Tension of the Lower Abdominal Aorta as Influenced by Sympathetic Stimulation

Although tension of the vessel wall is generally accepted to be suitable to provide information on mechanical properties of blood vessels[1–3], direct data related to tension of the aorta in vivo, and the changes eventually occurring due to its constriction at sympathetic stimulation, are lacking. Thus experiments have been performed to evaluate the aorta tension both at steady state and during sympathetic stimulation.

Methods. Experiments have been carried out on 10 dogs, of both sexes, anaesthetized with thiopental (70 mg/kg body wt). The sympathetic trunc was cut bilaterally at the LG_1–LG_2 level and the peripheral parts were stimulated by means of platinum electrodes with rectangular impulses of supramaximal voltage, 5 msec duration and 1–15 imp/sec frequency.

The diameter of the lower abdominal aorta 15 mm proximally to the bifurcation was monitored by an inductive transformer[4] modified to the aorta dimensions.

The blood pressure was measured by an Electromanometer Elema through a catheter introduced to the site of diameter measuring via the left femoral artery. Diameter and blood pressure transducers operated a direct writing jet recorder (Mingograph 81). The blood pressure, if necessary, was maintained constant by means of a pressure stabilizer connected to the right femoral artery.

To exclude the possible effect of suprarenal-borne catecholamines, both suprarenal veins were temporarily clamped during stimulation.

Calculating the vessel wall tension, BURTON[1] and RUSHMER[2] estimated tension, according to Laplace's law, to be directly related to the product of pressure and radius ($t = P \cdot R$); in their opinion, tension should be considered per length unit. BENNINGHOFF[5] and PETERSON[3], however, taking tension for force per unit area, pointed out that tension is inversely proportional to the thickness of the vessel wall ($t = P \cdot R/W$). The latter equation has been used in this study.

No method to record the wall thickness in vivo being available, the wall thickness in steady state was calculated according to BERGEL[6] as 10.5% of the external radius of the aorta. Estimation of the wall thickness during sympathetic stimulation was based on the following assumption: Due to incompressibility of the vascular wall[7,8] under the conditions used, no change of the cross-sectional area should occur

$$A_o = A_s \qquad (1)$$

A_o = cross-sectional area of the vessel wall during steady state,

A_s = cross-sectional area of the vessel wall during sympathetic stimulation.

Thus

$$\pi R^2oe - \pi R^2oi = \pi R^2se - \pi R^2si \qquad (2)$$

where

Roe = external radius of the aorta at steady state,
Roi = internal radius of the aorta at steady state,
Rse = external radius of the aorta at respective sympathetic stimulation,
Rsi = internal radius of the aorta at respective sympathetic stimulation.

By solution

$$Rsi = \sqrt{R^2se + R^2oi - R^2oe} \qquad (3)$$

and finally

$$W_s = Rse - Rsi. \qquad (4)$$

Results. Bilateral stimulation of the lumbar sympathetic trunc at the LG_1–LG_2 level usually ellicited an increase of the blood pressure, accompanied by an increase of the radius of the aorta and a thinning of the aorta wall. The calculated tension of the aorta wall kept increasing during the whole stimulation period (Figure 1).

If, on the other hand, the blood pressure was artificially stabilized, similar stimulation reduced the aorta radius and a thickening of the aorta wall occurred; consequently the tension of the aorta kept decreasing (Figure 2).

The range of the diameter changes of the aorta at sympathetic stimulation was revealed to be – similarly to the other vascular segments frequency dependent[4,9,10]. Plotting the decrements of aorta diameter to the frequency of stimulation illustrates the successively decreasing diameter with increasing stimulation frequency. It indicates the frequency-response to be steep up to 8 imp/

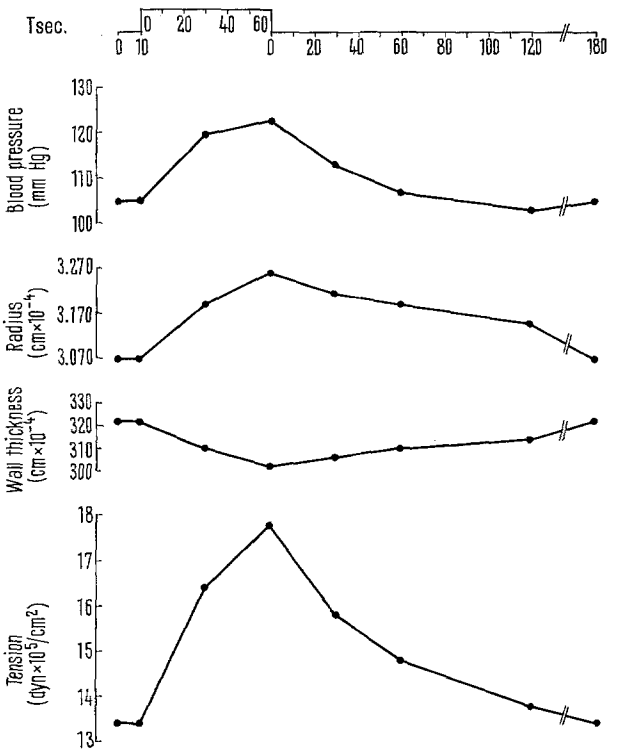

Fig. 1. Effect of bilateral stimulation of the sympathetic trunc at LG_1–LG_2 (rectangular impulses of 5 msec duration, amplitude 6.8 V and frequency 15 imp/sec) on blood pressure, radius, wall thickness and tension of the abdominal aorta.

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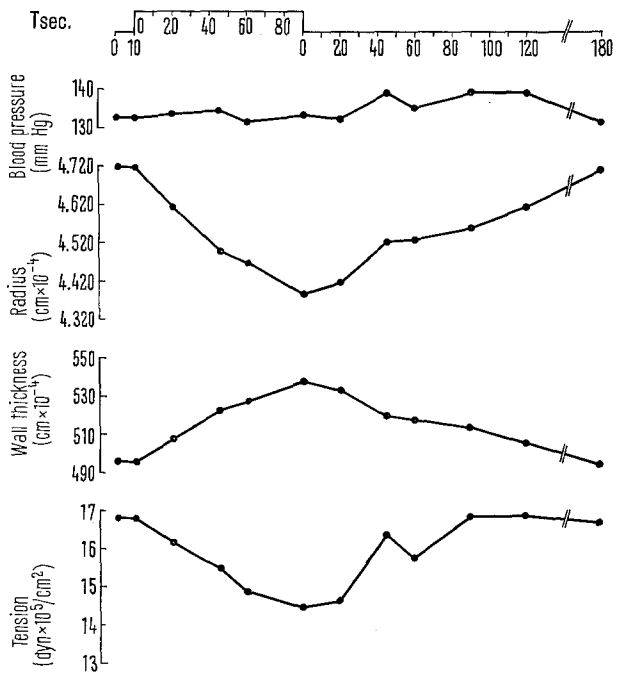

Fig. 2. Effect of bilateral stimulation of the sympathetic trunc at LG_1–LG_2 (rectangular impulses of 5 msec duration, amplitude 6.2 V and frequency 15 imp/sec) whilst blood pressure artificially stabilized on radius, wall thickness and tension of the abdominal aorta.

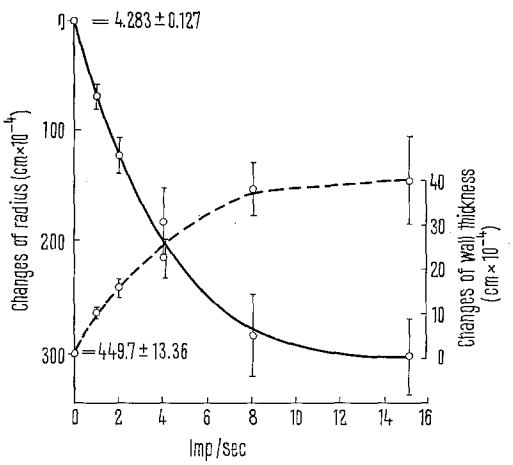

Fig. 3. Changes (mean values ± S.E.M. of 21 series of stimulations) of the radius (full line, scale left) and wall thickness (interrupted line, scale right) of the aorta plotted to the frequency of bilateral sympathetic stimulation. Numerals relative to zero frequency = the respective resting values before stimulation.

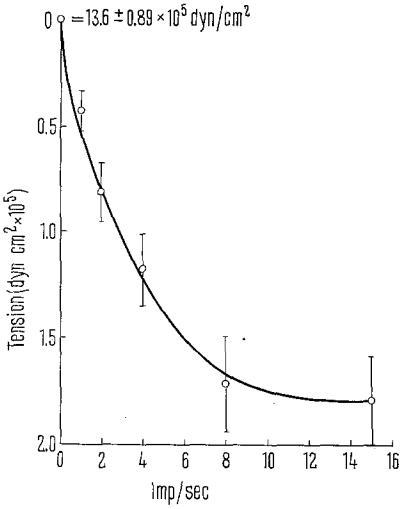

Fig. 4. Changes of the tension of the aorta (mean values ± S.E.M. of 21 series of stimulations) plotted to the frequency of bilateral sympathetic stimulation. Numerals relative to zero frequency = the respective resting value before stimulation.

sec stimulation frequency, maximal constriction being attained at 15 imp/sec. The wall thickness, on the other hand, is simultaneously increasing (Figure3). Consequently, changes in tension due to smooth muscle contraction are shown to be frequency dependent also (Figure 4), tension being indirectly related to the stimulation frequency.

Conclusions. The diameter and blood pressure of the abdominal aorta were monitered directly and wall thickness and tension were calculated both at steady state and sympathetic stimulation. The blood pressure being stabilized, sympathetic stimulation (LG_1–LG_2 level) ellicited a decrease of radius associated with wall thickening and with a decrease of the wall tension; all of the above parameters being frequency dependent. If during sympathetic stimulation the blood pressure increased, an increase of tension occurred.

Zusammenfassung. Blutdruck und Durchmesser der Aorta abdominalis wurden in vivo bei Ruhe und während Sympathikusreizung zur Berechnung der Wandstärke und -Spannung direkt registriert.

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Untersuchungen zur Klärung der Funktion des Reissnerschen Fadens. In-vitro-Bindung von Noradrenalin, Adrenalin und Serotonin

Das Subcommissuralorgan (SCO) des Wirbeltiergehirns sezerniert ein Glykoproteid, das als Reissnerscher Faden (RF) Ventrikelräume und Zentralkanal des Rückenmarkes bis zu dessen kaudalen Ende durchwächst[1–3].

In der Untersuchungsgeschichte des SCO-RF-Komplexes ist mehrfach die Ansicht vorgetragen worden, dass das von den Drüsenzellen in den Ventrikel entlassene Sekret Liquorkomponenten bindet und etwa als Detoxikator[2] oder hirneigenes Exkretionssystem[1] Substanzen eliminiert, die die normale Tätigkeit des umgebenden

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