroreceptor changes accompanying normal development and spontaneous genetic hypertension, normal aging, or rapid resetting. In the present study, the equivalence of strain properties together with the differences in pressure threshold between low salt and high salt for Dahl baroreceptors satisfies the basic tenets of this simple hypothesis.

Based on a simplified model of baroreceptor function, the differences in baroreceptor function between DR and DS might be attributed to differences in vessel wall distensibility or in coupling of the baroreceptor endings to the vessel wall. Differences in coupling would likely be most apparent during dynamic pressure inputs. Thus, coupling would be expected to have played a minimal role in the recorded differences in this study since quasi-steady-state responses were measured with slow.
ramps. Since global measures of the mechanical properties of the whole aortic arch were similar, the changes in the vessel wall responsible for the differences in baroreceptor pressure characteristics must be confined to the local environment of the sensory endings. It is apparently the relationship of these local mechanical properties to baroreceptor distortion which is altered by high salt.

Comparison of Dahl to Normal Rats

The Sprague-Dawley rat results suggest that baroreceptors in the two Dahl rat strains represent two extremes from normal baroreceptor function. DS tend to be less pressure responsive (high pressure threshold, low pressure sensitivity) than normal (SD), and DR tend to be somewhat more responsive to pressure than normal. Thus baroreceptors from the Dahl strains may represent opposite poles of a continuous distribution of baroreceptor properties in the more general, “normal” population (“normal” being equated to SD). The use of the term “defective” may be less than strictly appropriate in describing DS-low salt baroreceptors. Certainly all test groups had some baroreceptors with “normal” low pressure threshold and high pressure sensitivity characteristics, but these were relatively under-represented in the DS baroreceptor population and over-represented in DR rats compared with SD. A recent study suggests that adults, DS baroreceptors become highly variable. This may reflect a reduced capacity by DS to cope either with developmental changes in the vessel wall or with changes in blood pressure, so that DS baroreceptors track blood pressure very poorly. Unfortunately, we know too little about the factors influencing baroreceptors to determine whether the population differences we have observed are due to developmentally, trophic, or other unidentified factors.

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