Effect of Pressure on Aortic Hydraulic Conductance

Ann L. Baldwin, Lisa M. Wilson, and Bruce R. Simon

This study was performed to determine whether the transmural hydraulic conductance ($L_p$) of the rabbit aortic wall depends on its distension. In 19 rabbits, the aorta was cannulated in situ and perfused at a given pressure with a physiologically buffered solution containing 4% bovine serum albumin. The output cannula was then occluded to limit fluid flow to that traversing the artery wall. External diameter and transmural fluid flow were measured at three pressures (eight rabbits, group 1) or at four pressures (12 rabbits, group 2) in each vessel. Transmural fluid flow was determined by monitoring the velocity of an air bubble within a buffer-filled tube leading to the input cannula. From group 1 measurements, $L_p$ values (mean ± SD) at 50, 100, and 150 mm Hg were calculated to be 3.8 ± 2.8, 3.5 ± 1.3, and 4.1 ± 1.2 x 10^{-8} cm/sec/mm Hg, respectively. Group 2 measurements gave values of 4.2 ± 1.6, 3.8 ± 1.1, 3.8 ± 1.1, and 4.2 ± 1.1 x 10^{-8} cm/sec/mm Hg at 75, 100, 125, and 150 mm Hg, respectively. Paired Student's $t$ tests indicated no significant change in $L_p$ with pressure. However, linear regression analysis demonstrated a weak correlation between $L_p$ values obtained at 50 and 100 mm Hg ($r^2=0.30$) and at 75 and 100 mm Hg ($r^2=0.36$). Values of $L_p$ at 100 and 150 mm Hg and at 125 and 150 mm Hg were closely correlated in each case. These results suggest that between 50 and 100 mm Hg the structural properties of the aortic wall change so as to alter $L_p$ but not in the same way in each vessel. $L_p$ may increase or decrease depending on which structural change predominates in a particular vessel. (Arteriosclerosis and Thrombosis 1992;12:163–171)

The goal of this study was to quantitatively determine how the changes in arterial mechanical strain caused by distension influence the transport of water through the arterial wall. Many factors to which arteries are exposed in vivo, such as elevated blood pressure and changes in smooth muscle tension, alter the distension and hence the mechanical strain within the arterial wall. Aortic distension significantly alters the morphology of the endothelium and the structural configuration of the media. Thus, it is likely that distension also affects the vascular transmural hydraulic conductance ($L_p$).

It is important to investigate the details of convective fluid motion through the walls of the aorta because convection promotes the transport of macromolecules such as lipoproteins into the artery wall, possibly through leaky endothelial cell junctions in regions of high endothelial cell turnover. Such transport is increased by increasing the transmural pressure. Schwenke and Carew demonstrated that only if an increased low density lipoprotein (LDL) influx into the intima is accompanied by a reduced LDL efflux from the inner layers of the artery wall does LDL tend to accumulate in the intima. Thus, if an increase in arterial pressure altered the configuration of the aortic media such that its sieving properties were enhanced, it is possible that lipoproteins would accumulate within the intima. Excessive lipoprotein accumulation is one of the first stages of arteriosclerosis, and so study of the mechanisms that govern this process might help in the prevention and control of this disease.

Measurements of the $L_p$ of large arteries have been made by several authors. In four of these studies, two with the aorta and two with the common carotid artery, the effects that possible vessel distensibility would have on the values of $L_p$ obtained were investigated by the following procedure. The artery was prepared for perfusion, mounted on a rig, and pressurized through one cannula that was connected to a capillary manometer and saline reservoir while the outlet cannula was closed. The amount of extra saline entering the vessel to replace the fluid...
lost by transmural filtration and to accommodate arterial distension was indicated by the manometer meniscus shift. After each pressurization, sufficient time was allowed for distensibility and wall consolidation at the new pressure to occur so that the manometer meniscus shift resulted only from transmural filtration and could then be used to calculate transmural \( L_p \). The studies of the aorta described above provided valid estimates of arterial \( L_p \) but not over a wide range of hydrostatic pressures in a single vessel.

Tedgui and Lever measured \( L_p \) at two different pressures but not in the same aorta. Their data suggest that \( L_p \) may be a function of the strain within the interstitial matrix, but their data are complicated by the variation between vessels. In the present study, we designed experiments in which \( L_p \) could be measured in the same aorta at different pressures so that we could determine unequivocally whether \( L_p \) was influenced by vascular distension and hence, by mechanical strain.

**Methods**

**Surgical Procedure**

Experiments were performed on 24 male New Zealand White rabbits (2.0–2.5 kg) anesthetized with pentobarbital sodium (30 mg/kg i.v.). The trachea was intubated, and the lungs were mechanically ventilated. The abdomen was opened by a midline incision, and the sternum was split longitudinally to the level of the third costochondral junction. Next the aorta was exposed, and the fascia surrounding the vessel, from the origin of the innominate artery to the origin of the celiac artery, was carefully dissected away. Then the pairs of intercostal arteries and veins within this region were ligated close to the aortic wall. Heparin (1,000 IU) was administered intravenously. During the entire operative procedure, physiologically buffered saline solution (PBS) containing 4% bovine serum albumin (BSA), with pH adjusted to 7.4, was applied continually to the surface of the aorta to prevent drying.

A technique that has been described previously was used to cannulate the aorta without depressurizing it, thus avoiding endothelial damage. In brief, a 16-gauge needle, connected via a stopcock to a pressure transducer and reservoir containing PBS at pH 7.4 and 37°C that was 80 cm above the rabbit, was quickly inserted retrogradely into the aorta at the distal site and secured. The needle was tied in place, a second ligature was tied around the proximal distal site and secured. As the needle was tied in place, a second ligature was tied around the proximal site, distal to the ligature at the arch, and secured. The heights of the upstream and downstream reservoirs were adjusted to 100 cm and 30 cm, respectively, and the screw clamps were opened to allow the blood to be flushed from the segment. The flow rate was approximately 1 ml/sec corresponding to an estimated wall shear stress of 0.1 dyne/cm².

Inclusion of Trypan blue dye in the perfusate allowed a visual check that ensured that all branches had been ligated. The rabbit was then killed with an overdose of anesthetic. When the inner surfaces of vessels treated in this way were later examined with a light microscope, no staining of endothelial cell nuclei with Trypan blue was seen, indicating that the endothelium was intact. After establishing the absence of leaks, the body cavity was filled with the solution that had been used to keep the vessel moist until the aorta was completely submerged. Both the perfusate and the suffusate solutions contained \( 10^{-3} \) M NaNO₃ to relax the arterial smooth muscle cells, eliminate any active strain from the arterial wall, and thus facilitate interpretation of the data obtained.

In initial experiments, 2% rather than 4% BSA was used in the suffusate. However, in four experiments in which we repeated measurements of \( L_p \) four or five times on the same vessel at the same pressure to test for reproducibility, the value of \( L_p \) decreased with each repetition. One possible explanation for this response was that the vessels gradually became hydrated due to the lower colloid osmotic pressure of the suffusate compared with that of the perfusate. Such hydration would cause tissue swelling and hence an increase in arterial wall thickness. When these experiments were repeated with 4% albumin in both solutions, the values obtained for \( L_p \) were reproducible. For this reason, the modified suffusate was used in all experiments contributing to this study.

**Pressurization Experiments**

The first step in the pressurization experiments was to precondition the arteries to reduce the hysteresis effects resulting from viscoelasticity (i.e., the tendency for the vessel to return to a diameter greater than its initial value after pressurization). This was achieved by lowering the aortic pressure to 25 mm Hg, raising it to 150 mm Hg, and repeating this process three times. Pressurization of the vessel was achieved by inflating a cuff around the inlet reservoir (an i.v. bag) and closing the vessel off from the outlet reservoir by turning the stopcock. This stopcock connected the aorta to a pressure transducer, and the pressure was recorded with a Gould chart recorder (Gould Inc., Cleveland, Ohio). Preliminary experiments in which the aortic external diameter was measured with mechanical calipers (accurate to 0.1 mm) at chosen pressures within the above range demonstrated that after such preconditioning, the aorta assumed a constant diameter at a given pressure.

After preconditioning, measurements of the external radius and transmural fluid flux were made at 50, 100,
where \( r \) is the radius of the capillary tubing. \( I_p \) was determined by dividing this flux by the product of the transmural pressure and the surface area of the aortic segment, which was determined from dimensional measurements, assuming cylindrical geometry.

To establish that bubble movement accurately reflected fluid flux, the following experiment was performed. A bubble was introduced into the capillary tubing, the end of which was connected to a pulled-glass micropipette rather than to the aorta. The position of the front edge of the bubble was noted. The input pressure was then raised from 25 to 50 mm Hg, and the perfusate issuing from the pipette tip was collected in a preweighed beaker that was covered with parafilm to prevent evaporation. After 1 or 5 minutes, the final position of the bubble was noted and the beaker was weighed. The volume of fluid collected was determined from the measured weight gain and was also calculated from the distance moved by the bubble, assuming a capillary tube of uniform internal diameter. This procedure was performed with three different pipette tips, which resulted in flow rates of 0.05, 0.10, and 0.57 ml/min. In all cases, the measured and calculated perfusate volumes agreed with each other to within 1%.

In initial experiments when \( I_p \) was measured at 50 mm Hg, the low transmural convective fluxes often produced at this pressure sometimes resulted in such slow movement of the bubble that it became caught on irregularities on the inside surface of the tubing. In such cases, bubble movement did not reflect true transmural convective flux and \( I_p \) was underestimated. However, if the tubing was previously coated with Sigmacote (Sigma Chemical Co., St. Louis, Mo.) by perfusing the tubing with Sigmacote and then with air, sticking of the bubble rarely occurred. For this reason, all data reported in the present article were obtained with Sigmacote-coated tubing.

**Light- and Electron-Microscopic Observations**

Two of the vessels for which measurements of \( I_p \) had been made, as described above, were perfusion-fixed at room temperature for 2 hours at 150 mm Hg with phosphate-buffered Karnovsky’s fixative, pH 7.4, containing enough dextran 40 to bring the colloid osmotic pressure to that of plasma (25 mm Hg). Fixative was also poured into the body cavity so that the aorta was covered. Each vessel was then excised and cut into annular segments about 1 cm long. The segments were washed in 0.15 M phosphate buffer, postfixed in 1% OsO₄ for 2 hours, dehydrated in alcohols, embedded in Spurrs resin, and sectioned for light microscopy. Sections from three segments of each vessel were examined by light microscopy to visualize how the vessel ultrastructure changed with pressure.

**Statistics**

To determine whether \( I_p \) varied with aortic transmural pressure, the values of \( I_p \) obtained at two different pressures were compared by paired Student’s \( t \) tests. Group 1 and group 2 data were treated
separately. Sufficient tests were applied so that the $L_p$ values obtained at a given pressure were compared with those obtained at all other pressures. If a $t$ test gave $p<0.05$, we considered the null hypothesis to be rejected. To determine the correlation between $L_p$ values obtained at two different pressures in the same vessel, linear regression analysis was performed on the data. Such analysis indicated whether $L_p$ was influenced by the same arterial properties at one pressure as at another pressure.

Results

Light- and Electron-Microscopic Observations

Transverse sections taken from two aortas that had been subjected to the protocol for measuring $L_p$ at different pressures did not show any signs of endothelial damage. An example of such a section is shown in Figure 1. These observations, by necessity, were performed only on a tiny fraction of the total endothelial surface of each vessel. However, it is unlikely that the endothelium was damaged elsewhere on the aortic surface because neither these vessels nor any of the other vessels included in this study showed staining of endothelial nuclei by the vital dye, Trypan blue, when viewed en face.

Transverse sections from the middle segment of aortas fixed at 50, 100, and 150 mm Hg (four at each pressure) were examined by light and electron microscopy to visualize how the arterial wall architecture changed with distending pressure. The mean wall thickness at each pressure was obtained by making 10 measurements at different positions on a single annulus of each vessel and averaging the pooled results. The mean wall thickness of aortas fixed at 50 mm Hg (125±12 μm, $n=40$) significantly exceeded that of vessels fixed at 100 mm Hg (100±14 μm, $n=40$), but no further thinning was noted at 150 mm Hg. Typical electron photomicrographs from vessels fixed at 50, 100, and 150 mm Hg are shown in Figure 2. In aortas fixed at 50 mm Hg, the internal elastic lamella was sometimes convoluted (see Figure 2, upper left panel), and in vessels fixed at 50 and 100 mm Hg, the intimal lamellae were often convoluted when present. In aortas fixed at 150 mm Hg, all lamellae were always straight.

Pressure–Diameter Relation

The pressure–diameter curves that we obtained from our aortic preparations during the preconditioning phase were similar to those obtained by other investigators. A typical example is shown in Figure 3. This figure demonstrates that the vessel is most distensible between 25 and 75 mm Hg and becomes stiffer at higher pressures. In initial studies to check that the aorta retained its in vivo mechanical properties throughout the experiment, comparisons were made between pressure–diameter curves obtained just after cannulation and those obtained 3 hours later. In all cases, the pairs of curves were almost identical.

Measurement of Hydraulic Conductance

To determine $L_p$ for a given pressure in a given vessel, a plot was constructed of the distance traveled by the bubble as a function of time. The plot was examined by eye to determine when it became linear (i.e., when the bubble had reached a constant velocity). A regression analysis was then applied to the data contributing to the linear part of the plot to determine its slope and hence, the velocity of the bubble. The fact that such regression analyses always demonstrated an excellent linear fit ($r^2>0.99$ in most cases) is evidence that our visual assessment of the time taken to achieve linearity was accurate. In these experiments, the mean time taken for the air bubble to attain a constant velocity and hence for the aorta to complete its distension after an increase in transmural pressure was 6.6±3.7 minutes (mean±SD, $n=24$). This distension time period was independent of pressure. A typical time course for bubble movement is shown in Figure 4. In this case, linearity was achieved by 4 minutes.
An initial experiment was performed to determine the experimental error in measuring $L_p$ at a given pressure (100 mm Hg) in the same vessel. The values of $L_p$ obtained were 4.151, 3.097, 4.499, and $4.129 \times 10^{-8}$ cm/sec/mm Hg, and the standard deviation as a percentage of the mean was 13.2%. A similar experiment was performed at 50 mm Hg. The $L_p$ values obtained were 3.264, 2.573, 2.686, 4.042, and $3.782 \times 10^{-8}$, with a standard deviation of 16.7%.

In two initial experiments to check that the transport properties of the aorta did not change during the experimental procedure, $L_p$ measurements were made at 50, 100, and 150 mm Hg. The pressure was then reduced to 25 mm Hg and raised to 50 mm Hg for the repeat measurement of $L_p$. The air bubble first traveled forward due to the aortic distension resulting from the pressure increase, but after 4 or 5 minutes the bubble stopped moving. During the next 15 minutes, the bubble remained stationary and the aorta became edematous, as judged by appearance and by touch (i.e., puffy and swollen). Next the vessel diameter decreased, the bubble proceeded backward, and the vessel no longer appeared edematous. After 10 minutes, the bubble reversed direction and traveled forward in the normal manner.

In subsequent "repeatability" experiments after the first set of $L_p$ measurements had been made, the...
pressure was lowered and the vessel was left for 30
minutes to adjust to the new set of mechanical
stresses before repeating the measurements. When
this procedure was followed, the bubble traveled
forward in the normal way when the pressure was
raised and soon attained a constant velocity. The
results of these experiments are shown in Table 1. In
all cases except one, the difference between the two
$L_p$ measurements obtained at a given pressure was
within the experimental error. These results show
that the transport properties of the aortic prepara-
tion were not altered by the experimental procedure
and demonstrate the reproducibility of $L_p$ data ob-
tained using the in situ perfusion technique.

Measurements of $L_p$ were obtained from eight ves-
sels at pressures of 50, 100, and 150 mm Hg (group 1).
The results are shown in Table 2 and graphically in
Figure 5. Mean values of $L_p$ obtained at 50, 100,
and 150 mm Hg were 3.8±2.8, 3.5±1.3, and 4.1±1.2x10^{-8}
cm/sec/mm Hg, respectively (mean±SD). Note the rel-
atively large variation in $L_p$ at 50 mm Hg compared with
the variation at 100 and 150 mm Hg. Group 2 measure-
ments are shown in Table 3 and graphically in Figure 6.
Mean values of $L_p$ obtained at 75, 100, 125, and 150
mm Hg were 4.2±1.6, 3.8±1.1, 3.8±1.1, and 4.2±1.1x10^{-8}
respectively. Examination of Figure 6 reveals the same trend as shown in
Figure 5, that is, an increased variation in $L_p$ at the
lowest pressure. Paired Student's $t$ tests performed on

both sets of data indicated no significant change in
mean $L_p$ with pressure.

Linear regression analysis demonstrated a positive
correlation between $L_p$ values (group 1) obtained at
100 mm Hg and 150 mm Hg (Figure 7, left panel).
When $L_p$ at 150 mm Hg was used as the independent
variable, a regression line with a slope of $0.82±0.28$
(mean±SEM) was obtained (dashed line) and when
$L_p$ at 100 mm Hg was used as the independent
variable, the slope was $1.37±0.46$ (dotted line). The
square of the coefficient of correlation ($r^2$) was 0.60,
which gives a $t$ value of 3.0. Thus, the correlation
between $L_p$ at 100 mm Hg and $L_p$ at 150 mm Hg was
significant ($p<0.05$). Very little correlation was seen
between $L_p$ values (group 1) obtained at 50 mm Hg
and 100 mm Hg (Figure 7, right panel). When $L_p$
at 100 mm Hg was used as the independent variable,
the slope of the line of regression was $-1.19±0.75$
(dotted line), and when $L_p$ at 50 mm Hg was used as
the independent variable, the slope was $-4.00±2.51$
(dashed line). The square of coefficient of correlation
was 0.30, which gives a $t$ value of 1.6, and hence an
insignificant correlation at the 0.05 level. These re-

### Table 1. Reproducibility of Hydraulic Conductance Measurements at Different Pressures

<table>
<thead>
<tr>
<th>Rabbit Set</th>
<th>Pressure (mm Hg)</th>
<th>75</th>
<th>100</th>
<th>125</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 First</td>
<td></td>
<td>6.4</td>
<td>5.2</td>
<td>5.3</td>
</tr>
<tr>
<td>2 First</td>
<td></td>
<td>3.1</td>
<td>2.7</td>
<td>2.8</td>
</tr>
<tr>
<td>1 Second</td>
<td></td>
<td>6.9</td>
<td>6.3</td>
<td>5.9</td>
</tr>
<tr>
<td>2 Second</td>
<td></td>
<td>3.3</td>
<td>2.9</td>
<td>3.0</td>
</tr>
</tbody>
</table>

Units of hydraulic conductance are $10^{-8}$ cm/sec/mm Hg.

### Table 2. Individual Values of Hydraulic Conductance (Group 1)

<table>
<thead>
<tr>
<th>Pressure (mm Hg)</th>
<th>50</th>
<th>100</th>
<th>150</th>
</tr>
</thead>
<tbody>
<tr>
<td>8.5</td>
<td>2.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7.6</td>
<td>3.5</td>
<td>5.2</td>
<td></td>
</tr>
<tr>
<td>6.1</td>
<td>1.9</td>
<td>2.9</td>
<td></td>
</tr>
<tr>
<td>2.5</td>
<td>3.1</td>
<td>4.6</td>
<td></td>
</tr>
<tr>
<td>2.0</td>
<td>2.5</td>
<td>3.7</td>
<td></td>
</tr>
<tr>
<td>1.6</td>
<td>5.9</td>
<td>5.4</td>
<td></td>
</tr>
<tr>
<td>1.4</td>
<td>5.2</td>
<td>5.8</td>
<td></td>
</tr>
<tr>
<td>1.1</td>
<td>3.6</td>
<td>2.9</td>
<td></td>
</tr>
</tbody>
</table>

Units of hydraulic conductance are $10^{-8}$ cm/sec/mm Hg. Values are arranged in order of decreasing hydraulic conductance at 50 mm Hg.

### Table 3. Individual Values of Hydraulic Conductance (Group 2)

<table>
<thead>
<tr>
<th>Pressure (mm Hg)</th>
<th>75</th>
<th>100</th>
<th>125</th>
<th>150</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.4</td>
<td>6.3</td>
<td>5.8</td>
<td>5.7</td>
<td></td>
</tr>
<tr>
<td>5.8</td>
<td>4.0</td>
<td>4.8</td>
<td>5.5</td>
<td></td>
</tr>
<tr>
<td>5.6</td>
<td>5.0</td>
<td>4.0</td>
<td>4.8</td>
<td></td>
</tr>
<tr>
<td>5.2</td>
<td>3.8</td>
<td>4.5</td>
<td>4.6</td>
<td></td>
</tr>
<tr>
<td>4.8</td>
<td>2.5</td>
<td>3.2</td>
<td>3.8</td>
<td></td>
</tr>
<tr>
<td>4.6</td>
<td>2.8</td>
<td>3.0</td>
<td>3.5</td>
<td></td>
</tr>
<tr>
<td>3.9</td>
<td>3.9</td>
<td>4.0</td>
<td>4.9</td>
<td></td>
</tr>
<tr>
<td>3.7</td>
<td>5.2</td>
<td>5.2</td>
<td>5.2</td>
<td></td>
</tr>
<tr>
<td>3.1</td>
<td>2.7</td>
<td>2.8</td>
<td>2.6</td>
<td></td>
</tr>
<tr>
<td>2.4</td>
<td>2.9</td>
<td>3.4</td>
<td>4.8</td>
<td></td>
</tr>
<tr>
<td>2.2</td>
<td>3.0</td>
<td>2.4</td>
<td>2.3</td>
<td></td>
</tr>
<tr>
<td>1.8</td>
<td>3.6</td>
<td>2.1</td>
<td>2.9</td>
<td></td>
</tr>
</tbody>
</table>

Units of hydraulic conductance are $10^{-8}$ cm/sec/mm Hg. Values are arranged in order of decreasing hydraulic conductance at 75 mm Hg.
results imply that for a given vessel, $L_p$ at 50 mm Hg is not a good predictor of its value at 100 mm Hg, but $L_p$ at 100 mm Hg is a good predictor of its value at 150 mm Hg.

Linear regression analysis when applied to group 2 data revealed a similar trend: $L_p$ values at 125 and 150 mm Hg showed a high correlation ($r^2=0.82$, $t=6.7$, $p<0.05$), and $L_p$ values at 75 and 100 mm Hg were not so well correlated ($r^2=0.36$, $t=2.4$, $p<0.05$). These results are presented graphically in Figure 8.

Discussion

This study has provided some new information regarding the effect of pressure-induced distension on convective water flux through the wall of the in situ-perfused rabbit aorta. First, the mean values of $L_p$ obtained at pressures of 50, 75, 100, 125, and 150 mm Hg do not differ significantly from each other according to the paired Student's $t$ test. In light of the changes in arterial wall thickness and in endothelial and medial morphology that are known to occur over this pressure range, this result is surprising. Tedgui and Lever, who used an in vitro preparation, found that $L_p$ was lower at 180 mm Hg than at 70 mm Hg. However, only one pressure was used for each vessel. Because there is a high degree of vessel-to-vessel variability in $L_p$, particularly at low pressure, these previous results do not provide definitive data on the variation of $L_p$ with transmural pressure.

Second, closer examination of the $L_p$ data obtained from the present study revealed that although the mean $L_p$ did not change significantly within the pressure range of 50–150 mm Hg, the individual $L_p$ values obtained at 50 and 75 mm Hg showed a much wider variation than did the values obtained at 100, 125, and 150 mm Hg. This trend was further demonstrated by the fact that the individual $L_p$ values obtained at 50 mm Hg were poorly correlated with those obtained at 100 mm Hg, whereas individual values obtained at 100 and 150 mm Hg were well correlated. The changes in $L_p$ produced by raising the pressure from 50 to 100 mm Hg were not systematic (see Figure 5); in some cases, $L_p$ was increased and in others it was decreased. This is probably the reason why these differences were not demonstrated by applying a paired Student's $t$ test.

In physical terms, the above result indicates that the parameters that may govern transmural flow, for example, endothelial and interstitial morphology and medial thickness, change with pressure in such a way that their combined effect on $L_p$ is to alter it in an as-yet-unpredictable way between 50 and 100 mm Hg but to leave it relatively unchanged between 100 and 150 mm Hg. In some vessels, changes in medial thickness with pressure may dominate the way in which $L_p$ changes between 50 and 100 mm Hg. In other vessels, changes in endothelial morphology, interstitial morphology, or in the relative contributions of the endothelium and media to flow resistance may dominate the response of $L_p$ to changes in pressure. Changes in endothelial and smooth muscle cell morphology with pressure have been measured and reported in a previous study. Both types of cells became significantly thinner and wider as pressure was increased. Experiments are presently being conducted to determine whether the varied response of $L_p$ to a pressure change from 50 to 100 mm Hg is caused by differences in the response of the endothelium or of the arterial wall as a whole. Preliminary results indicate that the variation in the pressure response of $L_p$ is reduced when the endothelium is removed.

The similarity of $L_p$ at pressures of 100, 125, and 150 mm Hg in individual vessels is unlikely to arise from the constancy of the parameters affecting transmural flow within this pressure range. Changes in medial thickness, convolution of intimal elastic lamellae, and endothelial junction length (as seen in transverse section) have all been reported to occur between 100 and 150 mm Hg. It is more likely that the various factors that contribute to $L_p$ are affected by pressure in different ways such that the combined effect on $L_p$ is negligible within this pressure range.
Further investigation may reveal pressure dependence of $L_p$ outside the physiological range.

Apart from providing new information concerning the effect of pressure on arterial hydraulic conductance, this study has also demonstrated an interesting phenomenon regarding arterial mechanics, that is, the time required for an aorta in situ to adjust to a new set of mechanical stresses produced by changing transmural pressure depends on whether the pressure is increased or decreased. An increase in pressure results in a fast response (about 6 minutes), and a decrease in pressure a slow response (30–40 minutes). Further data and perhaps theoretical modeling are needed before we can explain this finding in terms of changes in fluid distribution and pressure gradient across the artery wall. Tedgui and Lever17 reported a response time of 30–60 minutes for their in vitro preparation of the rabbit aorta, but they did not specify whether the pressure was raised or lowered.

In summary, this study has shown that although at first sight aortic $L_p$ seems to be independent of pressure within a physiological range, $L_p$ does in fact depend in a complex way on changes that occur in arterial morphology with pressure. Further studies are planned to determine the contributions of differences in endothelial permeability, vessel distensibility.
ity, and porosity to the varied response of $L_p$ to pressure changes between vessels.

References


Key Words • transmural pressure • transmural strain • hydraulic conductance • endothelium • media • rabbit aorta
Effect of pressure on aortic hydraulic conductance.
A L Baldwin, L M Wilson and B R Simon

doi: 10.1161/01.ATV.12.2.163

*Arteriosclerosis, Thrombosis, and Vascular Biology* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1992 American Heart Association, Inc. All rights reserved.
Print ISSN: 1079-5642. Online ISSN: 1524-4636

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://atvb.ahajournals.org/content/12/2/163

**Permissions:** Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Arteriosclerosis, Thrombosis, and Vascular Biology* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

**Reprints:** Information about reprints can be found online at:
http://www.lww.com/reprints

**Subscriptions:** Information about subscribing to *Arteriosclerosis, Thrombosis, and Vascular Biology* is online at:
http://atvb.ahajournals.org//subscriptions/