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In this article...

- How age affects the normal functioning of the cardiovascular system
- Age-related changes to vasculature, chemicals, heart muscles and blood pressure
- Conditions experienced by older people as a result of declining heart function

Anatomy and physiology of ageing 1: the cardiovascular system

Key points

1 An efficient cardiovascular system is essential for health and longevity, but age brings about changes that reduce its efficiency

2 Blood vessels, particularly arteries, lose their elasticity with age, and the arterial walls become stiffer and thicker

3 Age-related changes in the chemical signals produced by the body contribute to restricting blood flow

4 As the heart ages, it undergoes a redistribution of its muscle mass, which negatively affects its function

5 Older people should be encouraged to take regular exercise, which will support their cardiovascular function well into old age

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Abstract The cardiovascular system is the body's main transport system, and its efficiency is essential for health and longevity. As it ages, it becomes less efficient, which has a negative impact on all other organ systems. This article explores the normal age-related changes occurring in the cardiovascular system. This is the first of an updated article series on how age affects the main organ systems.

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The average lifespan of people in the UK is rising – mainly due to advances in healthcare – and many 60-year-olds can expect a further 25 years of healthy life. However, knowledge of the ageing process remains limited. This is the first article in an updated and expanded series examining the anatomy and physiology of ageing.

The ageing process is largely determined by genetic factors but it is also heavily influenced by environmental factors such as diet, exercise and exposure to micro-organisms, pollutants and ionising radiation. That is why people of the same age vary both in physical appearance and physiology. Gender also plays a part; in most developed countries, women typically outlive men by 7-10 years.

The cardiovascular system

The cardiovascular system is the body's main transport system. Its most important role is to deliver oxygenated blood, nutrients and chemical signals, such as hormones, to the organs and tissues. It also transports carbon dioxide to the lungs and waste products, such as urea and uric acid, to the kidneys for elimination. It plays a

major role in thermoregulation – distributing and dissipating heat throughout the body (Marieb and Hoehn, 2015). An efficient cardiovascular system is essential for health and longevity, but its efficiency reduces with age, which has a negative impact on all other organ systems.

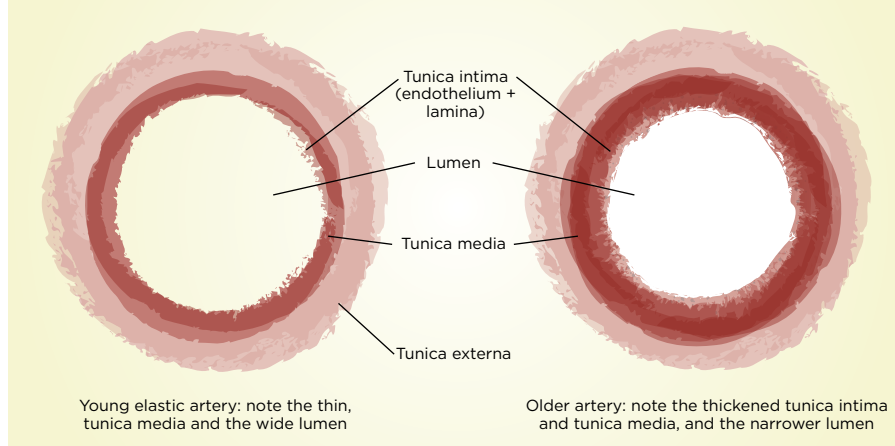
Vascular changes

We are born with arteries that are elastic, flexible and compliant, allowing optimal cardiac function and blood flow. During ventricular systole (contraction), blood is ejected into the pulmonary and systemic circuits and the larger elastic arteries stretch, reducing the resistance to blood flow. As the body ages, blood vessels, particularly arteries, lose their elasticity and the arterial walls become stiffer and thicker.

The tunica intima is the innermost layer of a blood vessel, and consists of two main regions:

- Endothelium – a single layer of cells;
- Lamina – a thin layer of connective tissue that anchors the endothelium to the tunica media (muscle layer) above. This mainly comprises elastin (elastic fibres) and collagen, and undergoes significant changes with age.

Fig 1. Age-induced arterial thickening



The larger arteries have a high elastin content as they need to stretch in harmony with the powerful ventricular contractions of the heart to cushion the force of the pulse wave, smoothing out the flow of blood entering the smaller arteries. These smaller arteries have much less elasticity and a greater proportion of collagen fibres in their vessel walls (Steppan et al, 2011).

The tunica media, which lies underneath the tunica intima, is made up of layers of smooth muscle cells. It is controlled by the vasomotor centre within the medulla oblongata (the lower part of the brain stem). The vasomotor centre plays an important role in regulating blood pressure by controlling vasodilation and vasoconstriction (Marieb and Hoehn, 2015).

With age comes a gradual thickening of the tunica intima and tunica media of large and medium-sized arteries (Fig 1). This is associated with an increase in the number and density of collagen fibres in the vessel walls (Ferrari et al, 2003). Collagen fibres also undergo a process of cross-linking, which makes them less compliant. With age and repeated stretching, elastin – which partly gives arterial walls their elasticity – undergoes fracture and fatigue (Greenwald, 2007). Ageing blood vessels may also display varying degrees of calcification. These events cumulatively result in a gradual loss of elasticity and stiffening of the arteries, which is often reflected by increased blood pressure (Bolton and Rajkumar, 2011).

Endothelium

The endothelium, the most delicate part of a blood vessel, is in direct contact with the circulating blood (Marieb and Hoehn, 2015). It is composed of a single layer of squamous epithelial cells that, in children and young adults, are regular and smooth, minimising resistance to blood flow.

As it ages, the endothelium develops irregularly shaped cells and is often thickened due to the presence of smooth muscle fibres that have migrated from the tunica media. This thickening contributes to a reduction in arterial elasticity and compliance, and reduces the lumen size (Fig 1), further increasing resistance to blood flow.

Atherosclerosis

Atherosclerosis, the most common form of blood vessel disease, is triggered by injury to the endothelium, which can have a wide variety of causes including:

- Toxins (for example, cigarette smoke);
- Hypertension;
- Hyperglycaemia.

The mechanism of atherosclerotic occlusion (fatty deposits) following endothelial damage involves monocytes (white blood cells) attaching to the damaged or irritated endothelium and crossing into the tunica media. These monocytes gradually grow and mature into much bigger cells called macrophages (Galkina and Ley, 2009). The macrophages absorb fat (including cholesterol) from the blood and ‘puff up’ to form foam cells. These foam cells form ‘fatty plaque’ that occludes blood vessels (Libby et al, 2011).

Atherosclerosis of the coronary arteries may result in coronary artery disease. The fatty plaque often ruptures, resulting in clot formation and heart attack (myocardial infarction). Similarly, atherosclerosis of the carotid or cerebral arteries dramatically increases the risk of stroke.

Chemical changes

Reduced nitric oxide production

Endothelial cells release various chemical signals that help to regulate blood flow by controlling the internal diameter of blood vessels. One of the most important of these

chemicals is nitric oxide, which is produced by endothelial cells from the amino acid L-arginine. This diffuses into the smooth muscle layer of the blood vessels, where it acts as a powerful vasodilator, expanding the vessels and ensuring good blood flow.

Damage to the endothelium – be it age-related or due to other causes – results in reduced nitric oxide generation and therefore blood flow (Greenwald, 2007; Bode-Böger et al, 2003). This contributes to, and may exacerbate, age-related blood vessel pathologies including peripheral vascular disease and angina pectoris.

Increased pro-inflammatory chemicals

The concentration of pro-inflammatory chemical mediators circulating in the blood increases with age. Many are implicated in blood vessel pathology, including atherosclerotic occlusion and blood vessel wall calcification (Harvey et al, 2015).

Delayed angiogenesis

After an injury or infection, new blood vessels can be rapidly produced in a process known as angiogenesis. This is orchestrated by a variety of chemical signals and growth factors. Angiogenesis slows with age, and is often significantly delayed (Sadoun and Reed, 2003), which may help to explain why wound healing generally occurs more slowly in older people.

Cardiac changes

To overcome reduced elasticity and increased resistance to blood flow of aged and occluded arteries, the heart’s ventricles have to pump with greater force. The myocardium (muscular layer of the heart) responds by becoming hypertrophied.

Earlier ultrasound studies suggested the thickness of the left ventricle increases by around 30% between the ages of 20 and 80 years, with an associated gradual increase in cardiac weight (Pearson et al, 1991). However, the validity of some of these studies has recently been questioned.

Examination of hearts removed during autopsy revealed little evidence of age-related ventricular thickening in women, while in men, there was often an apparent reduction in muscle mass. The hypertrophy observed on ultrasound scans seems to result primarily from a thickening of the intraventricular septum, rather than the left ventricle; a remodelling and redistribution of cardiac muscle tissue seems to occur with age (Strait and Lakatta, 2012).

The number of cardiac myocytes (muscle cells) in the myocardium decreases progressively through apoptosis

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(programmed cell death); the remaining myocytes undergo morphological changes, often becoming enlarged (cellular hypertrophy) or irregular in shape. The amount of collagen deposited in the myocardium also increases with age. Together with the redistribution of cardiac muscle mass, this typically results in an observable change in the shape of the heart from the classic elliptical shape to a slightly more spherical appearance (Strait and Lakatta, 2012; Ferrari et al, 2003).

Wear and tear to the heart's internal structure (which occurs more rapidly in patients with hypertension) can also lead to calcification and fibrous scar tissue on the heart valves. This commonly results in stenosis (a narrowing in the aperture of the valve), which restricts blood flow and reduces the heart's efficiency. Stenosed valves typically produce a turbulent blood flow, which may be detected through a stethoscope as a heart murmur (Bolton and Rajkumar, 2011).

Cardiac function

The changes to both the vasculature and the heart itself lead to a general reduction in the efficiency of the heart. The resting heart rate when a person is lying flat remains fairly constant as we age but, in a sitting position, this generally decreases (Bolton and Rajkumar, 2011).

One of the most striking age-related changes in cardiac function is a linear decrease in the maximal heart rate achievable during exercise. In young healthy children, a maximal heart rate of around 220 beats per minute (bpm) is normal following vigorous exercise. With age, this falls, roughly in line with the formula '220 minus age in years' so, by the age of 60, it is around 160bpm. It is thought this reduction is primarily due to changes in the heart's conductive system. The filling of the ventricles also slows with age, as the

increased collagen content in the ventricle walls leads to slower ventricular relaxation (Strait and Lakatta, 2012).

In addition to the age-related decline in cardiac function, the heart's ability to repair itself following injury or infection also declines (Strait and Lakatta, 2012).

Cardiac conductive system

By the age of 50, the sinoatrial node (the heart's natural pacemaker) has lost 50-75% of its cells. While the number of cells in the atrioventricular node remains relatively constant, there is fibrosis and cellular death in the atrioventricular bundle, also called the bundle of His (heart muscle cells specialised in electrical conduction).

These changes may reduce the efficiency of cardiac conduction and contribute to the decline in maximal heart rate (Ferrari et al, 2003). The reduction in pacemaker cells makes atrial and ventricular arrhythmias much more likely; an example of this is atrial fibrillation in older people.

Blood pressure

Systolic blood pressure gradually increases with age – the average in men is around 126mmHg at 25 years and 140mmHg at 60. This is thought to reflect the decrease in elasticity and lumen diameter within the arterial tree, and the associated structural changes to the heart. In addition, small arteries and arterioles become less responsive to vasodilators such as nitric oxide, further increasing peripheral resistance. Recent research has also demonstrated a general age-related up-regulation of the renin-angiotensin mechanism. This results in increased levels of the powerful vasoconstrictor angiotensin II, which elevates blood pressure (Harvey et al, 2015).

In the absence of any pathology, diastolic pressure (when the ventricles are relaxed) changes very little with age and may even be reduced (Steppan et al, 2011).

Reduced baroreceptor response

After a change in posture, such as moving from a sitting to a standing position, blood drains into the lower extremities and blood pressure falls. This hypotension is immediately detected by the baroreceptors (blood pressure sensors) in the aortic arch and carotid sinus, causing the cardiac in the medulla oblongata to increase the heart rate. The vasomotor centre, also in the medulla oblongata, initiates vasoconstriction to restore normal blood pressure, ensuring adequate blood flow to the brain and preventing postural hypotension and fainting (Marieb and Hoehn, 2015).

In older people, baroreceptor reflexes are blunted, which often results in an increased variability of blood pressure throughout the day and may reduce the ability to maintain blood pressure after blood loss (Monahan, 2007). It is thought that age-related thickening of the arterial walls may interfere with the ability of baroreceptors to accurately measure the degree of stretch (blood pressure) within the vessel. This can increase the risk of postural hypotension, increasing the risk of falls.

Conclusion

Ageing is often associated with a general reduction in activity and fitness but exercise can be beneficial at any age. It is a good idea to encourage older people to remain active and take regular exercise, as this will support their cardiovascular function well into their old age (Montague et al, 2005). **NT**

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