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Short Communication

Cardiovascular Complications in Elderly Patients

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DESCRIPTION

Age-related changes in the cardiovascular system are common, with aging being an independent risk factor for cardiovascular disease. However, many of the other changes common with aging are caused or exacerbated by modifiable factors. The effects of aging are manifold and can be identified at the molecular, cellular, tissue, organ, and systemic levels that contribute to impaired cardiovascular system function [1].

There is progressive degeneration of cardiac structures with loss of elasticity, fibrous valvular changes and amyloid infiltration. Age-related structural features have the most impact on the contractility of the left ventricular wall of the heart. The heart's pumping ability decreases with age due to various changes that affect the structure and function of the heart muscle [2].

Evidence suggests that between the second and seventh decades, an increase in left ventricular posterior wall thickness of about 25% occurs. This increase in myocardial mass was mainly due to an increase in the average size of cardiomyocytes, while a decrease in the number of cardiomyocytes [3]. Decreased left ventricular dilatation leads to increased workload on the atria, resulting in atrial hypertrophy.

Feature changes include;

Cardiac output at rest is not affected by age. Maximum cardiac output and aerobic capacity decrease with age.

Systolic ejection volume is less affected by age; at rest in healthy individuals may even increase slightly.

Blood pressure is a measure of the efficiency of the heart. In general, most older adults have moderately elevated blood pressure.

Cardiac conduction system

Cardiac conduction is affected by a decrease in the number of pacemakers in the sinoatrial node with age. From age 60, the number of pacemaker cells in the sinoatrial node decreases markedly, and by the age of 75, less than 10% of the cells are found in young adults. With age, there is an increase in elastic tissue and collagen in all parts of the conduction system. Fat accumulates around the sinoatrial node, sometimes creating partial or complete detachment of the node from the atrial musculature. The conduction time of the bundle of His to the ventricles did not change [2,4].

Valves

Age-related increases in valve circumference have been reported in all four valve types (aortic semilunar, semilunar, mitral, and tricuspid), with the most significant changes occurring. in the aortic valve (the valve between the left ventricle and the aorta). Calcium deposition usually occurs on one or more aortic leaflets. These changes usually do not cause significant dysfunction, although in some older adults, severe aortic stenosis and mitral insufficiency are associated with degenerative changes with age. Clinical murmurs are detected more frequently [3].

Myocardial subcellular changes

The nucleus, which contains DNA, grows and can induce invasion of its membrane. Mitochondria show changes in size, shape, crystal pattern and matrix density reducing their functional surface. The cytoplasm is marked by fat infiltration or degeneration, vacuole formation, and gradual accumulation of pigments such as lipofuscin. Combined age-related changes in the subcellular compartments of cells lead to decreased cellular activities such as impaired homeostasis, protein synthesis, and degradation rates.

Changes in the blood vessels

Decreased elasticity of blood vessels due to aging can lead to chronic increase or prolongation of blood vessel diameter and stiffness of blood vessel walls, impairing blood vessel function. Factors that contribute to increased wall thickness and stiffness with aging include increased collagen, decreased elastin, and calcification.

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The aorta becomes thicker, stiffer, and less flexible. This is probably related to connective tissue changes in the blood vessel walls. This increases blood pressure and forces the heart to work harder, which can lead to an enlarged heart muscle.

Arteries thicken and harden. In general, older adults have moderately elevated blood pressure. Receptors called baroreceptors monitor blood pressure and make changes to help maintain a relatively constant blood pressure when a person changes position or performs other activities. Baroreceptors become less sensitive with age, which may explain why many older adults develop orthostatic hypotension.

Thickening of the basement membrane of capillary walls means slower delivery of oxygen and nutrients in young adults; although there is evidence that this effect can be altered with moderate exercise high.

Vein walls can thicken with age due to increased connective tissue and calcium deposition and the valves become stiff and inefficient. Varicose veins may develop.

Heart rate

The decrease in maximal heart rate is thought to be due to changes in the autonomic nervous system, as well as an age-related decrease in the number of cells in the sinoatrial node.

Consequences of a decrease in maximum heart rate

The smallest possible aerobic workload-i.e. reducing the amount of stress the heart can tolerate over a period of time

Slower aerobic performance-e.g. 90-year-olds who complete the New York City Marathon usually do so in 7-8 hours

Reducing vasoconstriction of the vessel wall. This contributes to a slow heart rate, a decrease in VO2 maximal and leads to a reduction in possible aerobic workload [5].

Blood

The blood itself changes slightly with age;

Normal aging reduces the total amount of water in the body. As a result, there is less fluid in the blood, so the blood volume decreases.

Red blood cells are produced more slowly in response to stress or illness. This produces a slower response to blood loss and anemia.

Most white blood cells remain at the same level, although neutrophils decrease in number and in their ability to fight bacteria. This reduces the ability to fight infection.

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