Increased Resistance and Impaired Maximal Vasodilation in Normotensive Vascular Beds of Rats with Coarctation Hypertension

DAVID R. BELL, M.S. AND HENRY W. OVERBECK, M.D., PH.D.

SUMMARY To study the resistance of normotensive vascular beds in coarctation hypertension, we measured perfusion pressures of pump-perfused (blood), innervated, isolated hindlimbs of 12 rats (Group A) with 4 weeks of hypertension due to partial constriction of the abdominal aorta above the renal arteries, and of three control groups: 11 normotensive rats (Group B) with aorta sham-constricted, nine normotensive rats (Group C) with slight (5%) hindquarters atrophy due to partial constriction of the abdominal aorta below the renal arteries, and six rats with two-kidney, one clip Goldblatt hypertension (Group D). After aortic constriction, measured femoral arterial pressures in Group A rats remained normotensive. In hypertensive rats of Groups A and D, compared to normotensive Group B or C rats, hindlimb pressure-flow curves were displaced toward the pressure axis (p < 0.05). Compared to normotensive rats, drop in hindlimb resistance after acute local nerve section was increased in rats with coarctation hypertension. Residual resistance after maximal vasodilation with intraarterial sodium nitroprusside remained elevated in hypertensive rats of Groups A and D (p < 0.05), as compared to normotensive Group B or C rats; compared to Group B rats, this residual resistance in the coarcted rats of Group A was increased by 9%. Thus, in normotensive vascular beds of rats with chronic hypertension caused by aortic coarctation, resistance is elevated. The neurogenic component contributes to this high resistance, and structural vascular changes, indicated by impaired maximal vasodilation, may also contribute to the elevated resistance. It is most unlikely that these resistance changes are attributable to elevated hindlimb intravascular pressures. (Hypertension 1: 78-85, 1979)

KEY WORDS • vascular wall-to-lumen ratio • structural component of resistance • neurogenic component of resistance • limb vascular bed • pressure flow relationship • Goldblatt hypertension • plasma renin concentration

C OARCTATION hypertension is a useful model for examining the effects of intravascular pressure on vascular structure and function in hypertension, because the vascular beds below the coarctation are not exposed to elevated levels of pressure. Thus, abnormalities observed could not be said to be merely secondary to increased intravascular pressure. We have previously reported increased salt and water content in the walls of arteries from these normotensive vascular beds in rats with coarctation hypertension.1 It has been suggested that such “waterlogging” of arterial walls increases vascular resistance and responses.2 Therefore, it is of considerable interest to investigate the status of vascular resistance in these normotensive beds in coarctation hypertension.

In 1963 Nolla-Panades3 reported increased resting resistance and responses to norepinephrine in the normotensive hindquarters vascular beds of rats with coarctation hypertension. However, his experiments were conducted in hindquarters perfused with Krebs-Henseleit solution with no oncotic substitute. Thus, it is possible that the abnormalities in resistance he observed may have been artifacts, due, for example, to interstitial edema of the preparation. More importantly, Nolla-Panades did not document residual resistance at maximal vasodilation; this measurement is considered a prime indicator of the status in hyper-
tension of structural changes increasing vascular wall-to-lumen ratio. It is especially important to determine if such structural changes occur in resistance vessels in hypertension in the absence of increases in intravascular pressure. For these reasons, we felt Nolla-Panades' experiments should be extended with measurements in blood perfused beds of pressure-flow relationships, and resistance at maximal vasodilation.

Methods

Normotensive male Sprague-Dawley rats approximately 2 months old and weighing 150–200 g were randomly divided into four experimental groups. To create coarctation hypertension in 16 rats (Group A), we placed a partially constricting silver clip (diameter 0.813 mm) around the abdominal aorta upstream to the origin of both main renal arteries. In 21 rats (Group B) a clip (diameter ≥ 1.48 mm) too large to constrict the aorta was similarly placed; this group of rats served as sham-coarcted normotensive controls. In a third group of 15 rats (Group C), a partially constricting clip (diameter 0.610 mm) was placed around the abdominal aorta below the origin of both renal arteries. Because such infra-renal aortic coarctation reduces downstream intravascular pressure and flow but does not produce hypertension, this group of rats served as normotensive controls for any hindquarters atrophy occurring in the rats with coarctation hypertension (Group A). In a final group of nine rats, two-kidney, one clip Goldblatt hypertension was created by partially constricting the left renal artery with a silver clip (diameter 0.406–0.434 mm) with the opposite kidney intact.

Postoperatively, the rats were maintained on a diet of standard rat chow and tap water ad libitum. Systolic arterial blood pressures in the hindquarters of all rats were measured weekly by the tail plethysmographic method under light ether anesthesia.

Four to 5 weeks after surgery, we measured carotid and femoral arterial pressures directly with the rats anesthetized rats (n = 12, 11, 9 and 6 in Groups A, B, C and D, respectively), we then measured perfusion pressure in the vascularly isolated, innervated, pump-perfused hindlimb vascular beds, using the procedure described by Laverty. Briefly, we isolated one hindlimb from the body by severing and ligating the femoral artery of the isolated limb. To study pressure-flow relationships in the limb vascular bed, pump flow was then adjusted serially at 5- to 9-minute intervals to 0.125, 0.25, 0.5, 1.0, 1.5, and 2.0 ml/min, in that order. Steady-state perfusion pressures were recorded. Then the flow sequence was repeated and averaged values reported.

Pump flow was again returned to 1 ml/min, and, when a steady state was restored, the femoral and sciatic nerves to the perfused limb were severed. Ten minutes later the steady-state limb perfusion pressure was recorded. Calculated resistance was designated "resistance after acute nerve section." Then a supramaximal dose of sodium nitroprusside (0.15 mg/kg in 0.05–0.10 ml isotonic NaCl solution) was injected rapidly into the pump tubing upstream to the pump. Maximal vasodilation of the limb was tested by successively doubling the dose. In preliminary experiments in three rats, no further vasodilation was elicited by papaverine injections. Perfusion pressure 4 minutes after the final nitroprusside injection was recorded; calculated resistance was designated "resistance after maximal sodium nitroprusside vasodilation."

In all rats the perfusion pressure gradient across the outflow tubing and cannula was measured at each flow rate used; this value was subtracted from the perfusion pressures used to calculate all limb resistances. Limb resistances were calculated as the ratio of perfusion pressure to limb blood flow and expressed in terms of hindlimb wet weight.

After the perfusion study, blood was taken from all animals for measurement of hematocrit. In most animals plasma creatinine, sodium, potassium, calcium and magnesium concentrations were also measured. Plasma creatinine concentration was measured by auto-analyzer, plasma sodium and potassium concentrations by flame photometer (Beckman), and calcium and magnesium concentrations by atomic absorption spectrometry (Perkin-Elmer). Each rat was autopsied to verify clip type, placement, and general health. Kidneys and heart were weighed. Hindlimbs were removed and both wet and dry (after oven drying at 86°C for 7 days) weights obtained.

Thirteen additional rats of Groups A and B each were prepared. Four to 5 weeks after clipping, these rats were decapitated without anesthesia and aortic blood was collected for the first 2 seconds. Plasma renin concentrations were measured by radio-immunoassay.
Among groups, variables were compared with one-way analysis of variance and, if this indicated that significant differences existed, means were then compared with Student-Newman-Kuels test. Student’s $t$ test for paired replicates was used for within-group comparisons. Entire pressure-flow curves were also assessed with profile analysis. The null hypothesis was rejected at $p < 0.05$.

Results

All rats reported remained healthy with no evidence of cardiac or renal insufficiency or malignant hypertension at the time of study. At this time body weights (table 1) and serum sodium, potassium, calcium, magnesium and creatinine concentrations did not differ significantly among the experimental groups and were within normal limits. Hematocrits, also obtained after the perfusions, were within normal ranges.

Changes occurred in kidney, heart and limb weights in these rats (table 1). Absolute left kidney weight, as well as ratio of left kidney weight to body weight, were slightly (11%) but significantly ($p < 0.05$) reduced in rats with coarctation hypertension (Group A) compared to sham-coarcted controls (Group B). In the Goldblatt hypertensive rats (Group D) absolute weight of the clipped left kidney, as well as ratio of left kidney weight to body weight, were decreased by 27% ($p < 0.05$), whereas the weight of the untouched right kidney, and its ratio to body weight, increased by 19% ($p < 0.01$). Absolute heart weight and heart weight expressed in terms of body weight increased by 44 and 21%, respectively ($p < 0.01$) in rats of Groups A and C with partially constricted aortas compared to sham-coarcted control rats of Group B, suggesting that aortic constriction produced slight hindlimb atrophy.

As figure 1 indicates, tail systolic blood pressures measured indirectly under light ether anesthesia each week for the 3 weeks after clipping remained unchanged in rats with coarctation hypertension (Groups A and D) and in normotensive sham-coarcted controls (Group B), dropped significantly in rats with aortas coarcted below the renal arteries (Group C) and increased significantly in rats with Goldblatt hypertension (Group D). Mean carotid arterial pressure directly measured at the time of the perfusion study under light chloralose anesthesia was significantly increased in rats with coarctation and Goldblatt hypertension (Groups A and D) as compared to both normotensive control groups (fig. 2). As indicated by figure 2 and table 2, mean, systolic, and diastolic femoral arterial pressures in rats with coarctation hypertension (Group A) did not differ significantly from those in the normotensive controls of Group B, remaining within normal limits. Pulse pressure, however, decreased slightly in the femoral arteries of the Group A rats. Mean, systolic, and diastolic femoral arterial pressures of rats with aortas coarcted below the renal arteries (Group C) were significantly decreased and those of Goldblatt hypertensive rats (Group D) were significantly increased compared to sham-coarcted normotensive control rats of Group B. There was also a slight (1%), but significant ($p < 0.01$), increase in heart weight/body weight in normotensive rats with aorta coarcted below the renal arteries (Group C). Hindlimb wet weight, expressed in terms of body weight (table 1), was reduced 6 and 5%, respectively ($p < 0.01$) in rats of Groups A and C with partially constricted aortas compared to sham-coarcted control rats of Group B, suggesting that aortic constriction produced slight hindlimb atrophy.

Table 1. Body and Organ Weights*

<table>
<thead>
<tr>
<th></th>
<th>Group A (Coarctation hypertension)</th>
<th>Group B (Sham coarctation)</th>
<th>Group C (Infra-renal coarctation)</th>
<th>Group D (Goldblatt hypertension)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body wt. (g)†</td>
<td>353 ± 5(16)</td>
<td>353 ± 3(21)</td>
<td>359 ± 4(15)</td>
<td>359 = 10(9)</td>
</tr>
<tr>
<td>Lt. kidney wt. (g)†</td>
<td>1.154 ± 0.044(16)§</td>
<td>1.323 ± 0.037(21)</td>
<td>1.326 ± 0.040(15)</td>
<td>0.984 ± 0.106(9)§</td>
</tr>
<tr>
<td>Lt. kidney wt./body wt. × 10†</td>
<td>33 ± 1(16)‡</td>
<td>37 ± 1(21)</td>
<td>37 ± 1(15)</td>
<td>27 ± 3(9)§</td>
</tr>
<tr>
<td>Rt. kidney wt. (g)†</td>
<td>1.198 ± 0.056(16)§</td>
<td>1.308 ± 0.044(21)</td>
<td>1.331 ± 0.050(15)</td>
<td>1.554 ± 0.062(9)§</td>
</tr>
<tr>
<td>Rt. kidney wt./body wt. × 10†</td>
<td>34 ± 1(18)‡</td>
<td>37 ± 1(21)</td>
<td>37 ± 1(15)</td>
<td>44 ± 3(9)§</td>
</tr>
<tr>
<td>Heart weight (g)†</td>
<td>1.604 ± 0.050(16)§</td>
<td>1.176 ± 0.022(21)</td>
<td>1.207 ± 0.022(15)</td>
<td>1.446 ± 0.048(9)§</td>
</tr>
<tr>
<td>Heart wt./body wt. × 10†</td>
<td>481 ± 15(16)§</td>
<td>333 ± 6(21)</td>
<td>337 ± 7(15)§</td>
<td>403 ± 14(9)§</td>
</tr>
<tr>
<td>Perfused hindlimb wt. (g)†</td>
<td>20.107 ± 0.244(14)§</td>
<td>20.754 ± 0.234(20)</td>
<td>20.266 ± 0.287(15)</td>
<td>21.206 ± 0.589(9)</td>
</tr>
<tr>
<td>Perfused hindlimb/body wt. × 10†</td>
<td>574 ± 10(14)†</td>
<td>588 ± 6(20)</td>
<td>565 ± 7(15)</td>
<td>592 ± 10(9)</td>
</tr>
<tr>
<td>Opposite hindlimb wt. (g)†</td>
<td>19.221 ± 0.297(14)</td>
<td>20.597 ± 0.217(20)</td>
<td>19.825 ± 0.289(15)</td>
<td>20.701 ± 0.590(9)</td>
</tr>
<tr>
<td>Opposite hindlimb/body wt. × 10†</td>
<td>548 ± 10(14)†</td>
<td>583 ± 6(20)</td>
<td>552 ± 5(15)§</td>
<td>578 ± 7(9)</td>
</tr>
<tr>
<td>Perfused hindlimb dry wt. (g)</td>
<td>6.105 ± 0.131(16)</td>
<td>6.509 ± 0.084(21)</td>
<td>6.365 ± 0.129(15)</td>
<td>6.380 ± 0.194(9)</td>
</tr>
<tr>
<td>Opposite hindlimb dry wt. (g)</td>
<td>6.248 ± 0.141(16)</td>
<td>6.686 ± 0.087(21)</td>
<td>6.375 ± 0.110(15)</td>
<td>6.602 ± 0.225(9)</td>
</tr>
</tbody>
</table>

*Means ± SEM; Numbers of observations in parenthesis.
†Wet weights.
§p < 0.05, compared to Group B.
§§p < 0.01, compared to Group B.
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200 150 100 50
Group D

Group C

P<.05, vs Group B

tClipping

Weeks Postoperative

FIGURE 1. Mean (±SEM) tail systolic arterial pressures (by plethysmography) recorded at weekly intervals following clipping. Groups are identified on figure. Asterisk represents pressures significantly different (p < 0.05) from those in sham-coarcted control rats of Group B.

200 150 100 50

FIGURE 2. Mean (±SEM) carotid (CBP) and femoral (FBP) arterial pressures measured directly under light chloralose anesthesia 4 weeks after clipping. The p values are represented for comparison with pressures in the sham-coarcted control rats of Group B.

Table 2. Femoral Arterial Pressures*

<table>
<thead>
<tr>
<th>Group A (Coarctation hypertension)</th>
<th>Group B (Sham coarctation)</th>
<th>Group C (Infra-renal coarctation)</th>
<th>Group D (Goldblatt hypertension)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic (mm Hg)</td>
<td>116 ± 5 (13)</td>
<td>110 ± 2 (20)</td>
<td>78 ± 3 (13)†</td>
</tr>
<tr>
<td>Diastolic (mm Hg)</td>
<td>105 ± 5 (13)</td>
<td>95 ± 3 (20)</td>
<td>72 ± 3 (13)†</td>
</tr>
<tr>
<td>Pulse (mm Hg)</td>
<td>11 ± 1 (13)†</td>
<td>15 ± 1 (20)</td>
<td>6 ± 1 (13)†</td>
</tr>
</tbody>
</table>

*Means ± SEM; numbers of observations in parentheses; mean pressures provided in figure 2.
†p < 0.05, compared to Group B.
‡p < 0.01, compared to Group B.
Figure 3. Resting limb blood flow (ml/min/g limb wet weight). Pump perfusion pressure set to equal femoral arterial pressure. Groups are identified on the figure. The P values are represented for comparison with flows in the sham-coarcted control rats of Group B.

Figure 4. Pressure-flow relationships in the isolated innervated, pump-perfused vascular bed of the hindlimb. Groups are identified on the figure. The checks represent P values for comparison of points for Groups A (coarctation hypertension) and C (aortas coarcted below renal arteries). The asterisks represent P values for comparison of values in Groups A and B (sham-coarctation).

the normotensive sham-coarcted rats of Group B. There were no significant differences between decreases in Group B and those in Groups C and D.

Figure 5 also indicates that resistance after acute nerve section, with pump flow at 1 ml/min, in rats with coarctation hypertension (Group A) did not differ significantly from that in normotensive sham-coarcted rats of Group B. However, limb resistances after acute nerve section in rats with aortas coarcted below the renal arteries (Group C) and with Goldblatt hypertension (Group D) were 28% lower and 58% higher, respectively, than resistance in rats of Group B (p < 0.01).

Intrafemoral arterial injection of supramaximal doses of sodium nitroprusside dropped hindlimb resistance further in all rats. As in the case of nerve section, the magnitude of this drop differed among the groups. Expressed as percentage of resting resistance with nerves intact, the decrease in resistance evoked by sodium nitroprusside was 35.4 ± 2.2, 45.6 ± 3.1, 49.9 ± 2.1 and 46.8 ± 2.4% in Groups A, B, C and D, respectively. The drop in resistance was lower in Group A (p < 0.05) than in the normotensive rats of Group B. There were no significant differences between decreases in Groups B, C and D.

Resistances in the denervated hindlimbs of the four groups of rats at maximal sodium nitroprusside vasodilation (pump flow rate 1 ml/min) are also presented in figure 5. Compared to sham-coarcted normotensive control rats of Group B, residual resistance...
after maximal vasodilation was elevated by 9% 
\( p < 0.05 \) in rats with coarctation hypertension 
(Group A), and by 32% \( p < 0.01 \) in rats with 
Goldblatt hypertension (Group D). The difference 
between Groups A and D was highly significant 
\( p < 0.01 \). In contrast, residual resistance was 
decreased by 27% \( p < 0.01 \) in Group C rats with aor-
tas coarcted below the renal arteries. Again, the 
difference between Groups A and C was highly signifi-
cant \( p < 0.01 \).

Plasma renin concentrations in rats with coarcta-
tion hypertension (Group A) were increased by 81% 
\( p < 0.001 \) to 21.8 ± 2.0 ng angiotensin I/ml hr\(^{-1}\), as 
compared to 12.1 ± 0.8 in sham-coarcted normoten-
sive control rats (Group B).

**Discussion**

Abnormalities in vascular wall composition, struc-
ture, and function occur in hypertension, but it is often 
difficult to determine whether these changes reflect un-
derlying causative mechanisms or, on the other hand, 
are simply the result of the increased intravascular 
pressure. Experimentally induced coarctation hyper-
tension may be used to examine the effects of intra-
vascular pressure on vascular structure and function in 
hypertension, because the vascular beds below the 
coarctation are not exposed to elevated levels of 
pressure. In rats with this model of hypertension we 
found "waterlogging" in the walls of arteries from normotensive vascular beds.\(^1\) Furthermore, Nolla-
Panades,\(^3\) using a preparation similar to that of the 
present study, but perfused with artificial solution 
rather than blood, showed that resistance is elevated 
and norepinephrine responses are enhanced in the nor-
motensive vascular bed of the hindquarters in coarcted 
rats.

The results of the present investigation in blood-
perfused hindlimbs of rats with coarctation hyper-
tension confirm and extend Nolla-Panades' observations 
by indicating that resistance is elevated over a wide 
range of flows, that the major component of this 
elevated resistance is related to neural influences, and 
that a significant portion of the elevated resistance 
persists after maximal chemical vasodilation, suggest-
ing that vascular structural changes have occurred.

Both we and Nolla-Panades\(^3\) repeatedly measured 
hindquarters arterial pressures in these rats and never 
found them to exceed those in sham-coarcted normotensive 
control rats. However, continuous intra-
arterial pressures over the entire experimental period 
were not obtained, so there remains the remote 
possibility that there were slight transient pressure 
elevations. Nevertheless, it is most unlikely that these

![Figure 5](http://hyper.ahajournals.org/)
observed abnormalities in resistance are the direct effects of elevated arterial pressures. Nor may it be argued that these hypertensive rats had elevated hindquarters blood flows, with resistance changes resulting from "long-term autoregulation"; hindlimb blood flow was actually reduced. The changes we have observed in the present study cannot be artifacts caused by perfusion with artificial media. Nor can they be explained on the basis of hindquarters atrophy with decreased numbers of perfused vessels, because there was similar hindquarters atrophy in the normotensive rats with aortas coarcted below the renal arteries (Group C). In these latter control rats, resting hindlimb resistance was reduced, as was resistance after maximal sodium nitroprusside vasodilation, changes in the opposite direction from those we observed in the rats with coarctation hypertension. Indeed, findings in this control group of rats increase the significance of the changes in resistance we observed in the rats with coarctation hypertension.

Figure 5 may be useful in analyzing the components of the elevated resistance in the normotensive hindlimbs of these rats with coarctation hypertension (Group A). It may be seen that acute local nerve section evoked a greater \( p < 0.01 \) fall in limb resistance in these rats than in the sham-coarcted normotensive control rats (Group B), reducing resistance to levels not significantly different from those in the sham-coarcted rats. These data suggest that the component of resistance related to neural influences, the "neurogenic" component, accounts for the major portion of the elevated resistance of the hindlimb in the rats with coarctation hypertension. However, it cannot be said whether increased nerve traffic, modulation of nerve traffic by angiotensin, increased vessel responsiveness (on a structural and/or functional basis), or a combination, contributes to this elevated "neurogenic" component of resistance.

In contrast, the "humoral-myogenic" component of resistance (the difference between resistance after nerve section and resistance at maximal vasodilation expressed as percentage of resting resistance) was, if anything, reduced in the rats with coarctation hypertension. This finding suggests that the increased plasma angiotensin levels in the hypertensive rats with aortic coarctation did not directly contribute to the rise in limb resistance. Plasma renin activity was not measured in the rats with Goldblatt hypertension, so we are unable to comment on the contribution of angiotensin to the elevated hindlimb resistance in this form of hypertension.

The slightly but significantly greater magnitude of perfusion pressure, and, therefore, of residual resistance after maximal sodium nitroprusside vasodilation (the "structural" component of resistance) we observed in the hindlimbs of these rats with coarctation hypertension is especially intriguing. It is unlikely that this elevated residual resistance can be explained on the basis of increased passive vasoconstriction, because hindlimb perfusion pressure, and therefore transmural distending pressure, was higher in the rats with coarctation hypertension. This observation of impaired maximal vasodilation, in conjunction with the hyperresponsiveness to norepinephrine observed by Nolla-Panades, suggests that structural changes increasing wall-to-lumen ratio may have occurred in vessels of these hypertensive rats in the absence of increases in intravascular pressure or flow. Such structural changes in vessels in hypertension have been felt to be almost exclusively, if not exclusively, the result of elevated intravascular pressure or flow. This is because there is evidence that the magnitude of such wall thickening is directly proportional to the level of intravascular pressure. It has also been observed that such thickening may be prevented if the vascular bed is "protected" from the elevated arterial pressure by arterial ligation. Furthermore, there is evidence that vascular structural changes, once present, may regress if blood pressure is returned to normal levels. In the present investigation, the increase in this "structural" component of resistance was far greater in the Goldblatt than in the coarctation hypertensive rats, presumably reflecting the important causal role of elevated intravascular pressure.

On the other hand, there are also observations suggesting that additional factors may act in concert with intravascular pressure to produce structural changes in the vascular system in hypertension. For example, there is chemical evidence for structural vascular changes in young rats of the spontaneously hypertensive strain (SHR) before blood pressure rises. Folkow and co-investigators have reviewed homodynamic evidence suggesting that SHR may have a predisposition for vascular and cardiac wall thickening.

Assuming that the impaired maximal vasodilation we have observed, and the hyperresponsiveness Nolla-Panades observed, in the hindquarters of rats with coarctation hypertension does in fact reflect increases in vascular wall-to-lumen ratio, these structural changes can be attributed neither to a genetic predisposition nor to significant elevations in intravascular pressure. Instead they must be explained on the basis of abnormal neural or humoral stimuli. It has been suggested that adrenergic neurons may exert a trophic influence on vascular smooth muscle. Exposure to elevated levels of renin-angiotensin-aldosterone or other humoral factors might in some way alter the composition of vessel walls to increase wall-to-lumen ratio. Clearly, the influence of such systemic factors must be explored further.

The identification of factors other than pressure playing a role in the development of vascular structural changes in hypertension is most important, because it will provide additional insight into disease mechanisms and may eventually offer new therapeutic approaches.

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