# Introduction to Geriatric Anesthesia

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## Background

The word “geriatric” was derived from the Greek language, in the early twentieth century; *“*Geron*”* which means "old man" and “iatreia” which means "the treatment of disease."

*Geriatrics* is a sub-specialty that focuses upon the care of elderly people.
*Geriatricians* are the physicians who take care of elderly patients.
*Gerontology* specifies the study of aging.
*Biogerontologists* usually limit their scope to the physiological and biochemical, rather than the socioeconomic aspect of aging.

## Concept of Aging

**Life span** is an idealized, species-specific biologic parameter that quantifies maximum attainable age under optimal environmental conditions. Historical anecdote suggests that human life span has remained constant at 110 to 115 years for at least the past 20 centuries. In contrast, **life expectancy** describes an empirical estimate of typical longevity under prevailing or predicted circumstances. Advances in medical science and health care have improved life expectancy dramatically in industrialized societies and increased their relative "agedness" but do not appear to have altered human life span. The mechanisms that control the aging process and determine life span remain unknown.

Physiological age is more important than chronological age because aging process varies from person to person and from one organ system to another within a given person, therefore, all elderly patients present in different physical condition. There is little correlation between chronological age and biological age. People achieve peak physiological function by late 20s or early 30s and from then on it is, in general, a “down-hill course”. It has been suggested that persons, who age with minimum impairment of physiological function are known to have “successful aging” whereas other persons, who have deterioration of physiological function, are labeled as having undergone “usual aging”.

## Theories of Aging

In general, theories of aging fall into two major categories. One group can be described as stochastic because it is essentially time- and probability-dependent. The nonstochastic group includes theories, which proposes, that there are programmed or predetermined mechanisms that explain aging. Nonstochastic theories of aging share a common theme of a **"biological clock" or “life pacemaker**” for each species. In order to effect processes of aging throughout the organism, the pacemaker tissue or organ must itself have widespread interaction with all other organ systems. Therefore, this type of theory usually involves a neuroendocrine or immune mechanism.

The "**error-catastrophe**" theory of aging is a stochastic concept. It postulates that random

errors of protein synthesis due to faulty nucleic acid transcription or translation eventually accumulate to compromise cellular function and produce the physical signs of aging. However, there is little evidence that the individual cells of older subjects contain more defective protein than do young cells.

Although there is a general correlation between species longevity and nDNA repair capacity, there is no firm evidence that the ability to recover from random nDNA damage is, in fact, progressively or universally compromised in older human subjects.

However, investigations of oxidative phosphorylation in aging mitochondria suggest that progressive increases in the incidence of defects within mitochondrial DNA (mDNA) may lead to a decline in bioenergetic capacity and a progressive reduction in the efficiency with which free radical species such as superoxide, routinely produced in the mitochondria during aerobic metabolism, are scavenged from the cytosol of aging cells. Free radicals damage the unsaturated fatty acid and nucleic acid components of cells and cross-link protein molecules, eventually damaging cellular microarchitecture.

A relatively recent proposal suggests that cellular aging is due to a "vicious cycle" of diffuse bioenergetic failure in the mitochondria of metabolically-active tissues. This mechanism, which may be thought of as progressive failure of a genetically-determined capacity to clear random damage to mDNA by free-radicals, is compatible with both stochastic and nonstochastic theories and falls within the larger evolving concept that aging is a consequence of a lifetime of "**oxidative stress"**.

## Geriatric Anesthesia: Introduction

Increased life expectancy and reduced mortality from chronic age-related disease continue to enlarge that fraction of the surgical patient population considered elderly.

Surgical procedures in the elderly will continue to require a disproportionately large share of societal and institutional health care resources. Routine postoperative hospitalization and intensive care, especially after major trauma, are frequently protracted and may be further complicated by infection, poor wound healing and by multiple organ system failure for critically ill elderly patients. Of equal concern are recent findings that postoperative cognitive dysfunction may persist at least three months after otherwise uncomplicated surgery.

Currently, individuals 65 years of age or older represent only 12 percent of the United States population, and undergo almost one-third of the 25 million surgical procedures performed Annually, they consume about one-third of all health expenditures and fully one-half of the $140 billion annual U.S. federal health care budget.

The elderly population is expected to grow to 24% by 2040. Therefore, every practicing anesthesiologist will eventually become a subspecialist in geriatric medicine, with a special responsibility for delivering cost-effective health care to older adults.

People are never more alike than they are at birth, nor more different or unique than when they enter the geriatric era. As with pediatric patients, optimal anesthetic management of geriatric patients depends on the understanding of the normal changes in physiology, anatomy, and

response to pharmacological agents that accompany aging. In spite of many similarities between older and pediatric patients, elderly patients show a wide range of variations in their parameters and have a high frequency of serious physiological abnormalities. Therefore, precise assessment and appropriate perioperative management of the elderly surgical patient represents a great challenge to all medical health care providers.

Similarities: between elderly people and infants, compared to the general population.

- Decrease ability to increase heart rate in response to hypovolemia, hypotension, or hypoxia.
- Decreased lung compliance
- Decreased arterial oxygen tension
- Impaired ability to cough
- Decreased renal tubular function
- Increased susceptibility to hypothermia

## Anesthesia Risks

Compared to younger patients, elderly patients may be at greater risk for perioperative complications and mortalities, due to the following reasons:

1. Effects of concomitant disease and decline in basic organ functions.
2. Effects of emergency procedure.

## Age Related Concomitant Diseases

Multiple concomitant diseases are the rule, rather than the exception, in elderly patients. Most common co-existing diseases are given below and several studies show the significant increase in incidence of diseases in patients who are 65 years of age or older. For instance, pathological findings were observed in 92% of patients 81 year of age or older. Of these, cardiovascular diseases and hypertension, were seen in 78% of patients. French survey of 198,103 of anesthetics, demonstrate that rate of anesthesia related complication correlates with patients age but relates more closely to number of preexistent diseases the patient present with.
The Goldman study of cardiac risk factors also suggest that preexisting medical condition is more important than a patient's age when one predicts the risks associated with anesthesia and surgery.

Cardiac Risk Index

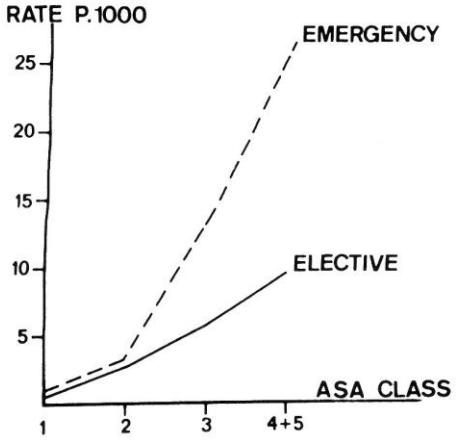
| CRITERIA | "POINTS" |
|---|---|
| Congestive heart failure | 11 |
| Myocardial infarction in previous 6 months | 10 |
| Rhythm other than sinus on ECG | 7 |
| Greater than 5 PVCs/min | 7 |
| Age greater than 70 yr | 5 |
| Emergency operation | 4 |
| General status | 3 |
| Intraperitoneal or intrathoracic operation | 3 |
| Valvular aortic stenosis | 3 |

(Goldman L, Caldera DL, Nussbaum SR *et al*: Multifactorial index of cardiac risks in non-cardiac surgical procedures. N Engl J Med 297:845, 1977.)

- Hypertension (essential)
- Ischemic heart disease
- Congestive heart failure
- Peripheral vascular disease
- Aortic stenosis
- Obstructive pulmonary disease
- Renal disease
- Diabetes
- Arthritis
- Dementia

## Effects of Emergency Procedures:

The risk of perioperative complication and death is greatly increased in elderly if surgery performed on an emergency basis. Gibson *et al* suggested that death is four times more frequent in elderly with coexistent diseases than healthy elderly and 20 times more frequently in those requiring emergency surgery. Possible explanation is, perhaps delayed seeking health care and may have more advanced pathologic conditions and also for emergencies allow very little time to optimize the preexisting condition. French study also shows that performing emergency surgery in elderly ASA III or greater significantly increases the risk of perioperative complication and death.




This figure shows complications Rate per 1000 anesthetics, related to the ASA physical status for elective and emergency surgical procedures.

## Pathophysiology of Aging

Processes of aging are usually distinguishable from age-related disease by the fact that they are universally present in all members of an elderly population and, in longitudinal studies of aging subjects, become progressively more apparent with increasing chronological age. Aging is a universal and progressive physiologic phenomenon characterized by degenerative changes in both the structure and the functional reserve of organs and tissues. It produces many physical manifestations due to reduced connective tissue flexibility and elasticity or the degeneration of highly structured molecular arrangements within specialized tissues.

The difference between maximum capacity and basal levels of function is organ system functional reserve, a "safety margin" available to meet the additional demands imposed by trauma or disease, or by surgery, healing and convalescence. Cardiopulmonary functional reserve, for example, can be quantified and assessed clinically using various exercise or maximal stress tests. However, there is at present no comparable approach to assessment of renal, hepatic, immune, or nervous system functional reserve. It is simply assumed that the functional reserve of these organ systems is reduced in elderly patients and that this is the mechanism by which the obvious susceptibility of elderly patients to stress- and disease-induced organ system de-compensation occurs.

### *Cardiovascular & Autonomic Nervous System and Aging:*

It is important to understand the differences between the changes in physiology that normally accompany aging and the pathophysiology of the diseases present in the elderly people. For

example, atherosclerosis is a pathologic disease, whereas reduction of arterial elasticity is caused by fibrosis of media of the vessel wall, is part of the normal aging process.

With advancing age, increasing arterial rigidity tends to elevate the systemic vascular resistance (SVR). Increased sympathetic nervous system (SNS) activity may also contribute to the increase in SVR, although this age-related change is controversial in its magnitude and importance. Hypertension in the elderly is characterized by a disproportionate increase in systolic pressure. In consequence, the left ventricle (LV) must work harder to eject blood into a more rigid aorta. This chronic strain eventually causes the LV to become hypertrophied. Also controversial is the degree to which aging is associated with decreases in cardiac output (CO) and stroke volume (SV) at rest. Decreases of upwards of 5% per decade have been described, but other studies show very little change with age.

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. Thus, aging can exaggerate the hypotension that results from blood loss, as well as from the peripheral pooling of blood with general or conduction anesthesia.

Increased stiffness of the (hypertrophied) elderly cardiac ventricle impairs diastolic filling causes a reduction in end-diastolic volume with an increase end-diastolic pressure. Severe diastolic dysfunction, caused by a stiffened ventricle, can be detected by Doppler echocardiography. In such cases, the elevated left ventricular filling pressures are reflected into the left atrium and the pulmonary vasculature and can lead to pulmonary congestion. Clinically important diastolic dysfunction likely involves poor ventricular relaxation in early diastole. In less affected elderly individuals, ventricular filling may be preserved without excessive increases in atrial pressure via the atrial kick to enhance late diastolic filling. Loss of the sinus rhythm, a common event during general anesthesia, may well depress cardiac output and arterial pressure more markedly in the elderly than it would in a normal younger patient.

The atrial enlargement predisposes the patient to supraventricular tachycardia, especially atrial fibrillation. LAE, which can develop as a consequence of diastolic dysfunction, is an independent predictor of coronary heart disease. These patients are at increased risk of developing congestive heart failure if IV fluid is administered too rapidly or in excessive volume.

On the following page, diagram A displays normal left ventricle diastolic filling recorded with a pulse Doppler.

Diagram B displays the left ventricle and left atrial inflow and pulmonary venous inflow, patterns that occur with age.

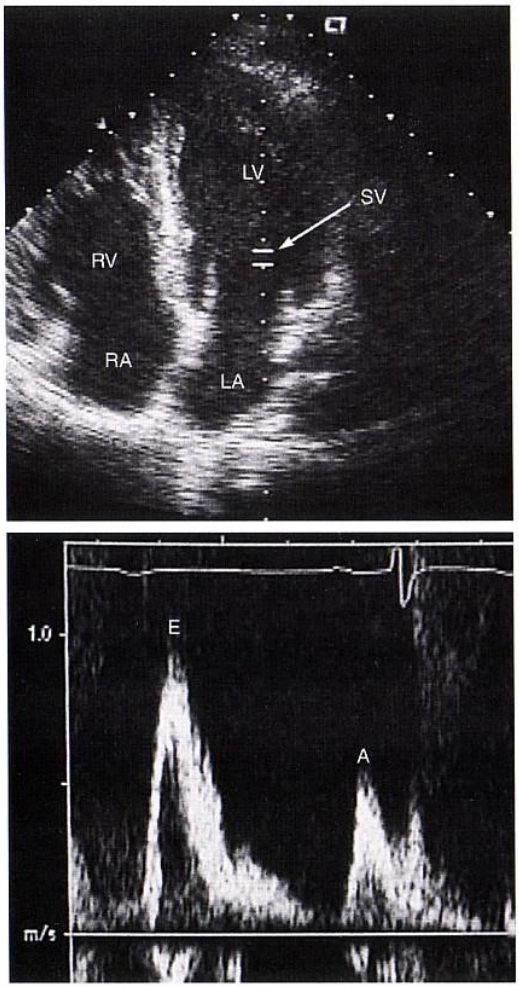
Table 1 shows the effect of age on diastolic left ventricular inflow pattern. In the age group 21-49, the majority of ventricular filling occurs in early diastole with prominent E velocity, and only a small contribution to ventricular filling due to atrial contraction (20% of total LV volume).

It can be determined from the table that with age, E velocity decreases and atrial contraction becomes more prominent, with equalization of E and A velocities at approximately age 60, and reversal of E/A ratio after that age in normal individuals. Early diastolic deceleration time also is

progressively prolonged with age, most likely due to gradual reduction in the rate of early diastolic relaxation.

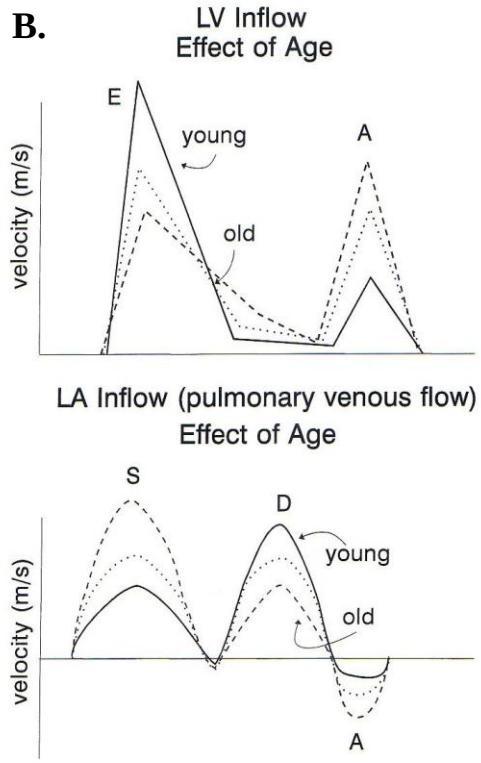
In patients with Diastolic Dysfunction (DD) impaired lusiotrophy limits rapid filling. The heart compensates by increasing the force of atrial contraction. With severe DD there are only two phase of LV filling: 1) slow passive filling of non compliant LV: and 2) atrial systole that can account for 50% of change in LV volume. Thus lower E/A ratio should describe DD well.

**A.**




Normal pattern of left ventricular diastolic filling recorded with pulsed Doppler in an apical four-chamber view. The sample volume is positioned at the mitral leaflet tips. In this normal young individual, the *E* velocity is high and the *A* velocity is low.

**B.**




Schematic diagram showing the changes in left ventricular *(top)* and left atrial inflow and pulmonary venous flow *(bottom)* patterns that occur with age. The typical pattern seen in younger (age <50 years) subjects *(solid lines)* is compared with middle-aged *(dotted lines)* and older (age >70 years) subjects *(dashed lines)*.

**Table 1**

**Effect of Aging on Parameters of Left Ventricular Diastolic Filling in Normal Subjects**

| | Mean (95% CI) | | |
|---|---|---|---|
| | **Age 21–49 y*** | **Age >50 y*** | **Age >70 y†** |
| *E*- velocity (m/s) | 0.72 (0.44–1.00) | 0.62 (0.34–0.90) | 0.44 (0.25–0.76) |
| *A*- velocity (m/s) | 0.40 (0.20–0.60) | 0.59 (0.31–0.87) | 59 (0.38–0.84) |
| *E/A* ratio | 1.9 (0.7–3.1) | 1.1 (0.5–1.7) | 0.8 (0.5–1.2) |
| Deceleration time (ms) | 179 (139–219) | 210 (138–282) | 140 (90–230) |
| IVRT (ms) | 76 (54–98) | 90 (56–124) | |

CI = Confidence interval, IVRT = isovolumic relaxation time.
*Data from Cohen GI, Pietrolungo JF, Thomas JD, Klein AL: A practical guide to assessment of ventricular diastolic function using Doppler echocardiography. *J Am Coll Cardiol* 27:1753–1760, 1996. Normal reference values were derived from 61 subjects age 21 to 49 years and 56 subjects over age 50 years.

In healthy young adults, the baseline autonomic tone is dominated by the parasympathetic system. With advancing age, tonic parasympathetic outflow declines, while overall sympathetic neural activity increases. However, elderly subjects generally manifest a reduced responsiveness to ß-adrenergic stimulation. Although resting heart rates do not change much with age, the maximal attainable heart rate, stroke volume, ejection fraction, cardiac output and oxygen delivery (DO2) are all reduced in healthy older adults. The administration of ß-adrenergic agonists elicits lesser inotropic and chronotropic responses in the elderly, while ß-blocking drugs retain their effectiveness.

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 DO2 are limited by aging.

The maintenance of hemodynamic homeostasis largely depends upon the baroreceptor reflex. Baroreceptors in the aortic arch and carotid sinus are actually stretch receptors; a decrease in distention of these receptors results in augmented SNS activity and inhibition of PNS 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.

Diminished cardiac reserve in many elderly patients may be manifested as exaggerated drops in blood pressure during induction of general anesthesia. A prolonged circulation time delays the onset of intravenous drugs but speeds induction with inhalational agents.

Although different individuals age in different ways and degrees, we can expect our older patients to require greater vigilance and more active interventions to guide them safely through surgery and anesthesia.

## Respiratory System

Because many of the intraoperative manipulations are performed by anesthesiologists, related to the respiratory system (e.g., assessing adequacy of patient ventilation, gas exchange, acid-base balance, delivery of inhalational agents), it is important for anesthesia providers to understand how aging affects the respiratory system. Knowledge of the age-related decrease in pulmonary capacity, combined with an understanding of the effects of the anesthetic process, will aid the practitioner in selecting appropriate supportive and prophylactic measures before and after surgery in the aged patient. With such information in hand, octagenarians and even centenarians should not be denied either elective or emergency surgery for fear of respiratory limitations.

Effect of the normal aging process on the respiratory system is a quite complex concept as it is difficult to separate the changes associated with age from those attributable to diseases of the aged.

Young adults can tolerate significant pulmonary morbidity during convalescence of major procedure; however, aging inexorably reduces the capacity of all pulmonary functions but the rate of loss of function is extremely variable among persons of the same chronological age. However, there are four hallmarks of the aging process:

1) Decline in elasticity of the bony thorax,
2) Loss of muscle mass with weakening of the muscles of respiration and reduced mechanical advantage,
3) Decrease in alveolar gas exchange surface and
4) Decrease in central nervous system responsiveness, which have anatomical, mechanical and functional consequences. There is very little information in the literature as to whether pulmonary mechanics under anesthesia are influenced by age.

There are a number of striking anatomic changes which occur in the respiratory system with age. As a consequence of a generalized loss of all muscular and neural elements (muscle fibers, mucosal receptors, nerve fibers, etc.), laryngeal structures undergo a slow but continual decline in function. Protective reflexes involved in the regulation of coughing and swallowing are diminished. The end result is chronic pulmonary inflammation from repeated aspirations with frequent contamination of the lower airway with oral and gastric organisms.

With aging, the larger and more central airways increase in diameter, as noted by an increase in anatomic and physiologic dead space. The trachea and large bronchi increase in size about 10 percent from youth to old age. Although beyond age 40, the diameter of the small airways which do not have cartilaginous support decreases significantly, the overall airway resistance does not appear to increase significantly. There is a small but measurable increase in dead space. Distally, there are more functional changes such as, the loss of elastic elements of the lung parenchyma and the dilation of respiratory bronchioles and alveolar ducts. When the alveoli become dilated, Kohn's pores become more numerous and larger and fine parenchymal tissue is lost with a loss of tethering support. 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 manifested functionally by air trapping, increased closing capacity, frequency-dependent compliance and gas exchange problems.

Parenchymal changes increases resting volume and the limitations imposed by a stiffer chest wall plus a decrease in motor power result in a change in the components of the total lung capacity. Vital capacity declines progressively with age. As a rough rule of thumb, there is a linear loss of 5 to 20 percent of functional ability per decade, which may be helpful in comparing an elderly patient's current capacity against normal values (Figure C). From age 20, vital capacity (VC) decreases progressively (-20 to -30 ml/yr) whereas residual volume (RV) increases (+10 to +20 ml/yr). In fact, the ratio of RV to TLC increases from 25 percent at 20 years of age to about 40 percent in a 70-year-old man, which gives the chest wall a somewhat barrel-like appearance. The total lung capacity (TLC) grows with age until puberty, where it reaches an average value of 6 to 7 liters, after which a slow loss of volume begins. With the age-related loss in total lung capacity (TLC), plus the very modest increase in FRC, the ratio of FRC to TLC tends to increase with age.

The reduction in motor power of the accessory muscles of breathing as well as the decreased expansion of the chest wall cause the dynamic lung volumes and capacities to decrease

progressively with age (e.g., FEV1). The FEV1 decreases with age by about 27 ml/yr in men but by only 22 ml/yr in women.

There is a clear age-related increase in the closing volume (CV) the volume of air at which small airways begin to close in dependent part of lungs and closing capacity ( CC), which is sum of CV and RV. Closing volume is the gas volume expelled during phase IV of the single-breath test and normally is approximately 10% of vital capacity, or 400 to 500 ml. (Fig. D) Even in normal persons, Closing Capacity exceeds Functional Residual Capacity (volume of air remaining in the lungs at the end of normal expiration) at age 45 in the supine position and age 65 in sitting position. When this happens especially postoperatively in recumbent position, some airways close during part of normal tidal breathing, resulting in mismatch of ventilation and perfusion.

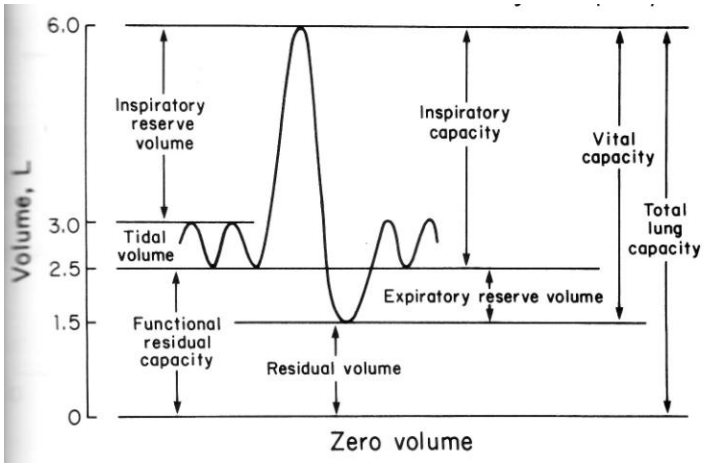



*Figure C.* Shows normal lung volume and lung capacity.

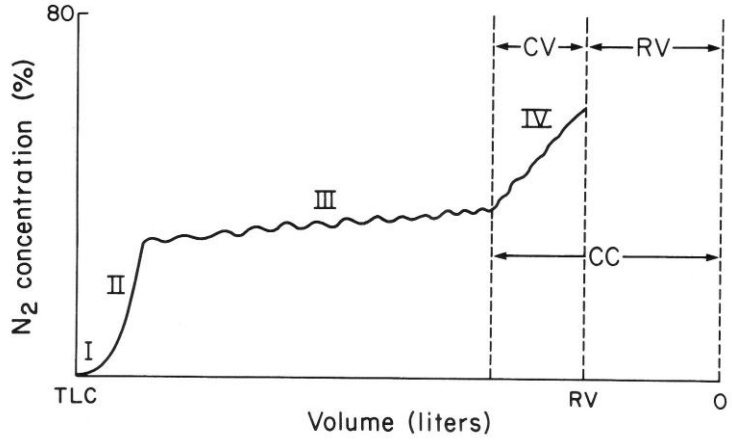



*Figure D.* Shows closing volume measurement by single breath, nitrogen test.

The flow-volume curve of an older lung is similar in shape (Fig. E, F), but shifted upward and to the left; in other words, the aged lung possesses less elastic recoil. This change in compliance is quite regional rather than being evenly distributed across the lung. The effect is to slow passive exhalation in some lung areas while other lung areas empty normally. The dynamic lung compliance (compliance measured during active breathing) becomes more frequency dependent with age. Thus as breathing rate increases, lung expansion is less effective particularly in some areas, thereby increasing the mal-distribution of ventilation to perfusion. During quiet breathing, inspired gases will preferentially go to the more distensible upper lung units leading to an uneven distribution of gases.

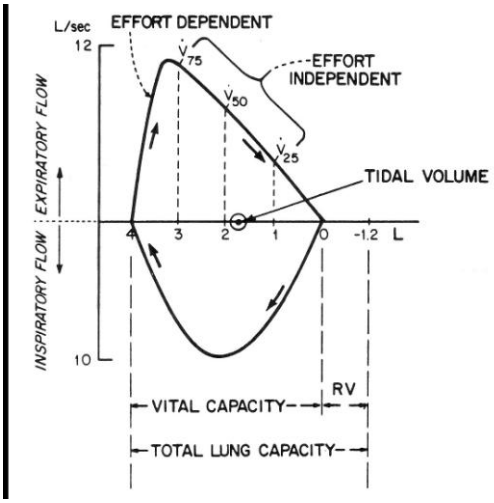



*Figure E.* Displays Flow-volume loop in normal subject.

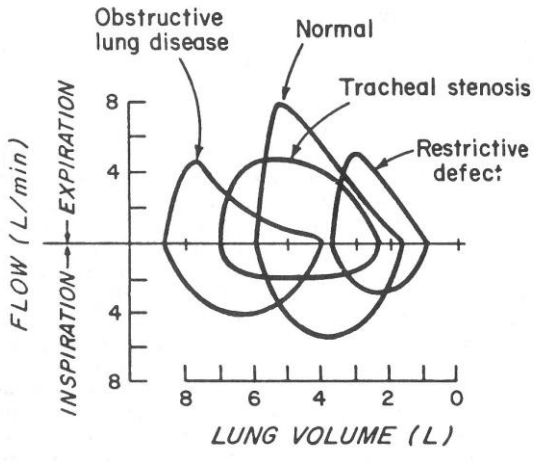



*Figure F.* Illustrates comparison between Flow-volume loop in normal subject and subject with pulmonary conditions.

The functional, or gas exchange capability, of the aged lung is affected by the anatomical and mechanical changes of age. The efficiency of alveolar gas exchange decreases progressively with age for a number of reasons. Alveolar surface area decreases with age from about 75 $m^2$ at age 20 years to about 60 $m^2$ at age 70 years.

Although blood volume does not change with age, the quantity of blood present in the pulmonary circulation decreases with age as well as distribution of pulmonary blood flow changes with aging. The change in blood flow and increase in alveolar dead space with age combined with the altered distribution of inspired gas, promotes even more V/Q mismatching which adversely affect the aged patient's blood gas values. A number of studies have demonstrated the mean PaO2 declines from 95 ± 2 mmHg at age 20 to 73 ± 5 at age 75 years. This decline in arterial oxygen tension is modest: approximately, 0.4 mm Hg/year. After age 75, however, PaO2 stays relatively constant at approximately 73 mmHg.

The efficiency of vascular distensibility and recruitment decreases with age. The increasingly rigid pulmonary vasculature probably blunts the hypoxic pulmonary vasoconstrictor (HPV) reflex. The loss of physical support of surrounding pulmonary elastic tissue surrounding both the small airways and pulmonary vessels may be a contributing factor. Thus the ability of the aged lung to respond to altered ventilation/perfusion matching is compromised.

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

In summary, the aged lungs have some but certainly not all of the features of chronic obstructive lung disease, e.g., increased RV and RV/TLC, reduced VC and FEV1 plus a

compliance that worsens as breathing rate increases. However, the fact that older patients have some of the features of chronic obstructive pulmonary disease (COPD) should not imply that they should be considered as having COPD.

The clinician should also realize that, together with these age-related decreases in reserve in the awaken state; the ventilatory responses to hypercapnia are reduced by narcotic premedication and by thiopentothal and narcotic and inhalational anesthetics in a dose-related manner.

Aged patients may be hypoxemic during normal spontaneous ventilation postoperatively because of the mechanical changes of the aged lung and chest wall. The risk is increased by the supine position and by the use of narcotic analgesics in an age group that already has blunted ventilatory reflexes to hypoxia and hypercapnia. Any residual anesthetic, delayed elimination of narcotics and muscle relaxants due to the impaired renal function and effects of large volumes of crystalloid infusion may also manifest in the recovery room.

Older subjects are less able to increase and maintain ventilation at high levels than young adults during periods of increased demand for oxygen. Ventilatory muscle fatigue is quite likely to occur early due to the altered physiology of voluntary muscle. Usually, older patients will develop ventilatory inadequacy earlier for any given ventilatory load and they complain of dyspnea therefore ventilatory impairment in the recovery room is more common in elderly patients.

The anesthetic technique and agents are of less importance than the degree of preparedness and the vigilance of the anesthesiologist taking care of elderly patients.

Finally, one should remember that, the overall age-related changes of the respiratory system essentially consists of a mix of restrictive and obstructive lung disease.

## **Renal Function**:

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

Between the ages of 40 and 80, the kidney loses approximately 20 percent of its mass, primarily from the cortex. 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. Vascular changes also occur in the aging kidney, and after the age of 30 years renal blood flow (RBF) declines progressively at a rate of 10 percent per decade. Most of the decline in RBF occurs in the cortex with a relative increase in blood flow to the juxtamedullary region. The glomerular filtration rate (GFR) decreases by approximately 1 ml/min/year beginning by age 40, normally 125 ml/min in young adult decreases to about 80ml/min at 60 years of age and 60ml/min at age of 80 years. However, this decline in GFR is accompanied by a gradual loss of muscle mass and is rarely associated with an increase in serum creatinine. Thus, serum creatinine is a poor indicator of GFR in the elderly patient while blood urea nitrogen gradually increases (0.2 mg/dL) per year. Dosing intervals for drugs that are excreted by the kidney, such as aminoglycoside antibiotics, digoxin and pancuronium need to be adjusted and drug levels closely monitored.

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. Elderly patient respond poorly to ADH and aldosterone. Glucose reabsorbtion is also decreased and are prone to develop hypo or hyperkalemia. Thus, fluid and electrolyte status should be carefully monitored in the elderly patient.

Renal dysfunction remains a serious complication during the perioperative period and is most likely to occur in critically ill patients undergoing major surgery. Typically, only after a patient has sustained renal injury, clinicians are focused on "renal protective strategies," and by then it is often too late. Despite significant advances in hemodynamic monitoring and hemodialysis during the past three decades, the mortality rate from acute renal failure has not changed significantly. Acute renal failure is in fact responsible for at least one-fifth of all perioperative deaths among elderly surgical patients. Perioperative renal failure accounts for one-half of all patients requiring acute dialysis. The precise mechanisms heralding the transition from compensated preserved renal function to uncompensated renal failure during the perioperative period remain poorly understood, in part because the methods used to assess renal function is insensitive and nonspecific. Acute tubular necrosis accounts for nearly 90 percent of the cases of perioperative renal failure.

## Central Nervous System:

Aging and age-related diseases are not the same. Those manifestations that are universally present in all elderly individuals and that increase in magnitude with advancing age, represent aging.

Effects of aging on the nervous system include:

1. Selective attrition of cerebral and cerebellar cortical neurons
2. Neuron loss within certain areas of the thalamus, locus ceruleus, and basal ganglia
3. General reduction in neuron density, with loss of 30 percent of brain mass by age 80
4. Decreased numbers of serotonin receptors in the cortex
5. Reduced levels of acetylcholine and acetylcholine receptors in several regions of the brain
6. Decreased levels of dopamine in the neostriatum and substantia nigra and reduced numbers of dopamine receptors in the neostriatum.

The association of serotonergic, cholinergic and dopaminergic systems, respectively with mood, memory, and motor function, may partially account for depression, loss of memory and motor dysfunction in the elderly.

## Afferentation

There is also a generalized reduction in afferentation, evident as progressively increased thresholds for virtually all forms of perception, including vision, hearing, touch, joint position sense, smell and peripheral pain and temperature.

## Sleep

Normal physiologic changes in sleep occur with advancing age, with probably the most common change being a decline in slow-wave, or delta, sleep. Delta sleep is thought to be the deepest level of sleep and perhaps the most restoring. Increased latency to sleep onset is often present, as well as increased awakenings and periods of wakefulness during the night. The timing of natural sleeping/waking cycles probably changes with age. In general, the usual bedtimes and awakening times of the elderly tend to occur earlier and are referred to as "sleep phase advancing". Two sleep disorders, sleep-disordered breathing (SDB) and periodic limb movements in sleep (PLMS), are commonly seen in the elderly.

## Memory

Memory and reasoning performance decline linearly with advancing age. Age-related decline in frontostriatal function, as supported by neuroimaging studies, most likely accounts for the majority of normal age-related decline in memory performance.

## Plasticity

Experience is the major stimulant of brain plasticity, which is the brain's ability to change structure and function. It is thought that an increase in dendritic growth and number of synapses with aging helps to compensate for the loss of neurons.

## Age-related Diseases

Age-related diseases such as cerebral arteriosclerosis, Alzheimer's and Parkinson's disease are all more common with advancing age. Most strokes affect those older than 70 years and the risk doubles every 10 years after age 55. The prevalence rates for dementia and Alzheimer's disease double approximately every five years from rates of 2 to 3 percent in the age category of 65 to 75 years to more than 30 percent in persons age 85 and older. Onset of symptoms in Parkinson's disease usually occurs between ages 60 and 69, although in 5 percent of patients the first signs are seen prior to age 40. About 1 percent of persons age 65 and older and 2.5 percent of those older than age 80 have Parkinson's disease.

## Postoperative Delirium

Postoperative delirium, a transient mental dysfunction, can result in increased morbidity, delayed functional recovery, and prolonged hospital stay for the elderly. The distinguishing features of this transient global disorder are impaired cognition, fluctuating levels of consciousness, altered psychomotor activity, and a disturbed sleep-wake cycle. It is usually seen on the first or second postoperative day and symptoms are often worse at night. The condition can be silent and go unnoticed, or it may be misdiagnosed as depression. Postoperative delirium

is defined as clinical situations in which patients think and speak incoherently, are disoriented and show impairment of memory and attention. The Mini-Mental Status Exam (MMSE) and other tests can assess speech, consciousness, perception, orientation, coherence, memory and motor activity. The MMSE is easy to conduct, reliable and can be used for serial testing in fluctuating conditions.

The reported incidence of postoperative delirium varies from 1 percent to 61.3 percent in different studies. One hypothesis for the mechanism behind postoperative delirium is a decrease in the oxidative metabolism of the brain, which results in the decline of neurotransmitter levels within the brain and causes mental dysfunction. Another hypothesis suggests that an increase of serum cortisol from the stress of surgery or anesthesia may be responsible for postoperative confusion.

Aging, pathologic states in the brain, polypharmacy and drug interaction, alcohol and sedative-hypnotic withdrawal, endocrine and metabolic problem, depression, dementia and anxiety, and gender are considered to be preoperative risk factors. Hypoperfusion and microemboli of air or blood cells in cardiac surgery, fat embolism in orthopedic surgical patients, regular use of anticholinergic drugs or drops and severe bilateral loss of vision in ophthalmologic patients may also contribute to the postoperative confusion. Anticholinergics, barbiturate premedication and benzodiazepines are implicated in the development of postoperative delirium. There is no difference in the effects of general, epidural or spinal anesthesia on postoperative confusion. Perioperative hypoxia, hypocarbia, and sepsis are also risk factors for postoperative confusion.

Preoperative assessment of the patient's physical and mental status and medications is very important. Pre-existing sensory or perceptual deficits compound a patient's chances of developing confusional states. The mainstay of intraoperative preventive measures is maintaining good oxygenation, normal blood pressure, correct drug dosage, and normal electrolyte levels. Drug cocktails should be avoided. Atropine, scopolamine, and flurazepam should be used only if necessary and the dose should be as low as possible. Glycopyrrolate may be a better choice than atropine as the former is a quaternary amine and should penetrate the blood-brain barrier less effectively than will atropine. earliest signs of delirium, which in the elderly may be withdrawal rather than agitation. The central nervous depressants, H2-antagonists, anticholinergics, digitalis, phenytoin, lidocaine and aminophylline should be used with discretion. In general, drugs with short elimination half-lives are preferable to long-acting drugs.

Once postoperative confusion has been diagnosed, the patient should be managed with extra vigilance. First, the underlying organic cause of the confusion should be found and treated. For acute control of delirium, doses of 0.25-2 mg oral haloperidol 1-2 h before bedtime is the preferred treatment.

A final concern that may be related to the phenomenon of postoperative delirium is the development of postoperative cognitive decline. Cognitive decline is not the same as delirium; patients who suffer cognitive loss are generally fully alert and oriented. A study that was performed with extensive neuropsychological testing, demonstrated significant decrements in cognition in 10% of the subjects at three months after surgery. Age was the only significant risk factor. It is unknown whether these changes are permanent and how to prevent them from happening.

## Endocrine Function:

Increasing insulin resistance due to aging leads to a progressive decrease in the ability to handle glucose load, emphasizes the importance of limiting glucose administration and monitoring glucose level perioperatively. In 1997 The American Diabetic association (ADA):

1. Eliminated the terms insulin-dependent DM and non-insulin dependent DM
2. Classified DM as either type I (destruction of insulin producing beta cells resulting in absolute lack of insulin or type 2 ( insulin present but impaired secretion or insulin resistance)
3. Lowered the diagnostic criteria for fasting serum glucose from >140 mg/dL to $\geq$ 126 mg/dL

## Metabolic and Thermoregulation:

Basal metabolic rate declines about 1% per year after 30 years of age, resulting in slowed metabolism of drugs and increased incidence of intraoperative hypothermia.
Normal autonomic responses to the decrease in core body temperature include vasoconstriction and shivering.

Elderly patients are unable to regulate their body temperature because their responses to changes in body temperature are altered. In general, geriatric patients neither vasoconstrict nor shiver in response to cold until their temperature has fallen to levels below that required for activation of these defense mechanisms in young adults. The relationship between impairment of thermoregulation and age is not linear and it does not occur in all aged patient. Younger patients will shiver at a temperature of 36.1°C; most patients over the age of 80 will not shiver until their core body temperature falls to 35.2°C, on average. Furthermore, the ability to vasoconstrict and reduce skin blood flow is also reduced with age, making obligatory heat loss in a cold environment greater than in young adults.

Anesthetics alter thermoregulatory responses in all patients. Hypothermia, in addition to being more pronounced, lasts longer in geriatric patients than it does in young patients. Recovery from mild hypothermia is accompanied by shivering in elderly patients. The shivering that does occur, though, is milder than it is in young patients. In elderly patients who shiver, body oxygen consumption only increases approximately 38% over non-shivering levels. Whether or not patients are shivering, there is an increase in their oxygen consumption that is proportional to the degree of hypothermia. Recovery from even mild hypothermia is prolonged in the elderly because their lower metabolic rate produces less heat.

Elderly patients are not immune to the adverse effects of hypothermia, which include bleeding, decreased immune function, and decreased wound strength. Bleeding is increased due to impaired platelet function and inhibition of the enzymes in the coagulation cascade. Decreases in temperature as little as 2°C will increase blood loss and transfusions. The vasoconstriction that accompanies hypothermia causes relative tissue hypoxia as less oxygen rich blood is brought to the vasoconstricted areas and the hypoxia results in decreased wound strength.

Hypothermia may exacerbate the decreased clearance of drugs in the elderly. This diminished clearance, accompanied by a decreased MAC in the elderly, means that anesthetic effects may be both pronounced and prolonged.

Elderly patients are more prone to have coronary disease than are younger adults. Hypothermia causes an increased incidence of myocardial ischemia in geriatric patients that is not related to shivering. Instead, ischemia is likely due to hypertension and increased plasma concentrations of norepinephrine. Consequently, it is not too surprising to note that hypothermia is associated with an increased risk of perioperative myocardial infarction.

The last major complication of hypothermia is an increased risk of infection. In a randomized study of colorectal surgery, patients assigned to routine care were almost 2°C colder at the end of surgery than patients who received aggressive intraoperative warming. Despite achieving normothermia in both groups by 6 hours postoperatively, the subjects receiving routine care suffered three times as many wound infections (19% vs. 6%) and remained hospitalized an average of two days longer than the patients who were more aggressively warmed.

As temperature regulation is altered in elderly patients, extra care must be taken to maintain their body temperature. This can be done by several consecutive measures, which include: warming the operating room until the patient is covered with drapes and warming blankets, prepping preoperatively and cleaning postoperatively with warmed solutions, not infusing cold IV fluids, and covering the patient with warm blankets at the end of a surgical procedure for transport to the post anesthesia care unit. Maintenance of temperature is extremely important as the elderly patients are more susceptible to all of the adverse effects of hypothermia, and it may be prolonged in this patient population.

## **Hepatic and Grastrointestinal Function**:

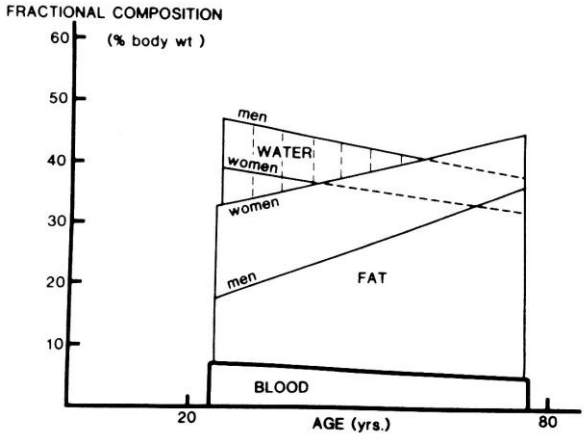
Liver mass declines as a person ages with a corresponding decrease in hepatic blood flow. Hepatic function declines in proportion to the decrease in liver mass. Thus the biotransformation and albumin production also decrease with age. Plasma cholinesterase levels are reduced. Gastric pH tends to rise, while gastric emptying is prolonged though gastric volume is low in elderly.

## **Airway Reflexes**:

Laryngeal, pharyngeal and airway reflexes are less active in elderly patients, making them prone for pulmonary aspiration.

## **Changes in Body Compartments**:

1 Loss of skeletal muscle mass and N M Jnc. are thickened. Skin is atrophied with age and prone to trauma from adhesive tape, ECG pads and Electrocautry pads.
2. Increase in body fat (volume of distribution increased for lipid soluble drugs)
3. Decrease in total body water (20-30% reduction in blood volume by 75 years of age, and a slowed rate of inter-compartmental clearance results in high initial plasma drug concentration)




This figure shows the changes in fractional body composition with age.

In conclusion, several studies have been published related to geriatric patients ranging from 65 to 115 years of age having undergone anesthesia and surgery and presently, with current understanding of medicine it is believed that, "elective surgery should not be deferred nor emergency surgery denied even for centenarians on the basis of chronological age". Therefore, it is very important to understand the relatively high frequency of serious physiological changes in elderly patients and evaluate them carefully preoperatively to minimize post-operative morbidity and mortality.

## Pharmacokinetics and Pharmacodynamics Differences in the Elderly

Pharmacokinetics has been described as what the body does to the drug, whereas pharmacodynamics has been described as what the drug does to the body. Pharmacokinetics factors include:

- The physiologic process of drug absorption (uptake)
- Tissue distribution
- Metabolism (primarily hepatic)
- Elimination (primarily renal)

The physiologic results produced by a drug concentration at the effector site describe the patient's pharmacodynamic response to a given drug.

Further details of pharmacokinetics and pharmacodynamics are beyond the scope of this presentation.

References:

1. Syllabus on Geriatric Anesthesia version 1/10/02
2. Clinical Anesthesiology, geriatric anesthesia, 3rd edition by G. Edward Morgan
3. ASA refresher course lectures 2007
4. Clinical Anesthesia by Paul Barash, 1992 edition
5. Textbook of Clinical Echocardiography 2nd edition, 2000 By Otto