DYNAMIC DEFORMATION EXPERIMENTS ON AORTIC TISSUE*

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Abstract—A simple experimental scheme is described for the determination of the dynamic stress-strain relations for fresh aortic tissue within the range of strain rates up to 3.5 sec⁻¹. This is the first such work known to the authors in this dynamic range, and is considered a necessary step toward a quantitative study of traumatic rupture of the aorta. The results indicate a definite stiffening of the tissue with increasing rates of strain; the stress-strain law exhibits an exponential character.

1. INTRODUCTION

THE NUMBER of fatalities due to automobile collisions in the United States has reached an annual level of 55,000, according to the statistics of the National Safety Council. Of these, 16 per cent have been estimated by Greendyke (1966) to be due to traumatic rupture of the aorta, on the basis of a sample of 1253 automobile fatalities in Monroe County, N.Y., over a four year period. Rindfleisch (1893) was among the first to observe rupture of the aorta, attributing the phenomenon to hypertrophy of the left side of the heart, and indirectly to an increase in intra-aortic pressure, particularly in the ascending portion of the aorta. Traumatic rupture of the aorta is generally associated with accidents characterized by violent and sudden deceleration. Taylor (1962), quoted by Gable et al. (1963), has demonstrated on pigs that, during acceleration, an emptying of the distal half of the thoracic aorta occurs with engorgement of the upper half and of the arch. This retrograde flow may increase the pressure sufficiently in the region of the arch to cause rupture there. Lundevall (1964) has classified the mechanical forces acting on the aortic wall into three types: (a) Longitudinal, radial and torsional forces due to motion of the adjacent thoracic organs, (b) Wave propagation in the aortic wall due to sudden stretching of the aorta, (c) Intra-aortic pressure field. Lundevall has indicated that geometric distortion of the aorta in the sagittal plane during deceleration may cause local longitudinal stretching of the aortic wall, particularly at two points of fixation: at the base of the heart, and at the isthmus. Traumatic rupture of the aorta is observed always to be transverse, normally indicating failure axially in tension. In addition, it is expected that radial expansion of the aorta will be limited by the restoring forces of the surrounding tissue. Under normal physiological conditions the excursions in vessel radius are limited to 10 per cent, while axial displacements of the wall are virtually negligible due to the tethering effect of the many intercostal arteries branching from the aorta (see Peterson, 1960).

In the present work, a simple experimental scheme is described for loading dynamically a fresh aortic segment so as to achieve very nearly a uniaxial state of stress. The results indicate a stress-strain relation of the same exponential character as observed for other soft tissues such as muscles by Hill (1953), Fung (1970) and numerous other researchers. In particular, it appears that the constitutive relations are definitely dependent on the magnitude of the strain-rate.

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2. DETERMINATION OF STRESS, STRAIN, AND STRAIN-RATE IN AN AORTIC SEGMENT

In order to determine the constitutive relations for aortic tissue under dynamic loading, it is proposed to subject the aorta to the simplest possible stress-state, this being one of uni-axial tension. Lawton (1953), as well as numerous other authors, have indicated that a Poisson ratio of 1 corresponds to most biological tissues. This implies deformation at constant volume.

Consider the longitudinal extension of a cylindrical segment of aorta of uniform cross-section, of gage length \( L \), volume \( V_o \), radius \( R \), and wall thickness \( h \), where \( h < R \). During a equivolumic extension of the aortic segment, the wall thickness \( h \) will vary as:

\[
h = \frac{V_o}{2\pi RL}
\]  
(1)

For a cylindrical shell closed at one end, and constrained radially, by a well lubricated outer collar as in Fig. 1: a force balance on the end plug yields

\[
m\ddot{X}(t) = p(t)\pi R^2 - 2\pi Rh\sigma
\]  
(2)

where \( \sigma \) is the aortic wall stress at the end, and \( X(t) \) the axial coordinate measuring absolute travel of the end mass. The wall stress \( \sigma \) is then obtained in terms of measurable quantities as:

\[
\sigma(t) = \frac{-m\ddot{X}(t) + \pi R^2(t) \cdot p(t)}{2\pi R(t)h(t)}
\]  
(3)

where the pressure \( p(t) \) is measured from the oscilloscope tracing of the signal from a miniature pressure transducer, \( \dot{X}(t) \) is obtainable from an accelerometer attached rigidly to the end mass, \( R(t) \) is estimated from high speed ciné-films, and the wall thickness \( h \) is calculated from equation (1) on the basis of a Poisson ratio of \( \frac{1}{3} \).

A gauge length \( L \) is defined (Fig. 1) on the distal end of the aortic specimen by the segment between two wall markers spaced nominally \( \frac{1}{2} \) in. apart. The strain \( \varepsilon \) is determined as a function of the extension of this gauge length \( L \), at which locations are measured the flow pressure and the acceleration of the end plug.

\[
\varepsilon(t) = \int_{L(t_0)}^{L(t)} \frac{dL}{L} = \log \frac{L(t)}{L(t_0)}
\]

and the strain rate \( \dot{\varepsilon}(t) \) may be calculated as the corresponding time derivative from ciné-film data.

3. EXPERIMENTAL PROCEDURE

The aortic specimens, approximately 5 in. long, were pared of their surrounding fat and bathed in a physiological saline solution to preserve their properties. The ends were dried and glued (Eastman 910 glue) to short segments of hollow tapered aluminum cylinders which were subsequently connected into the high pressure surge line as shown in Fig. 1.
1. The distal end of the aortic segment was closed by an end plug, and radial expansion of the aorta during impulsive injection of water was limited by a closely-fitting outer glass collar. A furled rubber liner placed inside the aorta prevented leaks through the numerous fine holes in the aortic wall formed as a result of excision of the branching intercostal vessels. This liner always remained furled, therefore not contributing to the strength of the aorta during dynamic loading.

The pressure tank (Fig. 2) was first half-filled with water and then pressurized with compressed air from a wall outlet. A solenoid-operated valve, located as shown, was controlled by a microswitch. The valve opened very rapidly—within 5 msec after contacting the switch. A high opening speed was important to allow the water ejected from the tank to enter the aorta very rapidly and thus produce the desired high axial strain rates in the aortic wall. One end of the aorta was closed with an end plug; a sensitive pressure transducer was mounted on the proximal face of this plug and its signals were recorded on a multichannel storage oscilloscope. The aorta was inserted into a glass collar in order to restrict its radial movement. The extension of the aortic gauge length (see Fig. 1) was filmed with a high speed camera (400 frames/sec). The following procedure was used to insure that the starting time on the pressure curve corresponded to the starting time on the film: first the camera was turned on; then the valve was opened by actuating the microswitch. The oscilloscope was triggered by the same microswitch, so that pressure was recorded as soon as the switch was on. The same microswitch also turned on a high intensity lamp whose short duration light pulse produced a reference marker on the ciné-film which corresponded in time with the opening of the valve.

A series of tests, using fresh pigs' aortae, was conducted in the strain-rate range \( \varepsilon = 1.0 \sim 3.5 \text{ sec}^{-1} \), by varying the chamber pressure. In addition, a set of 'static' tests (\( \varepsilon = 0.005/\text{sec}^{-1} \)) was carried out to evaluate the creep characteristics of aortic tissue, and to afford a direct comparison with the effects of dynamic loading.

In addition, preliminary qualitative tests were performed on specimens of human aortae to assess the effects of fixation of the aorta in localizing the rupture area. Sudden motion of the aorta away from the point of insertion of the ligamentum arteriosum just distal to the aortic arch (Fig. 3) resulted in a localized rupture which rapidly propagated circumferentially in the presence of a moderate axial tension.

4. RESULTS

Static tests were performed on fresh and frozen aortic specimens (thawed just before testing) in order to test the effects of freezing. Load-extension curves for specimens during both loading and unloading cycles portray, in general, identical shapes, and the effect of freezing appears to be small. Nor are the rupture loads influenced significantly by the freezing process, as indicated in Fig. 4. Examination of the three typical static tests on one aorta (Fig. 5) indicates the negligible
are depicted in Fig. 6. In the range $\epsilon = 1 - 3.5 \text{ sec}^{-1}$, all data appear to lie above the static stress–strain curve, indicating a dependence of the constitutive relationships on strain-rate in this range. The slope of the stress–strain curve is generally steeper for dynamic loading than for static, indicating that the aortic tissue acts as a stiffer material when strained rapidly.

The stress-strain relation is represented here in the form:

$$\sigma = (0.28 + 0.18 \epsilon)(e^{1.2\epsilon} - 1) \quad \text{for } (\epsilon < 3.5).$$

The aortic material clearly displays greater stiffness under dynamic loading. The same exponential character of the stress–strain relationship has been shown by Fung (1970) to hold for other biological tissues, such as muscles and visceral tissue. The results indicate a definite sensitivity to strain-rate within the range $\epsilon = < 3.5$. This fact may have particular relevance to automobile safety, where the proper design of passenger restraint systems must ensure limitation of stress levels in the aorta, if traumatic rupture is to be avoided. The departure from a uni-axial stress state is expected to be small since radial excursions were limited to a maximum of 10 per cent; the non-linearity of the stress–strain relation renders it difficult to estimate this contribution more precisely.

The most serious limitations of accuracy occur for the strain measurements. The aortic markers diffuse somewhat into the aortic wall, appearing as relatively large zones imbedded in the remaining fatty tissue surrounding the excised aorta. Significant errors can occur in measuring minute changes in distance between such markers, particularly since they move with the superficial fat whose motion may not be representative of the average longitudinal motion of the wall. The accuracy might be improved by sewing fine metallic threads into the aortic wall, and observing their displacements in a magnetic field.
5. DISCUSSION AND CONCLUSIONS

A general test procedure has been described and applied to the determination of the stress–strain relationship for aortic tissue under a limited range of dynamic loading. This procedure for loading aortic specimens dynamically appears to possess the advantage of minimal transmission of vibrations from the loading apparatus to the specimen. Further refinements are possible for improved accuracy; additional experimental data will account more fully for the expected scatter due to variations of material properties amongst different specimens.

The initial modulus of elasticity of pigs' aortae is estimated to be $4 \sim 10 \times 10^6$ dyn/cm² at strain-rates up to 3-5 sec⁻¹. These values lie within the range of elastic moduli ($1.2 \sim 18.1 \times 10^6$ dyn/cm²) estimated by Bergel (1960) for a dog’s aorta, and compare favorably with the measurements of Peterson (1960) of $3 \times 10^6$ dyn/cm² for elastin and $30 \times 10^6$ dyn/cm² for collagen. The present experiments indicate rupture stresses of the order of $30 \times 10^6$ dyn/cm² under static loading and $40 \sim 45 \times 10^6$ dyn/cm² for dynamic loading at strain-rates up to 3-5 sec⁻¹.

As the strain-rate increases, rupture occurs at a higher stress and at lower strain. It has been noted in Section 1 that the aorta is 'tethered' to inhibit relative longitudinal motion. This would indicate that the longitudinal strain is limited, and that tensile rupture may then occur only at higher rates of strain. Such information is of prime importance in the design of shock-absorbing material in the passenger compartment of vehicles. Passenger decelerations must be limited to the extent that the rupture stress will not be reached at the corresponding rate of strain. Restraint systems possessing some slackness may eventually produce a 'second impact' upon distension, with passenger decelerations exceeding those of the vehicle.

The present results, when supplemented with additional experimental data using human aortic specimens, will serve to establish working curves relating tensile stress to strain in the aortic wall at various rates of strain. These strain-rates may be related to tolerance levels of deceleration probably through experiments, thus providing meaningful specifications for safe vehicle design.

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