The Impact of Aging Physiology in Critical Care

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KEYWORDS

• Aging • Physiology • Critical care • Geriatric

KEY POINTS

- Managing the aging critically ill patient is highly complex and necessitates a comprehensive understanding of the normal physiologic changes that occur with aging.
- Age alone should not be considered a primary factor in the prognosis of critically ill patients.
- The practitioner must take comorbidities and functional disabilities into account, along with quality of life and the likelihood of recovery when determining care modalities in the geriatric population.
- All factors regarding the aging patient's plan of care, prognosis, and predicted quality of life should be openly and honestly discussed with patients and/or their families to ensure all decisions regarding care are fully informed.
- Future research surrounding the care of the older critically ill patient should include a comprehensive study of the impact of aging on diagnosis and treatment modalities that impact the underlying mechanisms of aging.
- Additional studies focusing specifically on the effects of aging and the critically ill patient are needed to add to this body of knowledge.
- In particular, the incorporation of evidence-based practice and nurse-driven protocols may have a positive impact on the care and outcome of these patients.

INTRODUCTION

The US population is aging at a rate faster than ever before.[1] The elderly represent a significant proportion of patients seeking care, who present more acute, get admitted more frequently, and account for as much as 50% of all intensive care unit admissions, and this number is only projected to grow.[1–4] These patients are at greater risk for complications during their admission, extended lengths of stay, and becoming chronically ill following their hospitalization.[1]

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The words of Justice Potter Stewart[5] "I don't know what it is, but I know it when I see it," can easily be applied to the definition of old. Scholars vary in their opinion of what defines old, but generally it is agreed that 65 and older is considered to be old age. Aging can be further subcategorized as young old (65–74), middle old (75–84) and old old (85+).[4] Regardless of how one categorizes age, it is recognized that significant changes in physiology related to the aging process are present in varying degrees by the age of 65.[1–4]

Aging does not accelerate over the life span; however, age-related changes have a greater accumulation in an older patient versus a middle-aged patient.[6] Progressive deterioration, which begins as subtle changes early on, continues throughout the aging process. The rate of change varies between individuals and among organ systems.[1] Factors contributing to the extent of aging physiology in elderly patients include genetics, lifestyle, and environment.[2] The impact of aging physiology in critical care is further compounded by the presence of comorbidities, which are more prevalent in the aging population.[3,7] Therefore, chronologic and biologic ages do not parallel one another, making the aging process patient specific, and the care of these patients of necessity individualized.[1,8]

An aging patient will not be able to mount the same hemodynamic and metabolic response to the stress of a critical illness as a younger patient.[7] This lack of resilience may lead to atypical presentation of severe illness, and may lead to delays in diagnosis and treatment.[9] For instance, an aging patient presenting with neurologic compromise, such as confusion, dizziness, and decreased level of consciousness, may not necessarily indicate a neurologic issue, but instead may mask infection, electrolyte imbalances, cardiac dysfunction, or drug toxicity, causing a delay in proper diagnosis.[9]

AGING PHYSIOLOGY

Cellular

Although the effects of aging may be visible at a macro level while observing a patient in the critical care unit, the true impact of what is occurring needs to be understood at the micro level. Changes at the cellular level impact all physiologic processes. Given that the effects of aging are mostly observed superficially, they begin and are at play on the cellular level well before being visualized. Before the first wrinkle is noted on a forehead or at the corner of the eyes, free radicals are bombarding cells from both within and outside the cell membrane, disrupting their ability to carry on with normal activity, communicate with other cells, and propagate new cells to take their place.[10,11]

Aging cells display a greater amount of damage compared with their younger counterparts.[10] An increase in cellular superoxide could be a significant factor in the ability of a cell to function.[2,10] In addition, certain enzymes have been identified to have protective properties for the mitochondrial wall of aging cells, and can slow the effects of aging in these cells.[10] Conversely, the lack of these enzymes in laboratory mice showed substantial acceleration in age and loss of skeletal muscle.[10] Wu and colleagues[11] provided evidence regarding mechanotransduction in the aging cell. The process of cellular mechanotransduction is defined as, "the ability of the cell to sense, process, and respond to mechanical stimuli and is an important regulator of physiologic function that has been found to play a role in regulating gene expression, protein synthesis, cell differentiation, tissue growth, and most recently, the pathophysiology of disease."[11(p1)] It is believed that with age the ability of the cells to correctly mechanotransduce information becomes damaged, or limited.[10]