J. Physiol. (1961), **156**, *pp.* 445–457
With 4 text-figures
Printed in Great Britain

THE STATIC ELASTIC PROPERTIES OF THE ARTERIAL WALL

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(*Received* 9 *November* 1960)

The mechanical properties of the arteries have been extensively studied in the past, but precise quantitative knowledge of this subject is still lacking. In addition, the available data are derived almost entirely from experiments on the thoracic aorta and it is not certain how the smaller vessels behave.

Much previous work describes the relation between pressure and volume in intact vessels. Pressure, radius and wall-tension in an elastic tube are all interdependent and in most cases insufficient data are given to calculate the exact behaviour of the wall itself. In addition, the retraction seen when an artery is removed from the body (Fuchs, 1900) has been neglected. It is consequently often difficult to infer the behaviour of the vessels *in vivo*, the more so as in some cases the inflated vessel was free to lengthen, while in others the specimens were held at some unspecified length. Studies have also been made on strips and rings cut from arteries but the properties of these specimens give but little information on the relation between pressure and radius in the intact vessel.

For these reasons it was felt that the question merited reinvestigation and an apparatus was developed to determine the pressure–radius relationships of arterial specimens. This was designed to enable both static and dynamic behaviour to be measured.

THE ELASTIC MODULUS

Classical elastic theory was developed to describe the relations between applied forces and the resulting deformations in relatively stiff materials such as metals. A perfectly elastic or Hookean substance shows a constant proportionality (within certain limits) between stress (applied force per unit area) and strain (relative change in dimension). This proportionality is expressed in the familiar elastic moduli such as Young's modulus (E), the ratio of tensile stress to tensile strain. Soft animal tissues are very much more extensible than metals and do not show a constant ratio between

stress and strain. To deal with these materials an entirely different theoretical approach has been advocated and these methods have been applied to the study of tissue elasticity with some success (King & Lawton, 1950). Nevertheless, this method has not been used here, largely because it rests on the assumption that such tissues are homogeneous in structure, which is patently not the case for blood vessels. The computations involved are also very lengthy.

The modulus used here is termed the incremental modulus (following a suggestion of Krafka, 1939), and strain is defined as the ratio of change in length to the mean length during the change.

The Young's modulus of an isotropic tube, which does not change in length on inflation, is given by Love (1927)

$$E = \frac{\Delta p}{\Delta R_0} \times \frac{2(1-\sigma^2)R_i^2 R_0}{(R_0^2 - R_i^2)},$$

where ΔR_0 is the change in external radius following a pressure change Δp, R_i is the internal radius and σ is known as Poisson's ratio. This is the ratio of transverse to longitudinal strain, all materials becoming narrower when they are stretched in length. If $\sigma = 0{\cdot}5$ no change in the volume of the material occurs for a very small strain. This is not strictly true when large strains are considered (Bergel, 1960), but σ has been taken as 0·5 for the arterial wall which has been shown (Lawton, 1954) to extend isovolumetrically. This equation assumes isotropy of the material, that is that the mechanical properties are identical in all directions. There is good evidence (Fenn, 1957) that the arterial wall is more extensible longitudinally than circumferentially, but when no change in length occurs the effective circumferential modulus (measured in these experiments) is a function only of the true radial and circumferential moduli (Lambossy & Müller, 1954).

With these assumptions the incremental modulus is

$$E_{\text{inc}}\, p_2 = \frac{p_3 - p_1}{R_{0_3} - R_{0_1}} \frac{2(1-\sigma^2)R_{i_2}^2 R_{0_2}}{(R_0^2 - R_i^2)},$$

where the subscripts 1, 2, 3 represent successive measurements of pressure and radius. If no volume change occurs in the wall, then $R_0^2 - R_i^2$ is constant. The units of stress are force per unit area. Strain is a ratio of length and is dimensionless, thus the elastic modulus has the same units as stress, here given in dynes/cm^2 for 100 % elongation. It is therefore necessary to measure the internal pressure, radius and wall thickness of the arteries used.

METHODS

The method chosen to measure radius changes was a photo-electric one: this has been described elsewhere (Bergel, 1958, 1961). The vessels used (thoracic, abdominal aorta, femoral and carotid arteries) were taken from the bodies of dogs which had been bled as donors for perfusion experiments; all specimens were removed within 2 hr of death and the experiments were carried out on the same day. Before and after excision the length of the specimen was measured. Chosen lengths of vessel, about 6 cm excised length, were gently cleaned of all surrounding tissue. The limit of the adventitia was arbitrarily taken as the zone reached in this way when the specimen appeared to be clean and without obvious fringes of connective tissue. Portions were selected with as few branches as possible, but all branches included were ligated and cut close to the parent vessel. Finally the artery was painted with Indian ink to reduce surface reflexions.

The cleaned specimen was weighed twice, in air and in water; thus its volume was obtained (immersion in water lasted a few seconds only and resulted in no detectable increase in weight). It was then attached at either end to a pair of tubular metal holders of suitable size, which could be screwed into two rigid horizontal supports. The specimen was then held vertically and could be stretched to its *in vivo* or natural length. In this position it lay in the path of a collimated light beam $\frac{1}{8}$ in. (3 mm) deep and of adjustable width. An accumulator supplied a 12 V 60 W filament bulb, which was separated from the specimen by two collimating slits 12 in. (25 cm) apart.

The shadow of the artery was thrown on a small diffusing screen formed by the flat surface of a length of cylindrical Perspex rod ($1\frac{1}{4}$ in. (3 cm) diam.) which had been longitudinally bisected. The flat face had been ground with fine emery powder; the curved surface acted as a lens and concentrated the light on the photo-cathode of a photomultiplier tube (R.C.A. 931 A) mounted 2 in. (5 cm) away in a light-tight box.

The photomultiplier was supplied from a stabilized E.H.T. source at a potential of 600 V (by under-running the tube somewhat the stability is improved). The last of the chain of electrodes, the anode, was connected to earth through a total load of 2·5 MΩ, and the potential of this point was taken to a high-gain oscilloscope. The maximum current drawn from the photomultiplier was 4 μA. The greater the radius of the specimen the less the amount of light falling on the photomultiplier window and the smaller the negative potential appearing on the anode. For the purpose of calibration this potential could be balanced against that from a ten-turn potentiometer which was graduated to 1000 units. Before and after each experiment the unit was calibrated with a series of metal rods of known radius mounted in the position of the artery; a linear relation between radius and output was obtained.

Procedure. While preparing the specimen the apparatus was switched on and left for 2 hr before making any measurements. Further to reduce drift, the battery supplying the lamp was under constant charge and the leads to the light were made of aluminium strips to lessen thermal resistance changes. It was not possible to eliminate drift and the apparatus was calibrated before and after each experiment. The over-all error in the measurement of radius was $\pm 4\%$ over 2 hr; the great majority of experiments lasted about 1 hr and none took more than $1\frac{1}{2}$ hr.

Having mounted the specimen the static pressure–radius relationships were measured. By 'static' is meant that the radius at each step of 20 mm Hg between 0 and 240 mm Hg was measured, with a pause of 2 min after each change before balancing the output against the graduated potentiometer. Usually no further change in size was perceptible after this interval, but occasionally a longer pause was necessary. These pressure changes were produced with a hand bulb and reservoir bottle connected to the specimen through the lower support and were measured with a mercury manometer. The apparatus was filled throughout with isotonic saline. All experiments were carried out at room temperature (18–22° C).

RESULTS

The mean values for the incremental moduli of four types of artery up to a pressure of 240 mm Hg are shown in Fig. 1. The vertical lines represent the standard errors of the means at pressures of 100 and 220 mm Hg for the thoracic aorta and the carotid artery, the errors for the other vessels were similar to those of the carotid. In Table 1 these mean values and

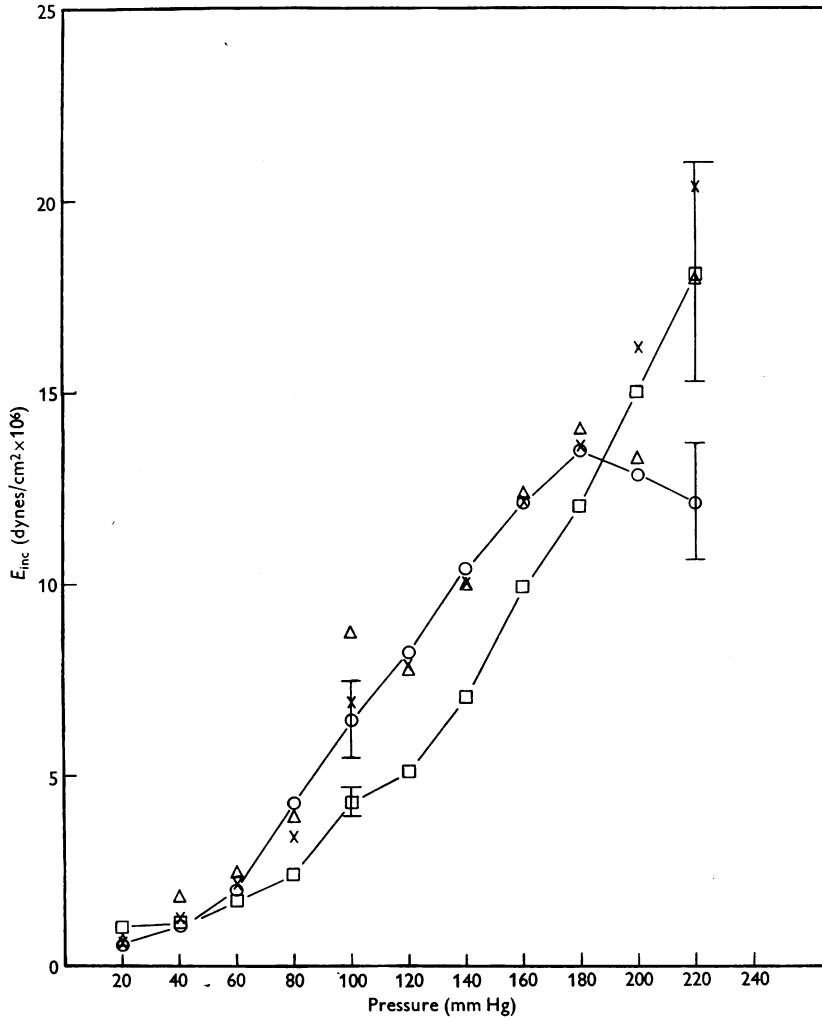

Fig. 1. Mean values for the static incremental modulus (E_{inc}) of four types of artery. Lines have been drawn connecting the points for the thoracic aorta and carotid artery; the standard errors of the means for these vessels are shown for pressures of 100 and 220 mm Hg. □ thoracic aorta; △ abdominal aorta; × femoral artery; ○ carotid artery.

standard errors are listed; the differences between the thoracic aorta and carotid artery at 100 and 220 mm Hg are probably significant ($P < 0.05$).

In all cases the vessel wall becomes progressively less extensible (higher elastic modulus) the more it is stretched. The increase in modulus is steady throughout the whole range: there is no suggestion that at some well defined pressure some less extensible material in the wall which had previously been slack is brought under strain. Up to pressures around 100 mm Hg this change is less marked in the thoracic aorta, but thereafter the modulus rises rapidly and becomes essentially the same as that of the other vessels. In the case of the carotid artery, but not of the other vessels,

TABLE 1. Mean values for static incremental modulus of elasticity (dynes/cm^2 $\times 10^6 \pm$ S.E. of mean)

Pressure (mm Hg)	Thoracic aorta	Abdominal aorta	Femoral artery	Carotid artery
40	1·2 ± 0·1 (6)	1·6 ± 0·4 (4)	1·2 ± 0·2 (6)	1·0 ± 0·2 (7)
100	4·3 ± 0·4 (12)	8·9 ± 3·5 (8)	6·9 ± 1·0 (9)	6·4 ± 1·0 (12)
160	9·9 ± 0·5 (6)	12·4 ± 2·2 (4)	12·1 ± 2·4 (6)	12·2 ± 2·7 (7)
220	18·1 ± 2·8 (5)	18·0 ± 5·5 (3)	20·4 ± 4·4 (6)	12·2 ± 1·5 (7)

The number of measurements is shown in brackets. Some additional specimens were studied at 100 mm Hg before making dynamic measurements, and these have been included.

a limiting modulus of the order of 13×10^6 dynes/cm^2 appears to be reached at extreme distension. (These figures are the differentials of a very steeply rising stress–strain curve and the small decrease may not be a real one.)

In all these experiments the length of the vessel was held constant; it is thus simple to construct a pressure–volume curve. A roughly linear relation between pressure and volume in the thoracic aorta was found up to pressures of 100–120 mm Hg. The pressure, wall thickness, and vessel radius determine the wall tension, but the pressure–volume curve will be a straight line if the wall modulus increases proportionally with the fourth power of the radius (Bergel, 1960). This is the situation in the thoracic aorta below 100 mm Hg, but for the other vessels the increase in modulus is steeper. At high pressures the volume distensibility ($\Delta V/\Delta p$) of the thoracic aorta becomes progressively less. The other vessels show steadily diminishing distensibility throughout the whole pressure range. The behaviour of these thoracic specimens contrasts with that reported by Roy (1880), Wagner & Kapal (1951) and, for young specimens, by Hallock & Benson (1937), all of whom found markedly sigmoid pressure–volume curves. The first two of these reports refer to experiments in which the vessel was allowed to lengthen on inflation, while Hallock & Benson appear to have held their vessels at excised length during a rapid (*ca.* 2 mm Hg/sec) inflation. In another experiment these procedures were

carried out on the same specimen of thoracic aorta. The vessel was inflated slowly (2 min between each increment of 20 mm Hg) while held at its natural length, and again while free to lengthen, and finally a rapid inflation (0–300 mm Hg in 3 min) was performed at the excised length. The experiments were performed in the order given and 2 hr separated them to allow the vessel to recover fully from the stretch.

The results (Fig. 2) show that the double curvature of the pressure–volume curve is much accentuated by allowing it to lengthen, or by rapid inflation. Roy (1880) and Wagner & Kapal (1951) have emphasized that

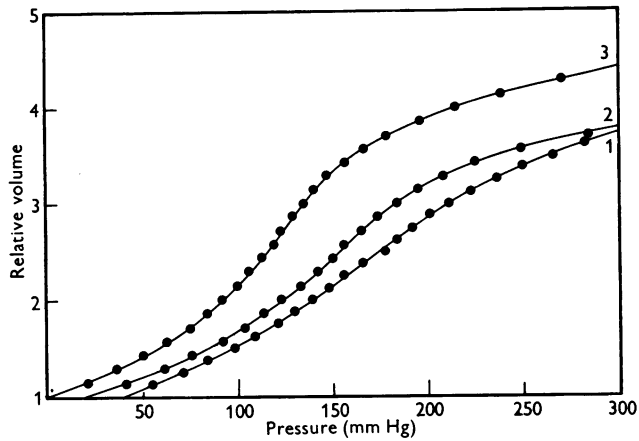

Fig. 2. Three pressure–volume curves for the same thoracic aorta. (For clarity, curves 1 and 2 are displaced horizontally.) 1, slow inflation, held at natural length; 2, fast inflation, held at natural length; 3, slow inflation, free to lengthen. Note the markedly sigmoid form of curve No. 3 which results from the vessel being free to lengthen as it is inflated. This curve also shows the greatest over-all distensibility.

the aorta is most distensible in the physiological pressure range. This effect, which is largely due to artifacts, can be seen in the high slope of the distensibility curve (No. 3) in this region.

During successive inflations all vessels became progressively larger. This tendency was particularly marked in the muscular vessels and it was normally reduced as far as possible by inflating the vessel to 250 mm Hg for a few minutes before each experiment, allowing the specimen to stretch and thus reducing the subsequent slow dilatation.

By omitting this preliminary stretch it was possible to make measurements on the same vessel over a wide range of diameter. The results of one such experiment are shown in Figs. 3 and 4. Figure 3 shows three pressure–volume curves obtained from the same specimen of femoral artery. These have been calculated from the measurements of external

radius. The first cycle of inflation and deflation was performed on a vessel which had not been under pressure since the death of the animal some hours previously. This cycle resulted in a wide loop, and at the end the zero-pressure volume had increased by some 50%. After being held at a steady internal pressure of 150 mm Hg for 20 min, during which time a further increase in size occurred, a second cycle was carried out. Again the vessel became larger. After dynamic measurements (mean pressure of 100 mm Hg for 20 min) a final slow inflation and deflation followed.

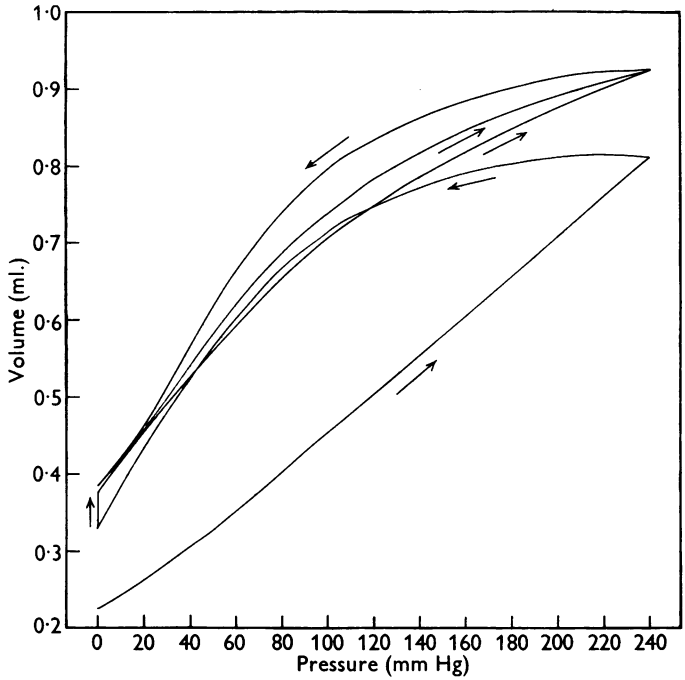

Fig. 3. Three consecutive pressure–volume curves of a femoral artery. The sequence of inflation and deflation is shown by the arrows (the last two deflations followed the same course). The vessel appears to become rather less distensible during the series of inflations (cf. Fig. 4).

This time the loop was closed (the course of deflation was identical in the second and third cycles) and no further increase in size occurred. This gradual increase in size accompanied by narrowing of the hysteresis loop has been observed by many workers (e.g. Remington, 1955).

The same data, for the inflation limbs only, are plotted as elastic modulus and radius in Fig. 4. All the points lie about a single curve, though the wall was somewhat stiffer on the first occasion. However, the modulus at any given pressure increased with each inflation. Points referring to 100 mm Hg are indicated by vertical lines and the modulus

increased from 1·1 to 4·8 × 10^6 dynes/cm^2. In the first cycle the vessel was less stiff because it was smaller. This experiment was repeated on other vessels with similar results.

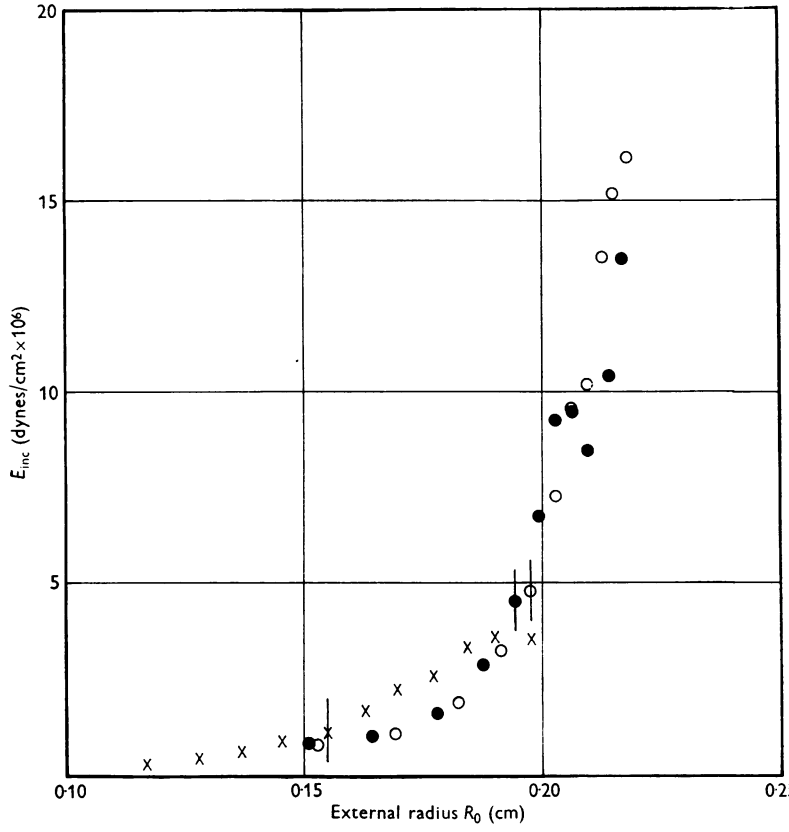

Fig. 4. The variation with radius of incremental modulus calculated from the inflation limbs of the three curves shown in Fig. 3. The vertical lines indicate points at 100 mm Hg pressure. × first inflation; ● second inflation; ○ third inflation. Note the consistency in behaviour seen when the results of the experiment illustrated in Fig. 3 are plotted in this fashion.

Measurements were also made of the amount of shortening on excision, and of the relative wall thickness (h/R_0) at an internal pressure of 100 mm Hg. Mean values are shown in Table 2. The wall-thickness figures were computed from measurements of external radius and the wall volume of the specimens. A few subsequent measurements on cut sections of vessels fixed while distended gave the same results.

TABLE 2. Mean values for relative wall thickness (h/R_0) and amount of retraction in different dogs' arteries

Vessel	h (% of R_0 at 100 mm Hg internal pressure)	Retraction (shortening as % original length)
Thoracic aorta	10·5 ± 0·46 (15)	31·9 ± 0·6 (22)
Abdominal aorta	10·5 ± 0·61 (9)	34·0 ± 0·6 (17)
Femoral artery	11·5 ± 0·96 (11)	42·0 ± 0·9 (22)
Carotid artery	13·2 ± 0·75 (13)	35·0 ± 0·5 (29)
Iliac artery	—	39·5 ± 0·7 (9)

The variations shown are the S.E. of means. Figures in brackets are the number of measurements.

DISCUSSION

The arterial properties illustrated in Fig. 1 are qualitatively similar to those reported by other workers. A full discussion of earlier studies has been published previously (Bergel, 1960) and only those reports which provide adequate quantitative data will be considered here. The longitudinal modulus of the thoracic aorta has been reported as 1·5 (Hardung, 1953) and 2–3 (Lawton, 1955) for strips, and as 2 (McDonald, 1960) for whole vessels; where all units are dynes/cm^2 × 10^6. The value reported here, 4·3 ± 0·4 is 1·5–2 times as great, but it has been estimated previously (Bergel, 1960) that this vessel is about 1·5 times as stiff circumferentially as longitudinally at 100 mm Hg internal pressure. Lawton's (1960) figures of about 2 × 10^6 dynes/cm^2 for the longitudinal modulus of a wide variety of arteries refer to somewhat retracted strips. It can be seen that all vessels have similar moduli at low strains.

The increase in modulus with increasing pressure depends both on the elastic properties of the collagen, elastin and muscle within the arterial wall, and on their arrangement and linkages. Various tissues have been taken to exemplify the mechanical behaviour of these wall constituents; the reported values of the elastic moduli of these tissues are presented in tabular form (Table 3). However, some caution is necessary when comparing these values with those found for arteries. In tissues such as the arterial wall the collagen forms, at moderate strains, a loose mesh. Only when fully distended, so that the collagen comes to lie more nearly parallel, would one expect the modulus of this material to approach that shown by the dense material which forms tendon. Ligamentum nuchae is known to contain some collagen. Hass (1942) extracted aortic collagen with formic acid and studied the properties of the remaining elastin, which were probably not entirely unchanged by this treatment. Burton (1954) has calculated the elastic modulus of elastin to be 3 × 10^6 dynes/cm^2 from Hass's data. This figure is based on the cross-sectional area of a desiccated specimen and should be nearer 1 × 10^6 (McDonald, 1960). It is

probable that the true value lies between this figure and that reported for ligamentum nuchae, which is 6.

Mammalian smooth muscle is an unsatisfactory material for mechanical studies, and molluscan unstriated muscle may well be considerably different from that found in mammalian blood vessels. Ducret (1931) estimated the maximal tension exerted by the muscle in a cat's mesenteric artery to be $0{\cdot}8 \times 10^6$ dynes/cm^2. This value is based on histological estimates of the amount of muscle present and cannot be very accurate. However, the elastic modulus of active muscle must be considerably greater than the tension it can exert.

TABLE 3. The elastic moduli (E) of arterial wall constituents

Tissue	E (dynes/cm$^2 \times 10^6$)	Author
Collagen (tendon)	100	Reuterwall (1921)*
	30–100	Krafka (1939)
	1000	Burton (1954) (30 % strain)
Elastin (ligamentum nuchae)	6	Reuterwall (1921)*
	6	Wöhlisch, de Rochement & Gerschler (1926)
	6	Krafka (1939)
Smooth muscle (resting)	0·1	Bozler (1936) (molluscan)
	0·1	Krafka (1939) (mammalian taenia coli)
	2·5	Reichel (1952)* (molluscan)

* Calculated by Bergel.

It is generally assumed that the collagen and elastin in the arterial wall function in parallel. This concept appears to have been first clearly formulated by Reuterwall (1921); it is supported by the histological studies of Benninghof (1930) and by the results reported here. Arterial moduli of the order of 20×10^6 dynes/cm^2 (see Table 1) can only result from the presence of some material as strong as collagen which is not in series with any weaker substance. The thoracic aorta contains about 1/6 collagen (Harkness, Harkness & McDonald, 1957); if this is arranged wholly circumferentially and bears all the load at full distension it is then exhibiting a modulus of 120×10^6 dynes/cm^2, which is similar to that reported for tendon by Reuterwall (1921) and Krafka (1939).

It is not easy to see why the carotid wall should have reached a limiting modulus of about 13×10^6 dynes/cm^2, for there is no sign that a similar limit has been approached in the other vessels. At a pressure of 220 mm Hg the tension on the collagen in the carotid wall is of the order of 10 kg/mm^2 and it is conceivable that the ground substance cementing the fibres might begin to flow. However, there was no sign that the vessels were in any way damaged by these pressures, which are not far outside the physiological range for the dog.

As the vessel is allowed to deflate the load is progressively passed from

the relatively inextensible collagen on to the elastin. It has been suggested that this tissue bears all the tension at pressures below 100–150 mm Hg (Reuterwall, 1921; Roach & Burton, 1957). However, the wall modulus of thoracic aorta at 100 mm Hg, $4{\cdot}4 \times 10^6$ dynes/cm^2, if due solely to the elastin present (30%, Harkness *et al.* 1957) would require the elastin to have a modulus twice as great as the values given in Table 3. Reuterwall's (1921) data show that the modulus of ligamentum nuchae at low strains is considerably smaller. It seems more likely, as McDonald (1960) has suggested, that even at the lowest pressures some material stronger than elastin, presumably collagen, contributes significantly to the arterial wall modulus. Elastin may be readily extended to 250% of its original length (Reuterwall, 1921) and it may be concluded that the difference in behaviour between the thoracic aorta and the other vessels between 60 and 100 mm Hg pressure (Fig. 1) is due to the great preponderance of elastin over collagen (2:1) that has been found (Harkness *et al.* 1957) only in the thoracic aorta, and to a relatively loose collagen network.

There is relatively little difference between the moduli of elastin and smooth muscle (Table 3) and similar arguments cannot be used to decide the disposition of these materials in the vessel wall. Histological observations (Benninghof, 1930) indicate that they are arranged in parallel, and this is supported by similar (unpublished) studies of my own. There can be no other explanation for the behaviour illustrated in Fig. 4 than that the material which was extended by a steady high pressure, presumably muscle, is arranged in parallel with the other elements. Since this specimen was rather stiffer during the first inflation (crosses) than subsequently it is possible to make an estimate of the modulus of resting smooth muscle. Assuming that the femoral wall contains 75% muscle, a value of about 1×10^6 dynes/cm^2 has been calculated. This figure is also based on the assumption that the stretched muscle subsequently develops no significant elastic tension (Abbott & Lowy, 1958); if this is not the case the figure should be higher. A similar value has been estimated from measurements of dynamic elasticity (Bergel, 1961). In view of these results, which are similar in value to those quoted in Table 3, the hypothesis of Burton (1954) that the muscle works through a complex system of linkages, conferring a mechanical advantage of about 100:1, is unnecessary. This suggestion stemmed from the assumption that the elastic modulus of smooth muscle was much lower, $0{\cdot}06 \times 10^6$ dynes/cm^2.

A parallel arrangement of the constituents implies that the properties of the arterial wall are more directly related to radius than to pressure, and this may be clearly seen in Fig. 4. For practical purposes it is more useful to relate the modulus to pressure, as has been done here. It should be remembered that, under normal conditions, arteries are prevented from

collapsing fully. Nevertheless, it takes a considerable time under pressure for a collapsed vessel to regain its original size, and significant changes in arterial elasticity are to be expected immediately following any substantial fall in blood pressure.

The amount of retraction seen in these vessels is very similar to that reported by Fuchs (1900) sixty years ago. It can be seen from Fig. 2 how greatly changes in length can alter the behaviour of arteries quantitatively and qualitatively. This shortening should obviously be taken into account in any study of arterial elasticity, particularly when longitudinal strips are used.

SUMMARY

1. The use of a simple incremental modulus of elasticity is proposed for description of the mechanical properties of the arterial wall. Measurements of this modulus were made on the thoracic and abdominal aorta, femoral and carotid arteries, of the dog. In all these experiments the vessel was held at the length it had in life.

2. The arterial wall becomes stiffer as it is extended. This increase is less marked in the thoracic aorta up to a pressure of 100 mm Hg. Mean values for the static elastic modulus (dynes/cm$^2 \times 10^6$) at 100 mm Hg pressure were thoracic aorta 4·3, abdominal aorta 8·7, femoral artery 6·9, carotid artery 6·4.

3. The elastin, collagen and smooth muscle in the arterial wall are arranged in parallel and each bears some load at all internal pressures.

4. The elastic modulus of resting vascular smooth muscle is probably in the region of 1×10^6 dynes/cm^2.

5. Values for the relative wall thickness and the retraction on excision are given.

I am very grateful to Drs D. A. McDonald and M. G. Taylor for advice and discussion; to Mr D. C. Moore and his staff for technical assistance; and to the Royal Society for the provision of a calculating machine used in this work.

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