Dependence of Wall Stress in the Human Thoracic Aorta on Age and Pressure

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ABSTRACT
The tangential wall stress and the tangential elasticity modulus at pressures from 0 to 200 mm Hg were calculated from pressure-volume diagrams of 27 human thoracic aortas aged 22 to 85 years. The relative volume increase of the aortas between 0 and 100 mm Hg decreased linearly from about 190% at age 20 to about 40% at age 85, whereas the elasticity modulus at 100 mm Hg increased linearly from 10 kg/cm² at age 20 to 42.5 kg/cm² at age 85. The decrease of the distensibility of the aortic wall with age was partly compensated by an increase of the internal volume of the aorta but at higher ages the distensibility was less than the theoretical values necessary for a proper buffering chamber activity. The tangential wall stress decreased linearly with age from about 2.5 kg/cm² at age 20 to about 1.2 kg/cm² at age 85. Since the pressoreceptors in the wall of the elastic vessels respond to the wall stress, this result means either that smooth muscles must increase the stress of the elastic fibers continuously, or the pressoreceptors must adapt their sensitivity throughout life to lower wall stress so that it will be kept constant. Weakness of the smooth muscles or a failure of the pressoreceptors to adapt themselves to lower stresses at higher ages may be the cause of essential hypertension.

ADDITIONAL KEY WORDS
volume-pressure relation
collagen fibers: smooth muscles
elastic fibers: life expectancy
pressoreceptors

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Accepted for publication January 26, 1967.
accomplished with a motor-driven 500-ml syringe. Its piston was connected to an optical kymograph so that its drum turned synchronously with the movement of the piston. The pressure was recorded with a membrane manometer to which a mirror was attached. It was thus possible to record volume-pressure diagrams reproducibly with one single light beam on a photographic paper. The pump rate was 5 ml/min. Four extension-release cycles were accomplished. During these cycles the aortas were floating freely in saline at 20°C. The fourth extension curve of each aorta was chosen for further calculations. The slope \((\Delta p/\Delta V)\) of the pressure-volume diagrams was obtained by drawing the tangent at intervals of 10 mm Hg on the recorded pressure-volume diagrams. The pressure at the inflection point was taken as that at which this tangent had the lowest value. At the end of the fourth extension-release cycle, the inside volume \(V_t\) at zero transmural pressure was measured by emptying the contents of the aorta into a graduated cylinder. The wall volume \(V_w\) of the aortas was measured by immersing the aorta in a known volume of water and measuring the volume increase.

Since the aortic wall is incompressible under physiological conditions (13) and the wall thickness of the aorta is relatively small compared to the radius (1) the ratio of inside radius \(r_i\) to wall thickness \(t\) at different pressures could be calculated within an error of less than 7% from the internal volume \(V_i\) at a certain pressure and the wall volume (14):

\[
r_i = \frac{2V_i}{t}.
\]

(1)

The tangential wall stress \(\sigma\) was calculated from the observed pressure \(p\) and the ratio \(r_i/t\) using Frank’s (8) equation for thin-walled tubes:

\[
\sigma = p \cdot \frac{r_i}{t}.
\]

(2)

(see Addendum).

The longitudinal wall stress in tubes is half the tangential wall stress (8).

The tangential elasticity modulus \((E_t)\) of the aortic wall at different pressures was calculated in the following way: According to Frank (8), the volume elasticity modulus

\[
\kappa = \frac{dp}{dV} \cdot V = \frac{\gamma_t \cdot \gamma_t}{2\gamma_t + \gamma_t}.
\]

(3)

\[
g_t = \frac{dp}{dt} \cdot \frac{r_i}{r_i} = \frac{2t}{r_i} \cdot \frac{E_t - (1 + \mu) p}{E_t - 2\mu}.
\]

(4)

\[
\gamma_t = \frac{dp}{dt} \cdot \frac{t}{E_t} = \frac{2t}{r_i} \cdot \frac{E_t - (1 + \mu) p}{E_t - 2\mu}.
\]

(5)

\[
[\mu = \text{Poisson’s ratio in the vessel wall (15, 16); } l = \text{length of the aorta; } E_t = \text{longitudinal elasticity modulus}].
\]

When 4 and 5 are inserted in 3, then

\[
\kappa = \frac{2t}{r_i} \cdot \frac{E_t - (1 + \mu) p}{2 - 2\mu - \frac{E_t}{E_t - 2\mu}}.
\]

(6)

or

\[
E_t = \left[ \frac{(2 - \mu) E_t}{E_t - 2\mu} + \mu \right] \kappa + (1 + \mu) p \cdot \frac{r_i}{t}.
\]

(7)

Poisson’s ratio \(\mu\) was determined by Kenner and Waldhäusl (16) as 0.2 for the aorta at physiological pressures. \(E_t\) was shown by Frank (10) to be about 1.2 at physiological pressures in the intact aorta (17). If these values are set in 7,

\[
E_t = (2.16 \kappa + 1.2 p) \cdot \frac{r_i}{t}.
\]

(8)

\(\kappa\) can be calculated from the pressure-volume diagrams by equation 3 and the ratio \(r_i/t\) by equation 1. In all calculations, \(p\) was expressed as kg/cm². For clarity, no points of measurements are shown in Figures 2 and 6; since the calculations were made with values taken from direct recordings of smooth pressure-volume diagrams, the calculated curves were also smooth.

Results

Figure 1 shows typical pressure-volume diagrams of six of the 27 human thoracic aortas of different ages. They are representative of all 27 aortas tested. The older the aortas, the larger was their inside volume at zero pressure. The radius of the aortas also increased at higher ages. At the diaphragm
Direct recording of pressure-volume diagrams of whole human thoracic aortas. The age of the aortas is given at the top of the curves. The start of each curve on the abscissa shows its internal volume at zero pressure.

The increase is nearly linear within the range tested and fits the regression

$$r_i (\text{mm}) = 5 + 0.064 A,$$  \hspace{1cm} (9)

in which $A$ is the age in years (coefficient of correlation $= 0.87, P < 0.01$; standard error of the estimate $= \pm 0.64$). Another characteristic of the pressure-volume diagrams is the decrease of the inflection point of the S-shaped curves with age. The pressures at the inflection point ($I$) at different ages fit a linear regression

$$I (\text{mm Hg}) = 106 - 1.32 A.$$ \hspace{1cm} (10)

(Coefficient of correlation $= -0.97, P < 0.01$; standard error of the estimate $= \pm 5.5$.) This means that the inflection point disappears at about 80 years. Since the pressure-volume diagram below the inflection point is mostly due to elastic fibers, and above the inflection point mostly to collagen fibers in the aortic wall (4), the disappearance of the inflection point at 80 years also indicates the disappearance of elastic tissue as a major support of the aortic wall.

Figure 2 shows the ratio $\frac{r_i}{t}$. This ratio is around 6.2 at zero transmural pressure for all ages and agrees very well with the measurements of Hieronymi (1). It varies, however, with different ages at higher pressures.

In general, the ratio $\frac{r_i}{t}$ increases with rising pressure, but the older the aorta, the lower the ratio $\frac{r_i}{t}$ at a given pressure. This means that aortas become more and more rigid with age and the relative volume increase is less with rising pressure. This result is similar to that published by Simon and Meyer (12). Figure 3 shows the percent volume increase of all tested aortas at 100 mm Hg. It fits a linear regression

$$V (\%) = 234 - 2.27 A.$$ \hspace{1cm} (11)

(Coefficient of correlation $= -0.9, P < 0.01$; standard error of the estimate $= \pm 17.4$.) The four oldest aortas tested by Simon and Meyer (12) were 80, 83, 83 and 92 years; their volume, measured at 100 mm Hg, increased 51, 40, 30 and 14%, respectively. These values are in line with the values shown in Figure 3. At the oldest age, the aorta is becoming a nearly rigid tube and has only the limited extensibility of collagen tissue.
Tangential wall stress ($\sigma$) of human thoracic aortas at 100 mm Hg related to age. The dotted line shows the wall stress of an inextensible tube with a ratio $\frac{r_1}{t}$ of 6.2 at 100 mm Hg.

The decrease in stress with age at 100 mm Hg is shown in Figure 4. The dotted line gives the stress of a rigid tube at 100 mm Hg with a ratio $\frac{r_1}{t} = 6.2$. The decrease of the tangential wall stress of the human aorta with age at 100 mm Hg follows a linear regression

$$\sigma \text{ (kg/cm}^2) = 2.86 - 0.02A.$$  \hspace{1cm} (12)

Transmural pressure of human thoracic aortas at a tangential wall stress of 2.5 kg/cm$^2$ related to age.

(Coefficient of correlation $= -0.79$, $P < 0.01$; standard error of the estimate $= \pm 0.2$.) If the regressions of $\sigma$ on age are calculated for different pressures, then regressions for the obtained parameters can be fitted and a general formula for the linear part of $\sigma$, depending upon the age ($A$) between 22 and 85 years and of pressures ($p$) between 40 and 200 mm Hg can be given. This formula is

$$\sigma = 0.04p - 0.00035Ap + 0.01A - 0.9$$

(13)

if $p$ is given in mm Hg.

Tangential elasticity modulus $E_t$ of human thoracic aortas at different wall stresses. The age is given at the end of the curves.
Figure 5 shows the pressures at which the tangential wall stress is 2.5 kg/cm² at different ages. This is about the stress of a 20-year-old aorta at 100 mm Hg. The pressure at which this wall stress is achieved increases progressively with age. The pressure follows a quadratic regression with age

\[ p = 111.6 - 0.82 A + 0.024 A^2. \]  

(Standard error of the estimate = ± 12.3.) An inextensible tube with a ratio \( \frac{r_1}{r_0} \) of 6.2 reaches a tangential wall stress of 2.5 kg/cm² at a pressure of 300 mm Hg.

The tangential elasticity modulus \( E_t \) at different tangential wall stresses is shown in Figure 6. \( E_t \) is similar at zero stress for all ages. Its mean value is 1.41 kg/cm². The \( E_t \) curves show two different parts. They rise slowly until they reach a stress which is achieved at the inflection point of the pressure-volume curve. From there on, the slope becomes steeper. These results are similar to those observed by Frank (10) on strips of the human aorta. The flat part of the curve is similar for all ages up to their stress at the inflection point of the pressure-volume curve. The regression of \( E_t \) on \( \sigma \) for the flat part is

\[ E_t = 1.41 + 3.15 \sigma. \]  

(Coefficient of correlation = 0.76, \( P < 0.01 \); standard error of the estimate = ± 0.31.) This regression shows a similar slope, as reported by Carton, Dainauskas and Clark (18) for the elasticity modulus of single elastic fibers, but the absolute values for \( E_t \) are about 4 to 5 times greater for the single fiber. The elastic tissue contributes about 20 to 30% to the aortic wall. Since the whole cross section is taken into account for the calculation of \( E_t \) in the aortic wall, the values of \( E_t \) are expected to be 3 to 5 times lower for the aortic wall than for the single fiber. But whereas the \( E_t \) curves of the elastic single fiber (18) and the elastic tissue (ligamentum nuchae) (20) remain flat at least up to a tension of 5 kg/cm², the \( E_t \) curve of the young aorta starts to become steeper at a wall stress of about 1.5 kg/cm² (Fig. 6). The stress where the steeper part begins lowers as the aorta ages, as the inflection point decreases with age.

Figure 7 shows the increase of \( E_t \) with age at a pressure of 100 mm Hg. Apparently, \( E_t \) at 100 mm Hg increases linearly with age with the simple relationship

\[ E_t = \frac{A}{A^2}. \]  

(Coefficient of correlation = 0.87, \( P < 0.01 \); standard error of the estimate = ± 4.45.) \( E_t \) would reach 50 kg/cm² if this regression is extrapolated to 100 years. This value is within the range of \( E_t \) for collagen fibers (21), when one takes into consideration that collagen tissue contributes 20 to 25% to the aortic wall (19) and, therefore, the value of \( E_t \) in the aortic wall has to be multiplied 4 to 5 times to obtain the elasticity modulus for the collagen tissue in the aortic wall.

### Discussion

The results show two striking facts: (a) the aorta at age 85 is becoming an almost rigid tube, and (b) the wall stress decreases linearly with age.

The thoracic aorta furnishes about half of the total buffering capacity of the arterial system (22); nearly half of the stroke volume...
is stored in the aorta during systole and is ejected into the circulatory system during diastole (12, 17). This is one of the major factors that keeps the arterial pressure at 100 ± 20 mm Hg throughout the cardiac cycle. The buffering chamber activity is mostly due to the elastic tissue in the aortic wall (4). The elastic tissue breaks down with increasing age and its task is taken over by the much less extensible collagen tissue. This is emphasized in Figure 7, which indicates that $E_t$ of the aortic wall at 85 years approaches the elasticity modulus of collagen fibers. The increasing stiffness shown by Figure 3 is compensated by an increase of the internal volume of the aorta (4) to keep the volume elasticity $\frac{dp}{dv}$ at the same low level. The volume elasticity $\frac{dp}{dv}$ remains constant in the aorta up to an age of about 60 years, as shown earlier (11, 12, 23). From there on it increases, which means the volume increase no longer compensates for the decrease of the extensibility, and the heart work has to increase. The volume elasticity of the thoracic aorta is about 0.0335 kg/cm² between 20 and 60 years (23). If this value is set in equation 8, an average 100-year-old aorta with an $E_t$ of 50 kg/cm² has to have a theoretical internal volume of roughly 1.0 liter to work properly as a buffering chamber. Such a large blood reservoir in the chest, which is continuously under high pressure would represent a tremendous hazard in any respect. Actually, the aorta follows the theoretical values closely up to an age of 50 years but lags behind the theoretical values from there on (Fig. 8). It seems the volume of the aorta does not increase after age 60. A similar observation was published by Simon and Meyer (12).

The decrease of the buffering capacity of the aorta with age could only be compensated by increasing the buffering capacity of the peripheral vessels. But these vessels increase their stiffness with age as does the aorta (24-27). Therefore the pulse pressure continuously increases with age because of an increase of the systolic pressure. This causes an increase of the tension-time integral of the aged heart, which will be larger the stiffer the aorta becomes.

Pressoreceptors, which regulate the blood pressure, are located in the wall of the elastic vessels (aorta, carotid sinus). The pressoreceptors appear as a very fine network of neurofibrils that nestle flat against the elastic fibers (28, 29). According to Hauss and associates (6) and Heymans and Neil (7) these pressoreceptors respond to the stress of the vessel wall. As shown in Figure 4, the wall stress decreases linearly at a given pressure with age, or, as Figure 5 shows, the blood pressure has to increase progressively to keep the same wall stress. There are two possibilities to keep the blood pressure at the same level. (a) The smooth muscles continuously increase the stress of the elastic fibers in the vessel wall. The aortic smooth muscles are able to increase the pressure in the aorta from 100 mm Hg to about 120–140 mm Hg (4). This means an increase of $\sigma$ of about 0.5–1.0 kg/cm² according to equation 13. This should be sufficient to keep the wall stress constant up to an age of around 50 years. (b) The pressoreceptors adapt their threshold (30) to a lower stress throughout life. It is tempting to consider that an increase of blood pressure in a certain group of humans may be caused either by a
weakness of the smooth muscles or an inability of the pressoreceptors to adapt themselves to lower wall stresses at higher ages. The clinical diagnosis in either case would be essential hypertension. This theory is not entirely new. Similar ideas have already been put forward (7, 30-32).

Up to the age of puberty, the percent change in volume with change in pressure is less the younger the child is (12). This means, also, the ratio \( r_t \) and the wall stress \( \sigma \) at 100 mm Hg are lower the younger the child is, since the ratio \( \frac{r_t}{t} \) remains around 6 at zero pressure at all ages (1). The wall stress never remains constant at a given pressure at any time of the life span. It increases up to the age of puberty and continuously decreases from there on.

Addendum

The wall stress in thick-walled tubes is defined by Müller (33) as

\[
\sigma = p \frac{r_t^2}{r_o^2} \left( 1 + \frac{r_o^2}{z^2} \right) \tag{17}
\]

\( r_t = \) inside radius; \( r_o = \) outside radius; \( z = \) radius of the layer in the vessel wall at which the stress is measured. Then the stress at the inside of the vessel wall is

\[
\sigma_i = p \frac{r_t^2 + r_o^2}{r_o^2 - r_t^2} \tag{18}
\]

and the stress at the outside is

\[
\sigma_o = p \frac{2r_t^2}{r_o^2 - r_t^2} \tag{19}
\]

When equation 19 is subtracted from 18, then

\[
\sigma_i - \sigma_o = p \tag{20}
\]

which means, \( \sigma_i \) is the closer to \( \sigma_o \) the larger the ratio \( \frac{r_t}{t} \) is. When Müller’s equation 17 is set in relation to Frank’s equation 2,

\[
p \frac{r_t^2}{r_o^2 - r_t^2} \left( 1 + \frac{r_o^2}{z^2} \right) = p \frac{r_t}{t} = p \frac{r_t}{r_o - r_t} \tag{21}
\]

then

\[
z = \sqrt{r_t \cdot r_o} \tag{22}
\]

This means the wall stress in thin-walled tubes like the aorta calculated with Frank’s equation 2 is, for all practical purposes, roughly halfway between the inside and the outside stress. When equation 1 is set in Frank’s equation 2 then

\[
\sigma = p \frac{2V_t}{V_w} = p \frac{2r_t^2}{r_o^2 - r_t^2} \tag{23}
\]

which is equation 19. Therefore the values for the wall stress given in this paper are the values for the stress at the outside of the aortic wall at the region where the pressoreceptors are mainly situated (28). The stress at the inside is, according to equation 20,

\[
\sigma_i = p \frac{2V_t}{V_w} + p \tag{24}
\]

The tangential elasticity modulus used in this paper is defined (8, 34) as

\[
E_t = \frac{d\sigma}{dr} \tag{25}
\]

Acknowledgment

I am indebted to Mr. Algie F. Trussell, Jr., Computer Center, University of Mississippi School of Medicine, for the statistical work.

References

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Circ Res. 1967;20:354-361
doi: 10.1161/01.RES.20.3.354

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

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