Morphometric Analyses of Rabbit Thoracic Aorta After Poststenotic Dilatation

U. Kukongviriyapan and B.S. Gow

The aim of this study was to quantify the morphological changes in the arterial wall resulting from poststenotic dilatation (PSD). PSD was produced by placing a split nylon ring around the thoracic aorta of the rabbits at a level of T_{6.7} during a sterile thoracotomy done under pentobarbital anesthesia. After period of PSD ranging from 1–51 months these rabbits were anesthetized, as were the control animals, and the descending thoracic aorta from the fourth to the eleventh ribs was removed following perfusion fixation with Karnovsky’s solution at a constant pressure of 80 mm Hg. The extent of PSD development was variable even though the stenotic ring was the same size in all rabbits. Ultrastructural findings showed degenerative changes of the wall components in the PSD region and were more prominent in the aortas with greater dilatation. Morphometric measurements showed that the PSD was accompanied by a decrease in volume density of both smooth muscle cells (SMCs) and elastin and an increase in collagen and ground substance. These changes were well correlated with degree of dilatation and ratio of internal radius to wall thickness (hence, mean wall stress) but not with duration of PSD. While the number of SMCs per unit volume in the PSD aortas was significantly less than normal (p<0.05), there was no significant change in mean cell volume. Although the reduced muscle mass might be expected to lower the capacity of the vessel to maintain tone, previous results show that this does not occur. (Circulation Research 1989;65:1774–1786)

Poststenotic dilatation (PSD) is a fusiform swelling distal to an arterial stenosis. It has been generally accepted that the vibration of the arterial wall in vivo, produced by turbulent flow through the stenosis, leads to dilatation and alterations of wall structure,1–11 but this is still unproven. While the presence of turbulence and wall vibration is well established, there is little information about the morphological changes that accompany a PSD. Trillo and Haust12 showed that there were degenerative changes of the elastic elements with a concomitant increase of the intracellular fibrous tissue after 1–6-month periods of PSD in dog femoral and carotid arteries. However, these changes were not quantified nor were changes in smooth muscle cells (SMCs). Legg and Gow13 found that endothelial cells just distal to a stenosis had marked loss of normal longitudinal orientation and change in the shape of some cells from elongated to polygonal. In addition, Potter and Roach14 noted enlarged fenestrations in the internal elastic laminae.

Although morphometric methods have been widely used to quantify the changes of structural components of the vessel wall, especially in hypertensive arteries,15–19 to our knowledge, no such study has been carried out on arteries with PSD. We were, therefore, interested to use this methodology to quantify morphological changes occurring in the PSD region. In a preliminary report,20 it was shown that there were tunica media alterations in the PSD region of the stenosed rabbit thoracic aorta at 3 months and beyond, and the volume density, V_v, (volume per unit volume), of SMCs of the PSD was lower than normal. Because this earlier study showed that the mean tensile stress in the PSD region was greatly elevated and not matched by a compensatory increase in wall thickness as is seen in hypertension, it became the aim of the present study to support the earlier data and quantify the changes in connective tissues and in SMCs by use of morphometric analyses relating these changes to age, degree of dilatation, and mean wall stress.

Materials and Methods

Nineteen male New Zealand White rabbits (Castle Hill strain) were used in the present study. Six rabbits served as unoperated controls; the remain-
TABLE 1. Characteristics of Rabbits Studied Under Control Conditions and With Poststenotic Dilatation

<table>
<thead>
<tr>
<th>Number</th>
<th>Weight (kg)</th>
<th>Age (months)</th>
<th>Duration of PSD (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>6</td>
<td>2.90±0.06</td>
<td>6–18</td>
</tr>
<tr>
<td>PSD</td>
<td>13</td>
<td>2.99±0.08</td>
<td>7–57</td>
</tr>
</tbody>
</table>

Values are mean±SEM. PSD, poststenotic dilatation.

der comprised the stenosed group. The rabbits were anesthetized with sodium pentobarbital administered intravenously (Sagatal, May and Baker Australia, Melbourne, Australia; 30 mg/kg) and positively ventilated (1.5 l/min). Under aseptic conditions, the descending thoracic aorta was exposed through a left thoracotomy in the region of the sixth and seventh ribs. A split nylon ring (2.6 mm i.d., 2 mm long) was placed around the aorta midway between the sixth and seventh intercostal arteries. The bore of the ring was such that an approximate reduction of 70% in luminal diameter occurred. The overlying tissues were then sutured, and the pneumothorax was reduced. The animals were allowed to recover and were maintained on a stock pellet diet with water ad libitum. Poststenotic dilatation was usually observed downstream to the stenosis about 2 weeks after the stenotic ring placement. Age, weight, and period of PSD of the animals are summarized in Table 1.

Perfusion Fixation and Specimen Preparation for Microscopy

The rabbits were anesthetized, intubated, and positively ventilated at various times after the stenoses were produced. The descending thoracic aorta from the fourth to the eleventh ribs was exposed through a left thoracotomy, and the left intercostal arteries were ligated by ties around the ribs. A region of the aorta at the level of T4 and T11 was isolated for perfusion by double cannulation with stainless steel tubes (3.5 mm o.d., 3 mm i.d.). The isolated segment was flushed with Dulbecco’s physiological phosphate buffer solution for 2 minutes, followed by Karnovsky’s fixative, containing 2.5% glutaraldehyde and 2% paraformaldehyde in 0.1 M phosphate buffer. Perfusion was maintained at a physiological distending pressure of 80 mm Hg for 1 hour using techniques as previously described. Following perfusion, the aorta was excised and washed in three changes of Dulbecco’s solution, and embedded in Spurr’s resin. For light microscopy, 0.5-μm thick sections of the whole vessels were cut with a glass knife on a Reichert-Jung Ultracut E ultramicrotome. The sections were placed on glass slides, heat fixed, stained with 1% azur II and 1% toluidine blue in 1% aqueous borax, and counterstained with 0.01% aqueous safranin O. For electron microscopy, ultrathin transverse and longitudinal sections (60–70 nm) were cut with a diamond knife. The sections were stained with uranyl acetate and Reynolds’ lead citrate and then examined and photographed using a Philips EM 400-HMG electron microscope (Philips, Eindhoven, The Netherlands).

Morphometric Protocol

Morphometric determinations were carried out at both light and electron microscopic level. Since the accuracy is influenced by the amount of the section compression during microtomy, an average compression factor (Fc) was calculated from 10 different tissue blocks by dividing the length of the section by the original length of the uncut block face. The compression factor values for thick and thin sections were found to be 0.978±0.003 and 0.958±0.004 (mean±SEM), respectively. Uncompressed tissue areas obtained from light microscopic images and electron micrographs were derived by dividing the measured areas by corresponding Fc values.

Light microscopy. The circumference of each aortic ring was measured from 0.5-μm transverse sections with a computer-assisted, light microscopic image analyzer at x20 magnification. The distance around the external elastic lamina and the intima within the aortic ring were taken as the aortic and luminal circumferences, respectively. Aortic diameter and wall thickness, exclusive of the adventitia, of normal and PSD aortas were calculated from the compression-corrected circumferences, assuming a circular shape and the cross-sectional area of the wall (A) calculated as follows:

\[ A = \pi (R_1 - R_2)^2 \]

where \( R_1 \) and \( R_2 \) are the external and internal radii of the aorta, respectively.

The degree of dilatation (\( D^\circ \)) was obtained from changes in the external diameter of the aorta at the PSD region (\( D_1 \)) relative to the corresponding location in the normal aorta (\( D_2 \)).

\[ D^\circ(%) = 100(D_1 - D_2)/D_2 \]

Arterial wall stress (\( \sigma \)) was calculated by using the Lamé approximation,

\[ \sigma = P(D - 2h)/2h = PR_3/h \]

where \( P \) and \( h \) are the distending pressure and the wall thickness, respectively, at the time of perfusion fixation.

In addition, the number of elastic laminae throughout the entire wall for normal and PSD aortas was counted under high magnification.
amount of dilatation was not surprising in view of the very small variation in stenosis diameter (2.26–2.39 mm), the latter being expected considering that identical rings were used in all animals.

**Morphological Appearance**

The thoracic aortas of normal adult rabbits were examined from 6–18 months of age and had structural features typical of those reported previously. None of the normal aortas from which specimens were obtained presented recognizable abnormalities, except for the 18-month-old aorta, which, compared with the normal aorta (Figure 2A), showed an irregularity of the smooth muscle surface with numerous plasmalemmal vesicles (Figure 2B).

Changes in medial architecture, confirming previous studies, were observed in the PSD aortas by 3 months and beyond and were more prominent in the aortas with greater dilatation. Typical examples of the light microscopic appearance of the aortic wall from the two groups are shown in Figure 3. Medical thickness of PSD aortas (Figures 3B, 3C, and 3D) was less than that of the normal aorta (Figure 3A) and corresponded to a decreased space between adjacent elastic laminae. The most striking changes in the media of the aortas dilated for at least 3 months was a stretched appearance, a decrease in space occupied by SMCs and elastic laminae, and an increase in volume of collagen fibrils and ground substance, especially in those with the greatest dilatation (Figure 4B). Compared with the normal aorta, SMCs were extremely narrow and elongated (Figure 4). In addition, the outer surface of these cells was more irregular than normal with numerous projections. Compared with the normal aorta (Figure 5A) these projections as well as other regions were markedly rich in plasmalemmal vesicles (Figures 5B and 5C). As there was marked stretching of the arterial wall, some SMCs were broken up, the elastic laminae becoming thinner and fragmented. Also, some of the collagen fibrils had lost their fibrillar structure and had accumulated around the split regions of the elastic laminae, with the SMCs seemingly filling in the gaps in the intracellular space (Figure 6).

**Morphometric Analysis**

The amounts of the structural components within the medial layers of the thoracic aortas and the number and the volume of SMCs are shown in Table 3. The volume densities of the various wall components in the PSD aortas were significantly different from normal aortas. V of SMCs and elastic laminae was significantly decreased whereas V of collagen and ground substance was increased.

In the PSD aortas, the calculated number of SMCs per unit volume of aortic wall was significantly less than normal (p<0.05), but the mean SMC volume in PSD aortas did not change significantly, presumably because the decrease in V of SMCs here paralleled the decrease in number of the cells over the same period as demonstrated by the linear correlation (r=0.814, p<0.0001) between V and the number of SMCs in the medial layers of the PSD aortas (Figure 7). Interestingly, when the degree of dilatation was plotted against the V of SMCs (Figure 8A), elastic laminae (Figure 8B), and collagen and ground substance (Figure 8C), it was apparent that the decrease in V of SMCs and elastin of the PSD aortas with increasing dilatation was paralleled by increases in the relative amount of collagen and ground substance. The correlation coefficients for these plots were -0.898 (p<0.0001), -0.698 (p<0.005), and 0.856 (p<0.0005), respectively. In addition, it could also be seen that the reduction in V of SMCs is negatively correlated to mean wall stress (r=−0.931, p<0.00005) but not with duration of PSD as the data obtained from PSD of different durations are scattered (Figure 9). Previous studies in this laboratory (see Table 4)
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FIGURE 2. Transmission electron micrograph of normal thoracic aorta 6 (A) and 18 (B) months old. Notice the outer surface of the smooth muscle cells (s) in the older rabbit is more irregular with numerous plasmalemmal vesicles (arrows). ×19,000.
FIGURE 3. Light micrographs of representative transverse sections of the wall of the thoracic aorta from a control (A) and PSD after periods of 3 (B), 9 (C), and 36 (D) months. Notice the considerable decrease in the arterial wall thickness. X400.

showed only a small pressure drop across the stenosis (4.0%, 3.6%, and 5.9%, n=4, 10, and 13, respectively) that was much less than the increase in the ratio of internal radius to wall thickness (R₂/h) of the PSD in this study (30.5%); therefore, the Lamé wall stress, the product of mean pressure and R₂/h, would be greater in the PSD region in vivo. It appears that the degree of dilatation and the consequent elevated mean wall stress and not duration of the PSD is perhaps the determining factor in the reduction in Vₐ of SMCs.

Discussion

The aortic wall is a highly organized structure consisting of concentric fibromuscular layers or lamellar units. Between the elastic lamellae lie collagen fibers that, combined with elastin, provide resiliency and distribute tensile stress. The smooth muscle and ground substance within the interlamellar space in addition to these scleroproteins account for the complex time- and frequency-dependent behavior of the aortic wall. It has been suggested that the vibration associated with turbulence within the PSD weakens the structure and changes the elastic properties of elastin possibly by breaking down links between the collagen fibers so that the wall becomes more distensible. Just what causes the dilatation and its rapid reversal when a stenosis is removed remains unclear, but it seems reasonable to postulate that smooth muscle contraction is involved in the restoration of normal geometry particularly if the reversal takes less than 24 hours.

Our findings indicate that the amount of PSD is variable even though the stenosis diameters are essentially the same in all rabbits. This is consistent with the observation of Roach, who found that the amount of PSD was unrelated to the size of the stenosis. Presumably, differences in amount of dilatation relate to individual differences in flow and turbulence and the varying responses of the aortas to hemodynamic stress. A satisfactory answer to this question would require detailed hemodynamic studies on conscious animals with implanted instrumentation. Regression analysis showed that the severity of the dilatation had a strong negative correlation to the wall thickness. Although the thinning of the wall with dilatation is associated with the marked narrowing of the space between adjacent elastic laminae, the thinning is not significantly different from that predicted by the theoretical relation for an expanding cylinder having constant wall volume. As the wall becomes thinner with dilatation, the mean increase in the ratio of internal radius to wall thickness (Table 2) can be five times greater than that of the pressure drop across the stenosis (see Table 4). Therefore, one can conclude that the wall stress is elevated in the PSD aortas compared with normal aortas irrespective of what given distending pressure is used to calculate it. Because R₂/h increases predictably with degree of dilatation, the number of SMCs and the amount of elastin would also show strong negative correlation to wall stress. Moreover, it seems a reasonable hypothesis that the elevated mean wall stress is causal in the loss of SMCs and elastin and the gain in collagen and ground substance. The peculiar hemodynamic conditions associated with PSD are the most likely cause of the amount of dilatation and loss of the muscle cells. Although SMCs were particularly elongated in the PSD region, their volumes were normal. Whether the cells are
FIGURE 4. Transmission electron micrographs of a control (A) and PSD after periods of 3 (B), 6 (C), and 51 (D) months. Note the alternating layers of elastic laminae (e) and smooth muscle cells (s) in the media. Compared with normal smooth muscle cells (s), those in PSD rabbits tend to be narrow and elongated. It is apparent that the space occupied by smooth muscle cells (s) and elastic laminae (e) was decreased, while the amount of collagen fibrils (c) was markedly increased. ×3,100.
FIGURE 5. Transmission electron micrographs of 6-month-old normal (A) and PSD aortas with 9-month-old (B) and 36-month-old (C) stenosis. Note the outer surface of the smooth muscle cells (s) is more irregular than normal with numerous branched processes and plasmalemmal vesicles (arrows). $\times 31,800$. 
Figure 6. Transmission electron micrographs of normal (A) and PSD aortas with the greatest dilatation after periods of 3 (B) and 9 (C) months. Note the collagen fibrils (c) accumulate between the split regions of the elastic laminae (e) and are increased around the smooth muscle cells (s) (arrows). x5,000.

Lengthened in exact proportion to the increase in circumference cannot be ascertained unless their lengths are measured. However, observations in this laboratory clearly show these cells collectively are able to generate comparable forces to normal ones despite their reduced numbers. Surprisingly, there is a lack of cellular hypertrophy in response to elevated wall stress as is seen in chronic hypertension. The reason for the apparent failure of the PSD region to follow this adaptive response is unclear, but one possibility is that vibration interferes with cellular metabolism. Alternatively, while
TABLE 3. Volume Densities of the Components in Aortic Tunica Media, Number of Smooth Muscle Cells, and Cell Volume of Normal and Poststenotic Dilatation Aortas

<table>
<thead>
<tr>
<th>Component</th>
<th>Normal</th>
<th>PSD</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volume density (μm²/μm³ medial wall)</td>
<td>0.390±0.008</td>
<td>0.359±0.011</td>
<td>*</td>
</tr>
<tr>
<td>SMC</td>
<td>0.023±0.003</td>
<td>0.013±0.001</td>
<td>†</td>
</tr>
<tr>
<td>Nucleus</td>
<td>0.367±0.007</td>
<td>0.346±0.011</td>
<td>NS</td>
</tr>
<tr>
<td>Cytoplasm</td>
<td>0.389±0.007</td>
<td>0.348±0.011</td>
<td>‡</td>
</tr>
<tr>
<td>Elastic laminae</td>
<td>0.221±0.003</td>
<td>0.291±0.020</td>
<td>*</td>
</tr>
<tr>
<td>Collagen and ground substance</td>
<td>3.073±0.156</td>
<td>2.333±0.089</td>
<td>†</td>
</tr>
<tr>
<td>Number of nuclear profiles x 10⁻³/μm²</td>
<td>1.550±0.013</td>
<td>1.544±0.010</td>
<td>NS</td>
</tr>
<tr>
<td>Shape coefficients of nuclei for ellipsoidal shape</td>
<td>7.271±0.266</td>
<td>6.519±0.178</td>
<td>*</td>
</tr>
<tr>
<td>Number of nuclei (SMCs) x 10⁹/mm³</td>
<td>539.380±13.440</td>
<td>551.980±9.860</td>
<td>NS</td>
</tr>
<tr>
<td>Mean smooth muscle volume (μm³)</td>
<td>539.380±13.440</td>
<td>551.980±9.860</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are mean±SEM. PSD, poststenotic dilatation; SMC, smooth muscle cells.

p values determined by t test are denoted as follows: *p<0.05; †p<0.001; ‡p<0.01. NS, not significant.

mean wall stress is elevated in PSD, pulsatile pressures, and with them pulsatile stresses, are attenuated. It is not unlikely that the combination of reduced pulsatile stress combined with elevated mean stress provides an inadequate stimulus for cell hypertrophy.

There seems little doubt that PSD damages the arterial wall. The alterations in the media described above confirm the findings in canine muscular arteries by Trillo and Haust, and our morphometric measurements show that these changes were associated with the degree of dilatation and not with age of the PSD. Of the multiple suggestions put forward to explain the genesis of PSD it seems probable that turbulence, aortic vibration, and elevated mean wall stress are responsible for the alterations in the vascular wall and eventually lead to the structural fatigue of the arterial mural components, causing the breakdown in elastic fibers and loss of SMCs. As vibration has never been demonstrated to produce dilatation comparable to a PSD in vivo, it could be that endothelium derived relaxing factor or some other substance is involved in PSD as the result of the disturbed hemodynamic conditions. Just what causes the initial weakening and subsequent dilatation is unclear, but once the vessel has dilated, the increased mean wall stress can be assumed to overload both active and passive ele-

TABLE 4. Comparison of Blood Pressure Proximal and Distal to a Stenosis From Separate Studies in This Laboratory

<table>
<thead>
<tr>
<th>Reference</th>
<th>Number</th>
<th>MAP (mmHg)</th>
<th>Reference</th>
<th>Number</th>
<th>MAP (mmHg)</th>
<th>Reference</th>
<th>Number</th>
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</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>4</td>
<td>113.50±11.27</td>
<td>31</td>
<td>10</td>
<td>103.50±6.95</td>
<td>32</td>
<td>13</td>
<td>107.54±14.29</td>
</tr>
<tr>
<td>13</td>
<td>4</td>
<td>109.00±11.02</td>
<td></td>
<td></td>
<td></td>
<td>31</td>
<td>10</td>
<td>99.80±7.59</td>
</tr>
<tr>
<td>32</td>
<td>13</td>
<td>101.15±13.69</td>
<td>31</td>
<td>10</td>
<td>99.80±7.59</td>
<td>32</td>
<td>13</td>
<td>101.15±13.69</td>
</tr>
</tbody>
</table>

Values are mean±SD. MAP, Mean arterial pressure.

FIGURE 7. Plot of relation between volume density of smooth muscle cells and number of smooth muscle cells in the medial layers of the PSD aortas.
FIGURE 8. Plot of relation between degree of dilatation and volume density of smooth muscle cells (A), elastic laminae (B), and collagen and ground substance (C).
ments leading to their failure. It is apparent that fragmenting elastic laminae and lost SMCs are replaced with collagen and could be considered an accelerated aging phenomenon induced by abnormal wall stresses.

While a number of authors have suggested that PSD can progress to be an aneurysm and produce vascular complications, Roach reported that PSD is reversible after removal of the stenosis, suggesting that PSD is different from an aneurysm. However, both PSD and aneurysmal dilatation are subject to elevated tensile mural stress and possibly share in common the phenomenon of degenerative change. Interestingly, Zarins et al. reported that an increase in collagenase activity is an early change in the PSD region, preceding the pronounced changes in the vessel diameter and before significant modifications of matrix fiber content. It is possible that the increase in collagen and ground substance content is in response to the changes in enzyme activity stimulated by one or more factors such as turbulence, aortic vibration, and elevated mean wall stress.

**Acknowledgments**

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**References**

18. Olivetti G, Melissa M, Marchetti G, Anversa P: Quantitative structural changes of the rat thoracic aorta in early spontaneous hypertension: Tissue composition, and hyper-

**Figure 9. Plot of relation between aortic wall stress (calculated at fixation pressure of 80 mm Hg) and volume density of smooth muscle cells of PSD aortas at different times after the stenotic rings were applied.**

Key Words • aortic wall stress • smooth muscle cell • elastin • collagen
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