Two Directional Delayed Compliance in the Canine Abdominal Aorta

With respect to structure and mechanical behavior the abdominal aorta has certain distinctive features: the highest connective tissue content of the aortic sites with a consequent reduction in compliance[1,2] together with a negative longitudinal strain during cardiac systole[3]. In view of these characteristics and the paucity of investigations of the time dependence of artery wall compliance in more than one direction[4], in-vitro experiments have been performed to evaluate the abdominal aortic wall stress induced by longitudinal and tangential step functions of strain encompassing the ranges of strain existing in-vivo. From these relationships a measure of the delayed compliance was derived as a function of strain.

Methods. Sections of abdominal aorta caudal to the renal arteries were removed from 10 mongrel dogs of average weight 20.0 $\pm$ 1.1 kg. The arterial segments were removed seconds after the death of the animal via a captive bolt, avoiding modulation of vascular properties by a general anesthetic. The apparatus, treatment of arterial segments, the procedures to obtain the stress-strain relationships and to maintain the arterial segments in a viable state, have been previously described in detail[4].

From the resulting stress-strain relationships for each experiment, stresses were tabulated at strain increments of 0.05, that is, 5% for strains of 0.05 to 0.70. The static elastic moduli were calculated as the secant moduli over the above strain increments and tabulated. Delayed compliance was assessed by the ratio of the stress induced in the arterial segments immediately following the application of strain (T_A) to the stress remaining after 3 min of stress decay (T_B).

Results. The static elastic moduli of the abdominal aorta in the longitudinal and tangential directions are shown in Figure 1 for ranges of strain corresponding to those found in-vivo. The maximum longitudinal mean strain existing in-vivo has been estimated as being approximately 0.47[5]. The pulsatile reduction of the mean strain during cardiac systole is small[3], with little extension during increases of mean intra-aortic pressure[6], thus the range of in-vivo longitudinal strain was taken to be 0.47 $\pm$ 0.05.

From relationships relating arterial stresses, geometry and pressure previously described[7], it can be calculated that the range of mean blood pressure of 60 to 170 mm Hg corresponds to tangential wall stresses of 462 to 1309 g/cm^2. From the experimental stress-strain relationships in the tangential direction the strains corresponding to the above stress and pressure ranges were approximately 0.325 to 0.575; corresponding to a range of strain of 0.470 $\pm$ 0.050 for the longitudinal direction. Over these ranges of strain the static elastic modulus is higher in the tangential direction: increasing with increasing strain in both directions.

The degree of delayed compliance increases with the level of initial strain, being greater in the tangential direction. The ratio T_A/T_B as a function of strain is shown in Figure 2 for the two directions. This ratio is greater in the tangential direction for all strain levels but in both directions decreases to an asymptotic level: being approximately 1.2 and 1.3 in the respective longitudinal and tangential directions. In the latter direction T_A/T_B becomes asymptotic at a higher strain level than in the longitudinal direction.

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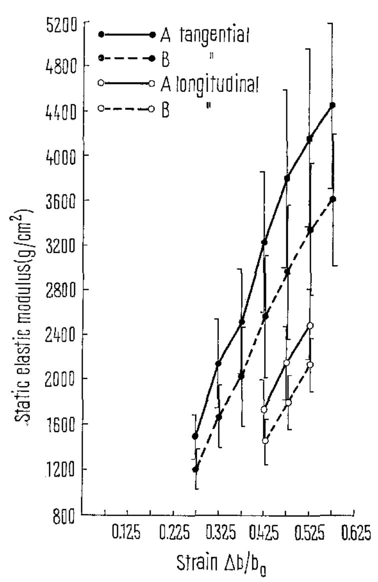

Fig. 1. Relationship between strain and the static elastic modulus for levels of longitudinal and tangential strain corresponding to those found in vivo. The solid lines represent the static elastic modulus immediately following step functions of strain; the broken lines 3 min after the application of strain.

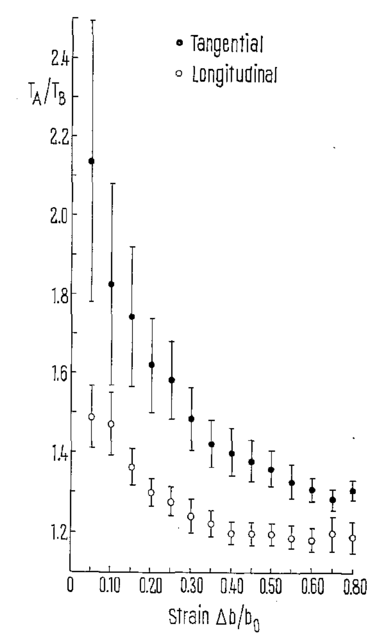

Fig. 2. Ratio of the tension developed in abdominal aortic segments immediately following the application of longitudinal and tangential step functions of strain, T_A, to the tension 3 min after the application of strain, T_B, as a function of the applied strain.

Discussion. Figure 2 shows that the delay in compliance as assessed by T_A/T_B becomes asymptotic at strain levels corresponding approximately to the upper limits of strain found in-vivo in the two directions. For high frequencies the ratio T_A/T_B can be shown to be equivalent to the ratio of the dynamic elastic modulus (E*) to the static elastic modulus (E), namely, the modulus ratio[8].

The dynamic elastic modulus E* can be expressed in terms of the static elastic modulus[9].

$$E^* = E \cdot \frac{(1+\lambda_2 s)(1+\lambda_4 s)}{(1+\lambda_1 s)(1+\lambda_3 s)} \cdots \frac{(1+\lambda_n s)}{(1+\lambda_{n-1} s)}$$

where $\lambda_1 - \lambda_n$ are constant coefficients and s represents the Laplace operator. For zero frequency

$$E^* = E^*(o) = E\,.$$

For infinite frequency

$$E^*(\infty) = E\left(\frac{\lambda_2}{\lambda_1}\right)\left(\frac{\lambda_4}{\lambda_3}\right)\cdots\left(\frac{\lambda_n}{\lambda_{n-1}}\right) = kE$$

$$\therefore \frac{E^*(\infty)}{E} = k$$

where k is a constant, whose magnitude depends upon the level of the initial strain (ε_i). In this investigation the tension (T) resulting from a step input of strain can be related to the strain, that is:

$$T(s) = E^* \mathfrak{L}\{\varepsilon(t)\} = \frac{1}{s} \cdot E^*(s).$$

For functions of time (t) where $t \to \infty$ then by the final-value theorem[10],

$$\lim_{t\to\infty} f(t) = \lim_{s\to 0} s.\, F(s)$$

$$\therefore T(t \to \infty) = T_B = s \cdot \frac{1}{s} \cdot E^*(0) = E\,. \quad {}_{s\to 0}$$

Similarly for a function of time where $t \to 0$, then by the initial value theorem[10],

$$\lim_{t\to 0^+} f(t) = \lim_{s\to\infty} s. \cdot F(s)$$

$$\therefore T(t \to 0) = T_A = s \cdot \frac{1}{s} \cdot E^*(\infty) = E^*(\infty)$$

$$\therefore \frac{T_A}{T_B} = \frac{E^*(\infty)}{E} = k.$$

The only error in equating these ratios is the extent to which $T(\infty)$ equals T_B, the latter being the residual tension 3 min after the application of strain. It was found that the decay of tension from $t = 2$ min to $t = 3$ min represented less than 2% of the tension decay from $t = 0$ to $t = 2$ min.

Thus, the ratio T_A/T_B becomes equivalent to the modulus ratio E*/E when the frequency becomes sufficiently high such that E* approximates $E^*(\infty)$.

The delayed compliance in the abdominal aorta was greater in the tangential direction than reported for the femoral artery[4] but in contrast to the latter was more pronounced in the longitudinal direction. This is consistent with the concept of a preponderance of serial arrangements between the elastic and viscous elements of the arterial wall in the tangential direction: with, however, a significant fraction of parallel arrangements in the longitudinal direction. These findings coincide with the histological observations of Benninghoff[11] and Attinger[5] who found that abdominal aortic cross sections of recoiled samples showed, at the outer border of the media, evidence of smooth muscle bundles which were cut transversely. Conversely, longitudinal sections showed longitudinally oriented muscle bundles which changed direction to run circumferentially.

Conclusion. The static elastic modulus and delayed complicance of the canine abdominal aorta were found to be a function of tangential and longitudinal strain; both variables being greater in the tangential direction. The delayed complicance was shown to be equivalent to the modulus ratio for high frequencies[12].

Résumé. Le module statique élastique et l'extensibilité (compliance) retardée de l'aorte abdominale du chien est une fonction des tensions tangentielle et longitudinale; ces deux variables prédominant dans la direction tangentielle. On a constaté que l'extensibilité était égale au rapport du module pour les fréquences élevées.

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Rôle du ganglion frontal sur le rythme cardiaque chez *Locusta migratoria* L.

Le ganglion frontal (GF) est classiquement connu pour intervenir sur une réaction nutritionnelle, paraissant différente suivant les espèces, et sur une réaction ovarienne uniforme[1]. Ces réactions, étudiées en détail chez *Locusta*[2–7], mettent en cause les corpora allata (CA)[8,9] et les cellules neurosécrétrices de la pars intercerebralis (PI)[9,10]. Connaissant l'importance de l'action des CA sur la valeur du rythme cardiaque chez *Locusta migratoria*[11–13], nous envisageons ici l'effet de l'ablation du GF sur le rythme du cœur de cet Insecte.

L'ablation du GF, qui est une opération facile, se solde fréquemment, dans nos conditions expérimentales, par une mortalité élevée, plus spécialement en fonction de l'époque de l'intervention.

Le Tableau I indique qu'il est préférable d'attendre au moins le premier jour de la vie imaginale (c'est-à-dire plus de 24 h après la métamorphose) pour réaliser l'ablation de GF si l'on veut avoir des chances de survie raisonnables jusqu'à J 15. L'opération est donc effectuée à J 1 ou J 2 sur des mâles et des femelles adultes et le rythme cardiaque mesuré 5, 10 et 15 jours après l'opération.

Les résultats, indiqués dans le Tabeau II, donnent des différences significatives à la fois chez les mâles et les femelles, à une exception près (chez les mâles, 15 jours