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Physiology Considerations in the Geriatric Patient

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Synopsis

A person's physiology is ever-changing at the structural, functional, and molecular levels as they age, and every major organ system experiences physiologic change with time. The changes to the nervous system result mostly in cognitive impairments, the cardiovascular system result in higher blood pressures with lower cardiac output, the respiratory system result in a reduction of arterial oxyhemoglobin, the gastrointestinal system result in delayed gastric emptying with a reduction of hepatic metabolism, and the renal system experiences a diminished glomerular filtration rate. All these changes are variable from patient to patient; however, combined, they create a complex physiological condition. This unique physiology must be taken into consideration for a geriatric patient undergoing general anesthesia.

Keywords

Geriatric; Physiology; Cardiovascular aging; Neurological aging; Aging

Introduction

A person's physiology is a complex, ever-changing state with aging-related changes occurring at the structural, functional, and molecular levels.¹ The process of aging is complex and multifactorial with multiple hypotheses broadly categorized into either the

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‘programmed’ theory or the ‘error’ theory.¹ The ‘programmed’ theory states that delineated biological alterations in homeostatic state and natural defense will occur over time.¹ The ‘error’ theory focuses on free radical accumulation secondary to reactive oxygen species generated during mitochondrial energy production, causing oxidative damage to DNA, protein, and lipids.¹ No matter the theory, aging is defined as the normal progressive decline in function and ability to respond to intrinsic (e.g., catecholamines, inflammation) or extrinsic (e.g., infection, surgery) stimuli.²

A patients’ age is a strong correlate of risk of morbidity and mortality.³ For non-cardiac surgery, 30-day mortality is expected to increase by a factor of 1.35 per decade of age.³ Age itself is also an independent risk factor for a long list of diseases, injuries, hospitalization, length of hospitalization, and adverse drug reactions,⁴ and almost every organ system is affected by aging. We will therefore examine the most recent evidence and understanding of how aging affects the major organ systems.

Nervous System

The aging brain is accompanied by a change in structure, function, and metabolism (Figure 1).⁵ The volume and weight of the brain declines at a rate of approximately 5% per decade after age 40.⁶ Once the brain is 70 years old, the rate of decline is thought to increase.⁶ The changes in neuronal volume and affected areas may be related to gender.⁶ Brain atrophy starts earlier in men but is more rapid in women once it has started.⁵ There are longitudinal studies using MRI imaging and reviews of cross sectional studies that show the prefrontal cortex as the most affected region of neuronal cell death.⁶ The medial temporal lobes are also very sensitive to age,⁵ and additional areas also include the cerebellar vermis, cerebellar hemispheres, and hippocampus.⁶

Upon analysis of post-mortem brains, there is a greater loss of white matter than grey matter with aging,⁷ and granular degeneration of myelinated axons is observed regularly by the age of 40.⁷ Neuronal cell death is believed to be the main reason for the loss of grey matter; whether this is the only reason, however, is unclear.⁶ There may be additional changes in dendritic arbour, spines, and synapses, with dendritic sprouting occurring to help maintain a similar number of synapses and compensating for any cell death.⁶

Along with a reduction of brain volume, there are cognitive changes associated with aging. Memory declines are one such cognitive change, with decline in episodic memory being most common.⁶ This type of memory is defined as ‘a form of memory in which information is stored with mental tags, about where, when and how the information was picked up’ and is thought to decline starting around the 4th and 5th decades onward.⁶

Neurotransmitter changes also occur with age. Dopamine levels decline by around 10% per decade starting in early adulthood.⁶ This decrease has been associated with declines in cognitive and motor performance.⁶ Serotonin and brain derived neurotrophic factor levels also decrease with age, and decreases in these neurotransmitters have been associated with reduced synaptic plasticity regulation and neurogenesis.⁶ Monoamine oxidase, an important substance in the homeostasis of neurotransmitter levels, increases with age and may liberate free radicals from reactions that exceed inherent antioxidant reserves.⁶

The blood-brain barrier protects the central nervous system from systemic insults through selective permeability. Increasing age is associated with increasing blood-brain barrier permeability,⁸ thereby allowing inappropriate passage of mediators from the plasma into the central nervous system. This likely results in an increased inflammatory response and structural damage in the brain as well as altered patterns of neuronal activity by modulating synthesis of neurotransmitters and changing expression of neurotransmitter receptors.^{9,10}

Vascular distribution in the brain also changes every decade of life. Capillaries are densely packed in areas of the brain that have higher processing demands, and these dense areas of capillaries tend to decrease.⁷ Starting around the 5th decade of age, every decade of life shows an increase in the degree and number of micro-vessel deformities.⁷ Cerebroarterial change begins mostly in the intima with approximately 50% of the vessels showing intimal thickening by the 4th decade and up to 80% by the 8th decade.⁷ These changes are often the precursors to arteriosclerosis which increases vascular resistance and decreases perfusion pressure, thereby compromising neurocognitive function.⁷ Studies using functional imaging techniques to evaluate the effects of aging have found a reduction in cerebral metabolic rate of oxygen consumption with decreased cerebral blood flow in grey matter but preservation of blood flow to the white matter.⁷

With an aging population, there will be an increase in elderly patients having surgery.¹¹ With this, the prevalence of postoperative cognitive disorders in the aging brain is likely to increase.¹¹ All the changes (Figure 1) seen in the brain increase the likelihood of post-operative cognitive disorders including delirium in the acute setting and postoperative cognitive dysfunction in the long term.¹¹

Cardiovascular System

The geriatric population tends to have higher blood pressures, similar heart rates and ejection fractions, and lower left ventricular end-diastolic volumes, stroke volumes, and cardiac outputs compared to younger populations (Figure 2).³ These aging-related changes in the cardiovascular system primarily start with changes in connective tissues. Connective tissue stiffens within the arteries, veins, and myocardium, causing them to become less compliant.³ This stiffening is secondary to a cessation of elastin production in the 4th decade of life.³ Also, collagen turnover is a slow process, and both elastin and collagen proteins accumulate free radical damage over time.³ As elastin is damaged, it is then replaced with less flexible collagen protein.³

Arterial stiffening leads to systolic hypertension, impaired impedance matching, and myocardial hypertrophy.^{3,4} The stiffening within the aorta causes an increase in systolic blood pressure and a decrease diastolic pressure.^{3,4,12} The diminished diastolic pressure leads to a decrease in coronary blood flow.¹² Under normal circumstances, most of the stroke volume is contained within the thoracic aorta; as the aorta stiffens, the pressure to transfer this volume increases.³ Thus, chronically elevated left ventricular afterload leads to left ventricular thickening.⁴ There is also an increase in impedance matching between the declining strength of the myocardial contraction and the increases in pressure within the aorta.³ When this pressure wave travels down the arterial tree, the wave will reflect off both

the vessel walls and branch points, returning to the thoracic aorta.³ This pressure reflection creates a poor impedance match and causes more strain on the myocardium—serving as a significant stimulus for myocyte hypertrophy.³

The combination of myocyte hypertrophy and elevated left ventricular afterload prolongs myocardial contraction.⁴ This extended contraction leads to a delay in ventricular relaxation and results in early diastolic filling rates declining by approximately 50% between the 2nd and 8th decade.⁴ The end diastolic volume is typically preserved secondary to late diastolic filling and becomes more reliant on atrial contribution for effective filling.⁴ Ventricular myocardial stiffening and hypertrophy, therefore, render the heart dependent on atrial filling pressures.³ It will also increase susceptibility to diastolic heart failure,³ for diastolic dysfunction is a key factor in the development of heart failure with preserved ejection fraction.¹³

Venous stiffening with age decreases ability to buffer changes in blood volume and blood distribution.³ Upwards of 80% of blood volume is stored within the venous network.³ This reservoir, therefore, is important in maintaining a stable preload to the heart, and venous stiffening will impair the ability to keep preload constant.³

Aging results in an increase in sympathetic nervous system activity and higher levels of circulating norepinephrine, resulting in an increase in arteriole constriction and systemic vascular resistance.³ Evidence for this increase in norepinephrine include an increase in norepinephrine release from nerve terminals, an increase in the percentage of norepinephrine reaching the general circulation, and a decrease in the metabolism and reuptake.³

The myocardium's beta-receptor is also affected with age. The response of the receptor to stimulation is reduced, resulting in a decrease in heart rate and contractile response to hypotension, exercise, and catecholamines.³ This does not seem to be secondary to a decline in the number of beta-receptors but rather a diminishment in the intracellular coupling with adenylate cyclase.³ The diminished chronotropic and inotropic response of the heart to beta-receptor stimulation changes the heart's ability to respond to either intrinsic or exogenous catecholamine stimulation.³ This beta-receptor limitation increases the dependence of the Frank-Starling relationship to maintain cardiac output.³ Other changes in the cardiovascular system include a diminished baroreflex stimulation, lower vagal nerve tone, and reduced oxygen extraction.³ The impaired baroreflex system and vagal tone result in less heart rate variability and ability to control a constant cardiac output.³

Endothelial dysfunction is a leading theory to the mechanism of vascular aging. Endothelial function, including nitric oxide release, has vasoprotective and cardioprotective properties due to inhibition of platelet aggregation and inflammatory cell adhesion to endothelial cells, disruption of pro-inflammatory cytokines, apoptosis inhibition, and tissue energy metabolism regulation.¹² There is considerable evidence published showing an increase in reactive oxygen species within the heart and vasculature with age,¹⁴ and the accumulation of oxidative stress results in altered nitric oxide production.¹² Furthermore, there is also a lower production of nitric oxide with age.¹² These factors diminish the bioavailability of

nitric oxide in the coronary and peripheral circulation in the elderly, resulting in impairment of flow in the microvasculature and increasing risk for organ dysfunction.¹²

The stiffening of the connective tissue, impaired impedance matching, the myocardial hypertrophy, venous stiffening, increase in sympathetic nervous system, altered nitric oxide production, and the diminished beta receptor response are the changes (Figure 2) in the cardiovascular system that cause there to be more hypotension and greater blood pressure lability during anesthesia in elderly patients versus young adults.³ This affects the depth and type of anesthesia required and the sympathetic nervous system response to changes in surgical stimulus and surgery in general.³

Respiratory System

The lungs continue to develop throughout life, and maximal functional status is achieved in the early part of the 3rd decade, after which lung function gradually declines.¹⁵ Changes with aging include alteration of the mechanical properties of the respiratory system, reduction of arterial oxyhemoglobin saturation, and impaired response to hypoxia (Figure 3).¹⁵

The parenchyma of the lung undergoes significant structural alterations with aging, with the most important one being a reduction in number and crosslinks of elastic fibers resulting in a reduction of elastic recoil.^{15–17} This creates inward forces that promote a decrease in lung volumes at an average rate of between 0.1–0.2 cm H₂O per year, and this decrease is most pronounced after the 5th decade.¹⁵ Homogenous enlargement of the air spaces also causes a reduction in alveolar surface area from 75 m² to 30–60 cm² by the age of 70—increasing lung compliance and representing functional emphysema.^{15,17}

In addition to decreased elastic recoil, there is also a decrease in compliance with age due to structural changes with intercostal muscles, joints, and rib vertebral articulations that decrease compliance of the chest wall.^{15–18} Age-related development of osteoporosis results in a reduction of height of the thoracic vertebrae causing further restriction.¹⁸ In addition, a reduction of respiratory muscle mass may contribute to a decrease in the force produced by the respiratory muscle activity.¹⁵ This loss of chest wall muscular function, however, does decrease the outward force requirements such that the total lung capacity remains unchanged.^{15,18}

With these changes in the properties of the connective tissues, there are significant changes to the mechanics of the lung. The functional residual capacity increases by 1–3% per decade.¹⁵ The residual volume increases by 5–10% per decade.¹⁵ Because the total lung capacity remains unchanged, there is a decrease in vital capacity.¹⁵ This decrease in physiological reserve increases geriatric patients' vulnerability to infection and impairment.¹⁶ After the age of 20 years, the vital capacity will decrease from 25% to 40% by 70 years of age.¹⁵ There are progressive decreases in both forced vital capacity (14–30 mL per year) and forced expiratory volume in one second (23–32 mL per year).¹⁵ At the age of 65, there is a decrease in FEV1 of approximately 38 mL per year.¹⁵ These changes can make a normal FEV1/FVC ratio as low as 55% in the elderly.¹⁵

Aging also affects gas exchange properties. Arterial oxygenation gradually declines with aging,^{15,17} likely secondary to an increase in ventilation/perfusion heterogeneity caused by a decrease in alveolar surface area and the premature closure of small airways.^{15,16} To predict the effect of age on arterial oxygenation, several equations have been proposed.¹⁵ Between the ages of 40–75 years, the best equation that takes into account both PaCO₂ and body mass index (BMI) is: $\text{PaO}_2(\text{mmHg}) = 143.6 - (0.39 \times \text{age}) - (0.56 \times \text{BMI}) - (0.57 \times \text{PaCO}_2)$.¹⁵ Once over the age of 75 years, arterial oxygen tension does not correlate with BMI and PaCO₂ and instead remains stable at approximately 83 mm Hg.¹⁵

Elderly subjects will have a lower tidal volume and a higher respiratory rate than younger subjects.¹⁵ There is an approximately 50% decrease in their response to hypoxia and hypercapnia¹⁵ and a decrease in diffusion capacity of the lung.¹⁶ Increased ventilation is often required to compensate for this decreased efficiency of gas exchange.¹⁵

Along with these changes in the lung, there are important changes seen in the upper airway. There is a loss of muscular pharyngeal support making the elderly more susceptible to upper airway obstruction.¹⁵ There is also a decrease in respiratory effort in response to upper airway occlusion.¹⁵ Protective mechanisms of coughing and swallowing also diminish with time, making a geriatric individual more at risk for aspiration.¹⁵ A presumed explanation for these changes includes peripheral differentiation along with decreased central nervous system reflex activity.¹⁵

These changes in the respiratory system (Figure 3) contribute to the pulmonary complications that can be seen post-anesthesia.¹⁵ The decrease in chest wall compliance results in an increase in a geriatric patients work of breathing post-anesthesia.¹⁵ The changes in lung mechanics will impair a geriatric patient's gas exchange and the tendency for small airway closure leading to atelectasis.¹⁵ Several studies have shown that age alone is a significant independent predictor of risk for perioperative pulmonary complications.¹⁵

Gastrointestinal System

Age-related changes occur along most of the gastrointestinal track (Figure 4). There is a decrease in the amplitude of esophageal contractions and a decrease in the number of peristaltic waves that occur with a standard swallow.¹⁹ When studied, there was found to be an increase in the number of disordered contractions in the body of the esophagus.¹⁹ Geriatric individuals are also subject to prolonged gastric emptying.¹⁹ One study demonstrated a standard mixed meal took double the time to empty when compared to younger subjects.¹⁹ Thus, elderly patients are at higher risk for aspiration at anesthetic induction or in the postoperative period. Gastric acid secretion decreases with age at both a basal rate and when fixed doses of exogenous gastrin are given to stimulate production.¹⁹ This is secondary to the development of atrophic gastritis that causes a decrease in secretion of acid and intrinsic factor (not enough, however, to result in vitamin B-12 malabsorption and pernicious anemia).¹⁹ Atrophic gastritis may affect calcium bioavailability because of a limited ability to dissociate from food complexes.¹⁹

Pancreatic function does not seem to be diminished with age, for there does not seem to be any diminished response to stimulation by secretin or cholecystokinin.¹⁹ The mucosal

surface of the small intestine is slightly diminished in the aging human.¹⁹ Despite this, the ratio of mean surface area to volume of jejunal mucosa does not change with age.¹⁹

The liver does experience significant change with age (Figure 4). There is a reduction of liver volume, ranging from 20–40% across the human lifespan.^{2,20} With the loss of volume, there is also age-related decline in hepatic blood flow.^{2,20} The age-related loss of surfaced endoplasmic reticulum causes a strong negative correlation between age and hepatic microsomal Phase-I drug metabolizing activity.^{2,20} This decline in total capacity of the liver to metabolize drugs can increase the incidence of adverse drug reactions.²⁰ This decline can be very variable, from drug to drug and from person to person.²⁰

Renal and Volume Regulation System

Renal function declines related to age have been well documented within multiple geographic settings, patient populations, and using a wide range of methods and parameters (Figure 5).²¹ There is a cumulative increase in patients with end stage renal disease with increasing age.²² Age-related renal decline is affected by gender, with males more affected than females secondary to increased damage from vascular changes and androgen production.²¹ Aging causes both creatinine clearance and glomerular filtration rates to decline, resulting in not only a sharp increase in chronic renal impairment and end-stage renal failure but increased susceptibility to acute insults.²¹

Electrolyte homeostasis is also affected with age. There is a slower homeostatic responsiveness to sodium changes and a decreased ability to maximally dilute or concentrate urine.^{21,23} There is also a global impairment of movement of other electrolytes and ions transport across the tubular epithelium.²¹

There are significant changes to the renal vascular network in the geriatric population. This includes a reduction in the actual and proportional renal blood flow with age.²¹ After the 4th decade, there is a 10% decrease in renal blood flow with each decade thereafter.²³ With this diminished renal blood flow, there is an accompanied decrease in responsiveness and autoregulation of volume status.²¹ The infrarenal arterial changes are similar to the systemic changes—arteriolosclerosis, intimal and medial hypertrophy.²¹

There are also significant interstitial changes to an aging kidney. When looking at the size of the kidney, it was found that they increase in size up to the 5th decade and then start to decrease.²¹ This volume loss is thought to be due to tubule-interstitial changes including infarction, scarring, and fibrosis, resulting in decreased clearance capabilities.²¹

The renin-angiotension-aldosterone system is also affected with age, resulting in substantial declines in plasma renin activity.²⁴ The most significant decreases are found in the 6th decade onward.²⁴ Age has a greater effect on the decrease in plasma renin and aldosterone level compared to angiotension II.²⁴

The geriatric patient will have pharmacokinetic changes in the absorption, distribution, metabolism, and excretion of anesthetic drugs.²² There is a reduction in systemic clearance of drugs that are eliminated unchanged by the kidney due to age-related changes in

glomerular filtration rate and tubular function.²² There is also a higher incidence of chronic kidney disease; this fact, along with the diminished blood flow and changes to autoregulation, leads to an increase in prevalence of perioperative acute kidney injury.^{21,22}

Endocrine System

There is a decline in endocrine function with age that includes decreased tissue responsiveness and a reduction in hormone secretion from peripheral glands (Figure 6).²⁵ Examples include reductions in thyroxin (T4) and triiodothyronine (T3). Age also results in a dampening of circadian hormonal and non-hormonal rhythms.²⁵

Impaired glucose tolerance develops in over 50% of individuals older than 80 years of age.²⁶ There is a decrease in insulin production by beta cells, an increase in insulin resistance related to poor diet, an increase in abdominal fat mass, and a decrease in lean body mass. These all contribute to the deterioration of glucose metabolism²⁶ and make elderly patients at higher risk of poor glycemic control in the perioperative setting.

Women typically experience menopause in the 6th decade of life when serum estradiol concentrations are lower and follicle-stimulating hormone concentrations are higher than in younger women.²⁵ Luteinizing hormone does not change like follicle-stimulating hormone.²⁵ These changes, along with the fall of estrogen, increase the risk of cardiovascular events, rapid loss of skeletal mass, vasomotor instability, psychological symptoms, and atrophy of estrogen-responsive tissue.²⁵

Male gonadal steroid production also changes with age – a change termed andropause.²⁵ There is a marked decline in free testosterone levels from an increase in sex hormone-binding globulin levels.²⁵ The age of this decline is variable, and the physiological consequences are unclear.²⁵ There is also a decline in total serum testosterone concentrations due to a decrease in production rates as men age.²⁵

Summary

No matter the mechanism, aging affects the physiology of every major organ system. The nervous system experiences cognitive decline and volume loss. The cardiovascular system changes result in lower cardiac output and higher blood pressures leading to significant changes to the structure and function of the heart. The respiratory system changes lead to impaired oxygenation, diminished ventilation/perfusion matching, and an increase risk of atelectasis. The gastrointestinal system experiences a delay in emptying along with diminished hepatic metabolism. The renal system changes result in a diminished glomerular filtration rate and a diminished ability to control electrolyte hemostasis. The endocrine system results in hormonal changes that lead to variation in the patients' condition. Importantly, all these changes can have major effects on the perioperative course of a geriatric patient, and anesthesia providers require an understanding of these changes and how they will affect the management of their geriatric patients.

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Key Points

1. Changes in structure, function, metabolism, and blood flow in the aging brain lead to cognitive impairments, most frequently episodic memory changes, and an increased risk of delirium in the acute setting.
2. The geriatric population tends to have higher blood pressure with lower cardiac output and diminished chronotropic and inotropic responses to beta-receptor stimulation.
3. Respiratory aging results in changes to mechanical properties of the respiratory system, reduction of arterial oxyhemoglobin saturation, and impaired response to hypoxia.
4. Gastrointestinal changes with aging include altered esophageal motility, delayed gastric emptying, and reduction in hepatic metabolism.
5. There is a reduction in renal function with age, and changes also occur to the endocrine system, including diminished tissue responsiveness and reduction in hormone secretion from peripheral glands.

Changes with age of the neurological system			
Brain volume	▼	Blood brain barrier permeability	▲
Dopamine levels	▼	Arterial wall thickness	▲
Cerebral metabolic rate	▼	Monoamine oxidase activity	▲
▲augmented;▼ diminished			

Figure 1.
Changes in the neurological system with age

Changes with age of the cardiovascular system			
Cardiac changes		Vascular changes	
Heart weight	▲	Arterial wall thickness	▲
Cardiomyocyte number	▼	Elastin	▼
Collagen cross-linking	▲	Elastin fragmentation	▲
Early diastolic filling	▼	Arterial distensibility	▼
End-diastolic filling	▲	Pulse wave velocity	▲
Chronotropic responsiveness to beta-agonists	▼	Total peripheral resistance	▲
Inotropic responsiveness to beta-agonists	▼	Endothelial function	▼
▲augmented,▼ diminished			

Figure 2.
Changes in the cardiovascular system with age

Changes with age of the respiratory system			
Ventilation-perfusion mismatch	▲	Protective cough	▼
Chest wall rigidity	▲	Residual volume	▲
Respiratory muscle strength	▼	Vital capacity	▼
Work of breathing	▲	Closing volume	▲
Respiratory muscle endurance	▼	Gas exchange	▼
Functional alveolar surface area	▼	Response to hypoxemia and hypercapnia	▼
▲augmented;▼ diminished			

Figure 3.
Changes in the respiratory system with age

Changes with age of the gastrointestinal and hepatic system			
Gastrointestinal changes		Hepatic changes	
Esophageal motility	▼	Hepatic blood flow	▼
Gastric acid secretion	▼	Hepatic volume	▼
Gastric emptying time	▲	Microsomal demethylation pathway	▼
Small intestine surface area	▼	Drug metabolism	▼
▲augmented;▼ diminished			

Figure 4.
Changes in the gastrointestinal and hepatic system with age

Changes with age of the renal system			
Number of nephrons	▼	Tubular secretions	▼
Glomerular filtration rate	▼	Ability to conserve sodium	▼
Renal blood flow	▼	Total body water	▼
Ability to concentrate urine	▼	Thirst perception	▼
▲augmented;▼ diminished			

Figure 5.
Changes in the renal system with age

Changes with age of the endocrine system			
Hormone secretion	▼	Female estrogen	▼
Tissue responsiveness	▼	Female estradiol	▼
Glucose metabolism	▼	Female follicle-stimulating hormone	▼
Serum thyroxin (T4)	▲	Female luteinizing hormone	▬
Serum triiodothyronine (T3)	▬	Male free testosterone	▼
▲augmented;▼ diminished; ▬ unchanged			

Figure 6.
Changes in the endocrine system