Arterial distensibility in normal and hypertensive man

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Summary

1. The pulse-wave velocity along the brachial radial artery was measured in 38 subjects and volume distensibility values were calculated.
2. Measurements made at each subject's resting blood pressure showed that those with high blood pressure had higher pulse-wave velocity values and hence less distensible arteries.
3. An acute rise in arterial distending pressure was accompanied by a rise in pulse-wave velocity.
4. When pulse-wave velocity was remeasured in all subjects at identical arterial distending pressures, no difference was found between hypertensive and normal subjects.
5. It appears that poor distensibility of large arteries in hypertensive subjects is a consequence of the elevated distending pressures and not due to irreversible structural changes in the arterial wall.

Key words: arteries, hypertension.

Introduction

The existence of morphological changes affecting the arteries in hypertension is well known, and widespread medial hypertrophy of the arterial system as well as an increased incidence of degenerative vascular disease occurs (Pickering, 1968). These changes in arterial wall structure may be expected to influence the mechanical properties of the arterial wall and in particular to reduce arterial distensibility. However, in 1880 Roy showed that arterial distensibility falls as the distending pressure rises, and this is explained by the applied load shifting with increasing pressure from the extensible elastin to the relatively inextensible collagen component of the wall (Roach & Burton, 1959; Bergel, 1961; Learoyd & Taylor, 1966). Because of this pressure-related change in arterial distensibility it is possible that the reduced distensibility of larger arteries in sustained hypertension could be explained solely on the basis of arterial distension, without irreversible changes in the structure and mechanical properties of the wall.

In subjects with very different blood pressures, we have studied the effect of an abrupt change in arterial distending pressure on an index of arterial distensibility (pulse-wave velocity), and related this index to each subject's resting blood pressure. We have also compared the arterial distensibility of each subject over the same range of distending pressures.

Subjects and methods

Subjects

We studied 38 subjects (17 females and 21 males) whose mean blood pressures ranged from 70 to 150 mmHg. In 21 the mean arterial pressure was less than 100 mmHg (arbitrarily taken as the upper limit of normal), these being normal volunteer subjects aged 19–72 years (mean 32 years). Seventeen subjects selected from outpatients and ward inpatients were hypertensive (mean arterial pressure > 100 mmHg). Informed consent to the study was obtained from all. The hypertensive subjects were aged 8–92 years (mean 55 years). Most of the hypertensive patients had essential hypertension but one had coarctation of the aorta and one chronic glomerulonephritis (demonstrated by open renal biopsy). Six hypertensive patients
were receiving diuretics or methyldopa (Aldomet, Merck, Sharp and Dohme Ltd) at the time of study.

**Pulse-wave velocity**

The velocity of the pulse wave along an artery depends on the distensibility of the artery. Pulse-wave velocity is related to the properties of the arterial wall by the equation derived from experiments on thin-wall elastic tubing by Moens & Korteweg (quoted by McDonald, 1974).

\[ C^2 = \frac{Eh}{2\rho} \]  

(1)

where \( C \) is pulse-wave velocity (m/s), \( E \) is elastic modulus of wall tissues (N/m²), \( h \) is wall thickness (m), \( r \) is vessel radius (m), and \( \rho \) is density of fluid (kg/m³).

Pulse-wave velocity has also been used to derive volume distensibility (the percentage change in volume/mmHg rise in pressure) from the equation:

\[ \text{Volume distensibility} = (3.57/C)^2 \]  

(2)

where \( C \) is pulse-wave velocity (m/s) (Bramwell & Hill, 1922).

**Experimental procedure**

All subjects were studied supine. Arterial pressure was measured in the left arm, either by an intra-arterial cannula and strain-gauge transducer (17 subjects), or by sphygmomanometric readings taken every 2 min during the experiment. Sphygmomanometric comparison of blood pressure between the two arms revealed no differences. Pulse waves were always recorded at two points along the right brachial–radial artery by Pixie strain-gauge transducers (Endevco Ltd), enclosed in a plastic mount and extended by a metal finger, which was applied to the skin over the most prominent part of the right brachial artery in the antecubital fossa, and over the radial artery in front of the lower end of the radius. The transducers, held in position by elastic straps, formed one arm of a Wheatstone bridge, which was balanced at the beginning and several times during the course of each experiment. The transducers were linear up to 147 N applied force, with a frequency response flat to at least 50 Hz, and identical phase characteristics.

The distending arterial or transmural pressure was regulated by placing the right arm with the transducers in an air-tight box (Fig. 1), in which various steady pressures were produced. Both positive and negative box pressures were assumed to be transmitted through the arm to the depth of the arterial wall. Thus if the mean arterial pressure was 80 mmHg and the box pressure was –40 mmHg then the mean transmural pressure was assumed to be 120 mmHg. Box pressure was measured by a mercury manometer, values between –80 and +80 mmHg being achieved.

**FIG. 1. Pressure box. Two transducers are applied to the skin over the artery.**
The two external pulse waves and the intra-arterial pressure were displayed on an oscilloscope, and clear wave forms were recorded on magnetic tape. The record and replay speeds of the tape recorder were identical. Records were made with the arm in and out of the pressure box, and also with the box open to air and after box pressures were held at different 10 mmHg steps for 2 min after a 30 s delay to stabilize the box pressure. When available the electrically damped mean intra-arterial pressure was also recorded once during each recording, and in the other studies mean intra-arterial pressure was calculated as diastolic blood pressure + \( \frac{1}{3} \) pulse pressure. The distance between the two skin marks was measured along the surface markings of the artery (Johnston & Whillis, 1954).

**Method of analysis**

For each pressure stage at least eight selected and artifact-free pulse pairs were replayed on to u.v. paper at 250 mm/s, the pulse-wave velocity being calculated from the time taken for a point 10% up the wave front to travel the known distance between the two recording sites.

Pulse-wave velocity was also used to calculate the volume distensibility by eqn. (2), and in 36 subjects pulse-wave velocity and volume distensibility values were available over the transmural pressure range 70–120 mmHg.

Statistical comparisons were made by unpaired t-tests and variables related by linear regression analysis. Values are given as mean ± 1 s.d.

**Results**

**Effect of transmural pressure change on pulse-wave velocity and volume distensibility values**

The pulse-wave velocity for the brachial–radial artery in all 38 subjects ranged from 7.4 to 15 m/s; in 21 normotensive subjects it was 7.4–12.5 m/s (mean 9 m/s) and in the 17 hypertensive patients 9.1–15 m/s (mean 12 m/s).

The full range of transmural pressures obtained varied between subjects, partly from the difference in individual resting blood pressures. In all subjects, however, a rise in the mean transmural arterial pressure was accompanied by a rise in pulse-wave velocity (Fig. 2). Considering the subjects individually, the relationship between pulse-wave velocity and transmural pressure was described by linear regression equations, with correlation coefficients greater than 0.9 in 35 of the subjects, and in only one was this less than 0.7. Although mean transmural pressure has been chosen, the rise in pulse-wave velocity also correlated significantly with systolic and diastolic transmural pressures.

The relationship between the change in pulse-wave velocity and change in distending pressure did not alter with age, no difference being found between the slope of this relationship in elderly subjects (70 years or more, \( n = 6 \)) and that in the age group 20–40 years (\( n = 15 \)). Moreover, no correlation exists between the slope and age or resting blood pressure.

**Volume distensibility measurements in normotensive and hypertensive subjects**

There was a linear relationship between the volume distensibility values of all the group measured at their resting mean arterial pressure, and that pressure (Fig. 3) (\( n = 38, r = 0.6568; P < 0.001 \)). Thus, at their natural pressure, the hypertensive patients had less distensible arteries than

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**Fig. 2.** Data from all 38 subjects. Each subject's pulse-wave velocity at a transmural pressure of 70 mmHg is taken as 100%. The continuous line is drawn through the mean pulse-wave velocity values calculated over the pressure range 70–120 mmHg. The broken lines represent the limits of 1 s.d from each mean. \( n = 38 \).

**Fig. 3.** Effect of resting arterial pressure on volume distensibility. Each point represents one subject's results. The line is a regression line of volume distensibility on mean arterial pressure: \( y = -0.0016x + 0.2970 \) (\( n = 38, r = 0.6568, P < 0.001 \)).
normal subjects. However, this relationship was not significant ($P > 0.05$) when volume distensibility was compared in all subjects over the same range of arterial transmural pressures, 70–120 mmHg (Fig. 4). As would be expected, volume distensibility values in all the subjects were less at the higher pressures.

**Discussion**

The velocity with which a pulse wave travels along an artery is a good measure of arterial distensibility and it is accepted as the standard against which other non-invasive methods are judged (McDonald, 1974; Gow & Taylor, 1968; Patel, Janicki & Carew, 1969). The pulse-wave velocity values observed in this study are in accord with those previously found in man for the subclavian–radial artery, the femoral artery and the femoral–dorsalis pedis artery (Hemingway, McSwiney & Allison, 1928; Schimmler, 1965; Eliakim, Sapoznikov & Weinman, 1971). Although we have assumed that the box pressures were transmitted unchanged through the arm to the depth of the arterial wall, this assumption may not be entirely true, particularly for negative pressures (Ludbrook, Manci, Ferrari & Zanchetti, 1976). However, when we compared (in 13 subjects) the box method of changing transmural pressure with a method using vasoactive drugs, we found that a discrepancy of 10 mmHg may exist with high pressures (i.e. negative box pressures) (Gribbin, 1974). This would not materially alter the results of this study.

The rise of pulse-wave velocity which occurred in all subjects when the distending pressure rose has previously been described (Hemingway et al., 1928), although Weinman, Sapoznikov & Eliakim (1971) failed to show an association between pulse-wave velocity and diastolic blood pressure. They did not measure the blood pressure directly, however, but assumed that the pressure altered with pulse intervals. We have found that even in the subjects with very high resting pressure a further increase in pulse-wave velocity occurred with increasing transmural pressure. The one exception to this was a 92-year-old woman with a resting mean arterial pressure of 110 mmHg and a very high pulse-wave velocity (16.3 m/s), who showed no significant change in pulse-wave velocity as transmural pressure was raised above her resting value and a very small change over the transmural pressure range of 70–120 mmHg (0.7 m/s). Presumably her artery was so stiff that further elevation of pressure made little difference.

In the past there has been little unanimity of opinion about the effect of chronic hypertension on the mechanical properties of large arteries and studies *in vivo* in man have proved conflicting. Greene and his colleagues (Greene, Friedlander, Boltax, Hadjigeorge & Lustig, 1966) compared pressure–volume plots from surgically exposed, isolated and ligated brachial arteries and concluded that the mechanical properties of the wall were altered in the hypertensive patients, and yet some of the vessels which had least distensibility were in subjects with only slight elevation of the blood pressure. On the other hand Nye (1964), who studied arterial properties non-invasively in normal and hypertensive subjects and used drugs to change arterial pressure, found no evidence of reduced arterial distensibility in hypertensive subjects when they were studied at the same distending pressure as normals.

Our results provide further evidence that arterial distensibility is indeed reduced in hypertensive subjects, and that the least distensible arteries are in subjects with the highest resting pressures. We have clearly shown how the distending pressure governs...
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this difference in distensibility. Although six subjects were taking anti-hypertensive drugs at the time of study, their results do not differ from the others, and there is no reason to suppose that the drugs influenced arterial distensibility, except by lowering the blood pressure. However, our results refer only to large arteries and do not exclude changes of a more permanent nature in small arteries and arterioles.

The brachial-radial artery is classified as a muscular vessel whereas the central arteries are elastic. If, however, by lowering the blood pressure, the distensibility of the central arteries can be increased, then systolic blood pressure, input impedance and left ventricular work should fall, with perhaps less chance of subsequent cardiac failure. Further, as five of the six subjects over 70 years of age, whose mean arterial pressures ranged from 98 to 137 mmHg, had the same improvement in distensibility as in younger subjects, this might imply that more vigorous treatment of high blood pressure in the elderly would be worthwhile.

References


