Influence of vascular smooth muscle on contractile mechanics and elasticity of arteries

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DOBRIN, PHILIP B., AND ALLEN A. ROVICK. Influence of vascular smooth muscle on contractile mechanics and elasticity of arteries. Am. J. Physiol. 217(6): 1644-1651. 1969.—Segments of canine carotid artery were held at in situ length and studied in vitro after treatment with norepinephrine (NEp) and after treatment with potassium cyanide (KCN). Activation of the vascular muscle caused contraction and greatly increased pressure-radius hysteresis. The blood vessel muscle exhibited an active stress-strain curve with a maximum stress of \(0.88 \pm 0.10 \times 10^6\) dynes/cm\(^2\) for the whole wall, or \(2.73 \times 10^6\) dynes/cm\(^2\) for the estimated smooth muscle component of the wall. Activation of smooth muscle increased the elastic modulus when this was plotted as a function of strain. The active muscle exhibited a maximum elastic modulus of \(3.99 \pm 0.06 \times 10^6\) dynes/cm\(^2\) for the whole wall, or \(12.66 \times 10^6\) dynes/cm\(^2\) for the estimated smooth muscle component of the wall. Activation of smooth muscle decreased the elastic modulus when this was plotted as a function of pressure. This decrease was attributed to the reduction in radius brought about by the active muscle.

THE CALIBER AND DISTENSIBILITY of conduit arteries are influenced by the activity of the smooth muscle cells within their walls. Studies on arterial strips by Spedden (23), Sparks and Bohr (22), and Lundholm and Mohme Lundholm (15) indicate that vascular smooth muscle exhibits a length-tension relationship which resembles that shown by skeletal and cardiac muscle. However, strips may not be representative of arteries in situ which are cylindrical and constantly under longitudinal tension. Moreover the degree of shortening produced by active smooth muscle depends upon geometric factors which are different in strips and cylinders.

The influence of smooth muscle activity on arterial distensibility is controversial. Lawton (13) and Torrance and Shwatz (24) found that excitation of vascular smooth muscle decreases the distensibility of arteries studied in vivo or subjected to in vitro pressure-volume experiments. On the other hand, Wexler and Boger (26) and Wiggers and Wegria (27) reported that excitation of vascular smooth muscle causes an increase in the distensibility of similar preparations. Finally, Landgren (11) and Alexander (3) found that excitation of smooth muscle decreased arterial distensibility at low pressures, but increased arterial distensibility at high pressures. The present studies were undertaken to clarify these issues by determining the influence of active smooth muscle upon the contractile mechanics and elasticity of cylindrical segments of arteries held at in situ length.

METHODS

A water-jacketed, 100-ml Lucite chamber with a stainless steel floor was designed to hold the arterial segments at in situ length (Fig. 1A). Thermostatic control of water circulated through the water jacket maintained the fluid in the tissue bath at 36-37 C. Two upright Lucite blocks were set 5.3 cm apart in the tissue bath, and segments of polyethylene P-E 240 cannula were mounted through these blocks. Excised arterial segments were mounted on the cannulas between the upright blocks. The cannula at one end of the vessel segment was connected to a 100 % O\(_2\) supply controlled by a pressure regulator, and the cannula at the other end of the vessel segment was connected to a Statham P23Dc pressure transducer. Vessel segments were inflated with gas in order to follow the method used by Bergel (4). It also provided a means of immediately detecting leaks in the vessel wall. In a nonleaking vessel the luminal gas should become saturated with water and, therefore, should not dry the vessel wall.

The external radius of mounted vessel segments was continuously measured with a linear displacement transducer (Fig. 1B). This consisted of a Robinson-Halpern differential transformer coupled to a Lufkin model 613 flat rod, nonrotating micrometer depth gauge. A nylon rod, ground to the shape of a mushroom, was threaded into the hallow steel core of the displacement transformer to increase the effective length of the core. The nylon and steel core weighed 0.615 g when the disc portion of the mushroom was submerged to the depth employed during experiments. The area of contact between the disc and the arterial segment was .10 cm\(^2\). A Sanborn carrier amplifier was used to supply an exciting voltage to the primary windings of the displacement transformer, and to amplify the output from the secondary windings of the displacement transformer. The output was fed into a Grass model 7 polygraph which was used to record radius and pressure. The amplifier recording system was linear over a .10-cm displacement range. Whenever the limits of linearity were approached, the micrometer was adjusted to keep the apparatus within the linear range. During the course of each experiment only changes in radius were recorded. After the final measurement the foot plate was elevated until it just touched the bottom of the vessel. The micrometer was then adjusted to advance the coil downward until the core was centered

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precisely within the coil. This was indicated by a movement of
the polygraph pen to the center of the recording channel.
The micrometer reading was noted. The displacement trans-
ducer was then removed from the tissue bath, the mushroom
was permitted to fall onto the foot plate, and the micrometer
was again adjusted to advance the coil downward. When
the polygraph pen was again at the center of the recording
channel the micrometer reading was again noted. The
second micrometer reading was subtracted from the first
to provide the absolute diameter of the vessel at the final
level of inflation. The amount of sag caused by the weight
of the core assembly was determined and found to be less
than .005 mm at all pressures. This corresponds to less than
.5 % of the absolute external radius of the vessel.

PROCEDURE

Mongrel dogs were anesthetized with either 34 mg/kg
Nembutal intraperitoneally, or 2 mg/kg Sernylan (Pot-
Davis) intramuscularly and 80 mg/kg α-chloralose intra-
venously. A carotid artery was exposed in the neck, and a
5.3-cm segment of the vessel was measured and marked
with small nicks in the adventitia. The artery was ligated
above and below the measured segment. This segment was
excised, trimmed of loose connective tissue and immediately
mounted on the cannulas in the tissue bath. In mounting,
the retracted arterial segment was restored to its original
length of 5.3 cm. The tissue bath was then filled with
Krebs-Ringer dextrose solution, composed of 120 mm NaCl,
4.8 mm KCl, 2.5 mm CaCl₂, 1.2 mm MgSO₄·7H₂O, 15 mm
phosphate buffer-pH 7.4, and 11.0 mm dextrose (25). The
vessel segments were inflated with 100 % oxygen while
external radius was continuously recorded. Transmural
pressure was increased in 25-mm Hg steps to the highest pressure. At the highest pressure
the bath was drained, rinsed, and refilled with dextrose-
free Krebs-Ringer solution. Potassium cyanide (KCN) was
added to the bath to give a final bath concentration of 100
mg/liter, and the vessel was permitted to remain at the
high pressure level for 1 hr. The pressure was then de-
creased in 25-mm Hg steps to the lowest value, and finally
was elevated, to the highest value. Preliminary
experiments indicated that 2 mg/liter NEpi produced a
maximum contractile response, and that 100 mg/liter KCN
in dextrose-free Krebs-Ringer solution produced 95-100 %
of maximum dilation within 1 hr. No response was elicited
by NEpi or KCN from vessels in which the smooth muscle
had been inactivated by freezing, heating, soaking in dis-
tilled water, or prolonged inflation with nitrogen, indicating
that the action of NEpi and KCN was on the muscle.

Following completion of all experimental procedures, the
displacement transducer was removed and the tissue bath
was drained. A dilute suspension of lead oxide was applied
sparingly to the outer surface of the mounted vessel seg-
ment and then was washed away with fresh saline. The
negligible lead oxide that remained imparted discernible
radiopacity. A chip of dried lead oxide was also placed
on the outer surface of the vessel to indicate where the vessel
had been in contact with the mushroom. The cannula at
one end of the vessel was disconnected from the gas supply
and connected to a syringe containing liquid mercury, and
the vessel segment was filled with liquid mercury under a
number of pressures. A packet of dental X-ray film was
placed beneath the vessel segment, and radiographs were
obtained at a number of pressures with a GE 11CF2 den-
tal X-ray machine. X-ray dimensions were measured mi-
croscopically with a Cook AEI image-splitting eyepiece to
determine internal diameter and wall thickness. Steel rods of
various dimensions were used to calibrate the X-rays.
Measurements of identical rods by the displacement trans-
ducer and the X-ray method exhibited excellent agree-
ment.

RESULTS

Wall volume. The volume (V) of the wall material of
excised arterial segments was calculated as:

\[ V = \left( \pi r^2 - \frac{\pi r^3}{3} \right) \times L \]  

(1)
where $r_0$ is external radius, $r_i$ is internal radius, and $L$ is the length of the specimen. Figure 2 presents wall volume data for seven untreated arteries. The mean wall volume of each artery is designated as 100% and the points contributing to that mean are plotted as a function of diameter. Each arterial segment is represented by a different symbol. The abscissa is external diameter, expressed as the percent of each individual vessel segment's diameter at 50 mm Hg; the ordinate is wall volume, expressed as the percent of mean wall volume for each vessel segment. These data are presented in percentage form in order to compare the wall volumes of arteries obtained from animals of different sizes and weights. If the wall underwent volume compression with increased distention, then the points would show a general tendency to fall from left to right. If the wall underwent a volume expansion with increased distention, then the points would show a general tendency to rise from left to right. It is clear from Fig. 2 that neither trend was observed. Similar results were obtained from treatment with NEpi and after poisoning with KCN, although the total volume was increased about 6% after KCN. Knowledge of the wall volume of each artery permitted calculation of the internal radius and wall thickness which corresponded to each external radius.

**Retraction behavior.** Pressure-radius data for a complete experiment are shown in Fig. 3. At low pressures, the vessel exhibited large changes in radius with each step change in pressure, whereas at high pressures, it showed very slight changes in radius with each step change in pressure. This pattern was most pronounced after poisoning with KCN, whereas a much more gradual curve was observed after treatment with NEpi. The maximum active decrease in radius, as indicated by the difference between the pressure-radius curves after treatment with NEpi and after poisoning with KCN, occurred in this preparation at 50 mm Hg. Marked distention-retraction hysteresis always was evident after treatment with NEpi but was negligible after poisoning with KCN. It is evident from the location of the descending pressure-radius curves in Fig. 3 that although the arterial muscle had been activated at high pressure it exerted little effect until the pressure was reduced several steps. The difference between the NEpi and KCN curves results from the active muscular force which develops gradually over the course of the stepwise decrease in pressure and radius. For this reason, length-active stress relationships were determined only from descending pressure-radius sequences. If the descending and ascending curves had been identical, then elastic properties could have been determined meaningfully from either sequence. However the marked hysteresis which occurs after treatment with NEpi suggests that the descending sequence reflects the

**FIG. 2.** Wall volume data for seven arteries, each represented by a different symbol. Tendency for points to fall about mean indicates that wall volume remains constant.

**FIG. 3.** Pressure-radius curves in pretreatment condition and after treatment with NEpi and KCN. Note pronounced hysteresis only after treatment with NEpi.

**FIG. 4.** Derived data for vessel illustrated in Fig. 3. **A:** stress-strain curves for retraction behavior after treatment with NEpi and KCN. Difference between these represents contribution of active smooth muscle. **B:** stress-strain curves for distention behavior after treatment with NEpi and KCN. **C:** strain-elastic moduli for distention behavior after treatment with NEpi and KCN. Arrows indicate vessel strains at 100 mm Hg pressure under each condition. **D:** pressure-elastic moduli for distention behavior. These are same data plotted in Fig. 3C, plotted here as a function of pressure. (C and D on facing page.)
gradual development of active stress and that the ascending sequence reflects the resistance to extension of activated, contracted smooth muscle in association with connective tissue. For this reason vessel elastic moduli were determined only from ascending pressure-radius curves.

The external radius at 25 mm Hg after treatment with NEpi was designated as zero strain. Strain (ε) was calculated as:

\[ \varepsilon = \frac{\Delta C}{C_0} - \frac{\Delta r}{r_0} \]  

(2)

where ΔC is the change in circumference, C₀ is the original circumference, Δr is the change in external radius, and r₀ is the original external radius.

Circumferential stress (σ) was calculated as:

\[ \sigma = \frac{P_T \times r_i}{h} \]  

(3)

where P_T is transmural pressure, r_i is internal radius, and h is wall thickness. Figure 4 presents stress-strain curves for the pressure-radius retraction data described in Fig. 3. The difference between the stress after NEpi and the stress after KCN provides an estimate of the active stress for vascular smooth muscle.

Table 1 presents active stress data for 17 arteries. Maximum active stress, the strain associated with the maximum stress, and the pressure at which the maximum decrease in radius occurred are presented. Means and standard errors are included at the bottom of the table. The mean peak active stress was 0.88 ± 0.10 × 10⁶ dynes/cm². It occurred at a mean circumferential strain of 0.59 ± 0.03. The contraction by the active muscle was calculated at each pressure by subtracting the radius after treatment with NEpi from the radius after poisoning with KCN. Maximum active muscular contraction occurred at 63 ± 4.6 mm Hg. Figure 5 summarizes the magnitude of active muscular contraction for all the vessels studied. The contraction observed at 75 mm Hg represents 18.9 ± 1.81% of the radius of the KCN-poisoned vessel at that pressure; the contraction at 100 mm Hg represents 17.1 ± 1.69% of the radius of the KCN-poisoned vessel at that pressure. Although maximum active stress is generated at large strains, the maximum contractile effects are manifested at relatively low pressures.

![Graphs and figures](http://ajplegacy.physiology.org/Downloaded/from http://ajplegacy.physiology.org/ by 122.03.36.1 on April 3, 2017)
This occurs because the KCN-poisoned vessel remains large until the distending pressure is reduced to low values. This is illustrated by the difference between the descending pressure NEpi and KCN curves in Fig. 3.

**Distention behavior.** Figure 4B presents stress-strain curves for the pressure-radius distention data illustrated in Fig. 3. The slopes of these stress-strain curves were determined graphically and 75% of the value of the slope was used as a measure of the incremental elastic modulus and is based on the assumption that the wall is isotropic and exhibits a Poisson's ratio of 0.5. This method of calculation is comparable to those employed by Bergel (4) and Patel et al. (17). The elastic moduli are presented in Fig. 4C, plotted as a function of strain. It may be observed that the elastic modulus of the artery was higher after treatment with NEpi than after poisoning with cyanide at all but the largest strains. Figure 4D presents the incremental elastic moduli plotted as a function of transmural pressure. Contrary to the strain-elastic modulus data, the pressure-elastic modulus curves indicate that arteries are less stiff at any given pressure after treatment with NEpi than after poisoning with KCN. However, very different strains are associated with each pressure when the smooth muscle was active and when the smooth muscle was relaxed. This is emphasized by the location of the arrows in Fig. 4C which indicate the strains associated with 100 mm Hg after treatment with NEpi and after poisoning with KCN. It is clear that NEpi decreased the strain at physiological pressures, and also decreased the elastic modulus. Figure 5 presents the mean incremental elastic moduli and standard errors for 16 arteries, plotted as a function of pressure. It is evident that the elastic moduli gradually increase with increasing pressures, and that treatment with NEpi decreases the elastic moduli at all but the lowest pressures. The data of Bergel (4) for untreated cylindrical segments of carotid artery held at in situ length are also included and suggest that his vessels were probably fairly relaxed. The elastic moduli after NEpi and KCN were compared at each pressure using the Wilcoxon matched-pairs signed-ranks test (21). The differences at the two lowest pressures were found to be insignificant, but the differences at all other pressures were statistically significant ($P < .025$).

If it is assumed that the muscle and connective tissue elements are in parallel then it is possible to estimate the elastic modulus of the active muscle by subtracting the elastic modulus of the KCN-treated vessel from the elastic modulus of the NEpi-treated vessel at equivalent strains. Table 2 presents the maximum elastic modulus of the active smooth muscle and the strain associated with that maximum. Means and standard errors are included at the bottom of the table. The mean peak elastic modulus of the active muscle was 3.99 ± 0.08 x 10^6 dynes/cm^2. It occurred at a mean strain of 0.46 ± 0.05.

### Table 2: Elastic modulus of active carotid smooth muscle: elastic modulus after NEpi minus elastic modulus after KCN

<table>
<thead>
<tr>
<th>Artery</th>
<th>Maximum Active Elastic Modulus, x 10^6 dynes/cm^2</th>
<th>Strain at Maximum Active Elastic Modulus</th>
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<tr>
<td>1</td>
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<tr>
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<td>.6</td>
</tr>
<tr>
<td>16</td>
<td>3.8</td>
<td>.3</td>
</tr>
<tr>
<td>Mean ± SE</td>
<td>3.99 ± 0.08</td>
<td>.46 ± 0.05</td>
</tr>
</tbody>
</table>

**Discussion**

**Wall volume.** The present results indicate that the volume of the arterial wall in fixed-length preparations remains constant over a wide range of physiological pressures. These results confirm the findings of previous studies (6, 12, 16) in which the volume of arteries was determined in fluid-filled chambers. Changes in the level of the fluid in a capillary tube projecting out of the chamber were used to indicate changes in the volume of the arterial specimen. However, it is important to note that the arterial wall is also permeable to water. The imposition of a strong compressive force across the wall might force fluid from the wall tissue into the surrounding fluid. Such a transfer of fluid would not produce a change in the level in the capillary tube because this level reflects the volume of the total contents.

![Graph](http://ajplegacy.physiology.org/DownloadedFrom)
within the chamber. The present results agree with the findings of the above authors, but were obtained by a method that is less susceptible to error due to fluid transfer.

Retraction behavior. The observation that vascular smooth muscle exhibits a peaked strain-active stress curve agrees qualitatively with the length-tension curves obtained in studies on arterial strips (15, 22, 23). However, in only a few studies on smooth muscle of various types has the cross-sectional area of the tissues been determined. Abbott and Lowy (1) reported that various molluscan smooth muscles exhibited peak active stresses of .5 to 4.5 × 10^6 dynes/cm²; Aberg and Axelson (2) reported that guinea pig taenia coli exhibited a peak active stress of 1.6 ± .5 × 10^6 dynes/cm²; and Lundholm and Mohme-Lundholm (15) reported that strips of bovine mesenteric artery exhibited a peak active stress of 2,000 g/cm². However, these values are for whole tissue, and no attempt was made to estimate the active stress exerted by just the muscular component of the tissue. Goodford and Hermansen (8) determined that the intracellular space of guinea pig taenia coli is equal to 360 ml/liter. If this value is taken as an indication of the ratio of extracellular-to-total tissue space, then this ratio may be used to correct the active stress data of Aberg and Axelson (2). Thus corrected, the smooth muscle of taenia coli generates a maximum of 2.85 × 10^6 dynes/cm² active stress. Ducret (7) determined, by histological studies, that smooth muscle occupies about 75% of the cross-sectional area of mesenteric artery. If the mesenteric artery data of Lundholm and Mohme-Lundholm (15) are corrected for this proportion, a value of 2.62 × 10^6 dynes/cm² maximum active stress is obtained. Finally, Jones et al. (9) determined that the intracellular space of canine carotid arteries is 315 ml/liter. If the present experimental data are corrected for this proportion, a value of 2.73 × 10^6 dynes/cm² maximum active stress is obtained. Thus, all three measurements performed on different smooth muscle preparations and using different techniques show reasonable agreement when corrected for the estimated percent of the tissue that is actually smooth muscle. The three studies share one very important feature in common that each of them included a specific step to be certain that the muscle was truly inactive.

Both the NEpi-treated and KCN-poisoned vessels are capable of maintaining a stable radius at each pressure level. Under these equilibrium conditions the distending force is equal to the retractive force. The distending force (FD) may be calculated as the product of the distending stress and the area over which that stress is exerted.

\[ F_D = P_T \times 2r_i \times L \]

where \( P_T \) is transmural pressure, \( r_i \) is internal radius, and \( L \) is vessel length. The retractive force (FR) may be calculated as the product of the retractive intramural stress and the area over which that stress is exerted.

\[ F_R = \sigma \times 2h \times L \]

where \( \sigma \) is intramural stress, \( h \) is wall thickness, and \( L \) is the vessel length. Each step decrement in pressure reduces the distending force to a value that is lower than the retractive force. This permits the radius to decrease which, in turn, further reduces the distending force. Simultaneously, the retractive force tends to be elevated by the increasing wall thickness. Therefore, the achievement of a new equilibrium must be determined by how steeply the retractive stress declines as the radius decreases. If the retractive stress declines steeply, then only a small decrease in radius will lower the stress sufficiently to establish a new equilibrium between distending and retractive forces. However, if the retractive stress declines gradually, then a large decrease in radius must occur before the retractive force will again equal the distending force. The retractive stress exerted by connective tissue is described by the KCN curve in Fig. 4A. At large strains, any decrease in radius is associated with a very large decline in connective tissue stress, while at small strains an equivalent decrease in radius is associated with a negligible decline in connective tissue stress. Thus, the KCN-poisoned vessel exhibits very small changes in radius with each pressure decrement at large strains, and very large changes in radius with each pressure decrement at small strains (Fig. 5).

The constriction produced by active smooth muscle with each step pressure decrement depends upon how much additional retractive force the muscle can provide and how the slope of the stress-strain curve is modified. The retractive stress exerted by the combination of muscle and connective tissue elements is described by the NEpi curve in Fig. 4A. At large strains, the NEpi curve declines more gradually than the KCN curve, and at moderate strains the two curves decline at about equal rates. Thus, at large strains, each pressure decrement produces a greater retraction from the NEpi-treated vessel than from the KCN-poisoned vessel (Fig. 3). At small strains just the reverse occurs, and at moderate strains the NEpi and KCN-treated vessel retract at about equal rates with each pressure decrement.

The present data may also be related to the phenomenon of critical closure, i.e., the cessation of blood flow in spite of a positive-perfusion pressure. Burton (5) observed that critical closure occurs in active muscular vessels which exhibit no connective tissue stress, such as arterioles at a small radius. However, if the connective tissue and the smooth muscle behave as parallel, extended, elastic elements, then both of these elements may be expected to exert retractive forces. If this assumption of parallel retractive function is true, then one might anticipate a greater tendency for closure in vessels containing significant amounts of connective tissue stress rather than in those containing negligible amounts of connective tissue stress. However, in the present experiments it was observed that the carotid artery segments exhibited a smaller radius at each pressure level after treatment with NEpi than after poisoning with KCN, even at 0 mm Hg pressure. The KCN-poisoned vessel at 0 mm Hg is probably the best approximation of the unloaded, natural state, because the forces within the wall are due entirely to the connective tissue elements under longitudinal tension. Under these conditions the circumferential stress is effectively zero, and all the elastin and collagen fibers which tend to retract are just counterbalanced by those which resist compression or distortion. This might be termed the "zero state," and is probably the most logical designation of a true zero strain for a cylin-

\[ FD = P_T \times 2r_i \times L \]

\[ F_R = \sigma \times 2h \times L \]
The general nonlinear form of the stress-strain and strain-elasticity curves (Fig. 4, B and C) resembles those described in the literature for studies on rings, strips, and pressure-volume segments (10, 14, 18-20). The biphasic character of this curve has been attributed to a parallel arrangement of relatively extensible elastin and highly inextensible collagen. Differential removal of either substance by putrefaction (10, 18) or chemical and enzymatic treatment (20) causes the wall to assume the behavior of the remaining material.

Activation of smooth muscle with NEpi produces an increase in vessel elastic modulus at most strains, with a maximum elastic modulus of 3.99 ± .08 × 10^6 dynes/cm^2 for the active muscle (Table 2). This value is for the whole carotid arterial wall. When corrected for the proportion of the wall that is smooth muscle a value of 12.66 ± 10^6 dynes/cm^2 is obtained. Although activation of the smooth muscle increases the elastic modulus at almost all strains (Fig. 4C), it also produces a decrease in the elastic modulus when this parameter is plotted as a function of pressure (Fig. 4D). This paradox may be resolved by considering the consequences of NEpi-induced vessel contraction. Because of this contraction the artery was at a smaller radius than after poisoning with KCN. This is illustrated by the location of the arrows in Fig. 4C which indicates the strains associated with 100 mm Hg in the NEpi-treated and KCN-poisoned states. The elastic modulus of the small-radius, NEpi-treated artery is dominated by the mechanical characteristics of smooth muscle and unextended elastin, the relatively extensible properties of which persist up to high pressures. By contrast, the elastic modulus of the large-radius, KCN-poisoned artery is dominated by the mechanical characteristics of elongated elastic and collagen, the inextensible properties of which appear at relatively low pressures. Thus, when the elastic moduli of a vessel are compared at comparable strains (Fig. 4C) it is clear that the smooth muscle contributes significant resistance to distention over most of the range. However, when the elastic moduli of the vessel are compared as a function of pressure (Fig. 4D) it is also evident that the increase in stiffness provided by active smooth muscle is insufficient to offset the decrease in stiffness that results from contraction to a smaller radius. This was found for all the vessels that were studied (Fig. 6).

The present data are relevant to published studies concerning the influence of smooth muscle upon vascular distensibility. Those authors who have reported that active smooth muscle decreases arterial or venous distensibility presented their data as a function of strain or volume (14, 24), while those authors who have reported that active smooth muscle increases vessel distensibility presented their data as a function of pressure (26, 27). Finally, the slopes of the pressure-radius curves in Fig. 3 illustrate the source of the observation that active smooth muscle may decrease vessel distensibility at low pressures but increase vessel distensibility at high pressures (3, 13). This is due to the contrast between the sharply inflected pressure-radius curve of the relaxed vessel and the more gradual slope of the pressure-radius curve of the contracted vessel. However, pressure-radius relationships fail to account for all of the variables included in the stress and strain terms (equations 2 and 3) and are less exact than elastic moduli as an index of wall properties.

These observations indicate that the effect of smooth muscle upon vessel distensibility depends on 1) the extent to which a vessel is permitted to contract, 2) the manner in which the wall properties are calculated, and 3) whether the wall properties are plotted as a function of strain or of pressure.

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