Series Elastic and Contractile Elements in Vascular Smooth Muscle

By Philip B. Dobrin and Thomas R. Canfield

ABSTRACT

Segments of dog common carotid artery were excised, cannulated, and restored to in situ length. They were immersed in a Krebs-Ringer's bath and inflated with 100% O₂ under nonoscillating pressure. Diameter was continuously monitored with a linear displacement transducer. The vessel segments were relaxed and then treated isometrically with norepinephrine to excite the muscle. These continuously contracted vessels were subjected to 10- or 25-mm Hg quick release steps to 0 mm Hg. Then the muscle was inactivated with potassium cyanide, and the quick releases were repeated to study the parallel elastic elements. Finally, vessel wall volumes were determined radiographically. Computations were performed to compute series elastic element stiffness for both the Maxwell and the Voigt model of the arterial wall. These experiments indicated that the series elastic element stiffness was dependent on the applied stress and independent of muscle length provided that the vessels were excited at initial strains less than 0.70. Vessels excited isometrically at various initial pressures up to 150 mm Hg yielded identical stress—series elastic element stiffness curves. The computed series elastic element extension at an applied stress of 1.0 \times 10^6 dynes/cm² was 18.9% for the Hill model and 14.7–17.9% for the Voigt model; these values are percents of the vessel diameter at 0 mm Hg after application of potassium cyanide. The values were reduced to 11.2% for the Maxwell model and 8.8–10.5% for the Voigt model when they were expressed as a percent of the circumferential length associated with the peak in the length-tension curve. Vessels excited isometrically at pressures higher than 150 mm Hg exhibited greater series elastic element extensibility.

KEY WORDS series elastic element extension common carotid arteries
contractile element stiffness dog parallel elastic elements
quick release vessel wall volume vessel stress
length-tension curve muscle models

Isometric contraction of muscle is defined as the development of active tension with no overt shortening; however, this mode of contraction is probably accompanied by internal shortening of the contractile apparatus with concomitant extension of compliant, series-coupled structures. The functional sum of these undamped, series-coupled structures has been termed the "series elastic element," and this element has been studied extensively in skeletal muscle (1-5) and cardiac muscle (6-9). Recently, a number of studies have also described the series elastic element in smooth muscle (10-13). For example, Lundholm and Mohme-Lundholm (12) examined the series elastic element in isolated strips of bovine mesenteric artery. They found that the contracted muscle required passive retraction of 15–20% to reduce the contractile tension to 0. It is assumed that the viscous contractile system remained constant and that only the undamped series elastic element retracted during this rapid, passive shortening of the tissue. A series elastic element of 15–20% is much larger than that reported for various striated muscles, suggesting that either the series elastic element in smooth muscle is very much more compliant than that in striated muscle or the absolute length of the series elastic element in smooth muscle is much longer than that in striated muscle. Alternatively, the method of complete tension reduction may not be appropriate for studying smooth muscle.

The present study was undertaken to measure the properties of the series elastic element in cylindrical segments of dog common carotid artery held at their in situ length. A technique of quick pressure release was utilized to determine the stiffness of the series elastic element. The properties of the parallel elastic element were also taken into account,
permitting evaluation of the series elastic element stiffness over a wide range of muscle lengths.

**Methods**

Common carotid arteries were excised from anesthetized dogs immediately after death. Each artery was cannulated at both ends with polyethylene (PE240) tubing and mounted in a tissue bath. One cannula was mounted rigidly through a fixed upright block, and the other was sealed at the outer end and attached with a Lucite clamp to a Grass FT-10 force transducer. The force transducer in turn was suspended from a rigidly fixed micrometer. The retracted vessel segment was then elongated gradually by turning the micrometer until the force gauge indicated a force (dynes x 10$^6$) equal to the dog's body weight (kg). This procedure restored the vessels to their in situ length (14). The vessel segments were about 4 cm long once they were elongated to in situ length. The force transducer was sufficiently stiff (4.0 x 10$^7$ dynes/cm) so that the vessel segment at in situ length. Length changes occurring during individual quick releases were less than 1% of this value.

The tissue bath was filled with Krebs-Ringer's dextrose solution composed of 120 mM NaCl, 48 mM KCl, 2.5 mM CaCl$_2$, 1.2 mM MgSO$_4$ • 7H$_2$O, 15 mM phosphate buffer (pH 7.4), and 11.0 mM dextrose. This fluid surrounded the vessel segment. A water jacket surrounding the tissue bath. Thermostatic control of water in the jacket maintained the fluid in the tissue bath at 36-37°C.

Vessel diameter was measured in vitro with a linear displacement transducer consisting of a differential transformer with a lightweight, movable core. An adjustable foot was raised to limit movements of the lower surface of the vessel, and the movable core of the displacement transducer was used to measure displacements of the upper surface of the vessel. The core of this instrument weighed 500 mg. The area of contact between the core and the vessel was about 0.10 cm$^2$ so that the weight of the core reduced transmural pressure by about 4 mm Hg at the site of contact, the inflation pressure was not corrected for this 4 mm Hg. The frequency response of the displacement transducer was determined by connecting the movable core of the transducer to the oscillating piston of a mechanical pump while the core was inserted into its normal position within the coil. The core was driven with sinusoidal oscillations up to 60 Hz. The output from the displacement transducer was recorded on a polygraph. Frequency response was measured as the percent of that observed at 1 Hz. These data indicated that the response amplitude decreased 0.3%/Hz up to 40 Hz. Response amplitude declined steeply at frequencies between 40 and 60 Hz. Therefore, the measuring device was judged to be adequate for measuring the behavior of the vessels during quick release procedures, especially since the vessels tended to maintain relatively constant diameters immediately following quick releases (Fig. 1). The free-fall velocity of the displacement transducer core within the surrounding coil is comparable to "stray compliance" in striated muscle quick releases. Free-fall velocity was determined by permitting the core to drop without restraint through the Krebs-Ringer's solution while displacement was recorded on the polygraph. The measured initial free-fall velocity was 5.1 cm/sec.

The smooth muscle series elastic element was studied using a quick release procedure based on the method of Wilkie (5). The vessel cannula mounted through the upright block in the tissue bath was connected to a network consisting of a pair of Conoflow constant-pressure regulators, a pair of three-way valves, a single Statham P23DC pressure transducer, and interconnecting polyethylene tubes. This system permitted inflation of the cannulated vessel with gas under the control of one of the Conoflow pressure regulators. The inflation pressure was monitored with the Statham pressure transducer. It also was possible to set the second Conoflow regulator for a different pressure level by temporarily switching the pressure transducer out of the circuit supplying the vessel and into the second pressure circuit. This procedure was carried out without disturbing the pressure within the inflated vessel. Then, by rapidly switching a three-way valve, the pressure of the gas inflating the vessel was changed from that set by the first Conoflow regulator to that set by the second Conoflow regulator. This procedure generated a step-function pressure change of predetermined magnitude and was accompanied by an extremely rapid step change in vessel diameter. This method was used to generate quick releases to study the smooth muscle series elastic element. Pressure decrements used were small enough to ensure that the vessel retractions occurring during quick releases were approximately 0.7% of the absolute vessel diameter.

All force, pressure, and diameter measurements were recorded on a Grass model 7 polygraph. The diameter

*FIGURE 1*

Polygraph record showing quick release steps. Top: Vessel behavior during continuous excitation of muscle with nor-epinephrine (NEpI). Bottom: Vessel behavior after poisoning of the muscle with potassium cyanide (KCN).
PROCEDURE
All vessels were mounted in vitro at their in situ length. Excised vessels tend to retain some degree of muscle tone, but this tone could be reduced by slowly elevating the inflation pressure in 10–15-mm Hg steps to 300 mm Hg. The pressure was maintained at each level until the vessel exhibited a steady diameter for several minutes. When the vessel was relaxed, the pressure was reduced to 0 mm Hg and then raised to a predetermined treatment level for excitation of the muscle.

Graded Muscle Activation Experiments.—One group of vessels was relaxed and then brought to treatment pressures between 0 and 230 mm Hg. These vessels were treated with a low dose of norepinephrine (0.2 mg/liter tissue bath fluid) and permitted to contract either isometrically or isobarically. Isometric contraction was accomplished by adding norepinephrine to the bath and carefully monitoring the polygraph record. During the gradual excitation of the muscle, the pressure was elevated just enough to maintain the diameter at a constant level. It was observed that, once the muscle had contracted, the presence of norepinephrine in the bath sustained this degree of contraction for several hours. It was this continuously contracted vessel that was subjected to consecutive quick release steps. After each quick release, the pressure was held constant until the vessel exhibited a steady diameter for at least 2 minutes. Then another quick release was performed. This procedure was repeated until 0 mm Hg had been achieved. Then the pressure was elevated slowly to return the diameter to the initial pretreatment value. A second, stronger dose of norepinephrine (0.8 mg/liter tissue bath fluid) was given, and the quick release procedures were repeated. These sequences were performed several times with successively higher doses of norepinephrine (2.0 and 20.0 mg/liter tissue bath fluid). Vessels which exhibited a tendency to lose active muscle stress during the stepwise constriction process were discarded. When the final sequence of quick releases had been completed, the pressure was brought to 75 or 100 mm Hg; the tissue bath was then drained, rinsed, and filled with dextrose-free Krebs-Ringer’s solution. Sufficient potassium cyanide was added to the tissue bath to bring the final bath concentration to 200 mg/liter to poison the vascular muscle. After 40 minutes the quick release procedures were repeated to determine the properties of the vascular connective tissue.

Excitation at Various Initial Muscle Lengths.—A second group of arteries was relaxed and then subjected to isometric contractions at initial pressures between 50 and 300 mm Hg. This procedure was carried out to examine the influence of initial muscle length on series elastic element stiffness. Sufficient norepinephrine was added to the bath to bring the bath concentration to 2 mg/liter. When isometric contraction was complete, the pressure was reduced in quick release steps to 0 mm Hg to permit the continuously contracted vessels to constrict. Quick release steps of 25 mm Hg were used at pressures above 200 mm Hg, and steps of 10 mm Hg were used at pressures below 200 mm Hg. These steps produced vessel retractions that were approximately 0.7% of vessel diameter. Any vessels which exhibited a tendency to lose active muscle stress during the stepwise constriction process were discarded. When these procedures had been completed, the pressure was returned to 75 or 100 mm Hg, the tissue bath was refilled with dextrose-free Krebs-Ringer’s solution, and the vascular muscle was poisoned with potassium cyanide. Finally, quick release procedures were repeated after inactivation of the muscle.

The final procedure in all of the experiments was the determination of the cross-sectional area of the vessel wall. Each vessel segment was filled with barium sulfate, and a radiographic method was used as described previously (15). The cross-sectional area was obtained for each vessel and was used to compute the internal radius and the wall thickness at each external diameter observed in the tissue bath.

Results
Quick release data such as those illustrated in Figure 1 were used to compute the stiffness of the series elastic element. Circumferential strains and circumferential stresses were required for this analysis. Circumferential strain (ε) was computed as

\[ \varepsilon = \frac{\Delta D}{D_0} = \frac{D - D_0}{D_0}, \]

where D is the diameter observed at any time and D₀ is the diameter observed in the cyanide-poisoned vessel at a transmural pressure of 0 mm Hg. Thus, strain is the fractional change in diameter relative to that observed in the vessel when it is unstressed in the circumferential direction and held.
at in situ length. This condition was taken as the unstressed state. Circumferential stress \( (\sigma) \) was calculated as

\[
\sigma = P_r \times r_i / h,
\]

(2)

where \( P_r \) is transmural pressure, \( r_i \) is internal radius, and \( h \) is wall thickness. Transmural pressure values were obtained directly from polygraph recordings. Internal radius and wall thickness were computed using the external diameter recorded in the tissue bath and the wall cross-sectional area obtained from the radiographs of each vessel.

Figure 2 illustrates the computational steps performed in the analysis of the series elastic element for a single vessel subjected to successive quick release steps at progressively lower pressures. These releases were performed while the vascular muscle was continuously active. The top left of Figure 2 presents static strain–stress curves. The data points for these curves were obtained after each quick release when the vessel exhibited a steady diameter for 2 minutes at constant pressure. The curve obtained after treatment with norepinephrine reflects the static properties of the whole wall, i.e., connective tissue and continuously contracted smooth muscle, and the curve obtained after poisoning with potassium cyanide reflects the static properties of just the connective tissue. If it is assumed that the muscle and the connective tissues are arranged in parallel, then the difference between the two stresses (broken line) represents the static strain–active stress curve for just the vascular smooth muscle.

The top right of Figure 2 presents stiffness curves determined for quick release procedures. Stiffness was computed as

\[
E = \frac{\Delta \sigma}{\Delta \varepsilon},
\]

(3)

where \( E \) is stiffness, \( \Delta \sigma \) is the difference in stress observed immediately before and after each quick release procedure, and \( \Delta \varepsilon \) is the difference in strain observed before and after each quick release procedure. \( E \) is a measure of stiffness, but it cannot be treated as a true elastic modulus, because (1) the vessel was not in a true state of equilibrium at the end of each retraction accompanying a quick release although the undamped series elastic element was presumed to be at equilibrium, (2) the cross-sectional area of the series elastic element per se was unknown, (3) the original length of the series elastic element per se was unknown, and (4) the method of computation ignored the anisotropy of the carotid arterial wall although this omission was probably not a major source of error (14).

One may assume a Maxwell model for the whole vessel wall; in this model the viscoelastic properties of the continuously contracted smooth muscle are represented by the contractile element. An undamped elastic element is coupled in series with the contractile element, and another parallel elasticity spans both of these elements. The analysis required for this model is shown in the top right of Figure 2. For this model, the stiffness computed for quick releases after treatment with norepinephrine reflects the sum of the stiffnesses of the series elastic and the
parallel elastic elements. The stiffness after treatment with potassium cyanide represents the properties of the parallel elastic connective tissue elements. Therefore, the difference between the effects after treatment with norepinephrine and those after treatment with potassium cyanide represents the stiffness of just the series elastic element. However, the stiffness of the series elastic element depends on the degree to which it is extended by the applied stress. Therefore, the stiffness of the series elastic element was evaluated as a function of the stress developed by the contractile element. This procedure is shown in the bottom left of Figure 2. In this section of the figure, stiffness (the ordinate values of the top right of Fig. 2) is plotted against static active stress (the ordinate values of the top left of Fig. 2) at the corresponding strains.

One may also assume a Voigt model to describe the whole vessel wall, as shown in the bottom right of Figure 2. For a Voigt model, the series elastic element is in series with both the viscoelastic contractile element and the parallel elastic element. Consequently, the series elastic element is extended by the sum of the forces exerted by the contractile and parallel elastic elements. Thus, computation of the series elastic stiffness for the Voigt model is simply the stiffness observed on quick release after activation of the muscle with norepinephrine. The bottom right of Figure 2 shows the series elastic element stiffness for the Voigt model plotted as a function of the stress applied to it, i.e., the total wall stress. The two bottom sections of Figure 2 permit comparison of the series elastic stiffnesses for the Maxwell and Voigt models. It is evident that the data obtained for these two models were quite similar. The computations employed with these models assumed that each element occupied the entire wall and that their original length was the circumferential length of the unpressurized, cyanide-poisoned vessel. In addition, it was assumed that the contractile element was so viscous that it did not retract at all during the initial, rapid response phase of the quick release procedures. The computational steps illustrated in Figure 2 were performed on each vessel included in the present paper.

Graded Muscle Activation Experiments.—Experiments were performed to evaluate the assumption that series elastic element stiffness must be examined as a function of the applied stress. Eight arteries were excited isometrically with graded doses of norepinephrine to generate graded levels of sustained activation of the muscle at initial pressures between 50 and 230 mm Hg. These vessels were subjected to quick release. Figure 3 presents data for one representative artery treated isometrically at 60 mm Hg. The left of Figure 3 shows strain-stress curves following administration of four doses of norepinephrine and an inactivating dose of potassium cyanide. Increasing doses of norepinephrine elicited progressively more active stress. The data show that for both models series elastic element stiffness depends on applied stress and not on vessel circumferential strain.

FIGURE 3

Data for a vessel treated with four successively higher doses of norepinephrine (NEpi) followed by a muscle-poisoning dose of potassium cyanide (KCN). Static stresses and series element stiffnesses are shown. The data show that for both models series elastic element stiffness depends on applied stress and not on vessel circumferential strain.
stress. Recent microelectrode and mechanical studies (16, 17) have demonstrated that the multiunit smooth muscle cells of large arteries are activated in a graded, rather than in an all-or-none fashion. Therefore, the increasing doses of norepinephrine used in the present experiments probably elicited graded levels of activation from each of the carotid muscle cells, except perhaps with the lowest dose of the drug. Figure 3 (left) shows that, because of the graded effects produced by the drug, any given level of wall stress corresponded to several different vessel strains. The center and right of Figure 3 present the stiffness characteristics of the series elastic element for the Maxwell and Voigt models, respectively, each plotted as a function of their appropriate stress. It is evident that the series elastic element stiffness depends on the applied stress, regardless of the vessel strain at which the stress occurred. This finding was true for both the Maxwell and the Voigt model. Qualitatively similar results were found for six vessels treated at initial strains of less than 0.70. Two other vessels, treated at initial strains of 0.82 and 0.91, respectively, exhibited attenuated series elastic element stiffness at each stress level as the dose of norepinephrine was increased. The importance of this observation will be considered in a later discussion. It was hoped that these graded dose experiments also might shed light on which of the two models was more appropriate for the carotid arterial wall. Unfortunately Figure 3 indicates that such a discrimination was not obtained. However, the assurance that series elasticity is dependent on the applied stress is an important observation, for it permits comparison of series elastic element stiffness data without concern for vessel strain.

Excitation at Various Initial Muscle Lengths.— Each artery in this group was relaxed and then excited isometrically at some initial strain. When this initial phase of isometric contraction was complete and the vessel exhibited a steady, maintained, contracted state, the pressure was decreased in successive quick release steps to 0 mm Hg. Figure 4 presents series elastic element stiffness data for 84 arteries for both the Maxwell and the Voigt model. Fourteen vessels were subjected to successive quick release procedures following isometric contraction at 50, 75, 100, 150, 225, and 300 mm Hg. Vessels treated at initial pressures between 50 and 150 mm Hg were excited to the left of the peak in the isometric strain–active stress curve. Vessels treated at 225 and 300 mm Hg were excited at strains beyond the peak in the isometric strain–active stress curve. Vessels treated between 5 and 150 mm Hg exhibited very similar active stress–series elastic element stiffness curves, but vessels treated at 225 and 300 mm Hg exhibited somewhat less series elastic element stiffness. Consider, first, the large group of vessels treated between 50 and 150 mm Hg. The Maxwell model active stress series elastic element stiffness curves for vessels excited between 50 and 150 mm Hg were fitted using a least-squares analysis. This procedure gave the polynomial

\[ E = 2.3196\sigma^4 - 9.8576\sigma^2 + 21.943\sigma + 0.5417. \]
The curve for this polynomial fell within ± 2.0% of the experimental mean data points at all active stresses except $0.1 \times 10^6$ dynes/cm$^2$. At this low stress the curve for the polynomial was 5.6% greater than the mean experimental value. The Voigt model stress–series elastic element stiffness mean data also were curve-fitted using a least-squares analysis. The vessels excited at 100 mm Hg were approximated by the polynomial

$$E = 0.459\sigma^3 - 4.1626\sigma^2 + 15.677\sigma + 1.2908. \quad (5)$$

The curve for this polynomial fell within ± 1.8% of all the experimental mean data points. The Voigt model data for vessels excited at 150 mm Hg were approximated by the polynomial

$$E = 0.605\sigma^3 - 3.380\sigma^2 + 13.748\sigma + 2.605. \quad (6)$$

The curve for this polynomial fell within ± 5.0% of the experimental mean data points. Since the stiffness computed from the quick release procedures approximates the tangent elastic modulus of the series elastic element, these polynomials may be used to estimate the extension of this element during isometric contraction. Series elastic element compliance is the inverse of series elastic element stiffness. Therefore Eqs. 4, 5, and 6 were inverted and integrated numerically to obtain the extension of the series elastic element.

Figure 5 presents series elastic element load-extension curves for the Maxwell model data for vessels activated between 50 and 150 mm Hg and for the Voigt model data for vessels activated at 100 and 150 mm Hg. For the Maxwell model, an active stress of $1.0 \times 10^6$ dynes/cm$^2$ was associated with a series elastic element extension of 18.9%. For the Voigt model data, a total stress of $1.0 \times 10^6$ dynes/cm$^2$ was associated with a series elastic element extension of 17.9% for the vessels excited at 150 mm Hg and 14.7% for the vessels excited at 150 mm Hg. These extensions are based on a computation of strain assuming that $D_0$ is the vessel diameter at 0 mm Hg after inactivation of the muscle with potassium cyanide. In most skeletal and cardiac muscle studies, original muscle length usually is defined as the length corresponding to the maximum active isometric tension, i.e., the peak in the length–active tension curve. If $D_0$ in the present experiments is redefined as the vessel diameter corresponding to the peak in the strain–active stress curve for the vascular muscle (a strain of about 0.70), then the series elastic element extension may be corrected by dividing by 1.7. This correction reduces the series elastic element extension at $1.0 \times 10^6$ dynes/cm$^2$ to 11.1% for the Maxwell model data, to 10.5% for the 100-mm Hg Voigt model data, and to 8.6% for the 150-mm Hg Voigt model data.

Returning to Figure 4, it may be observed that vessels excited isometrically at pressures greater than 150 mm Hg exhibited decreased levels of series elastic element stiffness. Student’s t-test was used to evaluate these stiffness data. The tests indicated that, for the Maxwell model, vessels treated at 225 and 300 mm Hg exhibited significantly less series elastic element stiffness ($P < 0.05$) at active stresses between 0.2 and $1.0 \times 10^6$ dynes/cm$^2$ than did all the vessels treated at pressures between 50 and 150 mm Hg. Similar differences were found for the Voigt model data, although the range of total stress at which significant differences were found was somewhat different. It may be recalled that a similar decline in series elastic element stiffness was found in the graded muscle activation experiments when vessels were excited isometrically at strains greater than 0.70. The decline in series elastic element stiffness observed in vessels originating from large strains correlates with the observation that arteries excited isometrically at large strains and high pressures develop attenuated active muscle stress (18). This depression in active stress was attributed to a decrease in the strength or the number of effective contractile units within the muscle following...
SMOOTH MUSCLE SERIES ELASTICITY

isometric contraction at large strains. The observation that both active stress and series elastic element stiffness are attenuated following excitation at large strains suggests that at least some part of the undamped series elastic element may reside within the contractile system. Another possibility is that the series elastic element may undergo mechanical yielding or plastic changes when the muscle is excited at high pressures.

In the present experiments, the activated muscle was capable of continuously generating contractile force and increased vessel viscosity for several hours. In previous experiments (14, 15) it was observed that such sustained smooth muscle contraction increased the static elastic modulus of the vascular wall in the circumferential direction. Therefore, it was of interest to determine the stiffness of the continuously activated contractile element. This determination was most easily accomplished for the Maxwell model. In this model, the properties of the parallel connective tissue elements can be excluded by subtraction, as in Figure 2. Then for the remaining elements,

\[
\frac{1}{E_{MUS}} = \frac{1}{E_{CR}} + \frac{1}{E_{SE}},
\]

where \(E_{MUS}\) is the stiffness of the muscle when it is at static equilibrium, \(E_{CR}\) is the stiffness of the contractile element, and \(E_{SE}\) is the stiffness of the series elastic element. At equilibrium, the stress borne by each of these series-coupled elements is identical. Consequently the static stiffness of each of these elements must be related at comparable levels of active stress. \(E_{MUS}\) was obtained by determining the slope of the static strain-active stress curves at each tenth interval of active stress; \(E_{SE}\) was obtained from the Maxwell model data in Figure 4 at corresponding values of active stress. Eq. 7 then was rearranged to solve for \(E_{CR}\).

\[
E_{CR} = \frac{E_{MUS} \times E_{SE}}{E_{SE} - E_{MUS}}.
\]

Maxwell model contractile element stiffnesses are shown in Figure 4. Means ± SE are indicated at each tenth stress interval. The symbols denote the same vessel groups as those for the series elastic element data on the same figure. These data show that the Maxwell model contractile element is only one-fifth to one-sixth as stiff as the Maxwell model series elastic element. These contractile element stiffnesses were computed from series elastic element data and from static muscle stiffnesses obtained after the vessel had achieved equilibrium. Therefore, the contractile element data presented in this figure represent the purely elastic, nonviscous stiffness of the muscle force-generating system. Statistical analysis of these data indicated that the vessels excited at 300 mm Hg exhibited significantly less contractile element stiffness (\(P < 0.05\)) than did all of the other vessels at all comparable levels of active stress, whereas the vessels treated at 225 mm Hg exhibited significantly less (\(P < 0.05\)) series elastic element stiffness than did all of the remaining vessels at some, but not all, levels of active stress. The decrease in contractile element stiffness exhibited by vessels excited at high pressures provides further evidence for the view that there is a decrease in the strength or the number of effective contractile units in these vessels (18).

Discussion

Series Elastic Element.—Carotid smooth muscle series elastic element properties may be compared with values published for other muscles. Table 1 presents comparative data for striated, cardiac, and smooth muscles. Most authors have reported series elastic element extension at maximum observed isometric force. However, the maximum muscle force varied in these different muscle preparations. Therefore, the data shown in Table 1 were normalized by evaluating all of the series elastic element extension data at a single applied stress. Forces were converted to stresses by multiplying the force in grams by 981 cm/sec² and dividing this measure of force by the cross-sectional areas of the tissues. Load-extension curves in each paper then were examined to determine the extension of the series elastic element at an applied stress of \(1.0 \times 10^6\) dynes/cm², a value which fell within the stress range of most published studies. These data show that skeletal and cardiac muscle exhibit similar series elastic element extensions and that smooth muscle exhibits somewhat greater series elastic element extension at the same applied stress. Table 1 also shows variable agreement between series extensions within each muscle type. Some of this variability may reflect the temperatures at which the experiments were performed, and another source of variability may be in the methods employed. For example, Stephens and Kromer (13) found a normalized compliance of 6.0% for the series elastic element of dog trachealis muscle when it was computed by either the quick release method of Wilkie (5) or the \(dP/dt \times dt/dr/dL\) method of Sonnenblick (8). In the same study these authors also found that the trachealis muscle preparation
**Table 1**

Series Elastic Element Characteristics in Three Types of Muscle

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Temperature (°C)</th>
<th>Maximum isometric force (g)</th>
<th>Series elastic extension at maximum isometric force (%)</th>
<th>Cross-sectional area (cm²)</th>
<th>Maximum stress (X 10⁶ dynes/cm²)</th>
<th>Series elastic element extension at 1.0 Hill (%a)</th>
<th>Voigt</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frogsartorius (2)</td>
<td>0</td>
<td>124</td>
<td>3.5</td>
<td>0.066</td>
<td>1.62</td>
<td>3.1</td>
<td></td>
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<tr>
<td>Frogsartorius (5)</td>
<td>0</td>
<td>30</td>
<td>5</td>
<td>0.027</td>
<td>1.09</td>
<td>4.1</td>
<td></td>
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<tr>
<td>Frogsartorius (3)</td>
<td>2</td>
<td>38</td>
<td>2</td>
<td>0.019</td>
<td>1.63</td>
<td>1.3</td>
<td></td>
</tr>
<tr>
<td>Cat tenuissimus (4)</td>
<td>37</td>
<td>18</td>
<td>7</td>
<td>0.013</td>
<td>1.4</td>
<td>6-12</td>
<td></td>
</tr>
<tr>
<td>Rat gracilis anticus (1)</td>
<td>17.5</td>
<td>35</td>
<td>7</td>
<td>0.022</td>
<td>1.56</td>
<td>6.4</td>
<td></td>
</tr>
<tr>
<td>Cat papillary (9)</td>
<td>30</td>
<td>10</td>
<td>4-5</td>
<td>0.0008</td>
<td>1.00</td>
<td>3.3</td>
<td>4.4</td>
</tr>
<tr>
<td>Guinea pig taenia coli (10)</td>
<td>37</td>
<td>13.1</td>
<td>0.0048*</td>
<td></td>
<td>1.8</td>
<td>12.5</td>
<td></td>
</tr>
<tr>
<td>Rabbit taenia coli (11)</td>
<td>22</td>
<td>19-23</td>
<td>0.0075</td>
<td>2.53</td>
<td>22-26</td>
<td>6.0</td>
<td></td>
</tr>
<tr>
<td>Dog trachealis (13)</td>
<td>37</td>
<td>19</td>
<td>7.6</td>
<td>0.075</td>
<td>2.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bovine mesenteric artery (12)</td>
<td>37</td>
<td>15-20</td>
<td>0.075</td>
<td>2.1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dog carotid (present study)</td>
<td>37</td>
<td>8-11</td>
<td>0.040</td>
<td>1.1</td>
<td>11.2</td>
<td>8.6-10.5</td>
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</tbody>
</table>

*Cross-sectional area was provided by direct communication with Dr. A. K. G. Aberg.

Exhibited up to 15% compliance when it was measured by Hill’s (19) method of redevelopment of tension. For another example, Aberg (10) found a value of 12.5% for the normalized series compliance in guinea pig taenia coli when it was measured using a quick release method, whereas Gordon and Siegman (11) found a normalized series compliance of 22-26% for the same tissue when it was measured using the method of redevelopment of tension.

Examination of the data presented in Table 1 indicates that skeletal muscle and cardiac muscle exhibit similar series elastic element compliances and that, by contrast, smooth muscle exhibits a more compliant series elastic element. The greater compliance of the series elastic element in smooth muscle may indicate that the series elastic element may have a different morphological basis in smooth muscle than it does in skeletal or cardiac muscle. Indeed, it is possible that the absolute length of the series elastic element in smooth muscle is greater than that in striated muscle. Still another consideration concerns the viscosity of the contractile element. The measurement of series elastic element stiffness by quick release depends on the assumption that the dynamic, viscoelastic stiffness of the contractile element is so damped that the latter element undergoes negligible retraction during quick release. Therefore, a contractile element in smooth muscle less viscoelastic than that in striated muscles would also result in the appearance of a more compliant series elastic element. Such a difference in the properties of the contractile element has not been shown to date.

**Contractile Element Stiffness.**—There are no published data with which to compare the static contractile element stiffnesses computed for carotid smooth muscle. However, Little and Wead (20) published stress-relaxation data for rabbit papillary muscle that may be utilized for such a comparison. Stresses were measured immediately after rapid stretch and also after 5 minutes of stress-relaxation. If the first step is used as a measure of series elastic element extension, then the delayed step may be used as a measure of extension of both the series elastic and the viscous contractile elements. This procedure assumes a negligible parallel elastic element. The slopes of the stress-strain curves of these data were used to compute the stiffness of the contractile element. The maximum contractile stress generated by these papillary muscles was only $0.12 \times 10^6$ dynes/cm², and at this active stress the contractile element stiffness was $2 \times 10^6$ dynes/cm². This value is comparable to that determined for the smooth muscle contractile element in the present study at low levels of active stress (Fig. 4).

The relatively low stiffness of the Maxwell model contractile element raises the possibility that an applied load may extend the contractile element disproportionately more than it extends either the stiffer parallel elastic element or the vessel as a whole. This possibility is illustrated diagrammatically for a Maxwell model in Figure 6.
Diagram showing relative extension of each element of the Maxwell model, assuming a stiff series elastic element and a relatively compliant contractile element. L = absolute length, e = strain, PE = parallel elastic element, SE = series elastic element, and CE = contractile element.

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References


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