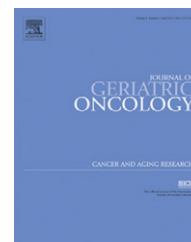


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Physiology of Aging

The changes in cardiac physiology with aging and the implications for the treating oncologist



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ABSTRACT

The link between cancer, cardiovascular disease, and aging is well documented. In this review, we highlight the physiologic and pathologic changes associated with the cardiovascular aging process, the role they play when interfaced with various cancer therapies and the implications for the treating oncologist.

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1. Introduction

The elderly have an 11-fold higher incidence of cancer and around 70% of cancer-related deaths occur in this vulnerable cohort [1,2]. A leading cause of death in the elderly is cardiovascular disease [3]. The proportion of elderly in the United States is rapidly increasing and is expected to exceed 20% by 2030, highlighting the growing burden of disease [1,2]. The management of cardiac-oncologic complications poses a clinical challenge due to the lack of clinical trial data and hence evidence-based guidelines.

1.1. The Concept of Aging

Aging is a physiologically complex and continuous process, which is largely irreversible. Age-related physiologic changes, the superimposed lifelong injury and reparative processes and the increased prevalence of cardiovascular and noncardiovascular comorbidities should be all viewed as a continuum in the aging process. Age-related changes in the cardiovascular system include decreased cardiac reserve, increased vascular stiffness and abnormal left ventricular diastolic function [4–6]. Age-associated changes in the cardiovascular system result in reduced aerobic exercise capacity and diminished ability to tolerate the physiological stressors associated with surgical intervention or critical illness. There is also a gradual decline in the function of other organ systems, resulting in changes in the metabolism of medications, reduced muscle mass and increased susceptibility to infections.

Additionally, there is age-related increase in prevalence of cardiovascular conditions including hypertension, left ventricular hypertrophy, left atrial enlargement, abnormal left ventricular ejection fraction (LVEF), atrial fibrillation (AF), heart failure with preserved ejection fraction, coronary artery disease, ischemic and thromboembolic stroke, peripheral and carotid arterial disease, aortic stenosis and regurgitation, mitral regurgitation, mitral annular calcification, hypertrophic cardiomyopathy, and pacemaker rhythm [7–9] (Table 1).

2. Structural Changes and Functional Mechanisms Associated with Cardiovascular Aging

2.1. Ventricular Structural and Functional Changes

Anatomical changes to the heart begin at a cellular level. With aging, there is a reduction in cardiac myocytes caused by increased myocyte apoptosis and necrosis. To compensate, the remaining myocytes increase in size [10–13]. Increased collagen, cross-linking of collagen and decreased elastin also contribute to the increase in myocardial thickness.

Left ventricular (LV) wall thickness increases despite the reduced myocyte concentration because of increased collagen matrix deposition and cross-linking as well as secondary interstitial fibrosis. In normal aging, there is no change in overall cardiac mass, LV cavity size nor LVEF. However, decreased LV compliance leads to significant changes in diastolic function with a 50% reduction in the early diastolic

Table 1 – Age-associated changes mechanisms and clinical consequences associated with normal aging.

Age-associated changes	Possible mechanisms	Clinical consequences
LV remodeling and increased LV thickness	Increased myocyte size Reduced myocyte number Increased collagen Increased fibrosis	Impaired diastolic filling Diastolic dysfunction Increased LV filling pressures
Changes in conduction	Reduced number pacemaker cells Increased SA node fat and collagen Increased calcification of AVN, LBB, RBB	Increased AV block Increased atrial arrhythmias
Increased arterial stiffness and wall thickness	Increased collagen Reduced elastin Endothelial dysfunction	Accelerated atherosclerosis Hypertension LV hypertrophy
Reduced CV reserve, oxygen consumption and max heart rate	Decreased β -adrenergic response Decreased myocardial contractility	Decreased exercise capacity Decreased threshold for heart failure

Abbreviations: AV, atrioventricular; AVN, atrioventricular node; CV, cardiovascular; SA, sinoatrial; LBB, left bundle branch; RBB, right bundle branch.

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