Effect of Sympathetic Tone on Pressure–Diameter Relation of Rabbit Mesenteric Veins In Situ

Keizaburo Ozono, Zeljko J. Bosnjak, and John P. Kampine

Although venous capacitance has been studied in the neurally isolated tissue or in the in vitro vein segment, this is the first study of sympathetic regulation of the pressure–diameter relation in mesenteric veins in situ, where innervation is kept intact. In 25 α-chloralose–anesthetized rabbits, mesenteric vein diameter (679±27 μm, ranges of 380–1,050 μm at initial state) and intravenous pressure were measured continuously at the same site by using videomicrometer and micropressure systems. Intravenous pressure was increased in a stepwise fashion from the baseline of 6–9 mm Hg to ~10, ~13, ~16, ~19, and occasionally to ~22 or ~26 mm Hg by occluding the portal vein with a pneumatic occluder. Each intravenous pressure was maintained for 90–120 seconds or 4–5 minutes until the diameter increase reached a plateau. Pressure–diameter curves were generated for the control state, during celiac ganglion stimulation, and during local tetrodotoxin or intravenous hexamethonium administration. Diameter was plotted as a function of pressure, and the curves were nonlinear or sigmoid. These results are different from the linear or curvilinear characteristics of the pressure–diameter or pressure–volume relation observed in the pharmacologically or chemically denervated preparation. Tetrodotoxin and hexamethonium attenuated the sigmoid shape of the pressure–diameter curve and shifted it toward the diameter axis of the curve. On the other hand, celiac ganglion stimulation did not change the sigmoid nature of the curve but shifted the curve toward the volume axis. Relative areas between celiac ganglion stimulation and control curves, tetrodotoxin and control curves, hexamethonium and control curves, and the two control curves were ~92.6±18.8 (p<0.01), 80.6±22.0 (p<0.05), 92.4±37.3 (p<0.01), and 65±4.9, respectively. These results demonstrate that the pressure–diameter relation is significantly controlled by sympathetic tone to these veins. (Circulation Research 1991;68:888–896)

Reflex control of venous capacitance plays an important role in regulation of cardiac output by affecting the filling of the right heart.1–3 Splanchnic circulation accounts for approximately 25% of the total blood volume and can be regulated by various reflex mechanisms.4–8 The capacitance of the splanchnic circulation depends on both the compliance of the system (slope of the pressure–volume curve) and the unstressed vascular volume (volume-axis intercept of the pressure–volume curve).3 Most of the studies of the pressure–volume or pressure–diameter (P-D) relation of the vein have been done in neurally denervated tissue9–12 or in the in vitro vein segment.13–15 These studies reported that the pressure–volume or P-D relation was linear or curvilinear in the range of 4–8 to 20–35 mm Hg. Neural control of the pressure–volume relation could not be determined in these preparations, although Vanhoutte et al14 showed the effect of electrical field stimulation. In an early work, Alexander14 reported the nonlinearity of the pressure–volume relation and the effects of various reflexes, but the results are difficult to interpret because only injected volume was measured and the stress-relaxation of the vein had not been overcome.

The purpose of this study was to demonstrate the P-D relation of small mesenteric veins and evaluate the effect of sympathetic tone in the preparation where sympathetic innervation was kept intact. We used a simultaneous recording system for the mesenteric vein diameter and intravenous pressure at the same site in the in situ preparation and found

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different characteristics of the P-D relation from that of the denervated preparation and could demonstrate the effect of changes in sympathetic tone.

Materials and Methods

The preparation and the measurement techniques used in this study are similar to those previously described.8

Surgical Preparation

Twenty-five male New Zealand White rabbits weighing 1.0–1.5 kg were anesthetized via the ear vein with thiamylal (25 mg/kg) and α-chloralose (40 mg/kg) after a 24-hour fast. A steady plane of anesthesia was maintained by a continuous infusion of α-chloralose (15–20 mg·kg⁻¹·h⁻¹). After tracheotomy, each animal was ventilated artificially (model 665, Harvard Apparatus, South Natick, Mass.) with room air. The Po₂, Pco₂, and pH were measured at intervals and maintained within the normal range (80–100 mm Hg, 30–40 mm Hg, and 7.35–7.45, respectively) by adjusting the ventilator setting and giving NaHCO₃ continuously (1 meq/hr). Rectal temperature was monitored with a thermistor probe and maintained at 36.5–37.5°C during the surgical and experimental procedures by using a water-perfused heating mat. Polyethylene catheters were placed in the abdominal aorta through the femoral artery to measure the aortic pressure and in the femoral vein for the injection of the anesthetics. An 8-cm laparotomy was performed via a midline incision. A pneumatic occluder was placed around the portal vein adjacent to the liver. In nine rabbits, a pair of silver electrodes was attached to the celiac ganglion for electrical stimulation. The incision was partially closed, leaving a 5-cm opening for exposure of a gut loop. After these surgical preparations, the animal was placed on a specially designed stage. As depicted in Figure 1, a segment of ileum —13 cm long was exteriorized and mounted gently in a superfusion chamber. The gut and the mesentery were superfused constantly with warmed physiological salt solution (37–38°C, pH 7.35–7.45). As described by Bohlen,16 the superfusate contained (mM) NaCl 118.4, KCl 5.9, CaCl₂ 3.3, and NaHCO₃ 25.0 and was bubbled continuously with 5% O₂–5% CO₂–90% N₂. The mesentery was pinned down to the clear Sylgard (Dow Corning Co., Midland, Mich.) base with 75-μm-diameter pins at several points along the vessels to prevent the transmission of gut movement. The tissue was allowed to equilibrate in the superfusate for 30 minutes before initial measurements were made. All measurements were taken within 6 hours after the start of the superfusion.

Measurement of Vein Diameter and Intravenous Pressure

Continuous, on-line video measurements of vein diameter were obtained with an RCA 2000 video camera mounted on the sidearm of a Reichert Zoom microscope (Reichert-Jung Optische Werke Ag, Vienna). The system was described in detail by Bell et al.17 The video-derived diameter was calibrated with the use of three different wires of known diameter. A Model 900 micropressure system (World Precision Instruments, New Haven, Conn.) was used to record the intravenous pressure at the same site as that of the vein diameter measurement. This is an application of the servo-null micropipette pressure-measuring system described by Wiederheilm et al18 and Intaglia et al.19,20 Glass micropipettes, whose tips were ~10 μm in outer diameter, beveled to a point with a diamond grinder and filled with 2 M NaCl, were

FIGURE 1. Schematic presentation of experimental setup for simultaneous recording of vein diameter and intravenous pressure in situ. Pneumatic occluder was placed around the portal vein adjacent to the liver. PSS, physiological salt solution. See text for details.
used as sensing electrodes. Continuous analog tracings of video-derived diameter and pressure transducer output were obtained on a Model MT-9500 recorder (Astro-Med, Inc., West Warwick, R.I.).

**Experimental Protocol**

**Controls.** After aortic pressure, heart rate, vein diameter, and intravenous pressure had stabilized, mesenteric venous pressure was increased from the baseline of 6–9 mm Hg to ~10, ~13, ~16, ~19, and occasionally to ~22 or ~26 mm Hg by occluding the portal vein with a pneumatic occluder. Each pressure was maintained for 90–120 seconds generally or for 4–5 minutes occasionally. Every effort was made to wait until the vein diameter had plateaued before making pressure increases, while keeping the time between the pressure jumps approximately the same for each animal. By plotting each plateau vein diameter as a function of corresponding intravenous pressure, the P-D relation curve was obtained. After acute deflation of the pneumatic occluder, the pressure inside the mesenteric vein returned to the baseline level instantly, while the dilated vein diameter returned very slowly (in 15–60 minutes). The vein diameter returned to the range of ±5% of the initial value before the next P-D curve was generated or the sympathetic ganglion stimulation was applied. If the dilated vein diameter failed to return to ±5% of initial value, the data were excluded from the analysis. To prevent large variations in baseline vein diameter caused by hysteresis, the first occlusion was not used. Two or three consecutive control curves were generated before sympathetic interventions to show the reproducibility of the P-D curve in nine rabbits.

**Sympathetic interventions.** Electrical celiac ganglion stimulation (CGS, 10 Hz) was performed to increase sympathetic tone to mesenteric veins in nine rabbits; tetrodotoxin (TTX, 50 μg) was administrated topically by applying it as a bolus to the outside of the mesenteric veins in nine rabbits to block local innervation of the veins. We found that this amount of TTX is necessary to be effective in blocking the response to sympathetic nerve stimulation over 10–12 minutes. TTX was added close to the mesenteric vein bathed with approximately 20 ml Krebs’ solution with a turnover rate of 15 ml/min, thereby decreasing the TTX concentration over time. An intravenous injection of hexamethonium (HEX, 20 mg/kg) was given in four rabbits to block ganglionic transmission. Effectiveness of each intervention was evaluated by the response of the vein diameter. Criteria for the effective intervention were >5% venoconstriction in CGS and >3% venodilation in the presence of TTX or HEX.

**Data Analysis and Statistics**

Vein diameters were expressed as percentages of maximum vein diameter in each experiment. To quantify the shift of the P-D curve caused by the change of sympathetic tone, the area between two curves (i.e., before and after sympathetic interven-

<table>
<thead>
<tr>
<th>Baseline Vein Diameter and Intravenous Pressure of Each Group Before Intervention</th>
<th>Vein diameter (μm)</th>
<th>Intravenous pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (n=11)</td>
<td>676±43</td>
<td>8.1±0.3</td>
</tr>
<tr>
<td>CGS (n=9)</td>
<td>739±54</td>
<td>8.9±0.3</td>
</tr>
<tr>
<td>TTX (n=9)</td>
<td>607±28</td>
<td>8.3±0.3</td>
</tr>
<tr>
<td>HEX (n=4)</td>
<td>737±127</td>
<td>9.0±0.3</td>
</tr>
<tr>
<td>Average (n=33)</td>
<td>679±27</td>
<td>8.5±0.2</td>
</tr>
</tbody>
</table>

Values are mean±SEM. n represents the number of observations. None of the values above are statistically different from each other by analysis of variance. CGS, celiac ganglion stimulation; TTX, tetrodotoxin; HEX, hexamethonium.

tion, or between two consecutive controls) was measured within a specified range of 8–22 mm Hg of intravenous pressure by using a digitizer (Sonic digitizer, Science Accessories Corp., Stratford, Conn.). This range was chosen because overall baseline intravenous pressure between groups were 8.5±0.2 mm Hg, and most curves reached plateau before 22 mm Hg. Areas were the product of intravenous pressure and vein diameter (percent of the maximum vein diameter). Left and upward shift was defined as a positive shift, whereas right and downward shift was defined as a negative shift. To characterize the P-D curve, initial slope, the steepest slope, and the vein diameter at resting state (at 8 mm Hg intravenous pressure) were measured and compared. Initial slope was defined as the slope before the P-D curve shows the dramatic change of compliance. In the curves that lack such a dramatic change in slope (see Figure 3, group A), the slope between the initial state and the change in slope (usually, but not always at 13–14 mm Hg) was used for comparison. Regardless of the method used, the steepest slopes were significantly different among the three groups. To quantify the sigmoid shape or dramatic change of compliance of the P-D curve, the difference of the initial slope and the steepest slope (S-I) was calculated and compared. Area between the two curves was analyzed by one-way analysis of variance and Duncan’s test. Values of the vein diameter at resting state, initial slope, the steepest slope, and S-I were analyzed by two-way analysis of variance and further individual comparison. The level of significance was taken at p<0.05.

**Results**

The averages for the diameters and the pressures of the veins studied were 679±27 μm and 8.5±0.2 mm Hg, respectively, at the initial state (ranges of 380–1,050 μm and 6.6–10.2 mm Hg) (Table 1). None of the initial diameters or pressures of any subgroup were statistically different from each other by analysis of variance.

**Pressure–Diameter Curve Without Sympathetic Interventions**

A typical example of P-D curve generation is shown in Figure 2. Mean aortic pressure decreased
(-14.7±2.0 mm Hg, n=29) and heart rate increased slightly (11.4±1.9 beats/min, n=25) as portal vein occlusion progressed. While intravenous pressure was increased from a baseline of 6–10 mm Hg to the maximum of 19–26 mm Hg, the amount of vein diameter increase and the shape of the P-D curve were quite variable. Therefore, as shown in Figure 3, all of the 37 P-D curves before sympathetic interventions are divided into three groups of equal number according to the initial state of vein diameter before portal vein occlusion; that is, group A, n=12; group B, n=12; and group C, n=13. Group A had the largest (88.7±0.8%), group B the intermediate (83.1±0.5%), and group C the smallest (75.5±0.9%) initial vein diameters (percent of the maximally dilated vein diameter). In group A, the initial diameter was a larger percentage of maximum vein diameter compared with those of the other groups. The absolute initial diameter for groups A, B, and C was 739±54, 667±43, and 607±28 μm, respectively. These subgroups probably indicate relative differences in resting sympathetic tone of the veins. In group C, the vein diameter increases were relatively small in the lower portion of the pressure range, vein diameter started to increase dramatically as a threshold pressure was reached, and then diameter plateaued. The resultant P-D curve was sigmoid in shape. In group A, for which initial vein diameters were larger, P-D curves tended to lose the sigmoid shape. Group B manifested the intermediate patterns between groups A and C. When HEX was given systemically or TTX was applied locally, group B and C curves did shift toward the pattern shown for group A. HEX and TTX administration also shifted the

![Figure 2](http://circres.ahajournals.org/) 

**Figure 2.** Pressure–diameter relation of mesenteric vein in situ. Changes in aortic pressure (AP), heart rate (HR), vein diameter, and intravenous pressure during stepwise occlusion of the portal vein in the control state.

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**Table 2. Areas Between the Two Curves (i.e., Before and After Each Intervention, or Two Controls), Vein Diameter at 8 mm Hg (Resting Vein Diameter), Initial Slope, Steepest Slope, and the Difference Between Slopes**

<table>
<thead>
<tr>
<th>Area between two curves (pressure×diameter)</th>
<th>Vein diameter at 8 mm Hg (%)</th>
<th>Initial slope (%/mm Hg)</th>
<th>Steepest slope (%/mm Hg)</th>
<th>S-I</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (n=11)</td>
<td>6.5±4.9</td>
<td>84.3±1.7</td>
<td>84.4±1.5</td>
<td>0.7±0.2</td>
</tr>
<tr>
<td>CGS (n=9)</td>
<td>-92.6±18.8*</td>
<td>84.0±1.8</td>
<td>75.6±2.6†</td>
<td>0.4±0.3</td>
</tr>
<tr>
<td>TTX (n=9)</td>
<td>80.6±22.0$§</td>
<td>80.1±2.0</td>
<td>89.5±1.6†</td>
<td>0.5±0.2</td>
</tr>
<tr>
<td>HEX (n=4)</td>
<td>92.4±37.3*</td>
<td>79.9±4.5</td>
<td>85.6±3.2†</td>
<td>0.0±0.1</td>
</tr>
</tbody>
</table>

Values are mean±SEM. n represents the number of observations. S-I, difference between the steepest slope and the initial slope; CGS, celiac ganglion stimulation; TTX, tetrodotoxin; HEX, hexamethonium.

* p<0.01 compared with control by one-way analysis of variance.

†p<0.05 compared with corresponding values of the group before intervention by two-way analysis of variance and simple effect test.

§p<0.05 compared with corresponding values of the group before intervention by two-way analysis of variance and simple effect test.

$ p<0.05$ compared with control by one-way analysis of variance.
P-D curve of group A, although the changes were much less than those seen for groups B and C. Figure 4 shows S-I, which indicates the sigmoid shape or dramatic change of compliance, as a function of initial vein diameter, according to groups A, B, and C. S-I values for groups B and C were significantly different from that of group A. Hence, initial tone of the vein plays an important role in the shape of P-D curves. Despite this variation in different vessels, however, P-D curves were reproducible in an individual vessel, as shown in Table 2. Areas between two consecutive P-D curves without sympathetic interventions were substantially small in 11 observations from nine rabbits, given the criteria for return to baseline described above.

**Effect of Celiac Ganglion Stimulation**

Continuous CGS at 10 Hz caused a significant decrease in vein diameter (−67±6 μm or −9.4±1.1%) with no significant change in intravenous pressure (0.5±0.3 mm Hg). Subsequent portal vein occlusion during CGS resulted in a P-D curve that was shifted to the right and downward (Figure 5B). Specifically, vein diameter was smaller than that of the control state at any given intravenous pressure except in the maximally dilated state. The area between the two curves (before and during CGS) was statistically different from that between the two control curves (p<0.01). CGS significantly decreased the resting vein diameter (p<0.05) but did not change the initial slope (Table 2). In addition, CGS enhanced the steepest slope and S-I.

**Effect of Tetrodotoxin**

As shown in Figure 6, topical application of TTX did not cause any change in aortic pressure, heart rate, or intravenous pressure but increased vein diameter significantly (67±12 μm or 11.8±2.6%, n=9). Subsequent portal vein occlusion resulted in a P-D curve that was shifted to the left and upward (Figure 5C). Vein diameter was larger than for the control state at any given intravenous pressure except in the maximally dilated state. The area between the two curves (before and after TTX) was statistically different from that between the two control curves (p<0.01). Furthermore, TTX attenuated the abrupt increase in the slope of the curve (S-I) (p<0.05). Finally, TTX significantly increased resting vein di-
Effect of maximal diameter

Curves were

5.

FIGURE

P-D

Heart pressure, not change (p<0.01) (-11

and downward

Injection curve (CGS) (panel B),

tion

5.

Typical examples of pressure-diameter curves in the control group (panel A), during celiac ganglion stimulation (CGS) (panel B), and during tetrodotoxin treatment (TTX) (panel C). Vein diameters were expressed as percent of maximal diameter before each intervention. Areas between the curves were measured in the range of 8 and 22 mm Hg. Left and upward shift was defined as a positive shift (+), whereas right and downward shift was defined as a negative shift (−). Measurements of the initial slope, steepest slope, and diameter at 8 mm Hg are also shown.

mm Hg, respectively), yet increased the vein diameter (59±16 μm or 10.2±4.3%, n=4). The effect of HEX on the P-D curve was similar to that of TTX. The area between the two curves (before and after HEX) was statistically different from that of the two control curves (p<0.01). HEX decreased the steepest slope and S-I (p<0.01); therefore, the shape of the P-D curve was less sigmoid. Resting vein diameters were significantly increased after HEX (Table 2). HEX did not change the initial slope significantly.

Figure 7 is a summary of the areas between the two curves obtained before and after each innervation, which indicates significant shifts of the P-D curve.

Discussion

We have shown that the P-D relation is not linear across the physiological range of 6–26 mm Hg in neurally intact mesenteric veins. With higher sympathetic tone, vessel compliance was relatively low in the ~6–14 mm Hg range. This stiffness persisted up to some threshold pressure at which point the vessel suddenly, or sometimes gradually, increased in diameter. At even higher pressures, veins became stiff again and reached a plateau. This morphological characteristic of the P-D curve was not observed in previous studies of neurally isolated tissue or in vitro vein segments.9–12 Moreover, functional denervation of the vein with TTX changed the P-D relation dramatically (Figure 5C). Vein diameter was larger in the resting state, and the diameter increase followed the pressure increase in a curvilinear fashion. The sudden dilation of the vessel or the drastic change of compliance was attenuated in the presence of TTX. The P-D curve tended to lose its sigmoid shape, and it became curvilinear. This corresponds to the observations obtained in the neurally isolated or in vitro preparation. Finally, the whole curve was shifted to the left and upward during sympathetic block with TTX or HEX. On the other hand, elevated sympathetic tone during CGS produced the same abrupt increase in vein diameter and the same sigmoid shape, yet the P-D curve was shifted to the right and downward (Figure 5B).

Even without sympathetic interventions, the patterns of P-D curves were variable, as shown in Figure 3, which indicates that the sympathetic tone to those veins was quite variable under control conditions. In spite of that variability of the control P-D curves, by pairwise comparisons (by measuring the area between the two curves, i.e., before and after intervention or two consecutive controls), we demonstrated that CGS, TTX, and HEX change the P-D curve significantly because the P-D curves were highly reproducible in individual veins.

The anatomic aspect responsible for such a sudden increase in venous compliance during the course of passive distension is not known. In the present study of the mesenteric vein, a sudden increase in venous compliance was seen at 16 to ~19 mm Hg, and occasionally at 21 mm Hg in the control group C and during CGS. It was not observed after TTX applica-
stimulation or administration of HEX. Therefore, sympathetic stimulation may play a major role in this phenomenon. Sympathetic innervation of smooth muscle in small veins may provide not only a mechanism to produce changes in stored volume but also a means of protecting against undesirable expansion of stored volume when transmural pressures rise in these thin-walled vessels.

Compliance and unstressed volume are the two important characteristics of the capacitance curve as it relates to the volume change at different pressures or in the presence of different vessel tones. Unstressed volume is defined as the volume of blood in a vessel at zero transmural pressure and is an important component of the blood volume in the vessel. This is a virtual volume estimated by extrapolating a linear portion of the capacitance curve to zero transmural pressure. Because the P-D curve could not be obtained in the pressure range lower than the initial state in the present study, we used the vein diameter at 8 mm Hg (resting vein diameter) as an index for evaluating the vessel volume at the resting state. CGS reduced the resting vein diameter, and TTX and HEX increased it significantly (*p<0.01) (Table 2).

Compliance is the ratio of the change in volume resulting from a change in transmural distending pressure. It is expressed as the slope of the pressure-volume relation at a specified point of the curve. In the present study, compliance varied substantially according to the pressure because the P-D curve was not linear. When the vessel dilates, compliance becomes maximum. After blocking sympathetic input to the vessel by TTX, sudden dilation was not observed in most cases. These changes resulted in the significant difference in the steepest slope and also in the S-I before and after TTX or HEX (Table 2). Before the vessel suddenly dilates, compliance is relatively small, and CGS, TTX, and HEX did not change it.

**FIGURE 6.** Effect of tetrodotoxin (TTX) on the pressure–diameter relation of mesenteric vein in situ. Changes in aortic pressure (AP), heart rate (HR), vein diameter, and intravenous pressure after local administration of TTX and during subsequent stepwise occlusion of the portal vein in the same vein as for Figure 2. Local administration of TTX increased vein diameter significantly, while intravenous pressure remained unchanged. Note the difference in vein diameter increase from Figure 2. PSS, physiological salt solution.

**FIGURE 7.** The areas between the two curves (i.e., before and after sympathetic intervention, or between two consecutive controls) are expressed as relative units obtained by multiplying intravenous pressure and vein diameter (percent of maximum vein diameter). Shift in the pressure–diameter curves by celiac ganglion stimulation (CGS), tetrodotoxin (TTX), and hexamethonium (HEX) were statistically significant as compared with the shift between two controls according to analysis of variance and Duncan’s test (*p<0.05, **p<0.01). Each bar represents mean±SEM. n represents the number of observations.
significantly (Table 2). Thus, in the lower range of intravenous pressure (6–16 mm Hg), which is considered to be more physiological, resting volume rather than the compliance seems to play a more important role in sympathetic regulation of the venous capacity.

A change in the diameter of the vessel would generally indicate a change in its volume except at the very low distending pressure at which the vein may be elliptical.\textsuperscript{21} We hypothesized that the vein diameter can provide a quantitative index of its volume in the present study because distending pressure was increased but never decreased from the resting level. If the radius of the vein changes from \( r_0 \) to \( r \) and the length is constant, the ratio for volume change is \( (r/r_0)^3 - 1 \). According to this formula, resting vein diameter changes of \(-9.4\pm1.1\%\), \(11.8\pm2.6\%\), and \(10.2\pm4.3\%\) produced by CGS, TTX, and HEX correspond to \(-17.9\pm1.9\%\), \(25.5\pm5.9\%\), and \(22.0\pm9.6\%\) changes in volume, respectively.

Creep is the gradual extension of tissue length at constantly applied load and is related to the viscoelastic properties of blood vessels. The magnitude of the creep correlates well with the amount and activity of smooth muscle in the tissue.\textsuperscript{22} To avoid this effect on the P-D relation, we waited for 90–120 seconds or for 4–5 minutes occasionally until the vein diameter reached a plateau each time after the new distending pressure was applied. In general, vascular tissues exhibit greater pressure at any diameter or smaller diameters at any pressure, during extension than during retraction. This is called hysteresis and is another phenomenon that is associated with the viscoelastic property of blood vessels. It is most prominent in the first cycle of pressure–volume generation.\textsuperscript{23} To minimize its effect, the P-D curve generated in the vessel for the first time was excluded from the analysis. In further repeated cycles, the ascending or loading limb of the loop is more shifted than in the descending or unloading limb of the loop.\textsuperscript{23} However, we used the ascending limb to study the sudden dilation of the vein during distension, which we found to be characteristic of the neurally intact preparation. Nonetheless, P-D curves were quite reproducible if they satisfied the criteria for the initial vein diameter described above. The area between the two consecutive control curves was relatively small (Figure 6 and Table 2).

Monos et al\textsuperscript{13} demonstrated the contribution of myogenic activity to capacity control in short-term pressure load in rat saphenous vein in vitro by analyzing stress, strain, and smooth muscle membrane potential. Although some myogenic activity could have influenced the generation of P-D curves in each condition, the significant shift of the P-D curve that we observed was probably not due to the change in myogenic activity because the stepwise pressure increase was applied in the same manner during each condition (CGS, TTX, and HEX).

In earlier investigations, it was shown that an elevation of the portal vein pressure increases the vascular resistance of the intestine (the venous-arteriolar response).\textsuperscript{2} The mechanism of this phenomenon is controversial. In the present experiment, this response did not affect the result directly because intravenous pressure was regulated independently from the arteriolar resistance.

In the present study, we demonstrated that the P-D relation of the mesenteric veins is nonlinear or sigmoid in the preparation where sympathetic innervation is kept intact. These results are different from the linear or curvilinear characteristics of P-D or pressure–volume relations observed in the denervated preparation. A decrease in sympathetic tone attenuated the sigmoid shape of the P-D curve and shifted it toward the diameter axis of the curve. On the other hand, an increase in sympathetic tone did not affect the sigmoid nature and shifted the curve toward the volume axis. These results suggest that mesenteric venous capacitance is well regulated by sympathetic tone.

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References


KEY WORDS • videomicrometer system • venous tone • tetrodotoxin • servo-null micropipette pressure-measuring system • venous capacitance
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