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MECHANICAL DESIGN IN ARTERIES

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Summary

The most important mechanical property of the artery wall is its non-linear elasticity. Over the last century, this has been well-documented in vessels in many animals, from humans to lobsters. Arteries must be distensible to provide capacitance and pulse-smoothing in the circulation, but they must also be stable to inflation over a range of pressure. These mechanical requirements are met by strain-dependent increases in the elastic modulus of the vascular wall, manifest by a J-shaped stress–strain curve, as typically exhibited by other soft biological tissues. All vertebrates and invertebrates with closed circulatory systems have arteries with this non-linear behaviour, but specific tissue properties vary to give correct function for the physiological pressure range of each species. In all cases, the non-linear elasticity is a product of the parallel arrangement of rubbery and stiff connective tissue elements in the artery wall, and differences in composition and tissue architecture can account for the observed variations in mechanical properties. This phenomenon is most pronounced in large whales, in which very high compliance in the aortic arch and exceptionally low compliance in the descending aorta occur, and is correlated with specific modifications in the arterial structure.

Key words: artery wall, elastic modulus, non-linear elasticity, elastin, collagen, lamellar unit.

Introduction

In the simplest functional terms, closed circulatory systems such as those of vertebrates, crustaceans and cephalopod molluscs can be regarded as a pump and a set of conduits that circulate the blood. In all cases, the pump is powered by cyclical contractions of linear actuators (muscle) and, consequently, the flow of blood from the heart is very pulsatile. Hence, blood pressure and flow velocity entering the arterial tree rise and fall during each cardiac cycle, reaching peak values that are much greater than their mean values. Ideally, however, blood flow to and through the peripheral capillary beds should be steady in order to minimise the hydraulic power requirements of the heart (pulsatile flow is more costly because the blood mass is accelerated and decelerated in each cycle) and to minimise the drag force imposed by flow on the endothelial cells lining the small vessels (Alexander, 1968). The solution to the pulsatility problem is an exquisite mechanical one that seems to be common to all circulatory systems examined so far: distensible elastic blood vessels.

This innovation provides a pulse-reducing effect on the flow of blood throughout the system by passive expansion and elastic recoil. In peripheral exchange vessels, flow is nearly continuous because the upstream arterial tree acts as an elastic ‘reservoir’. The major vessels expand with blood each time the heart contracts (systole) and then recoil elastically to continue blood flow to the small peripheral vessels and capillary beds while the heart is refilling (diastole). Flow waves become less pulsatile peripherally as the systolic peak decreases and the diastolic component increases. For example, in humans, the ratio of the flow pulse amplitude to the mean flow decreases from approximately 6 in the aortic arch to less than 2 distal to the femoral artery (Milnor, 1982). The elastic compliance of the arterial system also prevents the arterial pressure from falling abruptly after the heart valves close and reduces the pressure pulse, the pressure wave velocity and the hydraulic impedance faced by the heart. In turn, these factors influence the propagation and interaction of pressure and flow waves throughout the arterial tree and, consequently, the energetic demands placed on the heart. Thus, vascular elasticity is a profoundly important determinant of blood flow dynamics in any circulatory system.

Arteries must have non-linear elastic behaviour

When a material with linear elasticity is deformed, a plot of the applied stress (i.e. force/cross-sectional area) *versus* the resulting strain (i.e. Δdimension/initial dimension) yields a straight line whose slope is the Young’s modulus of elasticity. This is a measure of the elastic stiffness of the material, because one with high Young’s modulus is stiffer, and less easily deformed, than one with low Young’s modulus. But, like most soft biological materials, blood vessels do not have simple linear elastic properties. Instead, they exhibit non-linear behaviour when distended, so that elastic stiffness increases with the degree of loading, and it is more appropriate to

characterize these properties using an elastic modulus that is expressed as a function of the strain or distending pressure.

It turns out that non-linear behaviour is the key to elastic stability in any highly distensible pressure vessel, protecting against aneurysms and 'blowout' (Burton, 1954, 1962; Gordon, 1975; Bogen and McMahon, 1979; Gosline, 1980). The inflation behaviour of a cylinder is dictated by the so-called Law of Laplace: $T=PR$, where T is circumferential tension, P is pressure and R is internal radius. Now, the non-linear properties of an artery may be represented by a plot of T *versus* R (Fig. 1A), and onto this plot are placed a family of straight lines through the origin, each representing the Laplace relationship at a different pressure (note that the Laplace plot of T *versus* R for pressure P_i is a straight line with slope P_i). The result demonstrates that each Laplace line has only one point of intersection with the artery curve, and this point represents the equilibrium radius for the artery at that pressure. To maintain stable inflation, increasing pressures must all have equilibrium intersection points on the artery curve. For comparison, a similar operation on the tension–radius curve for a cylindrical rubber tube (Fig. 1B) shows that instability begins at the shoulder of the sigmoidal curve, where a small increment in pressure results in a large jump in radius, forming the aneurysm that is typically observed when a balloon is inflated. The instability occurs at the pressure at which the Laplace line is tangential to the inflation curve for the cylinder. This analysis illustrates that the tension–radius curve for the elastic cylinder must have a continuously increasing slope (i.e. non-linear elasticity) to be stable under increasing pressure. This requirement was also illustrated more quantitatively by Burton (1954), who defined relative volume distensibility as $D=(dV/V dP)$, where V is volume, and showed that this was inversely proportional to the expression $(Eh/R)–P$, where E is the elastic modulus and h is the wall thickness. So, as P increases, R will increase and h will decrease (this analysis applies primarily to the aorta and large arteries, where wall properties are not influenced significantly by vascular muscle activity). If E is constant (linear elasticity), then D will increase and become infinitely large as P approaches Eh/R, causing rupture. To guard against this, E must increase as a function of P, such that Eh/R is always greater than P. This condition is satisfied if the modulus increases as P^n, where n is greater than or equal to 3 (Bogen and McMahon, 1979; Gosline, 1980).

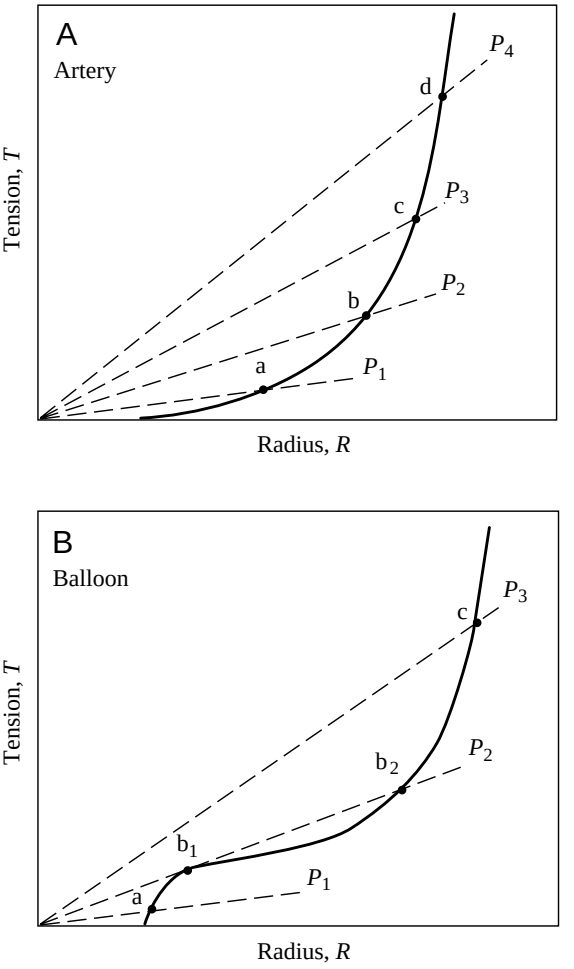

Fig. 1. (A) Plot of the mechanical response of an artery to inflation, showing the non-linear wall tension (T) *versus* internal radius (R) relationship (solid curve). The four broken lines are Laplace plots for four levels of pressure (P_1–P_4), each having a unique intersection point with the artery curve (a–d, respectively). As long as the Laplace lines are not steep enough to become parallel to the artery curve, there will be a stable inflation. (B) Plot of the mechanical response of a rubber tube to inflation, showing a typical sigmoidal response (solid curve) and a series of Laplace lines as in A. In this case, the tube is unstable between the points b_1 and b_2, where the Laplace line for P_2 lies tangential to the rubber curve.

Experimental description of artery wall properties

The earliest quantitative study of vascular elasticity appears to be the work of Charles Roy (1881). He constructed an ingenious gravity-driven apparatus that automatically performed *in vitro* inflation of blood vessel segments (from humans, rabbits and cats), measured instantaneous pressure and volume change, and plotted the resulting $P–V$ curves 'on-line' to a rotating smoked-drum kymograph (Figs 2, 3). Roy (1881) also performed *in vitro* uniaxial tests on strips of artery wall using a simple apparatus that plotted the force–length curves for the tissue as it was stretched. Using these experimental data, Roy correctly documented that the mammalian artery wall had non-linear elasticity and that the distensibility of the human aorta decreased as a function of age. Roy showed that arteries are subjected to a considerable level of distention by the resting blood pressure; thus, the circumferential stress is never zero *in vivo* (a related and much newer observation is that residual stress in the mammalian aorta at zero pressure makes the *in vivo* stress fairly uniform across the wall thickness; Fung, 1990). Roy also made the interesting observation that the greatest compliance of the aorta (dV/dP) coincides with the normal range of blood pressure (Fig. 3).

In other noteworthy experiments, Roy (1881) investigated

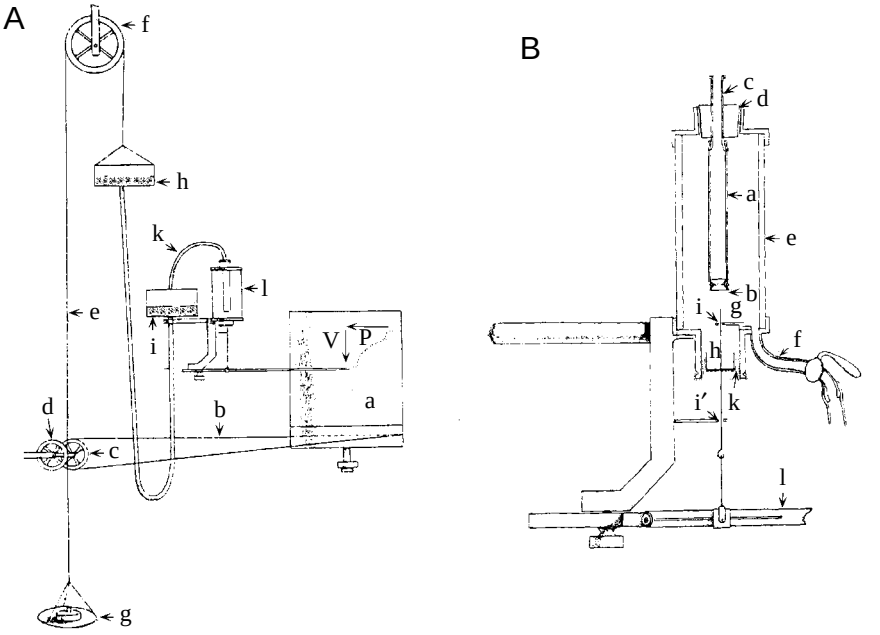

Fig. 2. (A) The artery inflation apparatus used by Charles Roy (1881). The kymograph drum, a, is wrapped with smoked paper on its upper three-quarters, while the lower quarter is left bare. Around this area passes a strong endless silk thread, b, which links the motion of the drum to a rotating pulley, c, mounted on the same axle as an identical pulley, d. Another silk thread, e, makes one turn around d and then attaches to a weight pan, g, used to counterbalance a mercury reservoir, h. The reservoir is suspended from the other end of e, passing over a large pulley, f. Connecting the open mercury reservoir, h, with the small pressure vessel, i, is a flexible tube. Another tube, k, of narrower diameter, brings the interior of the vessel i into communication with a cannula onto which the artery segment is mounted inside the chamber, l. The reservoir h and the tube leading from it are filled with mercury, which also half-fills the reservoir i. The upper half of i, the tube k and the artery segment all contain air. (B) Details of the cylindrical chamber, e (labelled l in A), that contains the artery segment, a. Through the rubber stopper, d, at the top passes a cannula, c, that is connected to the small tube shown as k in A. The artery segment is tied on the flared end of the cannula. The distal end of the segment is closed off by a grooved wooden plug, b. At the bottom of the chamber, e, is a drain port, f, to allow entry or exit of the olive oil that fills this chamber. In the large opening at the bottom of the chamber is a light piston, h, through the centre of which is fixed a fine steel wire, g, supported in the lateral directions by two guides, i and i′. A fine flexible membrane, k, makes a seal around the piston. The chamber acts as a plethysmograph or volume-measuring device. Changing the pressure in the artery by raising or lowering the mercury reservoir causes corresponding volume changes in the test segment which are accommodated by movements of the piston h. As h moves up or down, so does the wire g and the recording lever l to which it is attached. A stylus at the end of the lever inscribes a line on the rotating smoked drum. Thus, vertical displacement of the writing stylus corresponds on the drum to an axis of artery volume. Referring back to A, it can be seen that pressure in the artery segment is determined by the difference between the mercury levels in the reservoirs h and i. The experiment starts with h and i at the same level (i.e. pressure=0), and pressure is then increased by pulling the silk thread, e, down and raising h. The same motion causes rotation of pulleys c and d and of the drum, a. Thus, the horizontal axis on the drum corresponds to pressure changes in the artery, and the resulting plot inscribed is one of volume (*V*) *versus* pressure (*P*).

the thermo-elastic properties of arterial tissues using thermocouple devices. He showed that heat was released from the artery wall during extension, and that the application of heat to a piece of artery that was stretched by a suspended weight reduced the degree of stretch (i.e. heat increased the stiffness of the artery wall, so the applied stress produced less strain). These unexpected properties were contrary to the known behaviour of crystalline elastic solids, but Roy recognized that biological tissues such as artery wall had an elastic mechanism that was thermodynamically like that of natural latex rubber, although the physics of this type of elasticity was not understood at that time. Modern research on synthetic rubber-like polymers, as well as animal rubbers such as elastin, has revealed that the elasticity of such polymer networks is based primarily on changes in the entropy of the molecular chains that occur when the polymer is deformed (Gosline et al., 1988). An imposed strain increases order in the molecular network, thereby decreasing its entropy. The elastic force arises from the tendency of the kinetically free network to return to conformational states of higher entropy, or more disorder, according to the laws of thermodynamics. This mechanism is quite different from the changes in chemical bond energy that provide elastic restoring force to a deformed crystalline solid (Treloar, 1982).

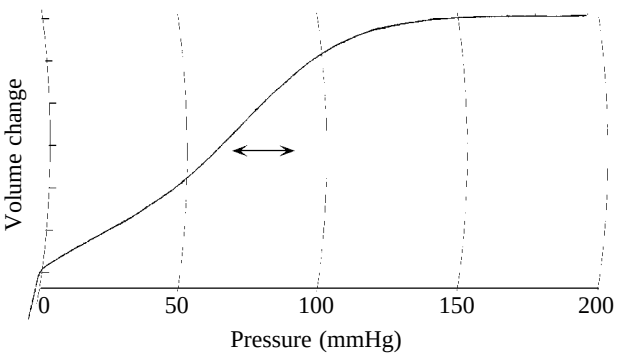

Fig. 3. A plot of volume change *versus* inflation pressure for a segment of rabbit carotid artery tested in the apparatus shown in Fig. 2. The curved vertical axis results from the arc drawn by the writing stylus. The slope of this inflation curve (d*V*/d*P*) represents the wall compliance, which is greatest from 70 to 90 mmHg, as indicated by the arrow. Above approximately 110 mmHg, the artery compliance is greatly reduced (redrawn from Roy, 1881). 1 mmHg=0.1333 kPa.

Arteries are composite structures

Arteries are composite structures that derive their non-linear properties from the combination of both rubbery and stiff fibrous constituents. The incorporation of extracellular elastic proteins into arteries has occurred independently in the evolution of several animal phyla. The most studied arterial elastomer is elastin, which occurs in nearly all vertebrates (Sage and Gray, 1979). This rubber-like protein forms a highly extensible tissue that has an elastic modulus of approximately 1 MPa, comparable with that of an ordinary rubber band (Aaron and Gosline, 1980). Collagen is relatively inextensible and acts as a stiff reinforcing component. The elastic modulus of collagen fibres has not been measured directly, but tendons that are predominantly composed of parallel collagen fibres have elastic moduli greater than 1 GPa. Thus, collagen is more than 1000 times stiffer than elastin. In lampreys and invertebrates, such as squids, octopuses, whelks, lobsters and crabs, there are elastin analogues (i.e. proteins with similar elastic properties to elastin, but which are chemically distinct), while collagen is also the stiff reinforcing element in the artery walls of invertebrates (Shadwick and Gosline, 1981, 1985a; Wright, 1984; Shadwick et al., 1990; Davison et al., 1995; McConnell et al., 1996).

The importance of the composite nature of the artery wall in providing the essential elastic non-linearity was clearly shown in a classic study by Roach and Burton (1957). By selectively digesting collagen or elastin from samples of human artery, the individual mechanical role of each was demonstrated (Fig. 4). They found that the initial stiffness of the artery wall represented the elasticity of the elastin, while the much higher stiffness at high strains represented the contribution of fully tensed collagen fibres. The upturning region of the stress–strain curve represented the transition between low- and high-stiffness behaviour and corresponded to the normal *in vivo* range of loading. This transition was proposed to represent a progressive straightening and mechanical recruitment of collagen fibres throughout the distended wall. This interpretation suggests that the elastic force in the artery wall at and above normal physiological pressures is dependent on contributions from both elastin and collagen, and that the transfer of load from elastin to collagen occurs over a broad range of pressure. Roach and Burton (1957) also showed using this technique that the so-called hardening of human arteries with age could be correlated not only to a relative increase in collagen but also to a loss of the initial low-compliance, elastin-dominated phase, causing collagen loading to occur at lower circumferential strains.

Bergel (1961) characterized the non-linear viscoelasticity of the artery wall in terms of the dynamic incremental elastic modulus on the basis of pressure–radius data for vessel segments subjected to sinusoidal pressure oscillations at different frequencies. Using this approach, the elastic stiffness of the vessel was quantified as it changed with distending pressure. Bergel showed that the incremental elastic modulus of the aorta increased sharply over the physiological pressure range, supporting the view that this is a transition region where both elastin and collagen bear the circumferential load. He also documented that the elastic modulus, at mean blood pressure, increases along the arterial tree with greater distance from the heart. This longitudinal change is correlated with a decrease in the elastin:collagen ratio from approximately 2 in the aortic arch to approximately 0.5 in the abdominal aorta (Milnor, 1982).

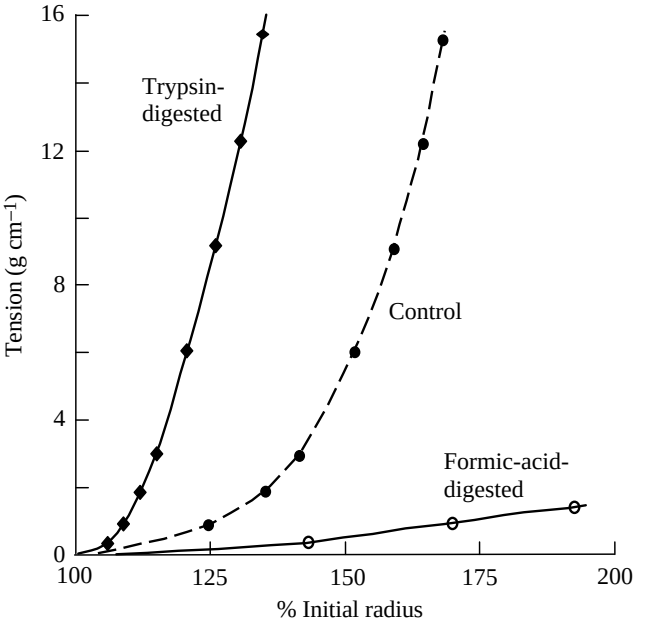

Fig. 4. The contributions of elastin and collagen to the tension–length response of human iliac arteries. When elastin is removed by trypsin digestion, the curve (diamonds) represents the properties of the remaining collagen. Alternatively, when collagen is removed by formic acid digestion, the curve (open circles) represents the properties of the elastin fibres. The broken curve (filled circles) is for an untreated artery (redrawn from Roach and Burton, 1957).

When an artery is subjected to inflation–deflation cycles, viscoelasticity will cause the pressure–volume curve for deflation to fall below that for inflation, forming a hysteresis loop. The area enclosed by this loop represents the energy lost in each cycle which, for most arteries, is of the order of 15–20 % of the total strain energy input. This means that most, but not all, of the strain energy is recovered elastically each time the artery wall is distended. Dynamic oscillation tests have been used to characterise arterial viscoelasticity in mammals, and the results show that the properties are fairly constant across a wide range of frequencies. The strain energy lost by viscoelasticity helps to attenuate travelling pressure pulses, which propagate along arteries as waves of circumferential distention of the vessel wall (Fung, 1984). Together with blood viscosity, the viscoelasticity of the artery wall prevents reflected pressure waves from resonating in the arterial system, as they would if there was perfect elasticity and inviscid blood.

Mechanical properties of arteries in non-mammalian species

The appearance of non-linear elasticity in blood vessels

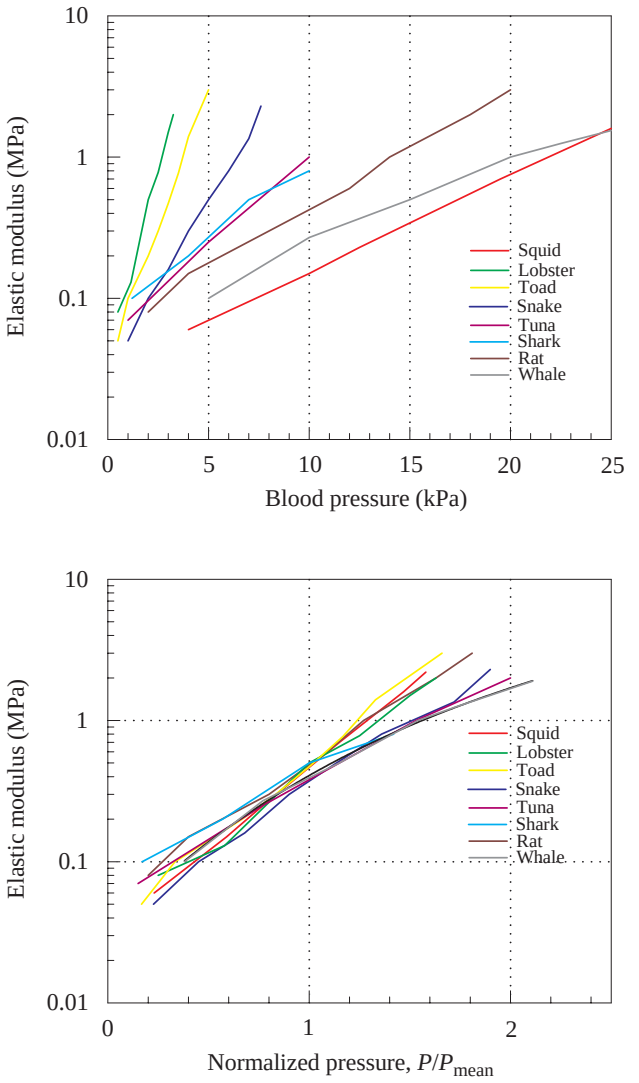

Fig. 5. (A) Plots of aortic elastic modulus as a function of inflation pressure for a variety of animal species. Note that the modulus data are plotted on logarithmic axes. Although substantial differences exist among these curves, in each case the aorta has non-linear elasticity and the modulus increases dramatically with pressure, as predicted for elastic stability. (B) The same data as in A, but plotted with pressure normalized to the mean blood pressure for each species. Data sources are as follows: cane toad (*Bufo marinus*), garter snake (*Thamnophis radix*) and rat, Gibbons and Shadwick (1989); lobster (*Homarus americanus*), Shadwick et al. (1990); fin whale (*Balaenoptera physalus*) aortic arch, Shadwick and Gosline (1994); squid (*Nototodarus sloani*), Gosline and Shadwick (1982); mako shark (*Isurus oxyrinchus*) and yellowfin tuna (*Thunnus albacares*), R. E. Shadwick, unpublished results.

throughout the animal kingdom has been documented in several recent studies on lower vertebrates and invertebrates with closed circulatory systems. In species of reptiles and amphibians (Gibbons and Shadwick, 1989), teleost and elasmobranch fishes (Satchell, 1971; R. E. Shadwick, unpublished observations), lamprey, hagfish and whelk (Davison et al., 1995), *Nautilus*, cuttlefish and squid (Gosline and Shadwick, 1982; Shadwick, 1994), octopus (Shadwick and Gosline, 1981, 1985b), crab and lobster (Shadwick et al., 1990) and horseshoe crab (Vreugdenhil and Redmond, 1987), the major arteries have non-linear viscoelastic properties that are similar to those of the mammalian aorta. That is, the initial low compliance gives way to a much-increased stiffness as inflation pressure increases over the physiological range; in all cases, the arteries are resilient and return at least 80 % of the strain energy in each cycle of deformation. These properties ensure that each vessel will act as an effective pulse-smoothing device *in vivo*.

All aortas show pressure-dependent increases in modulus, but the specific modulus–pressure relationships appear to differ substantially among the various animals (Fig. 5A; note that the change in modulus is so great across the range of pressures used that the data in Fig. 5 are plotted on a logarithmic scale). However, these differences essentially vanish if pressures are normalized to the mean blood pressure of each species (Fig. 5B). This was first pointed out in a comparative study of arteries from lower vertebrates (lizard, snake, toad), which have relatively low blood pressures compared with those of mammals (Gibbons and Shadwick, 1989). From this perspective, the aortas of these three species appeared to show the same functional behaviour at their respective *in vivo* blood pressures, as does the aorta of mammals. Thus, differences in the composition and micro-architecture of the artery wall must underlie these mechanical adjustments to specific pressure ranges. When mechanical data for the aorta of fish and invertebrate species are added to this comparison, the pattern is maintained (Fig. 5B) and the same conclusions hold. The elastic modulus of the aorta at the *in vivo* mean blood pressure for each species is approximately 0.4 MPa, be it rat, shark, squid or lobster. Since the velocity at which pressure waves propagate in an elastic tube is directly dependent on the elastic modulus, it turns out that the characteristic wave velocity in all these aortas is very similar, approximately 2–4 m s^{-1}.

Comparative artery wall structure

Histological examination of blood vessels shows that, generally, the elastic tissue is laid down in concentric layers, interspersed with collagen and circumferentially arranged smooth muscle cells. In animals as different as *Nautilus* and the rat, there is a striking similarity in the microscopic appearance of the aortic wall (Fig. 6). In the major arteries of mammals, the bulk of the vessel wall consists of the tunica media, which contains the elastic laminae. These are sandwiched between the inner tunica interna (a layer of endothelial cells and the underlying basal elastin layer) and the outer layer of predominantly collagen called the tunica adventitia. In an elegant study on segments of rabbit aorta fixed under various distending pressures ranging from 0 to 26 kPa, Wolinsky and Glagov (1964) described the three-dimensional architecture of the tunica media. This approach provided a clear picture of the architecture of the fibrous and cellular components and gave a structural basis to the model of strain-

dependent load transfer from elastin to collagen that Roach and Burton (1957) had postulated. When unpressurised, the elastin lamellae appear wavy and disorganized in longitudinal and transverse sections. With increasing pressure and distention, there is a progressive straightening of these lamellae and a decrease in the inter-lamellar distances. At 10 kPa (the low end of the physiological pressure range), the lamellae are straight and give the appearance of regular concentric cylinders with uniform thickness and radial spacing. Within the inter-lamellar spaces are smooth muscle cells and collagen fibres that become oriented in low-pitch helical paths around the circumference as the pressure is raised. In lower vertebrates and invertebrates, aortic elastic layers also appear wavy when unpressurised (Fig. 6; Gosline and Shadwick, 1982; Shadwick et al., 1990). As in mammals, these lamellae straighten with increasing pressure but, so far, this has only been documented directly in the toad. Using a series of pressure-fixed toad aortas, Gibbons and Shadwick (1991) found that elastin lamellae straightened at approximately the mean blood pressure of only 2.5–3 kPa (Fig. 6C,D), again demonstrating the functional similarity to the mammalian aorta and supporting the interpretation of Fig. 5B.

The circumferential straightening of elastin layers and the alignment of collagen fibres with distention under physiological pressures correlates with the increasing elastic modulus observed by mechanical testing and probably represents the basis for load transfer from compliant elastin to much more rigid collagen fibres. On the basis of these studies, the idea of a lamellar unit of structure, consisting of muscle–elastin–collagen, was formulated for mammals (Wolinsky and Glagov, 1964). In a refinement of this model based on scanning electron microscopy of fracture surfaces of pressure-fixed aortas, Clark and Glagov (1985) showed that the elastic tissue between concentric layers of circumferentially oriented smooth muscle cells actually consists of two layers of elastin fibres, each associated with adjacent muscle layers and containing interposed bundles of wavy collagen fibres. While there are no apparent connections between fibres of elastin and collagen, both appear to be linked to the membranes of adjacent muscle cells, from which they are synthesized. More recently, Davis (1993) showed that internal contractile filaments span the smooth muscle cells obliquely, linking to the membrane attachment sites of the adjacent elastin layers. This system of highly organised contractile-elastic units provides a mechanism for a direct line of tension transmission across the concentric elastic lamellae and suggests that the artery wall is designed to distribute uniformly the tensile stresses to which it is subjected. This conclusion is also supported by the histological evidence of Dobrin (1983), who showed that the radial elastic modulus is uniform across the wall thickness, so that the distribution of inter-lamellar strains matches the distribution of pressure-imposed stresses.

The modular nature of the artery wall structure is the key to how differently sized mammals have aortas with the same elastic properties. Larger mammals have larger arteries with proportionately more elastin layers. Wolinsky and Glagov (1967) showed this in a morphometric study of the lamellar unit in the thoracic aorta of adult mammals (from 28 g mice to 200 kg pigs). Their data revealed that diameter and wall thickness increase in nearly constant proportion and that the lamellar thickness remains constant at approximately 0.015 mm. This means that the number of lamellar units increases in direct proportion to the radius and wall thickness. Since the Laplace relationship defines circumferential wall tension per unit length as $T=PR$, and since the mean blood pressure in mammals is approximately 12 000 N m^{-1}, irrespective of body size, it appears that mean T should increase in direct proportion to R and, hence, to the number of lamellar units. Wolinsky and Glagov (1964) calculated that, while the total tension in the artery wall increased by 26-fold from mouse to pig, the tension per lamella for all aortas was similar, ranging from 1 to 3 N m^{-1}. This led to the conclusion that the elastin–muscle–collagen lamella is the basic structural and functional unit of the aorta.

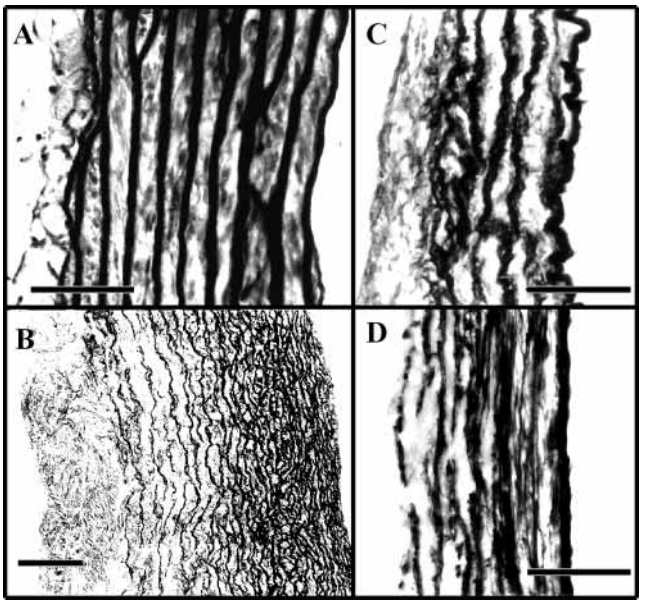

Fig. 6. Representative cross sections of aortas from rat (A), *Nautilus* (B) and toad (C) fixed while uninflated. A toad aorta fixed while inflated at 2.5 kPa is shown in D. Scale bars, 50 μm in A,C,D; 100 μm in B. (A,C,D are taken from Gibbons and Shadwick, 1991, with permission; B is taken from Shadwick, 1992, with permission).

While the idea of a lamellar unit is applicable to arteries of other animals, some distinctions are found (Table 1). For example, the wall thickness ratio h/R is lower in the aorta of reptiles and amphibians than in that of mammals, and these vessels have a larger proportion of collagen, thinner elastin lamellae and a tension per lamella of only approximately 0.4–0.5 N m^{-1} at mean blood pressure (Gibbons and Shadwick, 1989). These results suggested that the lamellar unit of the aorta is structurally and mechanically different in the lower vertebrates (and probably in invertebrates too) compared with that in mammals and that this is necessary to achieve the same elastic properties at low blood pressures in these animals that occur in mammals at higher blood pressures (Fig. 5B).

Table 1. *Structural properties of some vertebrate aortas*

					Whale	
	Toad	Lizard	Snake	Rat	Aortic arch	Thoracic aorta
Exernal radius, *R* (mm)	1.3	1.0	1.1	1.6	190	70
Thickness, *h* (mm)	0.06	0.06	0.05	0.13	20	2
h/*R*	0.05	0.06	0.04	0.08	0.10	0.03
Mean blood pressure (kPa)	3	3	4.4	12	12*	12*
Lamellar number	6	6	10	10	820	170
Tension per lamellar unit ($N\ m^{-1}$)	0.54	0.33	0.36	1.5	2.6	4.8

Dimensions are given at mean blood pressure.
*Assumed value.
Data for toad, lizard, snake and rat are taken from Gibbons and Shadwick (1989, 1991); data for whale are taken from Gosline and Shadwick (1996).

Novel arterial properties in marine mammals

Recent studies on whales have revealed unusual modifications of the arterial lamellar structure in these large mammals that correlate with specialised mechanical properties (Gosline and Shadwick, 1996). Cetaceans have an unusual expansion of the aortic arch that can be up to 3.5 times greater in diameter than the descending aorta to which it connects. Functionally, this aortic bulb increases the volume capacitance of the aorta and probably helps to maintain blood flow during diving bradycardia (Drabek, 1975; Shadwick and Gosline, 1995). In the extreme case of large cetaceans, such as the fin whale (*Balaenoptera physalus*), the aortic bulb represents virtually all the elastic compliance of the entire arterial tree. The haemodynamic consequences of this specialisation have been considered by Shadwick and Gosline (1994, 1995). Both the aortic arch and the descending aorta (i.e. thoracic and abdominal sections) have structural and mechanical properties that are very different from those of homologous vessels in other mammals. While the aortic bulb is greatly enlarged, it has essentially the same wall thickness/radius ratio (*h*/*R*) and elastic modulus at mean blood pressure as occurs in the proximal aorta of terrestrial mammals (Table 1). However, the whale aortic arch is unusual in that it can be distended to above-physiological pressures without much increase in stiffness, contrary to what typically occurs in the aorta of other mammals (but the increase in modulus is sufficient to prevent blowout). In contrast, the descending aortic segments have external diameters that are similar to those predicted by allometry, but they are relatively thin-walled and approximately 30 times stiffer than the aortic arch (Shadwick and Gosline, 1994). While some of the increase in vascular stiffness distal to the arch can be attributed to a four- to sixfold

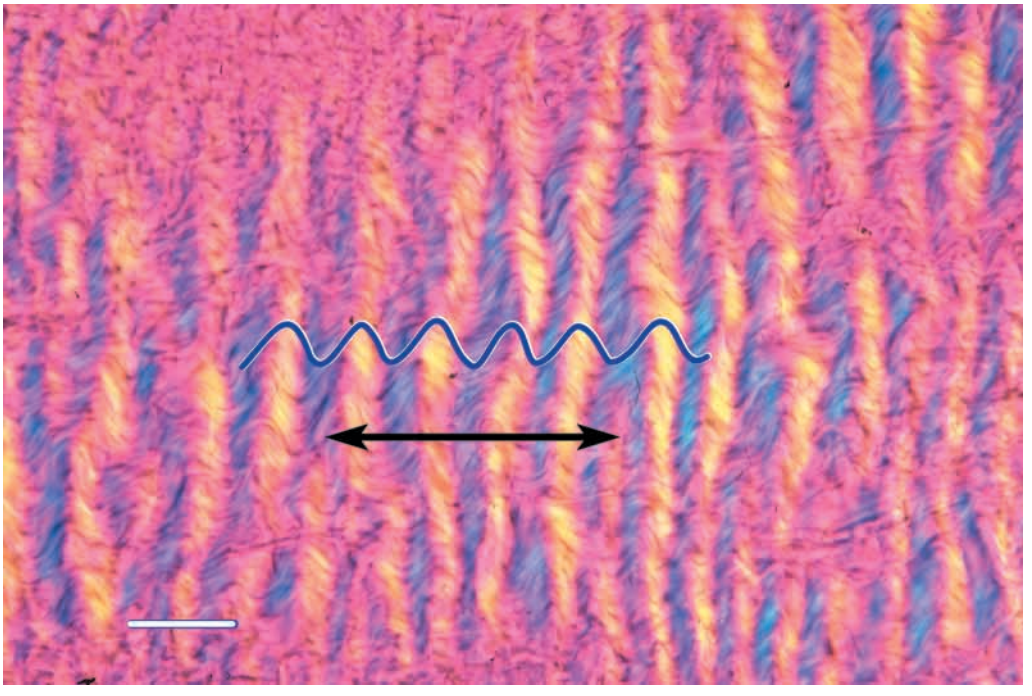

Fig. 7. A polarised light micrograph, taken using a first-order red filter, showing the organisation of collagen fibres in the fin whale thoracic aorta. This section was cut tangential to the circumferential elastic lamellae and reveals the tendon-like appearance of collagen fibres which lie between adjacent elastin layers. The wavy line and arrow indicate the crimp and the orientation of the collagen fibres, which in this example is circumferential. Scale bar, 20 μm (courtesy of J. M. Gosline).

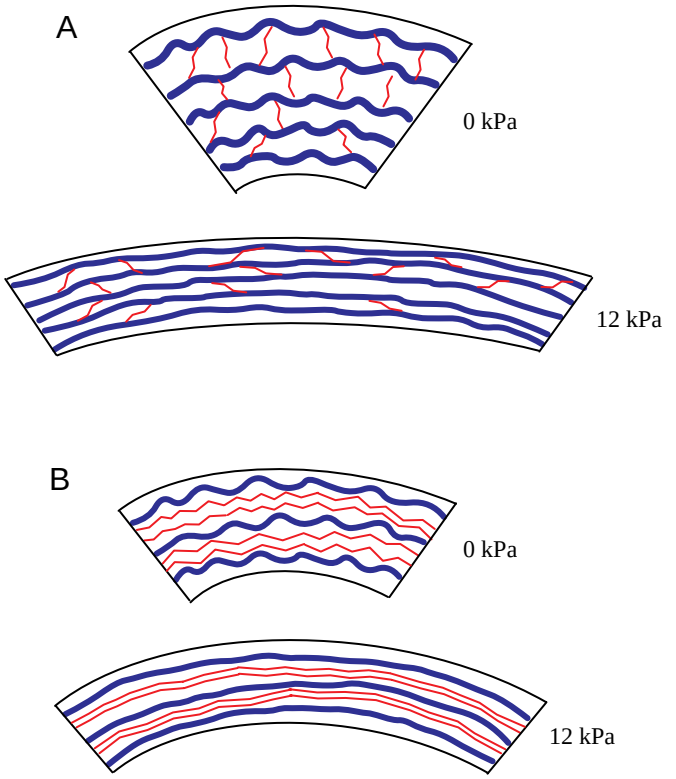

Fig. 8. The difference between the lamellar organization of the aortic arch (A) and thoracic aorta (B) of the fin whale. Each diagram represents a portion of a cross section of a vessel. For uninflated vessels (0 kPa), the drawings are based on light micrographs (from Gosline and Shadwick, 1996). In the aortic arch, the interlamellar collagen fibres (red) are predominantly radially aligned, whereas in the thoracic aorta the collagen occurs in circumferentially oriented sheets between layers of elastin (blue). With inflation to the estimated mean blood pressure of 12 kPa, the expected straightening of the elastin lamellae and the rearrangement of the collagen fibres are shown in the lower parts of the diagrams. The radial orientation of diffuse collagen fibres in the arch allows a large distention with only a small increase in stiffness. The near-circumferential arrangement of sheets of parallel collagen fibres in the thoracic aorta provides reinforcement that reduces distensibility and causes a sharp rise in modulus as the collagen is straightened.

increase in the collagen:elastin ratio, other structural differences also contribute. A quantitative analysis of these tissues suggested that, over the predicted physiological pressure range, stress in the aortic arch is resisted by elastin alone, allowing this vessel to be very distensible (Gosline and Shadwick, 1996). At the same time, the descending aorta is relatively inextensible and behaves as if the load were borne almost exclusively by collagen fibres. The key difference between these two segments of the whale aorta is the structure of their lamellar units.

In the thoracic aorta, the wall is very thin, so h/R is comparatively low (Table 1). Consequently, the number of lamellar units is approximately one-third of what would be predicted from scaling, and the tension per lamella is three times higher. To support this high load, the collagen fibres are much more uniformly oriented and in register than arterial collagen in other vessels. These fibres appear predominantly circumferential in tendon-like sheets that lie between adjacent elastin layers (Figs 7, 8). This suggests that the collagen will straighten and become the major load-bearing element after only a modest increase in circumference. Indeed, the thoracic aorta expands in diameter by only approximately 35 % between 0 and 10 kPa, and by only 2 % between 10 and 15 kPa. This novel vascular architecture transforms the whale descending aorta into a rigid conduit rather than a compliance vessel, as it is in other mammals.

The lamellar units of the whale aortic arch are quite different from those in the descending aorta. The elastin layers are thicker, the collagen fibre-lattice is more diffuse and, most importantly, has a predominantly radial orientation (Gosline and Shadwick, 1996). This allows the arch to undergo a large degree of expansion before the collagen is oriented in the direction of load (Fig. 8) and could account for both the lower modulus and the higher distensibility of the arch compared with the remainder of the aorta. Future studies on pressure-fixed vessels are necessary to explain fully the relationship between structure and function in these and other blood vessels.

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