Theme 1: Physiologic Changes in the Elderly
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This unit forms the second part of a series of teaching modules on Anesthesia for the Elderly. It will guide you step-by-step through some salient physiologic differences between the aged and the younger adult population. It is our hope that on completing this component you will appreciate why understanding physiologic differences between these age groups guides us in our anesthetic management.

Introduction

This section describes significant physiologic changes between the aged and younger populations as defined in longitudinal studies of healthy people. It will be seen that aging results in significant anatomic and functional changes in all the major organ systems. Aging is marked by a decreased ability to maintain homeostasis. However, within the elderly population there is significant heterogeneity of this decline.

This unit introduces facts in a systemic fashion, and then asks you some questions testing your mastery of the information. Each section is self-contained and leads to the next. One should not progress until one has fully understood the material in the previous section.

Goals

After completing this unit the resident should be able to:

- Understand the main structural and functional changes associated with normal aging
- Understand how these changes will impact the practice of anesthesiology
Cardiovascular System (CVS)

"In no uncertain terms, you are as old as your arteries." — M. F. Roizen, RealAge

It is controversial whether significant CVS changes occur with aging. Some authors claim there is no age-related decline in cardiovascular function at rest. Their position is that no age-related change is found in resting cardiac output (CO), end-diastolic or end-systolic volumes, or ejection fraction in the elderly. Cardiac tissue itself undergoes only small metabolic changes due to aging itself. Opposing this view are those who claim that there are decreases of upward of 5% in CO per decade. The truth probably lies in between (Figure 1).

![Diagram](image)

**Figure 1.** Physiological changes are not sufficient to explain CVS changes in the elderly. The CVS changes result from a combination of aging, pathology, and lifestyle.

Aging is marked by a significant deterioration in homeostasis. This is manifested in the CVS by a reduced ability to maintain hemodynamic stability. Although these changes represent a heterogeneous process, some aspects are characteristic of the group as a whole:

- A progressive replacement of supple, functional cardiac and vascular tissue by stiff, fibrotic material. The large arteries of the body lose their elasticity, with a stiffer aorta resulting in increased peripheral resistance. Increased sympathetic nervous system activity may contribute to the increase in peripheral resistance.

- The left ventricle must work harder to eject blood into a more rigid aorta. Left ventricular hypertrophy develops as an adaptive mechanism to the increased peripheral resistance. Increased ventricular wall thickness leads to increased ventricular wall stiffness in early diastole, impairing ventricular filling.
End-diastolic pressure may increase to overcome the noncompliant, stiffened ventricle. This elevated left ventricular filling pressures can be reflected into the left atrium and the pulmonary vasculature, leading to pulmonary congestion. Clinically important diastolic dysfunction likely involves poor ventricular relaxation in early diastole as well as the natural ventricular tissue stiffening from aging and hypertrophy. Loss of the sinus rhythm or sinus tachycardia, common events during anesthesia, may well depress cardiac output and arterial pressure more markedly in the elderly than they would in a normal younger patient.

Veins are also subject to progressive stiffening with age. The decreased compliance of the capacitance system reduces its ability to "buffer" changes in intravascular volume.

With advancing age, tonic parasympathetic outflow declines, while overall sympathetic neural activity increases. However, elderly subjects generally manifest a reduced responsiveness to beta-adrenergic stimulation. Although resting heart rates do not change much with age, the maximal attainable heart rate, stroke volume, ejection fraction, CO, and oxygen delivery are all reduced in healthy older adults. The administration of beta-adrenergic agonists elicits lesser inotropic and chronotropic responses in the elderly, while beta-blocking drugs retain their effectiveness. (In contrast, the vascular responses to exogenous alpha-adrenergic agonists do not appear to be much affected by age.)

The elderly respond to stress with less tachycardia, possibly due to the decline in the responsiveness of beta-receptors. Increases in CO in the elderly tend to be largely due to increases in stroke volume rather than heart rate.

As aging impairs both the diastolic filling and the chronotropic and inotropic responsiveness of the heart, the ability of the older patient to cope with perioperative stress is predictably impaired. Increased metabolic demands, such as those imposed by sepsis or postoperative shivering, may not be met when the maximal CO and oxygen delivery are limited by aging. While young adults can compensate for blood loss (exacerbated by anesthetic-induced vasodilatation) with increases in heart rate and ejection fraction, the elderly cannot so readily maintain their CO and are more dependent upon vasoconstriction to sustain adequate arterial pressures.

Blood vessels become less elastic with age. The “average” blood pressure increases from 120/70 to 150/90 and may stay slightly high even if treated. Elderly patients also respond with exaggerated rises in blood pressure to “stress” because of “stiff” vasculature.

The maintenance of hemodynamic homeostasis largely depends upon the baroreflex. Baroreceptors in the aortic arch and carotid sinus are actually stretch receptors; a decrease in distention of these receptors results in augmented sympathetic nervous system activity and inhibition of peripheral nervous system
outflow. Arterial stiffening may reduce the ability of the baroreceptors to transduce changes in pressure, diminishing the magnitude of the baroreflex. Both aging and hypertension are associated with increased arterial rigidity. It is therefore not surprising that, in general, both advancing age and chronic hypertension, alone or together, are associated with impairment of baroreflex responsiveness. This impairment likely contributes to the increased susceptibility of older adults to orthostatic hypotension, a problem that is exacerbated by the common administration of diuretic and other medications, such as those used to treat hypertension, depression, and parkinsonism.

Respiratory System

The effects of aging on the lungs are physiologically and anatomically similar to those that occur during the development of mild chronic obstructive pulmonary disease (COPD). Aging affects a number of parameters of lung function, such as ventilation, gas exchange, and compliance, as well as pulmonary defense mechanisms. Pure age-related changes do not, however, lead to clinically significant airway obstruction or dyspnea in the nonsmoker. As with the CVS, the presence of disease (i.e., damage from smoking) will lead to an acceleration of normal physiological decline and significant symptoms such as dyspnea.

In the young adult the respiratory system has significant reserve capacity. Aging, however, inescapably reduces the capacity of all pulmonary functions. This may lead to decompensation when the system is stressed (e.g., after major abdominal surgery). As with the CVS, the rate of loss of function is extremely variable among persons of the same chronological age.

There are 4 “core” characteristics of pulmonary aging:
- Reduction in muscle mass and power
- Changes in pulmonary compliance
- Reduction in diffusion capacity
- Decline in control of breathing

Each is discussed here in turn.

Reduction in Muscle Mass and Power

Because of a generalized loss of all neuromuscular elements, laryngeal structures undergo a slow continual decline in function. Protective reflexes are reduced, with resultant contamination of the lower airway through aspiration, silent or otherwise. The loss of an effective cough reflex occurs in >70% of elderly patients with community-acquired pneumonia (compared with only 10% of age-matched controls). Loss of the cough reflex is likely due to conditions associated with reduced consciousness in the elderly, such as sedative use and neurologic diseases. Dysphagia or impaired esophageal motility, also common in old age, may exacerbate the tendency to aspirate.
The reduction in motor power of the accessory muscles of breathing as well as the increased stiffness of the chest wall cause the dynamic lung volumes and capacities to decrease progressively with age (e.g., forced expiratory volume in 1 second, FEV$_1$). The FEV$_1$ decreases with age by about 27 mL/year in men but by only 22 mL/year in women. However, the percent change in the sexes is similar, because men start off with higher absolute values of these measurements. The annual decline in FEV$_1$ is small at first but accelerates with age (Figure 2).

![Figure 2. Decline in FEV$_1$ with age. Line (a) represents someone who has never smoked, line (b) represents a smoker, line (c) represents someone who stopped smoking at age 45, and line (d) represents someone who stopped smoking at age 65.](image)

Forced vital capacity (FVC) decreases as well, by about 14 to 30 mL/year in men and 15 to 24 mL/year in women. The decreases in FEV$_1$ and FVC that occur until age 40 are thought to result from changes in body weight and strength rather than from loss of tissue.

Airway collapse is prevented by elastic recoil of the lung tissue pulling on the airways and holding them open. Age-related loss of this elastic recoil results in early collapse of poorly supported peripheral airways, which in turn may result in decreased flow at low lung volumes, similar to the small-airway obstruction produced by long-term cigarette smoking.

**Changes in Pulmonary Compliance**

Dead space increases with age because the larger airways increase in diameter. However, expiratory flow changes very little. After the age of 40, the diameter of the small airways decreases, but again, there is no change in airway resistance.

Elastic elements of the lung parenchyma are lost with age. The end result is the smaller distal airways with a tendency to early collapse, dilated alveolar ducts, and fewer gas exchange surfaces. These changes are manifest functionally by air trapping, increased closing capacity, and frequency-dependent compliance and gas exchange problems.
Pulmonary compliance is the change in lung volume per unit change in elastic recoil pressure. Age-related changes in ventilation and gas distribution result primarily from changes in compliance of the lungs and the chest wall, as discussed below.

At about age 55 years, the respiratory muscles begin to weaken. In addition, the chest wall gradually becomes stiffer, probably as a result of age-associated kyphoscoliosis, calcification of intercostal cartilage, and arthritis of the costovertebral joints. Weakened outward muscular force combined with increased stiffness of the chest wall (decreased chest wall compliance) is counterbalanced by a loss of elastic recoil of the lungs (increased lung compliance), which probably results from a decrease in the number of parenchymal elastic fibers. Airway size decreases with age and the proportion of collapsible small airways increases.

With age, the diaphragm may weaken. Without concurrent disease, this weakening is not usually relevant. However, in the presence of disease that requires high minute ventilation, such as pneumonia, this weakening predisposes the elderly to respiratory problems.

The pressure-volume curve of an older lung is similar in shape, but shifted upward and to the left; in other words, the aged lung possesses less elastic recoil.

The tendency of the lung to assume a larger resting volume, the limitations imposed by a stiffer chest wall, and a decrease in motor power result in a change in the components of the total lung capacity (TLC) (Figure 3). The increased outward pull of the stiffer chest wall combined with the reduced ability of the lung to pull inward results in a small increase in functional residual capacity (the volume at which the lung comes to rest at the end of a quiet expiration) and residual volume (the volume that remains in the lung after a maximal expiration).

![Figure 3. Spirometric representation of lung volumes.](image)

The TLC grows with age until puberty, where it reaches an average value of 6 to 7 L, after which a slow loss of volume begins. With the age-related loss in TLC, plus the very modest increase in functional residual capacity, the ratio of functional residual capacity to TLC tends to increase with age.
Vital capacity declines progressively with age. There is a linear loss of 5% to 20% of functional ability per decade. From age 20, vital capacity decreases progressively (by $-20$ to $-30 \text{ mL/year}$) whereas residual volume increases (by $+10$ to $+20 \text{ mL/year}$). In fact, the ratio of residual volume to TLC increases from 25% at 20 years of age to about 40% in a 70-year-old man, which gives the chest wall a somewhat barrel-like appearance.

There is a clear age-related increase in the closing volume and closing capacity (Figure 4). By the age of 60 it enters into tidal volume. Both the closing volume and closing capacity also increase with recumbency, a common position perioperatively.

![Figure 4. Spirometric representation of closing volumes.](image)

**Reduction in Diffusion Capacity**

The efficiency of alveolar gas exchange decreases progressively with age, for a number of reasons:

- Alveolar surface area decreases from about 75 m² at age 20 to about 60 m² at age 70.

- Diffusing capacity (the ability of the lung to transfer gases between the lung and the blood) peaks in persons in their early 20s and then declines. From middle age onward, it declines at a rate of about 2.03 mL/min/mm Hg per decade in men and about 1.47 mL/min/mm Hg in women. This decline results from decreased surface area caused by destruction of alveoli, increased alveolar wall thickness, and small-airways closure. These changes also exacerbate ventilation and perfusion inequalities. Estrogen may slow this decline in women ages 25 to 46, presumably because of preserved vascular integrity; the effects of estrogen replacement therapy on this decline in postmenopausal women is unknown.
There is also evidence that the distribution of pulmonary blood flow changes with aging. The change in blood flow, combined with the altered distribution of inspired gas, promotes even more V/Q mismatching. Alveolar dead space, which is a good index of the distribution of pulmonary blood flow, increases with age. The increased V/Q mismatch plus the increased alveolar dead space adversely affect the aged patient's blood gas values.

The linear deterioration of the partial pressure of oxygen (PaO₂) that occurs with aging (about 0.3%/year) is estimated by the equation PaO₂ = 109 – (0.43 × age). After age 75, the PaO₂ level of healthy nonsmokers is stable at about 83 mm Hg.

The gradual decline in PaO₂ that occurs with age parallels the decrease in elastic recoil and the increase in physiologic dead space. These changes may lead to the collapse of peripheral airways, which decreases ventilation to distal gas exchange units but with much less effect on perfusion. This ventilation/perfusion imbalance accounts for most of the reduction in PaO₂. Also, lower cardiac output in the elderly results in increased tissue oxygen uptake, decreased mixed venous oxygenation, and, consequently, decreased PaO₂.

Decline in Control of Breathing

It is important to recognize that the ventilatory response to hypercapnia and hypoxia is blunted in the elderly patient. In a healthy 70-year-old, the ventilatory response (change in minute ventilation) to either a hypercapnic or hypoxic stimulus is half that seen in the 25-year-old.

Ventilatory responses to hypoxia and hypercapnia diminish with age because of diminished responsiveness of peripheral and central chemoreceptor function and integration of central nervous system pathways with age. Age also decreases neural output to respiratory muscles and lowers chest wall and lung mechanical efficiency. As a result, the ventilatory response to hypoxia is reduced by 51% in healthy men ages 64 to 73 compared with healthy men ages 22 to 30; the ventilatory response to hypercapnia is reduced by 41%. These reductions increase the risk of developing diseases that produce low oxygen levels (e.g., pneumonia, COPD, obstructive sleep apnea).

Renal System

Aging results in both structural and functional changes in the kidney that affect drug metabolism and kinetics, as well as predisposing the patient to fluid and electrolyte abnormalities.

Twenty percent of renal mass is lost between the ages of 40 and 80, mostly from the cortex (Figure 5). Microscopically there is a reduction in the number of functional glomeruli, but the size and capacity of the remaining nephrons increase to partially compensate for this loss.
Above 30 years of age the renal blood flow declines progressively at a rate of 10% per decade (Figure 6). The majority of this reduction occurs in the cortex, with a relative increase in blood flow to the juxtamedullary region.

Glomerular filtration rate (GFR) decreases by approximately 1 ml/min/year beginning by age 40 (Figure 7). This decline is accompanied by a gradual loss of muscle mass and is rarely associated with an increase in serum creatinine. Serum creatinine is therefore a poor indicator of GFR in these patients. Dosing intervals for drugs that are excreted by the kidney (e.g., pancuronium) need to be altered.
Under normal circumstances, age has no effect on electrolyte concentrations or the ability of the individual to maintain normal extracellular fluid volume. However, the adaptive mechanisms responsible for regulating fluid balance are impaired in the elderly, and the aging kidney has a decreased ability to dilute and concentrate urine. This problem is compounded by the fact that older individuals have a decreased thirst perception and fail to increase water intake when dehydrated.

Age also interferes with the kidney’s ability to conserve sodium. The geriatric patient excretes a sodium load more slowly and has a decreased ability to conserve sodium if dietary sodium is restricted, possibly predisposing the elderly patient to hemodynamic instability. Thus, fluid and electrolyte status should be carefully monitored in the elderly patient.

**Temperature Regulation**

Body temperature regulation is impaired in the elderly compared to younger adults. Elderly patients neither shiver nor vasoconstrict in response to cold until their temperature has fallen to a level below that required for activation of these homeostatic mechanisms in the younger adult population. Therefore, they are more prone to hypothermia. Such changes are mostly seen in patients over the age of 80, who can’t shiver until there is a significant fall in core body temperature. Anesthesia impairs thermoregulatory responses in all patients, but it produces even greater impairment in the geriatric population.

Perioperative hypothermia lasts longer in geriatric patients. Hypothermia is accompanied by milder shivering in the elderly than is seen in younger patients. The milder shivering produces less metabolic heat, therefore prolonging recovery to normal body temperature in the elderly.
Elderly patients are at greater risk than younger patients from the adverse effects of hypothermia, including bleeding, weakened immune function, decreased wound strength, increased infections after abdominal surgery, and myocardial infarction.

Additional care must be taken in the elderly to maintain their body temperature. Measures to be taken consist of warming the operating room before the patient comes in and maintaining this temperature until the patient is covered with drapes and warming blankets; prepping preoperatively and cleaning postoperatively with warmed solutions; avoiding cold intravenous fluids; and covering the patient with warm blankets at the end of a surgical procedure for transport to the post-anesthesia care unit.

Summary

- Homeostatic mechanisms deteriorate with aging; there is variability in this dysfunction.

- Changes in compliance of cardiovascular structures seem to be the primary defect in the CVS. The implication of this change affects many aspects of the circulation. There also seem to be some alterations within the autonomic nervous system. All these changes affect how elderly patients respond to anesthesia.

- The respiratory system undergoes both functional and structural changes with aging. These can be considered under 4 main headings: reduction in muscle mass and power, changes in compliance, reduction in diffusion capacity, and a decline in control of breathing. All of these changes have a profound influence on the response to anesthesia.

- Age-related changes take place in kidney structure, blood flow, and function. These renal changes have effects on the elimination of anesthesia drugs, and on water and electrolyte metabolism.

- Temperature control is impaired in the elderly. Anesthesia has a much more profound effect on temperature control in geriatric patients than in younger adults.
Quiz

1. Concerning changes in cardiac output (CO) with aging:
   a. Rises in CO with stress are solely due to increased heart rate.
   b. CO decreases by 10% per year above the age of 85 years.
   c. In healthy geriatric patients CO increases by 5% per year above the age of 65 years.
   d. In healthy adults there may be no significant change in cardiac function with aging.

2. With reference to changes in the peripheral vasculature:
   a. There is no significant change in the compliance of the major vessels with aging.
   b. Blood vessels become more compliant with aging.
   c. Only the arterial side of the circulation experiences significant changes in vasculature compliance.
   d. Both the arterial and venous sides of the circulation become “stiffer” with aging, and this is the main cause of the inability of elderly patients to maintain hemodynamic stability when receiving anesthesia.

3. Concerning cardiovascular autonomic function in the elderly:
   a. Tonic parasympathetic outflow increases, while overall sympathetic neural activity decreases.
   b. Elderly subjects generally manifest a greater responsiveness to beta-adrenergic stimulation.
   c. The administration of beta-adrenergic agonists elicits lesser inotropic and chronotropic responses.
   d. The effect of beta-adrenergic antagonists is impaired.

4. An 85-year-old man, with hypertension well controlled on beta-blockers and a mild diuretic, is undergoing a hernia repair using general anesthesia. There is a 200-cc blood loss, with no hemodynamic consequences. He is taken to the recovery room and is fully awake. The nurse sits him up in bed, and his blood pressure falls from 110/85 to 75/40, with no change in heart rate. Possible explanations of his hypotension include:
   a. Overdose of beta-receptor antagonist
   b. Excessive blood loss in the operating room
   c. Impairment of baroreflex responsiveness in the elderly
   d. Cardiac ischemia
5. Changes in the respiratory system in the elderly most closely represent which disease?
   a. Mild chronic obstructive pulmonary disease (COPD)
   b. Acidosis
   c. Myasthenia gravis
   d. Severe COPD

6. Please complete the text below using the given list of words. You can use each word or phrase once, more than once, or not at all.

   Dead space — 40 years — small airways — no change — increases in airway resistance — large airway — over 65 years — increase — expiratory flow

   In the lungs of people _________ of age, the _________ increases because of an _________ in _________ diameter. This is accompanied by _________ in airway resistance and _________ does not change. After the age of _________ the diameter of the _________ decreases, but again there is _________ in airway resistance.

7. Choose one or more correct answers. Above the age of 55 years:
   a. The respiratory muscles weaken.
   b. Chest wall compliance increases.
   c. Chest wall compliance decreases.
   d. Lung compliance increases.

8. Concerning renal function, which of the following are true?
   a. Twenty percent of renal mass is lost between the ages of 40 and 80, mostly from the cortex.
   b. Microscopically there is a reduction in the number of functional glomeruli, but the size and capacity of the remaining nephrons increase to partially compensate for this loss.
   c. Above 80 years of age the renal blood flow declines progressively at a rate of 10% per decade.
   d. Creatinine levels are an accurate reflection of glomerular filtration rate in the elderly.
   e. Glomerular filtration rate decreases by approximately 1 mL/min/year beginning by age 40.
9. Label the diagram below (A to E) using the following list:

- Functional residual capacity
- Closing volume
- Closing capacity
- Total lung capacity
- Residual capacity

10. Concerning perioperative hypothermia in the elderly, choose the correct statement:

   a. Body temperature regulation is unchanged in the elderly compared to younger adults.
   b. Corrective mechanisms for hypothermia (e.g., shivering, vasoconstriction) are only activated at lower body temperatures compared to younger adults.
   c. Geriatric patients both develop hypothermia and recover more quickly than younger adults.
   d. The adverse events associated with hypothermia (i.e., thrombocytopenia, increased rate of wound infections) are at least as severe in older patients as in their younger counterparts.

Congratulations! You have successfully completed this unit.