

Healthy lifestyle, healthy arteries

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Introduction

Today we consider arteries in terms of disease only, for instance the development of arterial hypertension, peripheral or coronary artery disease. However, some millennia ago, the arteries were considered to be 'the window to the heart'. Strategically positioned between the heart and the end organs, they both nourish and cleanse the body; subject to primary and secondary disorders, they may also reflect not only the health of the vascular system but also of the heart, aptly described thus: "The heart speaks out of the vessels of every limb" (Dawson, 1942).

In this article, we show how the function of the arteries can be easily assessed, particularly the speed at which pressure waves are transmitted to and from the heart and the form or shape of those waves. We can also derive other intrinsic arterial properties, such as amplification of pressure from the heart to the periphery, which also reflect the elasticity of arteries.

Put simply, the function of these large arteries is to dampen the pulsatile intermittent contraction of the heart in systole providing a steady blood flow into organs and during diastole recoil providing flow to the heart itself, which does not get perfused during systole. Large arteries can be likened to a rubber band, losing elasticity with ageing, becoming taught or stiff and subject to disruption. No longer do they fulfil their normal buffering capacity; thus, pulse pressure rises with detrimental consequences for end organ damage in terms of stroke, heart attacks, vascular disease and renal failure.

Assessment of arterial function is easy to perform and these measurements may allow us to ascertain the impact of lifestyle on vascular function. They may also enable us to document the beneficial effect of lifestyle changes in individual patients as well as in the whole community.

Arterial stiffness and wave reflection

The arterial tree is designed very elaborately in the form of elastic conduits, such as the aorta and its major branches which act as elastic cushions to dampen pulsatile cardiac outflow, the peripheral muscular arteries, the distributing channels and small arteries and arterioles, sites of vascular resistance. The large central arteries are the most elastic with a

gradual increase in stiffness as one traverses to the smallest peripheral arterioles. This elegant design serves to convert the pulsatile flow from the heart into a steady flow through the capillaries. The heart discharges the entire stroke volume during systole. Part of the energy of cardiac contract is dissipated as capillary flow during systole but the remainder is stored in the large arteries as potential energy by elastic stretch of the arterial walls of the large arteries.

During diastole, the elastic recoil of the arterial walls converts the potential energy into capillary flow. If arteries were rigid structures, capillary flow would cease during diastole with dire consequences, particularly for the endocardium of the heart itself as it receives no blood supply during systole.

The pressure wave generated by ventricular ejection is propagated down the aorta and its branches at a measurable speed — the pulse wave velocity (PWV). The PWV increases with ageing and hypertension and for the aorta ranges between 5 and 20m/second. Aortic PWV has been shown to be an independent marker of cardiovascular risk and of greater importance than a single measurement of blood pressure. Arterial PWV also increases from the aorta to the peripheral arteries, due to a higher stiffness in peripheral vasculature, resulting in higher pulse pressure in the peripheral than the central arteries. This phenomenon is known as pulse pressure amplification. This ranges between 12 and 20mmHg in healthy arteries and is reduced in stiff arteries; it is shown to be an important prognosticator of cardiovascular events in patients with end stage renal disease and hypertension.

The other important consequence of the human arterial geometry is the phenomenon of wave reflection. The pressure wave travels faster than blood itself and when it encounters the high resistance arterioles in the periphery, it is reflected back towards the heart. In healthy elastic arteries, the vascular resistance is low and the PWV optimal; therefore, the reflected wave arrives back at the heart during diastole. However, as arteries stiffen and the PWV increases, the reflected wave arrives back at the heart during systole, increasing left ventricular after-load and decreasing coronary perfusion pressure. The early and accentuated wave reflections augment

the systolic pressure, particularly in the aorta, and thus are the major determinant of pulse pressure.

The extent of this wave reflection is expressed as the augmentation index (AIx). There is evidence that the AIx is a significant predictor of coronary artery disease and an independent prognosticator of cardiovascular mortality in patients with end stage renal disease. Both PWV, reflecting large artery stiffness, and AIx, representing both small and large artery stiffness and vascular resistance, have to be measured to get a global picture of systemic arterial stiffness.

Effect of lifestyle on arterial function

In one of our earlier studies, we were struck by our ability to separate smoking from non-smoking healthy medical students by studying the performance of arteries. We also noted that the deleterious effect of cigarette smoking in men was mitigated if they took regular exercise, although clearly their arteries still perform less efficiently than those who do not smoke and take a similar degree of exercise. However, it does raise the possibility of a 'trade off' and for the intractable smoker or the one with the metabolic syndrome cardiovascular risk may be reduced by other unrelated lifestyle measures. The vascular tree would appear to perform in unison, whether endothelial dysfunction seen with minor changes at the arteriolar level or wave reflection at arteries, or structural changes, particularly in the carotid, measured by ultrasound, such as intimal medial thickness, all are measures of vascular prognosis. Our work complements

findings elsewhere but because of its non-invasive nature and low tech approach, this window on the vascular system can identify at-risk subjects before vascular events.

Figure 1 shows the chronology from the development of the earliest detectable abnormality within the vasculature, endothelial dysfunction; a failure of the smaller vessels to dilate in response to normal stimuli including exercise and endogenous vasodilators, through arterial abnormalities, damage to organs, cardiovascular events and finally death. The speed at which these processes develop are largely determined by our genetic make-up and the number of risk factors that are operating. Early changes can be seen in teenagers with hypercholesterolaemia or in those genetically predisposed towards hypertension. Fortunately for most of us the cycle takes many decades, providing a prolonged window of opportunity to reverse the early changes. Unfortunately, possibly due to major adaptive structural changes in the vasculature and in the heart, it reaches a stage where reversibility is no longer possible and our goal becomes one of damage limitation.

In the context of our expanding knowledge of the evolution of vascular damage, the presentation of patients for the first time with myocardial infarction, stroke or heart failure can be viewed as a collective indictment of the inadequacy of primary prevention and our reluctance to employ screening techniques with the ability to detect the earlier reversible stages of this process. Here lifestyle changes may obviate or delay the need for pharmacotherapy.

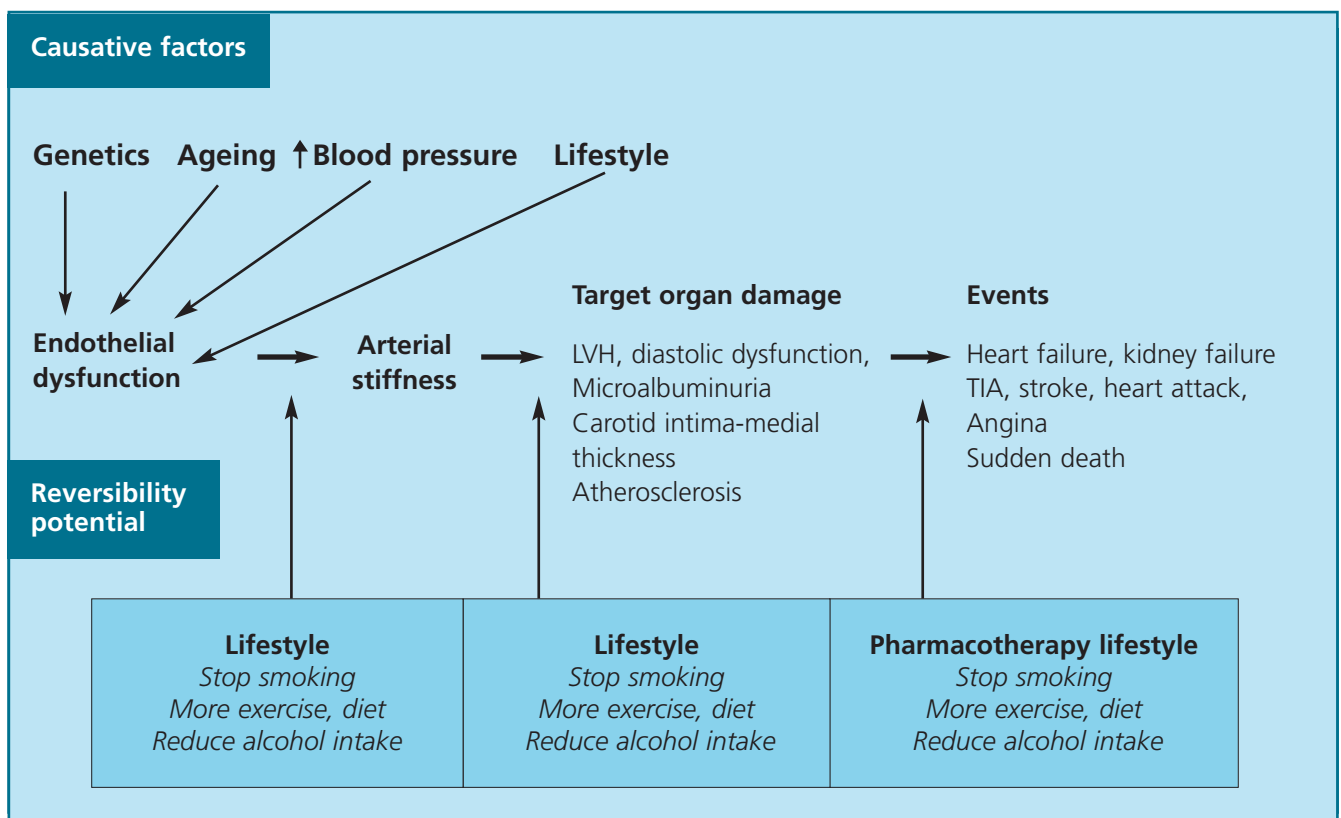


Figure 1. Evolution of vascular disease with the time-limited potential for reversibility of prevention.

Smoking

We have shown that smoking one cigarette can stiffen arteries and raise blood pressure in healthy adults. Furthermore, young healthy chronic smokers have markedly increased arterial stiffness and poor pulse pressure amplification than non-smokers even for the same blood pressure.¹ Other groups have confirmed these results and have shown the same detrimental effects with cigar smoking.

In addition, we have documented the effect of passive or second-hand smoke (see Figure 2); within 30 minutes there is a clear deterioration in the shape of the arterial pulse wave.² This change would be equivalent to being mildly hypertensive or being 15-20 years older. Preliminary data suggest that smoking cessation is associated with reduced systolic blood pressure in hypertensive patients³ but whether it will reduce arterial stiffness is not yet known. The introduction of the smoking ban precludes us from conducting further studies in this area.

Exercise

Aerobic endurance

There are cross-sectional data which show that older men who perform regular endurance exercise have lower PWV and AIx than their sedentary peers,⁴ age-related increases in arterial stiffness are absent in physically active post-menopausal women and that aerobic fitness was strongly associated with arterial stiffness.⁵ For sedentary middle-aged people, three months of aerobic exercise, including walking regularly, can reduce arterial stiffness particularly in the large elastic arteries; women may benefit to a greater extent than men.⁶

Regular exercise therefore appears to be an effective lifestyle measure that will reduce the rate of arterial stiffening even in the absence of any weight loss. However, similar exercise intervention programmes produce a rather blunted beneficial effect in those with elevated systolic blood pressure⁷ and for patients who have established isolated systolic hypertension.⁸ It may be that chronic degenerative changes leading to inelasticity of vessels are beyond repair by such lifestyle measures. The general applicability of exercise is emphasised by a study on some 684 female twins which showed that physical activity

reduced their genetic susceptibility to arterial stiffness through participation in regular leisure time physical activity.⁹

Strength training

In contrast to aerobic training, strength training including bench press, weight-assisted parallel bar, dumb-bell etc does not produce such benefits and, in most studies, increased arterial stiffness.¹⁰ This is possibly due to the marked increases in blood pressure associated with such exercises.

Weight reduction

Intensive weight reduction (approximately 10% of body weight) led to significant reduction in blood pressure and aortic stiffness both in hypertensive¹¹ and non-hypertensive subjects.¹² Sub-group analysis does show that in some individuals there is a significant reduction in stiffness without any changes in blood pressure; however, by and large, most of the benefit may be due to a reduction in blood pressure.

Dietary modifications

Salt

The Yellow Emperor (450BC) noted that salt hardens the pulse. In general, communities that have a lower salt intake tend to have lower aortic PWV¹³ and people who show sodium sensitivity with regard to their blood pressure have increased arterial stiffness compared to sodium-resistant subjects.¹⁴ Again where salt is restricted, there is usually a reduction in blood pressure and in arterial stiffness,¹⁵ which may, in part, be independent of the blood pressure changes. Another study has shown that these effects can be seen within two weeks.¹⁶

Alcohol

We found that, acutely, red wine containing alcohol reduces blood pressure and arterial stiffness in comparison with de-alcoholised wine in young healthy adults.¹⁷ These beneficial effects persisted even after taking into account the reduction in blood pressure. In contrast, we noted that hypertensive men who have a high alcohol intake (>21 units per week) have a greater degree of arterial stiffness than age-matched

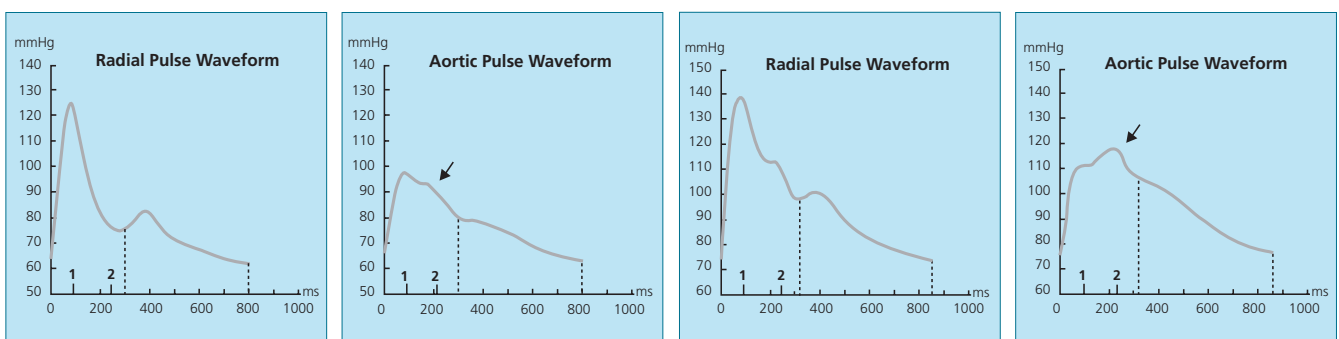


Figure 2. Radial and aortic pressure waveforms at baseline (two panels on left) and at 60 minutes following exposure to passive smoking (two panels on right). There is an increase in amplitude of the 'reflected wave' in the aortic pressure wave form.

hypertensives with lower alcohol intake.¹⁷ In addition, we and other groups have shown that coffee (in comparison to decaffeinated drinks) also stiffens arteries.¹⁸ However, interventional studies where these factors are removed from the diet have not been conducted.

Food

Cross-sectional studies suggest that those who have a high fish intake tend to have lower arterial stiffness.¹⁹ A high calorie, high fat meal can acutely increase arterial stiffness and the addition of antioxidant vitamins may have a beneficial effect in reducing arterial stiffness in short-term studies, although long-term studies are inconclusive.

Chocolate

One hundred grams of flavonoid-rich dark chocolate acutely decreases wave reflection but not aortic stiffness and improves endothelial dysfunction.²⁰ There is now evidence that such dark chocolate, but not white chocolate, may also reduce blood pressure when taken over a longer term.²¹

Conclusion

It is clear that arterial function is a sensitive barometer of vascular health. It may be used to identify asymptomatic 'healthy' but nonetheless at-risk individuals and also to demonstrate the positive beneficial effect of lifestyle changes. We are also hopeful that it may help inform people as they head into the non-reversible area of arterial stiffness that lifestyle adjustments may be their last opportunity for non-pharmacological intervention.

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