Hypertension, Transmural Pressure, and Vascular Smooth Muscle Response in Rats

By Timothy R. Hansen and David F. Bohr

ABSTRACT

The effect of transmural pressure on the responsiveness of vascular smooth muscle was studied using rats with chronic occlusion of one external iliac artery. The arterial pressure in the occluded leg was reduced to approximately half of that in the contralateral unoccluded leg. Helical strips from the low- and high-pressure femoral arteries of spontaneously hypertensive rats and rats with deoxycorticosterone acetate–induced (DOCA) hypertension were compared with corresponding tissues from normotensive controls. The sensitivity of both low- and high-pressure artery strips from the spontaneously hypertensive rat was greater than that of controls when strontium or lanthanum was used as the agonist. The sensitivity of strips from both low- and high-pressure arteries from the DOCA-hypertensive rat was greater than that of controls when potassium, epinephrine, or calcium was the agonist. There was no difference in sensitivity between strips from the low- and high-pressure arteries in any group of rats. Maximum contractile force (contractility) was reduced in femoral artery strips from both legs of all hypertensive rats. The KCl-induced contraction of vascular smooth muscle from both femoral arteries of either form of hypertensive rat was not as readily depressed by high calcium concentrations as was that from the normotensive rat. Changes in sensitivity and contractility associated with hypertension could not be reversed by lowering blood pressure in one leg of a spontaneously hypertensive rat or prevented by protecting one leg from high pressure prior to the induction of DOCA hypertension. The altered sensitivity and contractility of arterial strips in these models of hypertension are not, then, secondary to the increase in wall stress.

KEY WORDS smooth muscle contractility smooth muscle sensitivity spontaneously hypertensive rats potassium DOCA-hypertensive rats lanthanum strontium calcium depression of KCl contraction epinephrine

Extensive evidence from studies of perfused vessels (1) or vascular beds (2–7) as well as from studies of isolated vascular smooth muscle (8–13) indicates that vascular responsiveness is increased in hypertension. A reasonable inference from this evidence is that the increase in responsiveness causes the elevated total peripheral resistance that results in hypertension. However, in view of recent findings showing that the increased wall stress of hypertension can produce profound adaptive changes in the artery wall (5, 14–16), more definitive evidence is needed to determine whether the increased responsiveness of vascular smooth muscle in the hypertensive rat occurs in the absence of increased wall stress.

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This study attempted to determine whether changes in sensitivity (responsiveness to low concentrations of agonist) and contractility (maximum tension-developing ability) associated with deoxycorticosterone acetate–induced (DOCA) and spontaneous hypertension result from increased wall stress. To examine this question, we developed an animal model in which the arteries of one leg of a hypertensive rat were protected from high wall stress. We compared the sensitivity and the contractility of helical strips of vascular smooth muscle from the protected (low-pressure) and unprotected (high-pressure) femoral arteries of DOCA-hypertensive and spontaneously hypertensive rats with those of strips from corresponding low- and high-pressure arteries of normotensive rats. The special sensitivity and contractility characteristics of artery strips from the hypertensive rats appeared to be independent of wall stress.

Methods

EXPERIMENTAL MODEL

External Iliac Tie.—One external iliac artery of the rat was tied off completely at its distal end (Fig. 1, top); rats were under sodium pentobarbital anesthesia, and a
Effect of unilateral iliac occlusion on cannulation. Mean blood pressure in the two saphenous arteries of a normotensive rat. Each pair of points (occluded and control) after the day of occlusion represents pressures in a different rat.sure transducers recording on a Grass polygraph. After were cannulated and connected to Statham P23C pres-
sures due to the iliac tie. The pressure and structural
findings in a similar series of normotensive and hyperten-
sive rats have been reported previously (17).

DOCA-Hypertensive and Normotensive Rats.—Male Sprague-Dawley rats weighing 150–200 g were anesthe-
tized with sodium pentobarbital (ip). All of the rats were subjected to unilateral iliac artery occlusion. Half of the rats were unilaterally nephrectomized, and DOCA. 40 mg in wax pellets, was implanted subcutaneously. This DOCA group received 1% NaCl for drinking water; the control group received tap water. Two weeks after DOCA implantation, the rats had become hypertensive, and an additional 20 mg of DOCA was implanted in each. Finally, 4–5 weeks after initial treatment, paired normo-
tensive and DOCA-hypertensive rats were killed by a blow to the head, and both femoral arteries were removed from both rats.

Spontaneously Hypertensive and Normotensive Rats.—Female spontaneously hypertensive rats (Oka-
moto-Aoki strain) and male Sprague-Dawley rats 180–220 g in weight were subjected to unilateral iliac occlu-
sion. These rats were killed in pairs 2–3 weeks after the iliac tie, and their femoral vessels were removed.

SENSITIVITY AND CONTRACTILITY STUDIES
Helical strips approximately 400μ wide and 4–6 mm long were cut from low- and high-pressure femoral arteries from a DOCA-hypertensive or a spontaneously hypertensive rat and from its normotensive control. Four strips, one from each femoral artery of the hypertensive rat and its normotensive control, were mounted in a common muscle bath of physiological salt solution be-
tween a fixed base and a Grass FT-03 force-displacement transducer recording on a Grass polygraph. Strips were stretched with a resting force of about 300 mg and allowed to equilibrate for 2 hours before experimental procedures were begun. Two types of physiological salt solution were used in each experiment: (1) a standard bicarbonate-buffered solution whose millimolar compo-
sition was NaCl 119, KCl 4.7, KH₂PO₄ 1.18, MgSO₄ 1.17, NaHCO₃ 14.9, dextrose 5.5, sucrose 50, CaNa₂ versenate 0.026, and CaCl₂ 1.6 and (2) a low-bicarbonate physiological salt solution whose composition was NaCl 154, KCl 5.4, NaHCO₃ 6.0, dextrose 11.0, and CaCl₂ 1.6. This latter solution was used when strontium, lanthanum, or high concentrations of calcium were to be added to the bath to prevent precipitation of insoluble salts of these cations. The bath was aerated with a 95% O₂-5% CO₂ mixture and maintained at 37°C with a pH of 7.4.

After the equilibration period of 2 hours in standard physiological salt solution, the following procedures were carried out in the order listed (concentrations are ex-
pressed as final bath concentrations). (1) Potassium chloride was added cumulatively to give concentrations of 5 to 65 mM. (2) Calcium chloride was added cumulatively to give concentrations of 3.2 and 5.0 mM. (3) Epinephrine was added cumulatively to give concentrations of 10⁻¹² to 10⁻⁸ g/ml. (4) Standard physiological salt solution was changed to low-bicarbonate physiological salt solution. (5) Potassium chloride was added to give a concentration of 40 mM, and then calcium chloride

midline abdominal approach was used. The effects of such a tie on blood pressure are illustrated in Figure 1, bottom. Both saphenous arteries of a normotensive rat were cannulated and connected to Statham P23C pres-
ures in the saphenous artery of that leg fell from a mean pressure of 100 mm Hg to about 20 mm Hg, but within 10–20 minutes pressure rose again to 40–45 mm Hg. Blood pressures of other rats with similar iliac ties were measured on subsequent days. Within 3–5 days, the mean pressure in the saphenous artery of the occluded leg rose to 55–60 mm Hg; the pressure remained at this level for at least 5–6 weeks. Thus, in an otherwise normal rat, the mean blood pressure was about 100 mm Hg in one femoral artery and was chronically lower in the other. Occlusion of one iliac artery of a spontaneously hypertensive rat lowered the mean blood pressure in the saphenous artery of that leg from approximately 185 mm

Historic studies of cross sections of the femoral arteries below the tie revealed no reactive or pathologic

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1 Generously provided by the Upjohn Company.
2 Generously provided by the Parke-Davis Company.
was added cumulatively to give concentrations of 1.6 to 18.1 mM. (6) Strontium chloride was added to give a concentration of 5 mM. (7) Lanthanum chloride was added to give a concentration of 0.5 mM. After use of each agonist, the bath was rinsed repeatedly until the recording had returned to its previous base line, i.e., zero active force.

At the end of the equilibration period the length of the strip in the bath was measured with a calibrated ocular micrometer, and after the experiment the wet weight of each strip was determined on a Cahn electrobalance. From measurements of strip length and weight (and assuming the specific gravity of the strip to be 1), the cross-sectional area of each strip was calculated as strip volume (weight) divided by strip length. The cross-sectional areas calculated for these strips when the total force stretching the strip was 300 mg are given in Table 1. With the information in this table, the active force developed was normalized as tension (g/mm²) to eliminate variability in strip size.

Results

RESPONSES TO CALCIUM, STRONTIUM, AND LANTHANUM

An increase in calcium concentration from 1.6 to 3.2 mM produced measurable (at least 10 mg) contraction of strips from both high- and low-pressure femoral arteries from DOCA-hypertensive rats but rarely caused a response of strips from normotensive or spontaneously hypertensive rats (Fig. 2A). A further increase in calcium concentration to 5.0 mM (Fig. 2B) elicited little response in any of the strips and, in fact, caused relaxation in the strips that had contracted in 3.2 mM calcium. Both 5.0 mM strontium and 0.5 mM lanthanum produced contraction of high- and low-pressure strips from spontaneously hypertensive rats but rarely caused a response in strips from either leg of normotensive or DOCA-hypertensive rats (Fig. 2C and D).

POTENTIATION OF A KCl-INDUCED CONTRACTION BY CALCIUM

Strips were stimulated with 40 mM KCl; after the response had reached a plateau, the calcium concentration in the bath was raised in steps from 1.6 to 18.1 mM. Increasing the calcium concentration initially potentiated the contraction in response to KCl in all strips. In the spontaneously hypertensive rats (Fig. 3, top) the potentiation of contraction of strips from both high- and low-pressure arteries of normotensive, DOCA-hypertensive, and spontaneously hypertensive rats in response to 3.2 mM CaCl₂ (A), 5.0 mM CaCl₂ (B), 5.0 mM SrCl₂ (C), and 0.5 mM LaCl₃ (D). The number of strips responding over the total number of strips is given above each bar. N-H, N-L, D-H, D-L, S-H, and S-L designate strips from high-pressure (H) and low-pressure (L) arteries of normotensive (N), DOCA-hypertensive (D), and spontaneously hypertensive (S) rats.

TABLE 1

Cross-Sectional Areas (mm²) of Strips from High- and Low-Pressure Arteries of Spontaneously Hypertensive and DOCA-Hypertensive Rats and of Their Normotensive Controls

<table>
<thead>
<tr>
<th></th>
<th>High-pressure artery</th>
<th>Low-pressure artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normotensive controls</td>
<td>0.098 ± 0.012</td>
<td>0.077 ± 0.005</td>
</tr>
<tr>
<td>Spontaneously hypertensive rats</td>
<td>0.094 ± 0.007</td>
<td>0.067 ± 0.007</td>
</tr>
<tr>
<td>Normotensive controls</td>
<td>0.098 ± 0.011</td>
<td>0.086 ± 0.007</td>
</tr>
<tr>
<td>DOCA-hypertensive rats</td>
<td>0.076 ± 0.005</td>
<td>0.053 ± 0.008</td>
</tr>
</tbody>
</table>

All values are means ± se.
hypertensive rats (58.3 and 55.2%, respectively). Again when the calcium concentration had been raised sufficiently to reduce the potentiation of the strips from the high-pressure artery of the normotensive rat to near zero (10.1 mM), the responses of the strips from both the high- and low-pressure arteries of the DOCA-hypertensive rats were still strongly potentiated (49.3%, \( P < 0.001 \), and 43.7%, \( P < 0.01 \), respectively). There was no significant difference between high- and low-pressure artery strips from normotensive or DOCA-hypertensive rats.

In this study a striking characteristic of the difference between the vascular smooth muscle from either type of hypertensive rat and its normotensive control was the shift to the right of the concentration-response curve for the depressing effect of calcium. A high concentration of calcium was required to depress the response of the smooth muscle from either the low- or the high-pressure leg of the hypertensive rat. This shift was exhibited by strips from both femoral arteries of either type of hypertensive rat regardless of the pressure to which they had been exposed.

### KCl and Epinephrine Studies

Concentration-response curves to KCl of arterial strips from normotensive and spontaneously hypertensive rats are shown in Figure 4, top. For all groups of strips, maximum or near-maximum contraction was reached at a KCl concentration of approximately 50 mM. For the normotensive rats, the maximum strength of contraction of the low-pressure strips was not significantly less (\( P > \))

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**FIGURE 3**

*Effect of increasing calcium concentration on a KCl contraction of smooth muscle from high- and low-pressure arteries of normotensive (Normo) and spontaneously hypertensive (Spont. Hpt.) rats (top) and normotensive and DOCA-hypertensive (DOCA) rats (bottom). The response of each strip to 40 mM KCl with 1.6 mM CaCl\(_2\) in the bath is considered to be the initial active tension, and the effects of increasing the calcium concentration are plotted as percent changes from this tension. The height of each curve depicts the potentiation of the response. Each curve represents mean values of nine experiments except that for the low-pressure arteries from spontaneously hypertensive rats where \( N = 6 \).*

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than that of the high-pressure strips (3.34 and 4.56 g/mm², respectively). For the spontaneously hypertensive rats, the maximum tension developed by both low- and high-pressure artery strips (1.33 and 1.28 g/mm², respectively) was significantly less than that developed by normotensive rat strips ($P < 0.01$).

Similar studies on strips from DOCA-hypertensive rats (Fig. 4, bottom) showed that for the normotensive control rats the maximum contractile tension of the strips from the low-pressure arteries was not significantly less ($P > 0.20$) than that from the high-pressure arteries (2.76 vs. 3.44 g/mm²). Compared with these maximum tensions of strips from normotensive rats, those from the DOCA-hypertensive rats were significantly less ($P < 0.01$) for both low- and high-pressure strips (1.23 and 1.39 g/mm², respectively). There was no difference between the maximum tensions developed by the low- and high-pressure strips of the DOCA-hypertensive rats.

The data in Figure 4 were recalculated to show each response as a percent of the maximum tension achieved by the strip; these values are plotted in Figure 5. We used the percent of the maximum response elicited by low concentrations of the agonist as an index of the sensitivity of the artery strip. The sensitivity of strips from both low- and high-pressure femoral arteries of spontaneously hypertensive rats (Fig. 5, top) was essentially the same as the sensitivity of strips from low- and high-pressure arteries of normotensive rats ($P > 0.2$ for either high- or low-pressure artery strips from spontaneously hypertensive rats compared with normotensive rats). However, low concentrations (10 or 15 mM) of KCl elicited much larger responses from DOCA-hypertensive rat high- and low-pressure femoral artery strips (Fig. 5, bottom) than they did from normotensive rat strips ($P < 0.01$ and $P < 0.001$, respectively, at either dose). There were no significant differences between the responses of strips from high- and low-pressure arteries of normotensive ($P > 0.2$), DOCA-hypertensive ($P > 0.2$), or spontaneously hypertensive ($P > 0.2$) rats.

In similar studies in which epinephrine was the agonist rather than KCl, the results were similar to those of the KCl studies in terms of both sensitivity and contractility. For all groups of strips, maximum tension was reached at an epinephrine concentration of $10^{-6}$ g/ml (Fig. 6). For the normotensive controls of the spontaneously hypertensive rats (Fig. 6, top), the maximum strength of contraction of the low-pressure artery strips (4.92 g/mm²) was somewhat less than that of the high-pressure artery strips (6.52 g/mm², $0.05 > P > 0.01$). Maximum tensions developed by both groups of strips from spontaneously hypertensive rats (high-pressure 2.09 g/mm², low-pressure 2.41 g/mm²) were significantly less than those developed by strips from the normotensive rats ($P < 0.01$). There was no significant difference in maximum contractility between the strips from high- and low-pressure arteries of spontaneously hypertensive rats ($P > 0.20$). Similar studies on strips from DOCA-hypertensive rats (Fig. 6, bottom) showed that for normotensive control rats the maximum contractile tension of strips from the
low-pressure arteries was slightly less than that of strips from the high-pressure arteries (3.28 and 4.71 g/mm², respectively). This difference was not statistically significant (0.20 > $P > 0.10$). Compared with the maximum tension of the high-pressure strips from the normotensive rats, that from the DOCA-hypertensive rats was significantly less ($P < 0.001$) in both high- and low-pressure strips (2.53 and 2.05 g/mm², respectively). The difference between the two groups of strips from DOCA-hypertensive rats was not significant ($P > 0.20$).

The data in Figure 6 were recalculated to show each response as the percent of the maximum tension achieved by the strip; these values are plotted in Figure 7. The sensitivity in response to epinephrine of strips from both high- and low-pressure femoral arteries of spontaneously hypertensive rats was essentially the same as that of strips from either high- or low-pressure arteries of normotensive rats (Fig. 7, top). However, the sensitivity of both high- and low-pressure artery strips of DOCA-hypertensive rats (Fig. 7, bottom) as judged by their response to $10^{-10}$ g/ml of epinephrine (22.7 and 30.9% of maximum tension) was greater ($P < 0.05$) than that of comparable strips from normotensive controls (11.4 and 9.7% of maximum tension). Once again there was no significant difference between the responses of strips from high- and low-pressure arteries of normotensive ($P > 0.20$), DOCA-hypertensive ($P > 0.20$), or spontaneously hypertensive ($P > 0.20$) rats.

![Figure 6](image1.png)

**Figure 6**
Concentration-response curves to epinephrine of smooth muscle from high- and low-pressure femoral arteries from normotensive (Normo) and spontaneously hypertensive (Spont. Hyp.) rats (top) and from normotensive and DOCA-hypertensive (DOCA) rats (bottom). The position of the curves depicts contractility. Each curve represents the average value for nine experiments except the curve for the low-pressure arteries from spontaneously hypertensive rats where $N = 6$.

![Figure 7](image2.png)

**Figure 7**
Normalized concentration-response curves to epinephrine. These data are recalculated from those in Figure 6 to normalize differences in contractility. Each response is shown as a percent of the maximum tension achieved by the strip. The percent of maximum response produced by low concentrations of epinephrine is considered to be an index of the sensitivity of the strip. Each curve represents the average value for nine experiments except the curve for low-pressure arteries from spontaneously hypertensive rats where $N = 6$. Abbreviations are the same as they are in Figure 6.
Discussion

Results of the current experiments identify specific functional differences between vascular smooth muscle from normotensive and hypertensive rats. In the first place, there is evidence that at least with regard to some specific agonists the sensitivity of vascular smooth muscle from the hypertensive rat is greater than the sensitivity of the same muscle from the normotensive rat. On the other hand, the contractility (as indexed by the maximum tension-developing ability) of arterial strips from the hypertensive rat is consistently less than that from the normotensive control. The finding that fulfills the primary objective of the current study is that neither of these differences is dependent on the increased wall stress to which the vascular smooth muscle is subjected in the hypertensive animal.

Sensitivity

The term sensitivity is used to denote the ease with which the contractile process of vascular smooth muscle is activated. Our findings confirm extensive evidence (8–13) that there are differences in sensitivity between the vascular smooth muscle of hypertensive and normotensive rats. Muscle from arteries of DOCA-hypertensive rats differed from that of normotensive rats in its greater sensitivity to stimulation by calcium, KCl, and epinephrine. Muscle from arteries of spontaneously hypertensive rats differed from muscle from other groups tested in its greater sensitivity in response to strontium and lanthanum. This finding must be taken advisedly; since our groups of spontaneously hypertensive rats and their controls were not well matched, this difference may be based on genetic factors unrelated to the hypertension.

Calcium is known to have a dual effect on vascular smooth muscle (18): (1) it causes contraction by virtue of its affinity for the regulatory protein, and (2) it binds to the cell membrane, thereby stabilizing it and inhibiting contraction. Thus, if the amount of calcium bound to the membrane is lower than normal, the stabilizing effect of calcium will be lessened and the membrane will be more sensitive to any stimulus, such as the 40 mM KCl used in the current study. If this increased sensitivity is caused by a deficit in the number of membrane binding sites (MBS) for calcium and there is a mass action relationship between the calcium and these sites (Ca²⁺ + MBS = CaMBS), then a greater additional calcium concentration would be required to inhibit a KCl-induced contraction. Since we have observed that a higher concentration of calcium is required to depress the response of vascular smooth muscle from the hypertensive rat (Fig. 3), it is possible that the cause of the increased excitability associated with hypertension is a decreased number of calcium binding sites in the plasma membrane. The critical observation is that this increased calcium requirement was present in smooth muscle from both the low- and high-pressure legs and hence was not secondary to increased wall stress.

Jones (19) has presented evidence from his potassium studies that the plasma membrane of vascular smooth muscle from spontaneously hypertensive rats is "leakier" to potassium than is that of normal rats. This observation constitutes further evidence of an abnormality in the plasma membrane in this muscle from the spontaneously hypertensive rat.

Contractility

We used the term contractility to describe the force-generating ability of the muscle when it was fully activated, and we measured contractility by the maximum tension developed by a muscle in response to an agonist. We saw in this study that the contractility of vascular smooth muscle from the hypertensive rat was less than that from the normotensive rat. Clineschmidt et al. (20) have reported that vascular smooth muscle strips from different strains of normotensive rats show marked differences in contractility, and they have warned that care must be taken to employ the proper control animal, especially when the contractility of vascular smooth muscle of spontaneously hypertensive rats is being evaluated. However, in this study we demonstrated differences not only between the contractility of strips from spontaneously hypertensive rats and Sprague-Dawley normotensive controls but also between two groups of Sprague-Dawley rats that differed only in blood pressure (one normotensive and the other DOCA hypertensive).

The maximum contractility of a muscle is also dependent on the amount of preload or passive tension placed on it. Gordon and Nogueira (21) have presented evidence that the decreased contractility of vascular smooth muscle strips from the hypertensive rat is only apparent; when they increased the passive stress on this muscle to equal the greater stress to which it had been subjected in situ, muscle from the hypertensive rats developed at least as much active tension as did muscles from the normotensive controls. However, subsequent studies (10) have failed to confirm these findings.
and have indicated that, throughout the full range of the length-tension curve, vascular smooth muscle strips from the hypertensive rat are not capable of developing as much active tension as are those from the normotensive control. We have demonstrated (17) that the differences in contractility which we have observed in this study using only one preload accurately reflect the differences that exist between femoral artery strips from hypertensive and normotensive rats throughout the entire length-tension range.

The decreased maximum force-generating ability of smooth muscle strips from the femoral artery of the hypertensive rat has no obvious relationship to the cause of hypertension, nor can it be viewed as evidence opposing the observation of increased maximum pressor response in the perfused hindquarter of the hypertensive rat (5). These pressor responses reflect contractions of smooth muscle of resistance vessels that are smaller and functionally different from the femoral artery; furthermore, these pressor responses are influenced by the wall thickness in a way that does not affect the simple measurement of tension developed by a strip of muscle.

WALL STRESS AND VASCULAR RESPONSES

In the current study we have observed that characteristic alterations in artery strip sensitivity occur in the hypertensive rat regardless of whether the artery has been exposed to an elevated transmural pressure. It is possible that changes in sensitivity seen in the vascular smooth muscle of the spontaneously hypertensive rat are in fact produced in response to an increased transmural pressure but are irreversible and thus do not regress in the presence of lowered arterial blood pressure. However, in DOCA hypertension, muscle from femoral arteries never exposed to the high pressure had the same changes in sensitivity observed in the contralateral high-pressure artery. Since we could neither reverse nor prevent changes in vascular smooth muscle sensitivity by iliac ties, we must conclude that these functional changes are not the direct result of elevated transmural pressure. This conclusion in no way contradicts the findings of others indicating that structural changes in the vessel wall may be caused by the increase in transmural pressure. Folkow et al. (22) have observed that 3 weeks after occlusion of the lower abdominal aorta in the spontaneously hypertensive rat the structural resistance in the hindquarter is below normal. Both Lundgren (23) and Wolinsky (14) have observed that when hypertension in renal hypertensive rats is reversed by removing the renal artery clip structural changes in the aorta are also reversed. On the other hand, Shibata et al. (12) have reported that the increased responsiveness to the nonphysiological cations strontium, lanthanum, and manganese is present in the vascular smooth muscle before the onset of hypertension in the spontaneously hypertensive strain of rats. The differences in findings regarding the stress dependence of vascular changes in hypertension may be reconciled by postulating that structural changes are secondary to the increase in transmural pressure whereas certain functional changes in smooth muscle sensitivity are not.

Whether changes in contractility are stress dependent is not quite so clear. Even in the normotensive rat, the contractility of the smooth muscle from the low-pressure femoral artery appears to be less than that of the contralateral femoral artery. Although the differences are not striking, iliac occlusion, per se, is associated with a weaker contractile response from ipsilateral femoral artery strips. For this reason, although the contractility of the strips of both high- and low-pressure arteries of the hypertensive rat is equivalently depressed, it is not possible to distinguish between the decreased contractility due to iliac occlusion and that due to hypertension.

It must be concluded from these studies that there are differences in sensitivity and contractility between arterial smooth muscles of hypertensive and normotensive rats. Most important, however, is the finding that there are functional differences in sensitivity which are not caused by the increase in wall stress during arterial hypertension.

References

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