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Longitudinal Tension of Anterior Tibial Artery Segments during Constrictor Responses[1] (39355)

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We have previously shown (1, 2) that isolated segments of anterior tibial arteries of the dog underwent marked decreases in diameter when stimulated via their sympathetic innervation. The purpose of the present study was to determine if appreciable changes in longitudinal tension occurred in these vessels during vasoconstrictor responses. If longitudinal tension is increased during vasoconstrictor responses, appreciable changes in the architecture of the vascular bed could occur. Data on longitudinal tension changes occurring during vasoconstriction could also provide further insight into vascular smooth muscle mechanics.

Methods. Experiments were conducted on normally tethered and normally innervated anterior tibial artery segments of dogs. They were premedicated with morphine (3-5 mg/kg) anesthetized with sodium pentobarbital (20 mg/kg) and heparinized at 5 mg/kg. Arterial segments were isolated from the remainder of the limb vasculature by inserting nylon catheters upstream and downstream in the vessel and advancing the catheters until their tips were approximately 1 cm apart. This procedure produced a length of anterior tibial artery which was free of collateral channels except for vasa vasorum.

Segments were perfused with blood from the femoral artery of the contralateral leg with a constant output pulsatile pump. Outflow from the isolated segment was returned to the femoral vein of the contralateral limb. Resting intraluminal pressures were adjusted by a variable resistance on the outflow tubing. The perfusion circuit has been described in more detail previously (2).

Arterial segment inflow pressure was recorded through the side arm of a plastic T-tube with a P23G Statham pressure gauge. The T-tube was placed in the inflow circuit just upstream from the nylon catheter and was firmly positioned to prevent movement or stress from being exerted on the vessel segment. Outflow pressures were recorded through another plastic T-tube placed just distal to the outflow nylon catheter in the outflow circuit. The outflow T-tube was connected through a length of small wire to a Grass FT03 force transducer to record changes in axial tension. Springs were removed from the force transducer to provide maximum sensitivity. Care was taken to keep the blood vessel segment at its original length.

Vasoconstrictor responses were produced by stimulation of the superficial fibular nerve, by infusion of levarteranol at 1-2 μg/min into the inflow circuit, and by stimulation through platinum wire electrodes inserted into the blood stream at the inflow and outflow T-tubes. Succinylcholine chloride (2 mg/kg) prevented skeletal muscle contraction during superficial fibular nerve stimulation. Vasoconstrictor responses were recorded under constant inflow perfusion, under constant pressure perfusion, and at constant intraluminal pressure under constant pressure perfusion when the outflow circuit was occluded. Changes in longitudinal tension were also recorded when intraluminal pressures were passively changed by altering the outflow resistance under constant inflow perfusion.

Under constant inflow perfusion the entire output from the constant output pump was directed through the vascular segment. With inflow held constant, the inflow pressure and the pressure drop across the segment varied directly with vessel segment resistance. Under constant pressure perfusion a side arm containing a Starling resistor was opened to the inflow tubing at a site be-

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tween the pump and the vessel segment. As the resistance of the isolated vessel segment increased during vasoconstrictor responses, a greater portion of the pump output flowed through the parallel circuit containing the Starling resistor. Thus, small changes in vessel inflow pressure occurred during vasoconstrictor responses under constant perfusion. Complete occlusion of the outflow tubing under constant pressure perfusion directed all the output from the pump through the parallel circuit containing the Starling resistors with no flow or pressure change in the isolated vessel segment as it underwent constriction.

Results. Figure 1A shows inflow and outflow pressures and longitudinal tension changes of an isolated segment during superficial fibular nerve stimulation (15 Hz) under constant inflow perfusion. Sympathetic stimulation produced marked vasoconstriction as indicated by the increase in inflow pressure from a control level of 115 to 300 mm Hg at peak response. Outflow pressure, as would be expected under constant inflow perfusion, showed no significant change. The pressure gradient across the segment increased from 15 to 200 mm Hg as a result of increased vessel resistance. The longitudinal tension decreased by approximately 2 g.

Figure 1B shows the response of the same preparation to superficial fibular nerve stimulation under constant pressure perfusion at the same stimulation parameter as in Fig. 1A. Sympathetic stimulation produced a small increase in inflow pressure of 10 mm Hg. Outflow pressure decreased by 30 mm Hg, thus a small decrease in mean pressure occurred. A small decrease in longitudinal tension during the stimulation period was followed by an increase during the recovery period. Thus a relatively small change in longitudinal tension occurred during vasoconstriction when changes in intraluminal pressure were small.

Figure 2A illustrates the effect on longitudinal tension of passively increasing intraluminal pressure by partially occluding the outflow tubing under constant inflow perfusion. The increased intraluminal pressure produced a passive dilation of the vessel segment as indicated by the decreased difference between inflow and outflow pressures. Longitudinal tension decreased markedly with increased intraluminal pressure.

Figure 2B shows the response of the same segment to superficial fibular nerve stimulation under constant pressure perfusion, with the outflow tubing occluded to prevent any change in intraluminal pressure. Sympathetic stimulation then produced insignificant changes in longitudinal tension, although the diameter of the vessel was pre-

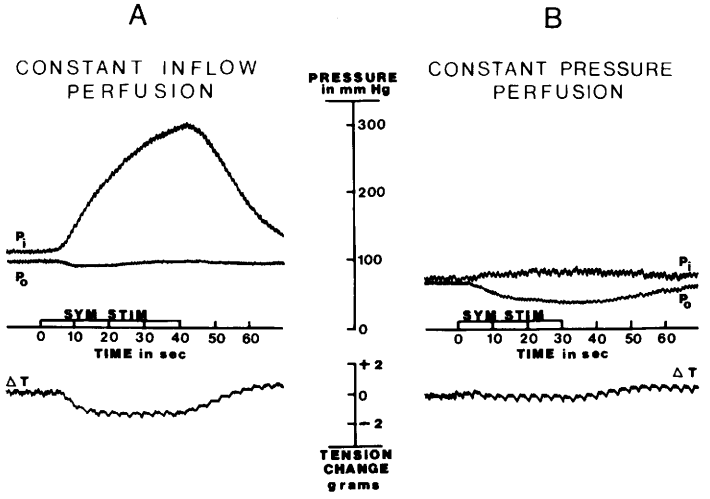

FIG. 1. Panel A shows response to superficial fibular nerve stimulation at 15 Hz, under constant inflow perfusion. Panel B shows response to same stimulus, under constant pressure perfusion. P_i and P_o represent inflow and outflow pressures, respectively. T represents changes longitudinal tension.

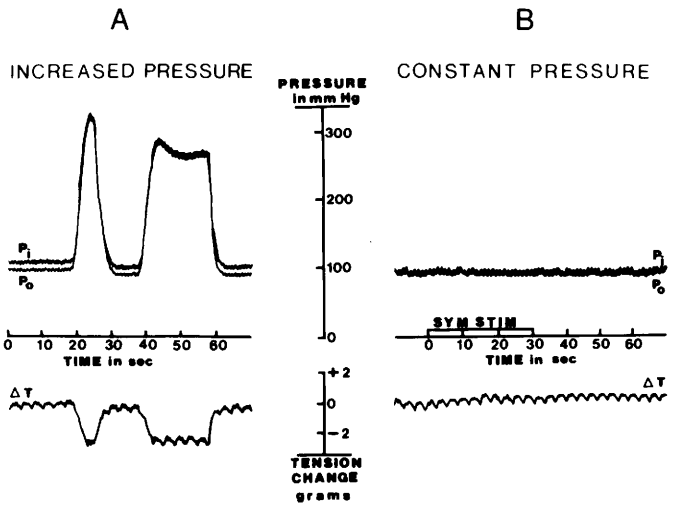

FIG. 2. Panel A shows response to partial occlusion of the outflow tubing under constant inflow perfusion. Tracings are arranged as in Fig. 1. Panel B shows response to superficial fibular nerve stimulation under constant intraluminal pressure. Constant intraluminal pressure was achieved by completely occluding outflow tubing under constant pressure perfusion.

sumably decreased as in the previous constrictor responses.

Five of the seven animals exhibited responses similar to those described for Figs. 1 and 2. The remaining two animals showed a greater tendency toward increased longitudinal tension during vasoconstrictor responses as shown by increases in longitudinal tension during constrictor responses under constant inflow and constant pressure perfusion. Changes in longitudinal tension of arterial segments from each animal to sympathetic stimuli, infusions of levarterenol, and increases in intraluminal pressure are shown in Table I. The mean of responses to each procedure for each animal are shown. Tension changes are expressed in grams for each procedure except for the increases in intraluminal pressure where directional changes were indicated. As will be described in the Discussion, these differences in response may depend on variations in orientation of smooth muscle elements in individual arteries. Similar changes in longitudinal tension as described for those obtained in response to superficial fibular nerve stimulation were obtained during infusions of levarteranol and during stimulation via electrodes in T-tubes upstream and downstream from the vessel.

Discussion. Relatively small changes in longitudinal tension occurred during vasoconstriction responses of arterial segments. Tension changes were in some cases positive and in other cases negative. Tension changes appeared to be the result primarily of changes in intraluminal pressure. The importance of intraluminal pressure changes was indicated by the relatively large decreases in longitudinal tension produced by passively increasing intraluminal pressure, and by the fact that during vasoconstrictor responses longitudinal tension changes varied with the magnitude of the intraluminal pressure change. During vasoconstrictor responses under constant inflow perfusion, with elevated intraluminal pressures, longitudinal tensions usually decreased. Under constant pressure perfusion and when intraluminal pressure was held constant, small changes in longitudinal tension occurred.

The results of the present study are substantiated by previous comparisons of circumferential and longitudinal stress relationships of blood vessels. Little information has, however, been available on longitudinal tension or length changes occurring during vasomotor responses. Most information applicable to this problem comes from comparisons of distensibility characteristics of relaxed and constricted blood vessels in *in vitro* preparations. Dobrin and Doyle (3, 4)

TABLE I. LONGITUDINAL TENSION CHANGES OF ANTERIOR TIBIAL ARTERY SEGMENTS.

	Sympathetic stimulation[a]			Levarterenol infusion[b]			Increased intraluminal pressure	
Dog number	Perfusion method[c]	Tension change in grams[d]	Number of times studied	Perfusion method		Tension change in grams	Number of times studied	Tension change in grams	Number of times studied
1	CIP	−2.3	3	CIP	−	2.5	3		
2	CIP	−1.4	2					−	3
	CPP	0	3						
3	CIP	+0.6	5	CIP	+	0.9	1	−	1
	CPP	+0.3	2						
4	CIP	−1.4	1					−	1
	CPP	0	3						
5	CIP	−0.9	2					−	1
	CPP	−0.3	3						
6	CIP	+1.3	1	CIP	+	1.8	1	−	1
		−0.7	1	CPP	+	1.8	2		
	CPP	+0.4	1						
		−0.6	2						
7	CIP	−0.7	3	CIP	−	0.5	2	−	1
	CPP	−1.0	2	CPP	−	0.3	3		

[a] Produced by stimulation of the superficial fibular nerve or by electrodes inserted into blood stream as described in text.

[b] Levarterenol infused into inflow circuit at rate of 1–2 μg/min.

[c] CIP denotes constant inflow perfusion; CPP denotes constant pressure perfusion.

[d] (−) denotes decreased longitudinal tension; (+) denotes increased longitudinal tension; and (0) denotes negligible change in longitudinal tension. Values represent mean for that animal.

from studies on excised dog carotid arteries reported that activation of smooth muscle elements did not significantly alter the longitudinal elastic modulus. They reported that total longitudinal stress could be calculated as the sum of two components. One was the longitudinal stress due to pressure. The other was attributed to longitudinally applied traction. Vonderlage (5) from studies on strips cut at different angles from abdominal aorta of rabbits reported that distensibility characterisitics of strips cut parallel to the longitudinal axis were changed little after treatment with arterenol.

In our experiments both the circumference and length of vessels were increased by passive increases in intraluminal pressure. Appreciable increases in circumference were indicated by the decreased difference between inflow and outflow pressures under constant inflow as intraluminal pressure was passively elevated. Increased vessel length was indicated by the abrupt fall in longitudinal tension with increased intraluminal pressure. Previous studies also substantiate these findings. Dobrin and Rovick (6) and Bergel (7) showed that circumferential strains over physiological pressure ranges could produce increases of approximately 100% in vessel radius. Although the problem seems to be still unresolved as to whether or not arterial vessel walls are exactly isotropic (4, 7, 8) these studies indicate that increases in intraluminal pressures produce increases in length as well as in circumference (9).

Little information is available on morphological changes of vascular smooth muscle elements during contraction. If smooth muscle elements were arranged in helical coils around the circumference of the vessel, shortening should produce a tension vector parallel to the long axis of the vessel with a resultant increase in longitudinal tension. On the other hand, circumferentially oriented smooth muscle elements may increase their width during shortening with resultant increase in thickness and a decrease in longitudinal tension. It has been reported that vascular smooth muscle is arranged in a more helical fashion in central large arteries and in a more radial fashion in smaller peripheral vessels (10). Differences in arrangement of smooth muscle elements from animal to animal may thus account for the difference in longitudinal tension developing during constrictor responses in these studies.

Summary. Longitudinal tensions were recorded from *in situ* isolated segments of dog anterior tibial arteries during vasoconstrictor responses. Two factors appeared to be responsible for longitudinal tensions occurring during vasoconstrictor responses. One was due to changes in intraluminal pressure and appeared to be the dominant factor. Increases in intraluminal pressures produced decreases in longitudinal tensions, indicating that the vessel segments were elongated. Thus, increases in intraluminal pressure passively stretched the blood vessel walls so that increases in both the circumferential and longitudinal axes of the vessel segment occurred. These findings agree with similar findings reported earlier (4, 7, 8). The other factor was apparently due to changes in longitudinal tension produced by vascular smooth muscle contraction. In some cases longitudinal tension changes were positive, in others they were negative. Increases in longitudinal tension were probably the result of contractions of helical smooth muscle elements. Decreases in tension may have been due to displacement of tissue parallel to the long axis of the vessel as smooth muscle elements contracted.

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