INTRODUCTION

Aging is a progressive process defined as maintenance of life with diminishing capability for adjustment. Senescence results in a progressive decline in cellular function, resulting in a loss of organ performance. Cells lose their capacity to respond to injury and eventually die. Senescence is associated with impaired adaptive and homeostatic mechanisms, resulting in an increased susceptibility to the effects of stress. Function may seem to be unchanged, yet physiologic reserve diminishes. Any disruption of homeostasis that is well tolerated by younger adults might precipitate functional decline in the elderly population ***(Banks and Lewis, 2013).***

Trauma is currently the fifth leading cause of death and it is calculated that one-third of the health care resources are being expended on patients older than 65 years of age ***(Abdelfatah et al., 2014).***

Trauma is defined in the Polytrauma Rehabilitation Centers Directive dated June 8, 2005 as "injury to the brain in addition to other body parts or systems resulting in physical, cognitive or psychosocial impairments and functional disability".

The geriatric traumatized patients are more likely to present in shock than younger patients matched for Trauma. Both diuretics use and reduced fluid intake predispose older

patients to chronic volume depletion, which is exacerbated by hemorrhage, immobility, and starvation in preparation for surgery ***(Silverstein, 2008).***

Early assessment and identification of vulnerable patients is an important determinant of outcomes in trauma patients. Mechanism of injury, Injury Severity Score, vital signs on presentation, comorbidities, and medication history are known to be associated with the development of in-hospital complications, longer hospital length of stay, and adverse discharge disposition in trauma patients ***(Hsia et al., 2011; Boyd et al., 2005; Foreman et al., 2007).***

Morbidity and mortality rates are higher in elderly patients undergoing emergency trauma surgery largely because of their diminished physiological reserve and their greater incidence and severity of concomitant disease compared with younger adults. The high prevalence of polypharmacy involved in the treatment of chronic disease increases the risk of adverse drug reactions. Additionally some medications used to manage preexisting disease can affect the injured elderly response to perioperative resuscitation common examples include insulin, oral hypoglycemic, chronic corticosteroid use, antihypertensive including both beta blockers, vasodilators and antiarrhytmic ***(William et al., 2007).***

Despite these considerations no patients are too old for anesthesia even though some surgical procedures will maximally stress the elderly and bring them to the edge of their

physiological reserve necessitating postoperative mechanical ventilation and critical care ***(Boyé et al., 2014).***

If an emergency surgery is going to be performed, the anesthetist will be presented with a number of challenging problems including an uncertain diagnosis, concomitant poorly controlled medical conditions, a full stomach, dehydration, hemorrhage, pain and hypothermia ***(Lermitte and Hudsmith, 2004).***

AIM OF THE WORK

The aim of this essay is to review the physiology of aging and how it affects the elderly patients and how to manage an elderly traumatized patient during anesthesia.

PHYSIOLOGY OF AGING

(I) Concept of Aging

Aging is a progressive physiological process characterized by "declining end organ reserve, decreased functional capacity, increasing imbalance of homeostatic mechanisms and an increasing incidence of pathologic processes" ***(Brown and Sieber, 2014).***

Several theories on the nature and control of aging have contributed most significantly to this debate. Aging theories are very different, each of them touches a particular aspect of the aging process.

The Programmed Theory

Considers aging as a genetic program that has evolved to specifically direct senescence and death, thereby benefiting future generations***(Longo et al., 2005).***

However, while the undisputed role of genes in regulating aging does imply genetic, and therefore, program like features, there is currently no evidence of any gene or process that evolved specifically to stimulate aging or eliminate older individuals, and no mutants in any organism have been found in which such genes/processes are disrupted aborting the aging program ***(Longo et al., 2005).***

The Evolutionary Theory

Its key concepts of mutation accumulation (MA) and antagonistic pleiotropy (AP) The theory posits that certain alleles could be selected and mutations could accumulate in the genomes over evolutionary timescales, if these alleles and mutations show beneficial or neutral effects on fitness in early life, but are detrimental in later life when selection is inefficient to remove them (***Gladyshev and Vadim, 2016).***

The Free Radical Theory

It offers an attractive mechanistic cause of aging, where in reactive oxygen species generated as a consequence of metabolism randomly damage cellular components, with this damage gradually accumulating resulting in senescence ***(Gladyshev and Vadim, 2016).***

Throughout adulthood, increasing levels of oxygen-derived free radicals, or reactive oxygen species (ROS), create oxidative stress within the mitochondria and disrupt the structural and enzymatic machinery of oxidative phosphorylation ***(Gladyshev and Vadim, 2016).***

The Disposable Soma Theory:

Further advanced the damage based aging by proposing the idea that organisms have limited resources that must be distributed between maintenance (e.g., processes that remove damage) and reproduction.

The inability to allocate all resources (energy, building blocks) to maintenance (because organisms must invest into reproduction or they become extinct) makes protection less than 100% efficient, leading to damage accumulation ***(Lemaître et al., 2014).***

The Hyperfunction Theory:

It proposes that continued development and overactivity of genes in the reproductive age cause hypertrophy resulting in aging. Molecular damage on the other hand, even if it accumulates, is considered a bystander that has no influence on the aging process, or perhaps it represents a secondary factor (i.e., hyperfunction and hypertrophy cause damage, not the other way around) ***(Blagosklonny, 2008).***

Organ-level changes with aging

These are common across all systems, but for anesthetists it is the changes in the cardiovascular, respiratory and central nervous systems that have most impact ***(Kanonidou and Karystianou, 2007).***

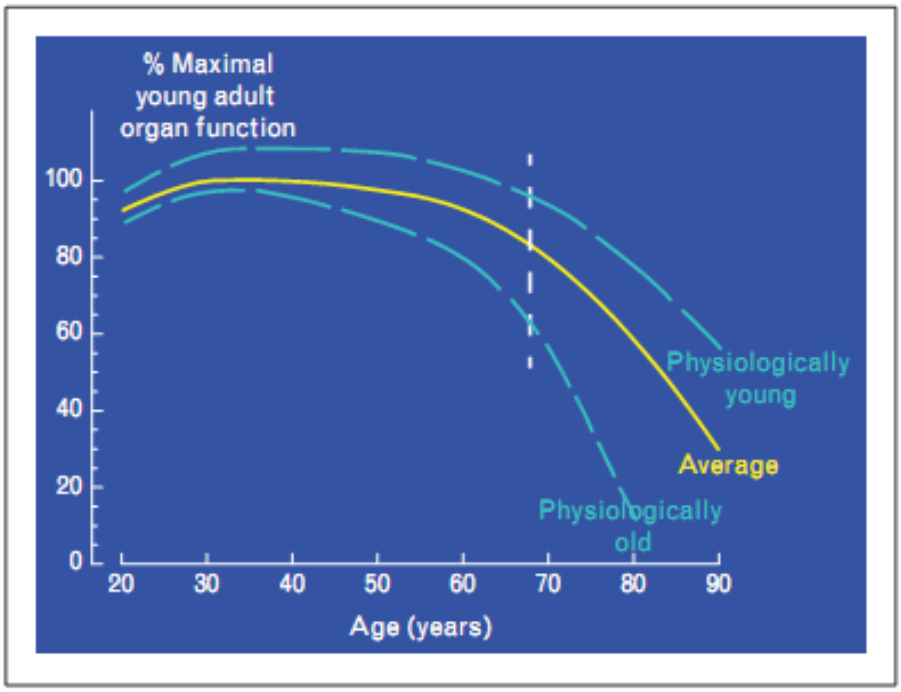

Figure (1): Age-related changes in major organ system function.

Cardiovascular system changes

a. Cardiac changes at rest and with exercise

Cardiac structural changes:

There are a number of structural and functional changes in the heart with aging and each of these can have significant implications for cardiovascular disease.

Structurally, there is a significant increase in myocardial thickness as a result of increased cardiomyocyte size**.** In addition, the heart changes its overall shape from elliptical to spheroid with an asymmetric increase in the interventricular septum more than the free wall**.** These changes in thickness and shape have important implications for cardiac wall stress and overall contractile efficiency ***(Hees et al., 2002)***.

Cardiac systolic function:

Despite a number of aging associated changes that may limit a person's functional capacity and promote vascular stiffening with consequent increased afterload, the overall resting systolic function of cardiac muscle does not change with healthy aging.

An overall decrease in exercise tolerance is evident in the progressive decline in V_{O2}max, starting at age 20–30 and falling by approximately 10% per decade.

Additionally, the rate of this decline progressively increases with age. The meaning of these changes and insight into the underlying factors can be clarified through a review of the Fick Equation: VO2max=CO× (A−V) O2 ***(Fleg and Lakatta, 2000).***

Table (1): Cardiovascular system changes ***(Fleg and Lakatta, 2007)***

Oxygen consumption	↓ (50%)
$(A\text{-}V)O_2$	↓ (25%)
Cardiac Index	↓ (25%)
Heart Rate	↓ (25%)
Stroke Volume	No Δ
Preload EDV	↓ (30%) (men>women)
After load Vascular (PVR) Cardiac (ESV)	 ↓ (30%) ↓ (275%)
LV Contractility	↓ (60%)
Ejection Fraction	↓ (15%)
Plasma Catecholamines	↑
Cardiac and Vascular responses to β-adrenergic stimulation	↓

EDV, End Diastolic Volume; ESV, End Systolic Volume; LV, Left ventricular; (A-V) O2, Arterial-Venous Oxygen concentration; PVR, Peripheral Vascular Resistance.

CO is known to fall 25% with aging, which by definition must be due changes in stroke volume (SV) or heart rate (HR)**.** Because SV is thought to be maintained throughout life the bulk of the CO decline is likely due to impaired heart-rate acceleration ***(Fleg and Lakatta, 2007).***

Overall, this reduction in cardiac reserve is a result of multiple factors including increased vascular after load, arterial-ventricular load mismatching, reduced intrinsic

myocardial contractility, impaired autonomic regulation, and physical deconditioning ***(Fleg et al., 2005).***

Cardiac diastolic function:

Despite maintenance of systolic function at rest, there are a number of changes in the diastolic phase of the cardiac cycle that occur with aging. Normal diastolic filling can be divided into two phases, passive and active. The heart fills with blood more slowly in older vs. younger healthy individuals resulting in a lower proportion of total diastolic filling occurring during this passive, early diastolic phase due mainly to an increase in the isovolumic relaxation time ***(James and Lakatta, 2012).***

Changes in cardiac conduction system and electrocardiogram:

Aging is associated with a generalized increase in elastic and collagenous tissue. Fat accumulates around the sinoatrial node, sometimes creating a partial or complete separation of the node from the atrial tissue. A pronounced decline in the number of pacemaker cells occurs after age 60; by age 75 less than 10% of the number seen in young adults remain. This can impact the atrioventricular node, atrioventricular bifurcation, and proximal left and right bundle branches leading to significant risk for atrioventricular conduction block ***(James and Lakatta, 2012).***

Table (2): Normal age-associated changes in resting ECG measurements ***(James and Lakatta, 2012)***

Measurement	Change with age	Effect on mortality
R-R Interval	No change	
P-wave duration	Minor increase	None
P-R interval	Increase	None
QRS duration	No change	
QRS axis	Leftward shift	None
Q-T interval	Minor increase	Probable increase
T-wave voltage	Decrease	None

Cardiac adrenergic responsiveness:

Adrenergic signaling is an important component of aging-associated cardiovascular change. Acute exercise and other stressors stimulate sympathetic modulation of the CV system, which increases heart rate, augments myocardial contractility and relaxation, reduces LV afterload, and redistributes blood to working muscles and skin to dissipate heat ***(James and Lakatta, 2012).***

However, with aging there is a diminishment of the autonomic modulation of heart rate, LV contractility, and arterial afterload related to a decline in the efficiency of post-synaptic **β**-adrenergic signaling.

Norepinephrine and epinephrine increase to a greater extent in older than in younger healthy individuals. This increase in plasma catecholamines appears to be a

compensatory response to the reduced cardiac muscarinic β-receptor density and functional decline with advancing age ***(James and Lakatta, 2012).***

b. Central arterial changes with aging

Central arterial microscopic and biochemical changes:

Aging is associated with a number of structural and functional changes of the arterial wall media (hypertrophy, extracellular matrix accumulation, calcium deposits) and the vascular endothelium (decrease in the release of vasodilators and increased synthesis of vasoconstrictors) that are associated with increased vascular stiffness ***(Ungvari et al., 2010).***

Central arterial function:

Blood Pressure

Systolic blood pressure (SBP), which is influenced by arterial stiffness, PVR, and cardiac function, rises with age even in normotensive. In contrast, diastolic blood pressure (DBP) rises with increased PVR but is lowered by arterial stiffness resulting in an increase in diastolic pressure till age 50, a leveling off between 50 and 60, then a decline after age 60 ***(Tanaka et al., 2000)***.

Thus, hypertension in the elderly is often characterized by isolated or predominant systolic BP elevation. Pulse pressure, the difference between SBP and DBP, is a useful

clinical index of arterial stiffness and the pulsatile load on the arterial tree and typically increases with aging. Pulse pressure is a more powerful predictor of future CV events than either SBP or DBP in older adults ***(Roman et al., 2009).***

c. Cardioprotective and repair processes

Aged heart is placed under increasing levels of stress due to its diminished functional reserve. Furthermore, increasing age results in the increased occurrence of numerous disease processes e.g. diabetes, hypertension.

While the heart has a number of protective systems in place to deal with such insult, these decline with age resulting in a lower injury threshold. ***(James and Lakatta, 2012).***

Ischemic preconditioning:

(IPC) is the hearts endogenous capacity to resist ischemic damage and its effects including suppression of ventricular arrhythmias and enhanced recovery of contractile function. The process is dependent on an initial, brief ischemic event usually lasting less than 5 minutes. It appears to have both an early protective function lasting about 1 hour after preconditioning (PC) and also a late-delayed action that returns around 24–96 hr later.***(Juhaszova et al., 2005).***

Respiratory system changes

Aging, in the absence of additional challenges, does not result in hypoxia or pneumonia. However, age-related anatomic and functional changes in the respiratory system contribute to the increased frequency of pneumonia, increased likelihood of hypoxia, and decreased maximum oxygen uptake in the older person ***(Sharma and Goodwine, 2006).***

These fundamental structural changes lead to the following physiological changes seen with aging process:

Table (3): Changes in respiratory function associated with aging and pathophysiologic mechanisms that explain preoperative complications ***(Ceba et al., 2008).***

Function alteration		Pathophysiology	Potential complications
Upper airway patency	↓	Hypotonia of hypopharyngeal and genioglossal Upper airway muscles, obesity (redundant tissues)	Upper airway obstruction and OSA
Swallowing reflexes and cough	↓	↓Clearance of secretions	Aspiration due to risk, inefficient expectoration, pneumonia, atelectasis, hypoxemia
Chest wall compliance	↓	Structural changes of the intercostal muscles and joints	↑Work of breathing; delayed weaning from mechanical ventilation
Airway resistance	↑	↓ Diameter of small airways	Air trapping, propensity for developing