Longitudinal Stress Relaxation in the Canine Aorta

As a result of in vivo and in vitro studies the arterial vascular wall is considered to be a visco-elastic material. One characteristic of many visco-elastic materials exhibited by the arterial wall is the progressive fall in stress following the application of a constant strain: this change of arterial mechanical properties with time is called delayed compliance or stress relaxation. In non-biological materials the phenomenon depends upon the structural coupling and relative moduli of the elastic and viscous elements of the material[1].

In the case of the arterial wall, the stress relaxation is probably related to the relative proportions of the constituent elements: collagen, elastin, smooth muscle and the mucopolysaccharides, the mechanical properties of these elements, together with their architecture and structural coupling.

Previous studies have demonstrated that arterial stress relaxation predominates in the tangential direction, compared with the longitudinal, at least in the canine femoral artery[2]; but that significant degrees of longitudinal stress relaxation can be demonstrated in the canine abdominal aorta[3].

Further experiments have been performed upon arterial sections taken from 5 aortic sites and some of the main aortic distributing arteries to evaluate the degree of longitudinal stress relaxation at these sites.

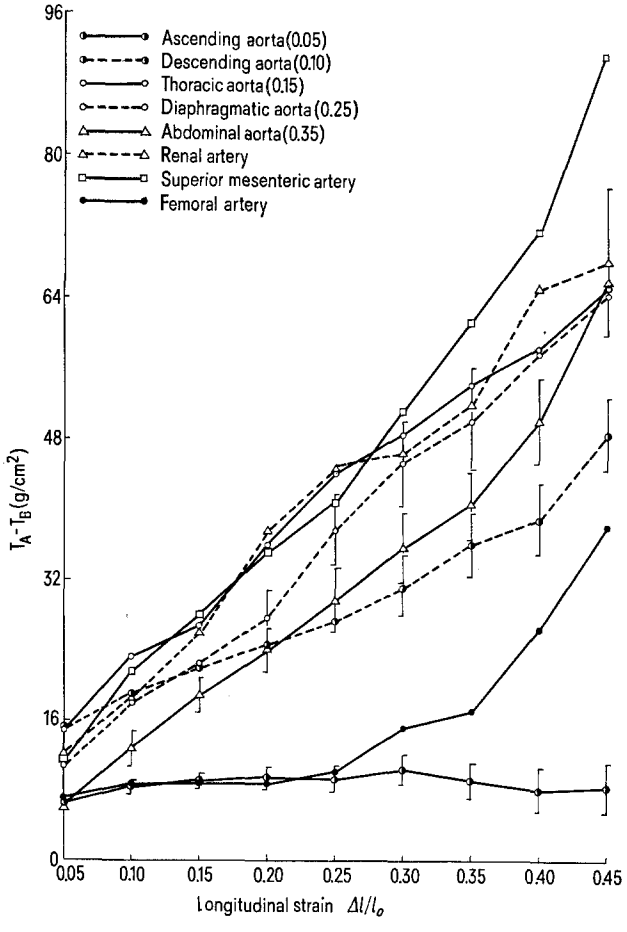

Fig. 1. Difference between the induced arterial stress immediately following step input of longitudinal strain T_A, and the stress remaining after 3 min of stress decay T_B, as a function of the applied strain $\Delta l/l_o$; for 8 arterial sites.

Methods. Arterial sections from 7 sites were removed from 12 mongrel dogs of average body weight 22.3 + 1.1 kg. The apparatus, technique to maintain arterial segments in a viable state, and the procedures to obtain the stress-strain relationship along the longitudinal axes of the vessels have previously been described[2]. Absolute measures of stress relaxation were obtained by subtracting the tension induced in the arterial segments immediately following application of strain (T_A) from the tension remaining after 3 minutes of tension decay, (T_B). These differences were calculated for strain increments of 0.05 for longitudinal strains of 0.05 to 0.45, where the strain is defined as the change in length (Δl) divided by the original length (l_o) of the blood vessel segment.

Results. Figure 1 shows the stress relaxation (T_A–T_B) as a function of longitudinal strain for 5 aortic sites together with the renal and superior mesenteric arteries. Values for the femoral artery, taken from ATTINGER[2], are included for comparison. All arterial sites considered showed stress relaxation in the longitudinal direction, and, with the exception of the ascending aorta, there was a progressive increase in stress relaxation up to the limits of longitudinal strain under investigation. The figures in parentheses following the aortic sites in the key indicate the degree of resting aortic longitudinal strain as measured in the dog in vivo by APTER et al.[4] using a retraction technique. Data are expressed as the means ± 1 s.e. Standard errors of the strain are very small and have been omitted. Also several standard errors in the vertical direction have been omitted in the interests of clarity.

Discussion. From the quantitative differences between stress relaxation in human umbilical artery (almost 100% smooth muscle) and canine carotid artery (approximately 30% smooth muscle), ZATZMAN et al.[5] concluded that stress relaxation was a phenomenon associated primarily with the smooth muscle component of the artery. Further evidence of the importance of smooth muscle and its state of contraction was demonstrated by ATTINGER[2], who demonstrated a large increase in stress relaxation when femoral artery smooth muscle was contracted by topical norepinephrine, and its diminution following pharmacological inactivation of the smooth muscle. GOTO and KIMOTO[6], using a universal tensile testing instrument, obtained stress relaxation curves for toad aortae which they divided into 3 phases from the distribution function of the relaxation curves. They enzymatically removed collagen and/or elastin and repeated the studies: the results suggested that in the course of stress relaxation collagen loses its tension first, smooth muscle next, and elastin last.

Whatever the primary determinant of stress relaxation, because arterial composition and arrangement vary with site, the degree and direction of stress relaxation should also be a function of arterial site. For a given site, it may be the degree of longitudinal alignment of arterial wall constituents which contributes to the degree of stress relaxation in a longitudinal direction.

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Histological evidence for some degree of smooth muscle orientation in the longitudinal direction has been demonstrated in the superior mesenteric artery[7] and some aortic sections[8]. These sites showed a relatively high degree of longitudinal stress relaxation compared with the femoral artery. As the longitudinal stress increases, the proportion of longitudinally aligned smooth muscle and/or connective tissue components presumably increases.

If the proportion of the longitudinal alignment of smooth muscle fibres is a function of the smooth muscle content for all arterial sites, then we would expect smooth muscle to relate to longitudinal stress relaxation as measured by T_A–T_B. However, no correlation was found between values of aortic stress relaxation at the initial longitudinal strain of 0.05 and the smooth muscle aortic content, taken from FISCHER and LLAURADO[9], derived by subtraction of the collagen and elastin percentage content from 100%.

The physiological longitudinal strain along the aorta is not equal at all sites. However, even if we consider the stress relaxation at the resting physiological longitudinal strains as given by Apter et al.[4] for the canine aorta, there is still no correlation with the smooth muscle content at these sites.

It has been shown that at and above physiological pressures collagen plays a greater tension bearing role in the arterial wall than other arterial constituents, whereas elastin predominates in this function at lower than physiological pressures[10]. From the above and the work of GOTO and KIMOTO[6] it would be expected that a function of arterial wall constituents which correlates well with stress relaxation at physiological strains would include an expression of either collagen content or its variation with respect to elastin content, that is the collagen-elastin ratio C/E[9]. Consequently at resting physiological strains longitudinal stress relaxation in the aorta was considered as a function of both the sum of smooth muscle and collagen content and the product of the smooth muscle content and the C/E ratio. Both these variables gave a very good correlation with longitudinal stress relaxation, particularly the sum of smooth muscle and collagen content. This relationship is shown in Figure 2.

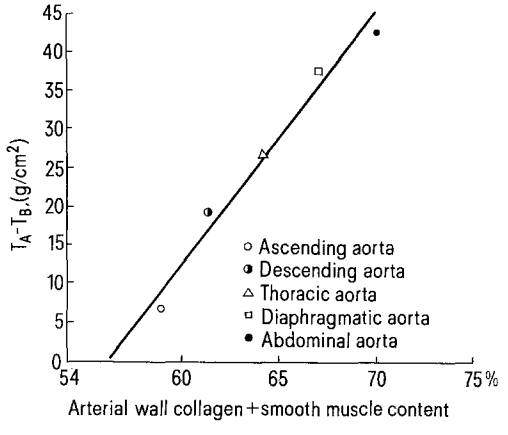

Fig. 2. Difference between the induced arterial stress immediately following step input of longitudinal strain T_A, and stress remaining after 3 min of stress decay T_B as a function of the sum of smooth muscle and collagen content for 5 aortic sites.

Conclusion. A significant degree of longitudinal stress relaxation occurs in the various regions of the canine aorta, renal and superior mesenteric arteries. The magnitude of such stress relaxation is strain dependent and differs from site to site. At longitudinal strains equivalent to those existing in vivo, longitudinal stress relaxation in the aortic sites considered increases progressively in a caudal direction and correlates well with the sum of collagen and smooth muscle known to exist at these sites.

Résumé. On a mesuré chez le chien la relaxation de la tension longitudinale en fonction de la déformation dans l'aorte, l'artère rénale et l'artère mésentérique supérieure. Dans les déformations équivalent à celles du vaisseau in vivo, la relaxation de la tension longitudinale est mise en relation avec les effets combinés de la teneur en collagène et des muscles lisses.

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Bobierrit-Newberyit-Konkremente in den Drüsensäckchen des Vormagens von *Lama lama*

Im Gegensatz zum Struvit kommen die Magnesium-orthophosphat-Minerale Bobierrit und Newberyit nur sehr selten als Komponenten pathologischer Konkremente vor[1]. Bei der Sektion eines 12jährigen Lamahengstes, der wegen senilem Marasmus getötet worden war, wurden in den sog. Drüsensäckchen des ersten Vormagenabschnittes (cranialer und caudaler 'Pansen'-Sack) zahlreiche jeweils solitäre, erbsen- bis kastaniengrosse, unregelmässig gestaltete Konkremente gefunden (Figur a und b), die an der Oberfläche pflanzlicher Futterbestandteile bzw. verschiedener Fremdkörper zur Abscheidung gelangt waren. Die im Inneren dieser Konkremente locker gefügten, kristallin inkrustierten Pflanzenfaserballen waren aussen von einer unterschiedlich dicken kompakten Mineralschicht umgeben.

Die röntgendiffraktometrische Untersuchung ergab als kristalline Komponenten: im Inneren der Konkremente: Newberyit $MgHPO_4 \cdot 3H_2O$; als dicht gefügte Schale: Bobierrit $Mg_3(PO_4)_2 \cdot 8H_2O$. Der kompakte Bobierrit-Anteil bestand aus faserigen Aggregaten von ~0,01 mm bis zu 0,1 mm dicken nach [001] gestreckten Kristallen.

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