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MYOGENIC ADAPTATION OF RABBIT EAR ARTERIES TO PULSATILE INTERNAL PRESSURES

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SUMMARY

1. The effect of sinusoidal internal pressures on the constriction of *in vitro*, pressurized segments of ear arteries from rabbits has been examined. All arteries were constricted against a static transmural pressure of 60 mmHg to 35–60% of maximal, using extraluminal noradrenaline, before being exposed to the sinusoidal pressures.

2. There was a short period of adaptation when active arteries were first exposed to physiological pulsatile pressures. This adaptation had two components: a small, largely transient distension, lasting about 1 min, and sustained suppression of the distension produced by individual pressure pulses.

3. Constriction of adapted arteries was insensitive to physiological changes in pulse frequency (3–5 Hz), mean pulsatile pressure (60–120 mmHg), pulse amplitude (20–40 mmHg) and to alterations in pulse shape (sinusoidal, triangular and ramp). Over-all distension was restricted to $3{\cdot}6 \pm 1{\cdot}0\%$ (S.D. of an observation) when the mean of a 3 Hz sinusoidal pressure of 30 mmHg amplitude was increased in steps from 75 to either 115 or 120 mmHg.

4. An initial distension was needed to initiate suppression of pressure pulse distension. Distension by individual pressure pulses, within a train of rectangular 60–90 mmHg pulses of 0·5 s duration, was maximally suppressed ($85{\cdot}6 \pm 1{\cdot}3\%$; S.E. of mean, $n = 9$) at a pulse interval of 0·7 s.

5. Active ear arteries possess a myogenic mechanism capable of minimizing changes in constriction over the full physiological range of pulsatile internal pressures.

INTRODUCTION

The way in which stability of constriction of arterial blood vessels is achieved against a pulsatile and variable internal pressure is an important unresolved problem. Stability of constriction is essential for adequate control over the distribution of the cardiac output because the resistance to blood flow is dominated by the fourth power of the internal radius of blood vessels (Poiseuille's formula). One possible explanation is that local active reactions of blood vessels against distension by pressure pulses (myogenic responses; Johnson, 1980) help stabilize constriction. Isolated segments of active ear arteries from rabbits react to distension by pressure jumps with a

counteracting constriction (Speden, 1984; Speden & Warren, 1986). However, the counteracting constriction took 1–2 min to achieve near-completion and, therefore, seemed too slow to suppress distension by pulsatile internal pressures at the physiological frequencies, for rabbits, of 3–5 Hz (Ludbrook, Graham & Potocnik, 1985; White, Traugott & Quail, 1985). The present experiments have shown that the myogenic mechanism does have the capacity to minimize distension of active ear arteries against pulsatile pressures of physiological frequency, mean pressure and pulse amplitude. A preliminary report of some of this work has been made (Speden & Warren, 1985).

METHODS

The general methods used were the same as those described in detail previously (Speden, 1984; Speden & Warren, 1986). The one new feature was the incorporation of a third pressure-generating system which was used to produce the sinusoidal pressures. An electrohydraulic piston was constructed that had a working range of 0–220 mmHg and was accurate to ±0·5 mmHg (Fig. 1).

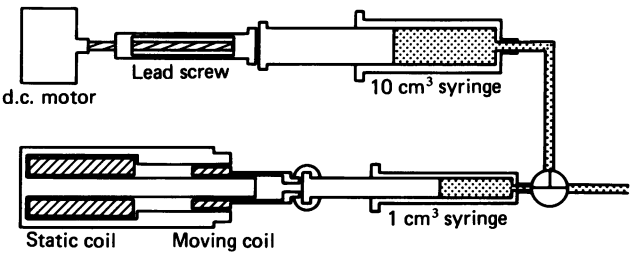

Fig. 1. Schematic diagram of the electrohydraulic piston. The stippling shows those parts of the apparatus filled with Krebs solution. See Methods for a detailed description of the piston and how it was controlled.

The low-volume displacement piston was a 1 cm³ glass syringe with the plunger being driven by an electromagnetic activator. A coil of 1700 turns of enamelled copper wire (0·4 mm diameter) was wound on a soft iron core 13 mm in diameter. This static electromagnet, 48 mm in length, was placed at one end of the core leaving 45 mm of free core protruding. A horseshoe of soft iron was wrapped around the coil to contain the magnetic field. A smaller coil, 17 mm long, of 200 turns of wire (0·25 mm diameter) was wound on one end of a free-moving nylon rod (15 mm diameter) which was fixed to the protruding core of the electromagnet. The other end of the hollow nylon rod was attached to the plunger of the 1 cm³ glass syringe. The output of the syringe was connected through two three-way valves to the artery lumen and to a 10 cm³ glass syringe. The large 10 cm³ glass syringe served as a reservoir of Krebs solution from which the 1 cm³ syringe was refilled. A geared d.c. motor was used to drive the plunger of the larger syringe with the motor being powered in a way that prevented sudden movements. The major loss of Krebs solution was through slow leakage between the plunger and barrel of the 1 cm³ glass syringe.

An electric current of 1 A was passed through the static coil creating a strong magnetic field with which the field from the moving coil could interact. A negative feed-back controller was used to compare the pressure recorded by the pressure transducer (P23 Db Statham, Puerto Rico) with a composite command signal. The command signal was the sum of a d.c. offset voltage, a d.c. computer-generated voltage and a signal-generator voltage (Hewlett–Packard 3110B Function Generator, CO, U.S.A.). Both the comparison and the sum were executed by a standard operational amplifier used in the difference mode with both high-gain and damping control. The resulting signal was amplified by a TDA 2030, audio-amplifier integrated circuit (Radio Spares, U.K.) and then passed to the moving coil on the nylon rod. In this way, any detected difference between the command pressure and the observed pressure resulted in a large corrective current being sent to the moving coil. There was a tendency for the glass plunger of the 1 cm³ syringe not to move freely. This sticking was overcome by imposing a low-amplitude, high-frequency (113 Hz) vibration on

the plunger. Transmission of the high-frequency vibration to the artery was damped by placing an air bubble (1 cm long) in the Teflon tubing (1·93 mm internal diameter) connecting the 1 cm^3 syringe to the artery.

Blood pressures were recorded by inserting a hypodermic needle into the lumen of the central ear artery using the method of Speden & Ryan (1982). The recordings were made at a room temperature of 28–29 °C and were stored on computer disks.

The results are expressed as the mean value ±S.D. of an observation (n, number of observations) unless otherwise stated. The Student's t test was used to assess statistical significance.

RESULTS

The arteries, which were not spontaneously active under the conditions used, were activated by infusing noradrenaline into the organ bath solution. All experiments were carried out using arteries which had been first constricted against a transmural pressure of 60 mmHg to 35–60% of maximal by extraluminal noradrenaline concentrations of 11–89 nM.

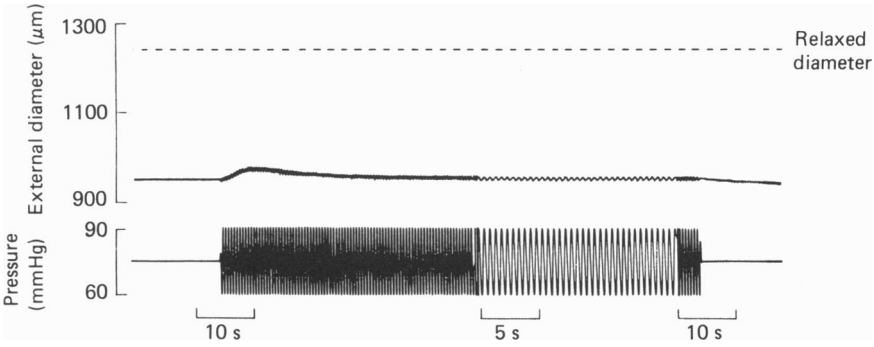

Fig. 2. Adaptation to sinusoidal internal pressures. Extraluminal noradrenaline (59 nM) was used to constrict an artery against a static pressure of 75 mmHg until a stable constriction of 45% of maximal was achieved. Exposure to a 2 Hz, 90/60 mmHg pulsatile pressure initiated a largely transient constriction, usually lasting < 1 min, and small oscillations in external diameter in synchrony with the sinusoidal pressure (seen better at the higher chart speed). Maximal constriction against 60 mmHg (51·8%) was determined by exposure to high noradrenaline concentrations at the end of the experiment.

Adaptation to a pulsatile internal pressure

The effect of changing from static to a pulsatile pressure of the same mean pressure was examined using a 2 Hz sinusoidal pressure of 90/60 mmHg (Fig. 2). Exposure to the pulsatile pressure had little sustained effect on the external diameter of the arteries. The arteries dilated transiently with a maximum distension of 6·5±2·8% (n = 11, five arteries) being reached in 5·2±0·9 s. This initial distension was largely counteracted by compensatory constriction with a stable residual distension of 1·0±0·8% being achieved by 44±16 s. Superimposed upon these slow diameter changes was a ripple in synchrony with the pulsatile pressure. Each pressure pulse produced a 1·1±0·5% distension of the arteries. These reactions to a pulsatile pressure were, in three experiments, unaffected by tetrodotoxin (1·5 μM).

Maintenance of constriction against different sinusoidal pressures

The range of pulsatile pressures used was based on direct recordings of ear artery blood pressures from six conscious rabbits. The rabbits had heart rates of 2·9–4·9 Hz (3·9±0·7 Hz), diastolic pressures of 84–104 mmHg (92±7 mmHg), systolic pressures of 110–133 mmHg (121±8 mmHg) and a pulse pressure of 25–33 mmHg (29± 3 mmHg). These pressures are not basal as the untrained rabbits were agitated and the blood pressure of rabbits is susceptible to environmental factors (Ludbrook *et al.*

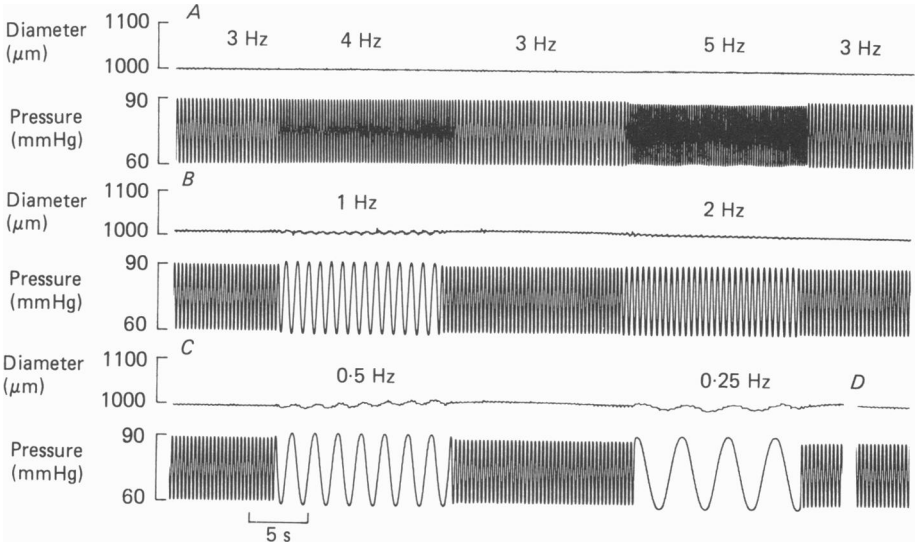

Fig. 3. The effect of different pulse frequencies on constriction of an adapted artery. The noradrenaline-constricted artery was exposed to a 90/60 mmHg pulsatile pressure of 3 Hz until constriction was again stable and returned to that frequency, after each frequency change, to maintain adaptation. Physiological changes in frequency had no effect on the mean constriction (*A*), but all arteries relaxed slightly at frequencies of ⩽ 1 Hz (*B*, *C* and *D*). *D* was recorded 8 s after *C*. Sinusoidal oscillations in external diameter became increasingly pronounced as the frequency was decreased. All records are from the same continuous exposure to pulsatile pressures and are in the sequence recorded. These diameter changes were reproducible as there was no alteration in the reactions of the artery when the series of frequency changes was repeated. Constriction at 3 Hz was 49% of maximal (22 nM-noradrenaline) with maximal constriction against 60 mmHg being a 53·0% reduction in external diameter. The relaxed external diameter at 60 mmHg was 1350 μm.

1985). Ludbrook *et al.* recorded a mean blood pressure of 81±1 mmHg (S.E. of mean) and a heart rate of 3·57±0·15 Hz when six rabbits were quiet or being petted.

Changes in pulse frequency. Fig. 3 illustrates the method used to examine the effect of pulse frequency on constriction. Six active arteries were exposed to a 3 Hz 90/ 60 mmHg pulsatile pressure until a stable constriction of 47±6% of maximal was attained. The frequency was then changed to one of the six different frequencies shown in Fig. 3 and returned to 3 Hz after 15 s. Constriction of the arteries was insensitive to frequency changes within the physiological range as no significant diameter changes were observed (Fig. 3*A*). At these frequencies of 3–5 Hz, diameter

pulsations were minimal, being either a just-detectable 5–10 μm distension (Fig. 4) or, with two arteries, undetectable (Fig. 3*A*). There was a large safety factor as constriction was well maintained down to the unphysiological low frequency of 1 Hz (Fig. 3*B*). Constriction was less well maintained at frequencies below 1 Hz, although the changes in external diameter were still small (Fig. 3*C*). The distension produced by each pressure pulse increased with decreasing frequency below 3 Hz from $0{\cdot}52 \pm 0{\cdot}38\%$ to $2{\cdot}2 \pm 0{\cdot}2\%$ ($P < 0{\cdot}01$; paired observations) at 0·25 Hz. The arteries also dilated slightly by 5–20 μm ($1{\cdot}1 \pm 0{\cdot}6\%$ increase in diameter) at frequencies below 1 Hz.

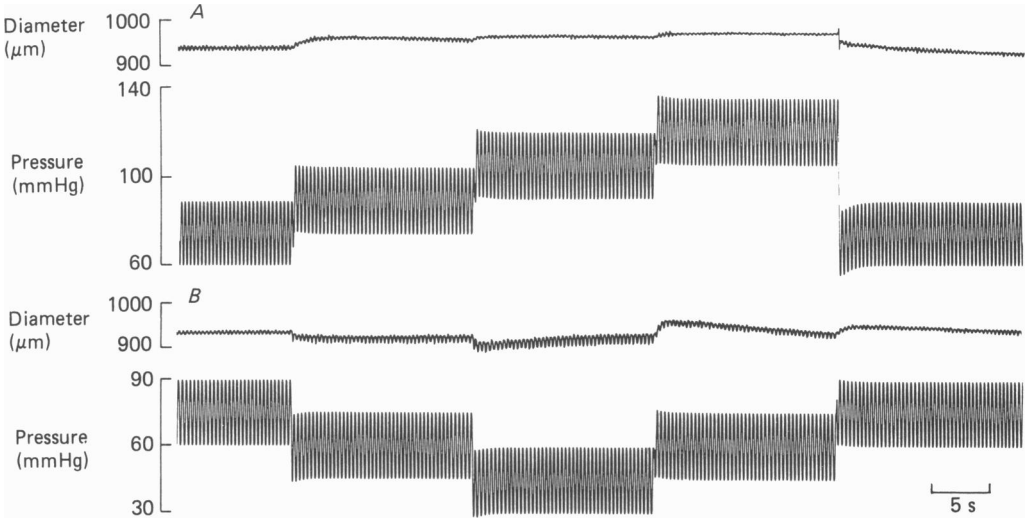

Fig. 4 Maintenance of constriction against different mean pressures. In *A*, the mean pressure of a 3 Hz sinusoidal pressure with a pulse amplitude of 30 mmHg was increased in steps of 15 mmHg from 75 to 120 mmHg. The artery distended by a maximum of 30 μm which was 10·7% of the maximum possible distension of 280 μm; the relaxed external diameter at 120 mmHg was 1215 μm. Arteries were more easily distended by increases in non-pulsatile pressure (see Speden, 1984, Fig. 2). In *B*, the mean pressure was lowered to 45 mmHg. Note the increase in diameter oscillations, the slow dilatation and the 'rebound' distension when the pressure was restored. Constriction against the 90/60 mmHg pulsatile pressure was 46·2% of maximal and was attained using extraluminal noradrenaline (30 nM). Maximal noradrenaline activation constricted the artery to 609 μm against 60 mmHg (a 49·9% reduction in diameter).

Changes in mean pressure. The mean pressure of a 3 Hz pulsatile pressure with a pulse amplitude of 30 mmHg was increased from 75 mmHg in steps of 15 mmHg to 105 mmHg and then to either 115 or 120 mmHg (Fig. 4*A*). An upper limit of 135 mmHg was placed on the peak pressure as a transmural pressure of 140 mmHg overloaded constriction of ear arteries *in vitro* (Speden & Ryan, 1982). Over-all distension of the five arteries was $3{\cdot}6 \pm 1{\cdot}0\%$ with the arteries tending to be more resistant to distension at mean pressures above 90 mmHg. The distension produced by increasing the mean pressure from 90 to either 115 mmHg (three arteries) or 120 mmHg was $1{\cdot}06 \pm 0{\cdot}45\%$ compared to $2{\cdot}55 \pm 1{\cdot}26\%$ for the increase from 75 to 90 mmHg ($P < 0{\cdot}2$; paired observations). This resistance to distension was due

primarily to increases in active, circumferential wall stress as the changes in passive stress were small (Fig. 5*C*). The active stress increased from 32·2±4·0% of maximal at a mean pressure of 75 mmHg to 60·4±4·2% at the highest mean pressures used (Fig. 5*B*). This active stress has been expressed as a percentage of the maximum available at that diameter when the arteries were activated by a maximally effective concentration of noradrenaline (29·6 μM).

The resistance to distension during a pressure pulse may be due to a combination of increases in active stress and stiffness of the series elastic component with the latter being unknown. Fig. 5*B* shows the changes in active stress during a 30 mmHg pressure pulse when the simple Maxwell model was used to calculate the active stresses (Speden & Warren, 1986). The active stress fluctuated from a minimum of

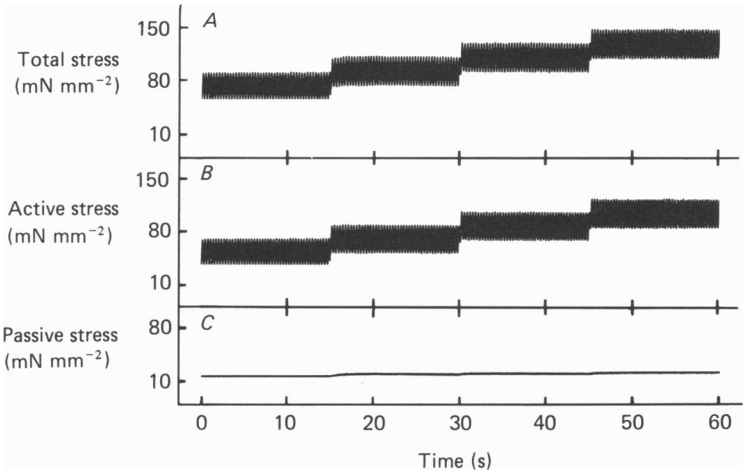

Fig. 5. Circumferential wall stresses at different mean sinusoidal pressures. Computer analysis techniques were used to convert the observations shown in Fig. 4*A* into circumferential wall stresses (*A*). The total wall stress was then separated into active (*B*) and passive components (*C*) using Maxwell's mechanical model of muscle. There was little change in the passive stress over the entire pressure range. The small changes in diameter were assumed to have no effect on the wall cross-sectional area (0·160 mm²).

23·7±3·2% of the maximum available active stress to a maximum of 42·8±6·2% when the mean pressure was 75 mmHg. The fluctuations in active stress during a pressure pulse increased slightly as the mean pressure was raised as a consequence of distension of the arteries (Fig. 5*B*).

The arteries were less able to resist distension at mean pressures below 75 mmHg. Diameter pulsations increased from 5±4 μm to 9±4 μm ($n = 5$) when the pressure was lowered to 60 mmHg, but the small increase in mean constriction of 1·2±0·5% was sustained (Fig. 4*B*). A mean pressure of 45 mmHg was an inadequate stimulus for distension activation; diameter pulsations increased to 19±4 μm ($P < 0·01$; paired observations at 45 and 75 mmHg), the initial constriction was not maintained and there was a pronounced transient increase in diameter of 4·7±1·6% when the pressure was subsequently increased to 60 mmHg (Fig. 4*B*). The active wall stress at 45 mmHg was 15±2% of the maximum force-generating capacity at that diameter.

Changes in pulse amplitude. Both the pulse frequency (3 Hz) and the mean pressure (75 mmHg) were kept constant when the pulse amplitude was altered. Constriction of five arteries was well maintained when the pulse amplitude was increased from 30 to 40 mmHg (Fig. 6*A*). There was an initial dilatation of $2{\cdot}2 \pm 1{\cdot}3\%$ which decreased to $1{\cdot}2 \pm 0{\cdot}9\%$ over 30 s. The distension produced by each pressure pulse also increased from $0{\cdot}62 \pm 0{\cdot}18\%$ to $1{\cdot}04 \pm 0{\cdot}58\%$. Constriction was, with one exception, not well maintained when the pulse amplitude was raised to an unphysiological 50 mmHg. The arteries dilated by $4{\cdot}7 \pm 3{\cdot}0\%$ with little subsequent constriction, and the constriction of two arteries became unstable (Fig. 5*B*). Lowering the pulse amplitude from 30 to 20 mmHg had no significant effect on constriction.

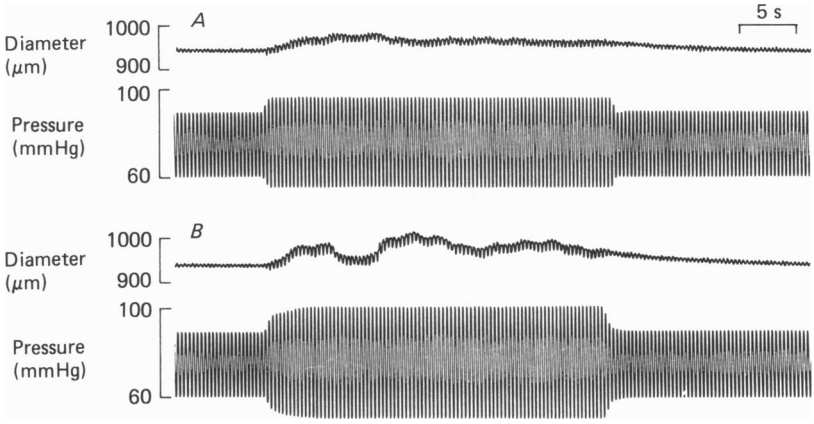

Fig. 6 The effect of increases in pressure pulse amplitude on constriction. Increasing the pulse amplitude of a 90/60 mmHg 3 Hz sinusoidal pressure from 30 to 40 mmHg increased the diameter oscillations and slightly dilated the artery (*A*). Further increasing the pulse amplitude to an unphysiological 50 mmHg accentuated these changes and constriction tended to become unstable (*B*). These records and those of Fig. 4 are from the same continuous exposure to different sinusoidal internal pressures. Note the stability of the reference constriction.

Changes in pulse shape. Changing the shape of the pulse of a 3–5 Hz pulsatile pressure from sinusoidal to triangular or to a fast-rising ramp (0·23–0·25 of the cycle in the rise phase) had no effect on the constriction of four arteries. The fast rising ramp was used to more closely mimic the shape of the blood pressure pulse.

Analysis of the adaptation to pulsatile internal pressures

The initiation and duration of the adaptation was examined further by varying the time between rectangular pressure pulses of 0·5 s duration.

Initiation. An initial distension was needed to activate arteries against distension by subsequent pressure pulses. One pressure jump from 60 to 90 mmHg for 0·5 s gave sufficient distension to fully activate adaptation. Such a pressure jump elicited a transient distension lasting 20–30 s that was reproducible provided the time between pulses exceeded 10 s (Figs. 7*A* and 8*A*). At shorter pulse intervals, the distension elicited by the second pressure pulse became increasingly suppressed as the pulse interval was decreased. A second pressure pulse 2 s after the first pulse was able to

cause more distension (Fig. 7*C*), but further distension by subsequent pulses was largely suppressed. At pulse intervals of $\leqslant 1$ s, distension by individual pressure pulses was reduced to a ripple superimposed upon a slower counteraction of the distension initiated by the first pressure pulse (Fig. 7*D* and *E*).

Duration. An estimate of the degree of adaptation and its persistence was obtained by comparing the distension produced by individual pressure pulses, within a train of pulses, with the reproducible distension initiated by the first pressure pulse. The

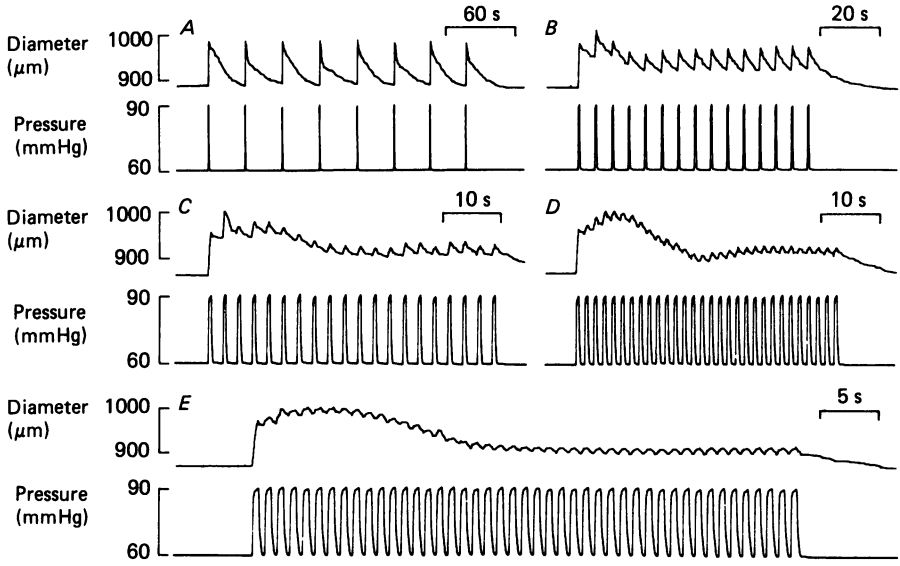

Fig. 7. An analysis of adaptation to pulsatile internal pressures. The onset and duration of the adaptation was examined by varying the time between 60–90 mmHg rectangular pressure pulses of 0·5 s duration. An initial distension was necessary to initiate resistance against pressure pulse distension. Pressure pulses elicited a reproducible transient distension provided the time between pulses exceeded 10 s (*A*). The same distension was produced by the first pulse of faster trains of pulses, but distension by subsequent individual pulses was increasingly suppressed as the pulse interval was reduced to 5 (*B*), 2 (*C*) and 1 s (*D*). At a pulse interval of 0·5 s (*E*), distension by individual pressure pulses was reduced to a ripple superimposed upon a slow counteraction of the distension initiated by the first pressure pulse. Exposure to the different trains of pulses was randomized. The rectangular shape of the pulses has been distorted by the inadequate response time of the pen recorder. The rectangular pulses were generated through an electropneumatic transducer to a standard of 98% completion in 80–90 ms when stored on floppy disk (Speden & Warren, 1986). Responses of the same artery to 3 Hz sinusoidal internal pressures are shown in Fig. 4. Constriction by extraluminal noradrenaline (44 nM) against 60 mmHg varied from 54 to 59% of maximal.

distension produced by the first pressure pulse was the unadapted response as the same distension was elicited when the time between pulses was more than 10 s (Figs. 7 and 8). Adaptation, as indicated by suppression of the initial distension, depended upon the time between pressure pulses in a biologically variable way. With five arteries, a uniform suppression of pressure pulse distension was achieved within 20 s, a suppression that increased with decreasing time between pressure pulses (Fig. 7). Counteraction of the initial distension also increased as the time between pulses was reduced. With the other four arteries, maintenance of adaptation was

erratic at intermediate time intervals as distension by pressure pulses fluctuated (Fig. 8). These fluctuations in distension decreased as the pulse interval was shortened. In Fig. 8*C*, alternate pulses caused more distension at the end of a train of pulses when the time between pulses was 2 s. Shortening the pulse interval to 1·5 s reduced the frequency at which more distension occurred to every third to fifth pulse (Fig. 8*D*). A more uniform suppression of distension was not achieved until the pulse interval was reduced to 0·5 s (Fig. 8*E*).

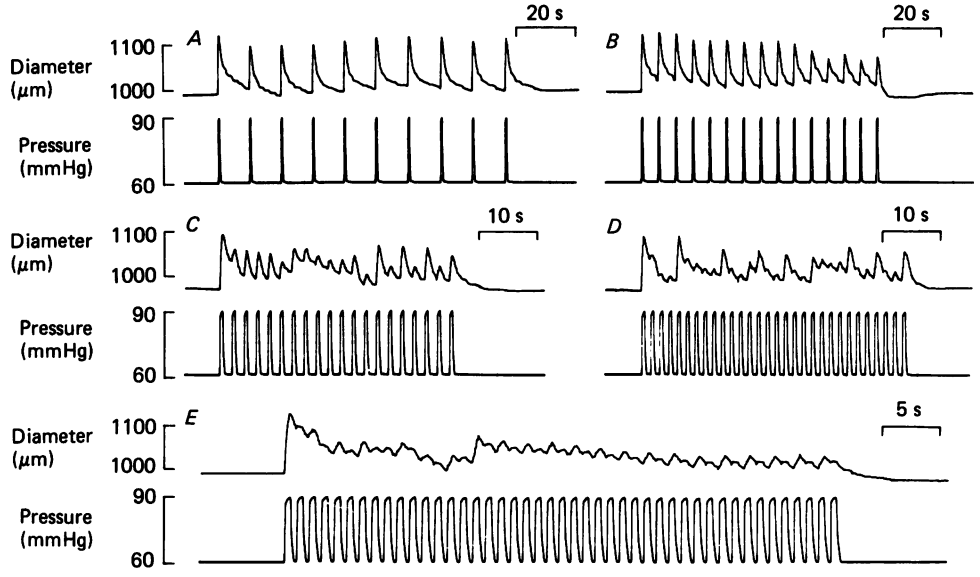

Fig. 8. Fluctuations in the adaptation of an active artery to pulsatile internal pressures. Resistance to distension by individual pressure pulses fluctuated when the pulse interval was 2 s (*C*) or less. These fluctuations were increasingly suppressed as the pulse interval was decreased further to 1 (*D*) and then to 0·5 s (*E*). The other pulse intervals were 10 (*A*) and 5 s (*B*). The noradrenaline-induced constriction (89 nM) varied slightly from 47 to 50% of maximal. Relaxed external diameter was 1320 μm and maximal noradrenaline activation (29·6 μM) constricted the artery by 52·7% against 60 mmHg.

The results of all nine experiments are summarized in Fig. 9 where the percentage suppression of pressure pulse distension has been plotted against the pulse interval. Such a plot indicates the degree of adaptation achieved against a pulsatile pressure. Adaptation was about half-maximal at a pulse interval of 5 s and reached a maximum of 85·6 ± 1·3% suppression at a pulse interval of 0·7 s.

DISCUSSION

These experiments have shown that the rabbit ear artery possesses a myogenic mechanism capable of minimizing changes in constriction when the active arteries were exposed to the full physiological range of pulsatile internal pressures. This insensitivity of constriction to physiological pulsatile pressures is also shown *in vivo* by the proximal arterial vessels (estimated internal diameters > 25 μm) of the cat hind limb (Grände, Lundvall & Mellander, 1977; Grände, Borgström & Mellander,

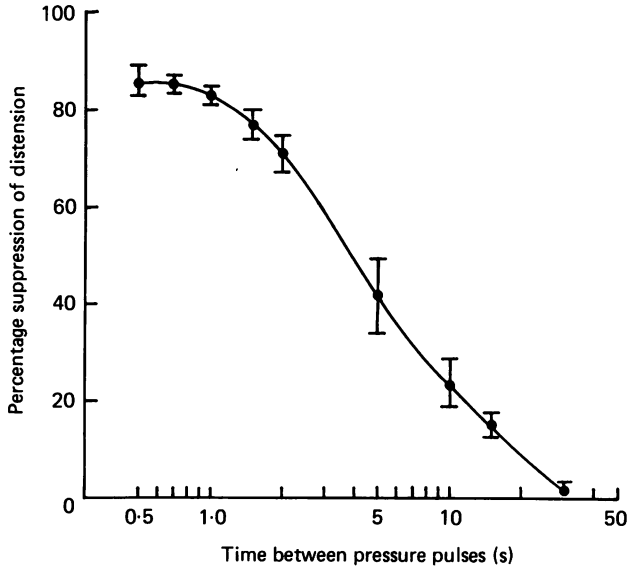

Fig. 9. The dependence of adaptation to pulsatile pressures on the time between pulses. The distension caused by individual pulses at the end of a train of pulses (Figs. 7 and 8) has been expressed as a percentage suppression of that produced by the first pulse. Distension was averaged over the last 10–20 pulses at the shorter time intervals ($\leqslant 2$ s). Each observation (●) is the mean $\pm$S.E. of mean for the adaptation of nine arteries which were constricted by an average of $50{\cdot}9 \pm 1{\cdot}0\%$ (S.E. of mean) of maximal. Maximal noradrenaline stimulation constricted the arteries by $51{\cdot}3 \pm 2{\cdot}0\%$ against 60 mmHg. Adaptation had a comfortable physiological safety factor with near-maximal suppression being achieved when the time between rectangular pulses was 1·0 s. The peak-to-peak time between pressure pulses within the ear artery *in vivo* is normally 0·2–0·4 s.

1979). Moreover, these proximal arterial vessels, like rabbit ear arteries, dilated only slightly when a static distending pressure was made pulsatile with the distension also being mostly transient and over within about 1 min (Grände *et al.* 1979). These similarities indicate that the type of myogenic response shown by the isolated rabbit ear artery does occur *in vivo* and is little affected, if at all, by our *in vitro* techniques. The main physiological function of this myogenic mechanism may be to enable remote control systems like the sympathetic nervous system to achieve adequate control over the constriction of those larger resistance vessels which are exposed to a highly pulsatile and variable internal pressure (Speden & Warren, 1986). Modest fluctuations of constriction may have pronounced haemodynamic effects as the resistance to blood flow is a fourth power function of the internal radius (Folkow, Kalström, Nilsson & Sjöblom, 1984).

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