Between 1960 and 1994, the population of those 85 years and older in the United States grew 274%. Similarly, the fastest-growing sector of surgical patients older than 65 years is those older than 85 years. These figures are critical because elderly persons have the highest mortality in the adult surgical population (5.8%-6.2% in those ≥80 years in 500 consecutive patients requiring general or regional anesthesia and 8.4% in those ≥90 years in 795 in-house operations). Why do elderly persons face such high surgical mortality rates? In addition to a higher incidence and prevalence of disease, elderly persons experience baseline physiological changes associated with senescence. It is vital for the modern surgeon to be aware of the physiological changes associated with aging to minimize morbidity and mortality in the aging surgical population.

Multiple theories of aging exist, all of which are controversial and largely unproved. In general, theories of aging are divided into either extrinsic (stochastic) or intrinsic (developmental-genetic) causes. The stochastic theories point to cumulative cellular damage from free radicals and radiation, errors in protein synthesis, and protein cross-linking. Developmental-genetic theories hypothesize intrinsic, preprogrammed, genetic control of cellular aging. The neuroendocrine and immunologic theory and the concept of aging genes fit into this latter category. Hayflick studied cellular senescence across multiple species, and noted that the maximum lifespan of a fibroblast varied among species. Specifically, there was a linear relationship between the maximum fibroblast population doubling and the maximum lifespan of a given species. Other cellular studies have introduced the concept of telomere shortening representing an internal cellular clock.

In this review, critical physiological changes of systems associated with aging are discussed. In addition, the blunted responses of each system in the face of perioperative stresses are examined.

ALTERATIONS IN CARDIOVASCULAR PHYSIOLOGICAL FEATURES

Normal physiological changes occur in the cardiovascular system with aging. There is a progressive loss of myocytes with a reciprocal increase in myocyte volume in both ventricles. The large vessels stiffen, as does the myocardium. As a result, afterload is increased and early diastolic filling is impaired. The β-adrenergic responsiveness of the heart decreases, limiting the maximum achievable heart rate (HR). In addition, the number of pacemaker cells in the sinus node decreases with age. The duration of myocardial contractility is lengthened, but force does not decrease significantly in elderly persons. The heart partially compensates for lower HRs and maximal left ventricular (LV) contractility with exercise-induced dilatation of the left ventricle. Despite these compensatory mechanisms, and because of the prevalence of cardiac disease, myocardial infarctions have been reported as the leading cause of postoperative death among 80-year-old patients.1
Cross-sectional echocardiographic studies\textsuperscript{17} suggest that LV wall thickness progressively increases with age independent of cardiovascular risk factors such as hypertension. Furthermore, a histomorphometric analysis\textsuperscript{18} has revealed that enlarging cardiac myocytes (hypertrophy) rather than an increase in number (hyperplasia) accounts for ventricular wall thickening; however, the absolute number of myocytes actually decreases over time. In addition, the local collagen concentration and its properties are altered in elderly persons. The number of collagen fibers increases along with increases in nonenzymatic cross-linking. Although the myocyte-collagen ratio remains relatively unchanged, this is mainly because of increased myocyte size; as a result, hypertrophy rather than hyperplasia is more prominent in elderly persons. These anatomical and structural changes contribute to an increase in myocardial stiffness and a decrease in compliance.

Previous reports\textsuperscript{17} have estimated that the LV early diastolic filling rate progressively decreases and reaches only 50\% of the peak rate by the age of 80 years. The LV end-diastolic volume does not reduce with age, but does mildly increase while at rest and during upright exercise.\textsuperscript{19} This is mainly because of increased atrial contraction during the latter phase of ventricular filling. The increased contractility of the atrium will lead to hypertrophy and enlargement of the chamber, which can be detected by an atrial gallop on auscultation.\textsuperscript{19} Although the LV ejection fraction ([LV end-diastolic volume - LV end-systolic volume]/LV end-diastolic volume) is relatively preserved with age, the maximum LV ejection fraction (the ejection fraction during exhaustive upright exercise) decreases. This is because of the dramatic reduction in ejection fraction reserve during aging.\textsuperscript{17} The stroke volume remains unchanged over time, and is achieved by increasing end-diastolic volume and maximally using the Starling curve.

The resting HR does not change with age, but the maximum achievable HR decreases, with the maximal HR that an 85-year-old person can achieve being approximately 70\% of that of a 20-year-old person.\textsuperscript{17} Because the stroke volume does not change over time, the maximum cardiac output (stroke volume \times HR) decreases during aging, indicating that the overall cardiac reserves diminish with age. The dysfunction of sympathetic modulation of the cardiovascular system with advanced age is consistent with increased spillover of catecholamine and impaired responses to \(\alpha\)-adrenergic receptor stimulation.\textsuperscript{20,21} This results in further reduction of the contractility of the myocardium.\textsuperscript{17} Aging also affects the LV afterload and vascular-ventricular load matching. This mismatch leads to failures of LV elasticity (contractility) in response to increased afterload.\textsuperscript{22} In addition, the diastolic pressure decreases with age, compromising myocardial perfusion and worsening overall cardiac function.

Normal aging affects the arterial system. Intimal hyperplasia and thickening, with a concomitant decrease in vascular compliance and increased stiffness, develop with advanced age.\textsuperscript{23,24} with the intimal thickness of the carotid artery increasing 2 to 3 times between the ages of 20 and 90 years.\textsuperscript{25} Multiple studies\textsuperscript{19,25} have demonstrated that intimal thickening is a risk factor for silent coronary artery disease, and it is, therefore, considered a component of subclinical vascular disease. Microscopically, there are alterations in the vascular media. Increased nonenzymatic collagen cross-linking, similar to collagen-associated changes in the myocardium, is seen. The elastin content in the media decreases with age. This contributes to decreased vascular elasticity (compliance) and increased stiffness. Pulmonary vessels of elderly persons have a lower elastin content and experience a decrease in collagen content by 1\% a year.\textsuperscript{26} Lower extremity arteries of patients older than 50 years commonly demonstrate medial calcifications and sclerosis.\textsuperscript{27} Dysfunctional endothelial vascular smooth muscle tone regulation (eg, a decrease in nitric oxide production with age) contributes to stiffer arterial walls independent of atherosclerotic changes.\textsuperscript{28,29}

Peripheral vascular resistance and central artery stiffness are 2 main determinants of arterial blood pressure. Increases in peripheral vascular resistance lead to an increase in systolic and diastolic pressure, while increases in central artery stiffness lead to an elevation in systolic pressure but a reduction in diastolic pressure.\textsuperscript{25} Although blood pressure in younger individuals is primarily dictated by peripheral vascular resistance, with aging central arterial stiffness becomes the main determinant of pressure.\textsuperscript{24} Investigators\textsuperscript{40} have demonstrated that systolic blood pressure increases in adults of all ages, well into the 80s, while diastolic blood pressure peaks in the 50s and subsequently declines. Thus, the overall effect of aging (disallowing for hypertension) is an increase in systolic pressure and a decrease in diastolic pressure, which manifests as a widened pulse pressure. The most common form of hypertension in adults older than 50 years is isolated systolic hypertension.\textsuperscript{31} Increased systolic pressure, although used as a screening variable for increased cardiovascular disease risk, is not as good a predictor of coronary disease as is increased pulse pressure.\textsuperscript{32,34}

**ALTERATIONS IN PULMONARY PHYSIOLOGICAL FEATURES**

Normal aging results in changes in pulmonary mechanics, respiratory muscle strength, gas exchange, and ventilatory control. Increased rigidity of the chest wall and a decrease in respiratory muscle strength with aging result in an increased closing capacity and a decreased forced expiratory volume in 1 second (FEV\(_1\)).\textsuperscript{35} The partial pressure of oxygen, arterial, decreases progressively with age because of the age-induced ventilation-perfusion mismatch, diffusion block, and anatomical shunt.\textsuperscript{36} Also, elderly patients have a diminished ventilatory response to hypercapnia and hypoxia.\textsuperscript{37}

The large airways grow slightly with age, but the resulting increase in dead space is insignificant.\textsuperscript{38} The respiratory bronchioles and alveolar ducts, on the other hand, increase in size significantly over time, particularly after the age of 60 years. The fraction of lung consisting of alveolar ducts increases progressively over time. As the alveoli grow, the cumulative surface area available for gas exchange decreases by 15\% by the age of 70 years.\textsuperscript{39}

Changes associated with aging result in a diminishing pulmonary elastic recoil pressure.\textsuperscript{40} The fusion of adjacent alveoli, which occurs with aging, decreases sur-
face tension forces and pulmonary elastic recoil. In addition, chest wall stiffness increases with advancing age, decreasing compliance. Chest wall stiffness increases because of the calcification of intercostal cartilages, arthritis of the costovertebral joints, and gradual atrophy and weakening of the intercostal muscles with advanced age. A patient with kyphosis or osteoporosis has an even more significant decrease in chest wall compliance. Elastin fiber concentration surprisingly increases with age, and is not believed to contribute to the age-related decrease in lung elasticity.

Gradual atrophy and weakening of the intercostal muscles during aging demands greater contributions from the diaphragm and abdominal muscles for breathing. Unfortunately, the strength of the diaphragm declines with age, as measured by the maximum transdiaphragmatic pressure.

Pulmonary function test changes are most affected by the age-related decreased compliance of the pulmonary system and muscle strength. Burr et al. showed that the FEV₁ and the forced vital capacity decline progressively with age. Knudson et al. estimated that the FEV₁ decreases by 30 and 23 mL/y in nonsmoking men and women, respectively, with an even greater decrease after the age of 65 years. This is equivalent to an 8% to 10% decline in FEV₁ each decade. The forced vital capacity in nonsmokers is estimated to decrease about 15 to 30 mL/y. The vital capacity progressively decreases and the residual volume gradually increases, leading to a relatively unchanged total lung capacity (vital capacity + residual volume). The functional residual capacity also increases with age, although it is not as significant because of the counteraction of the increased stiffness of the chest wall.

Immunohistochemical staining of type IV collagen in alveoli showed increased thickness of the alveolar basement membrane. Thickening of the alveolar basement membrane decreases gas-diffusing capabilities. The diffusing capacity of the lung is decreased about 2.03 mL/min per millimeter of mercury in men per decade and 1.47 mL/min per millimeter of mercury in women per decade. The resulting ventilation-perfusion mismatch leads to higher alveolar-arterial oxygen gradients. The partial pressure of oxygen, arterial, decreases during rest and exercise with age, becoming more prominent during exercise.

Collectively, the normal aging process changes the anatomical structures and tissue properties affecting respiratory physiological features in several aspects. Most important, expiratory flow rates decrease with age, as does the partial pressure of arterial oxygen. Both of these functional variables have been reported as risk factors for pulmonar...
are responsible for the most common age-related colonic disorder, diverticula. Functional alterations in secretion and absorption are predominantly found in the stomach (secretion) and small bowel (absorption), although it is difficult to consistently link these age-related changes with pathological features.

Changes in Neuromuscular Function

Changes in function with aging of the oropharynx and esophagus are primarily related to neuromuscular degeneration and subsequent alterations in the ability to coordinate the complex reflexes that lead to successful swallowing and propulsion of food along the esophagus. Aberrant contraction can also be caused by weakness in the muscles. Failure to coordinate the reflexes regulating proper motility can lead to numerous pathological features, including diffuse esophageal spasm, achalasia, and reflux. Because these age-associated problems with esophageal motor function can also be caused by primary neurological conditions, it is especially important to delineate deficits in neurological function caused by cerebrovascular accidents and true central nervous system degenerative processes from age-related changes.

The cricopharyngeus muscle, the primary muscle of the upper esophageal sphincter, is particularly susceptible to alterations in motility and may lead directly to problems such as aspiration, dysphagia, and pharyngoesophageal diverticula. Neuromuscular deficits of the lower esophagus include a decreased or even absent response to normal upper esophageal peristalsis, with a general weakness and slowing of contractions. Insufficient resting pressure in the lower esophageal sphincter can lead to gastroesophageal reflux and symptoms simulating hiatal hernia and achalasia.

The other portion of the gastrointestinal tract thought to experience altered neuromuscular function with age is the stomach. However, the exact effect that aging has is unclear. Studies investigating the gastric emptying times of young and elderly patients have demonstrated increases and decreases in the rate of emptying. Furthermore, it has been challenging to link any alteration in gastric emptying with patient complaint or pathological features. At best, these data suggest that there may be perturbations in the homeostatic mechanisms regulating gastric emptying in elderly persons. The small bowel has been demonstrated to have unaltered rates of transit.

Changes in Gastrointestinal Wall Structure

The upper alimentary tract does not experience significant structural changes as age progresses. As mentioned previously, weakening of the musculature of the oropharynx and upper esophagus can contribute to the formation of pharyngoesophageal diverticula and can cause functional problems. Similarly, although the gastric mucosa becomes atrophic with age, correlations between histologic change and disease processes, including atrophic gastritis, have been hard to establish. The primary structures affected by age in the small bowel are intestinal villi. Starting around the age of 60 years, there is a progressive decrease in the height of the villi, with a concomitant decrease in surface area available for absorption. Fibrous connective tissue can also be seen replacing normal mucosal parenchyma and smooth muscle cells.

The colon is the most consistently affected portion of the gastrointestinal tract with regard to age-related structural alterations. Mucosal changes are noted, but do not affect the absorptive capabilities of the colon. The primary process seen in the colon is a thickening of the muscular layers, particularly the muscularis propria and the muscularis mucosa. This thickening is due to elastogenesis and a buildup of elastin between myocytes in the bowel wall, not due to muscle cell hyperplasia or hypertrophy (although these may be caused by inflammation and fibrosis). Thinning is present throughout the life cycle, but becomes more rapid after the age of 60 years. The tinea coli are affected more than the circular muscle layers, and it is contraction in the longitudinal direction secondary to elastin accumulation that contributes to hard stool, constipation, and fecal impaction. Diverticular disease, the most common age-related colonic disease, occurs as a result of concomitant weakening of the muscularis propria at locations where arteries and veins cross the bowel wall.

Changes in Secretion and Absorption

It is a common misconception that age-associated xerostomia is due to a primary deficit in the production of saliva. Although the composition of saliva is altered in elderly persons (they have higher levels of mucin, resulting in a more viscous secretion), overall production and flow rates are normal. Diminished salivation is almost always attributable to secondary factors, such as medication. Secretion of gastric acid and pepsin declines with age. This is more noticeable in women, and is manifested as an attenuation of peak and basal acid secretion. However, serious problems, such as achlorhydria, are not usually attributable to age alone and, thus, the underlying pathological features in patients with this type of condition must be determined. Decreases in pepsin secretion correlate with those patients in whom age-related mucosal atrophy is present.

In the small bowel, a decrease in the height of villi and a reduction in surface area can lead to disorders of absorption. Specifically, absorption of calcium, carbohydrate, and D-xylose is impaired, although this may be inconsistent and may not be clinically significant. There are no significant changes in the absorptive capacity of the colon attributable to age.

ALTERATIONS IN HEPATOBIARY PHYSIOLOGICAL FEATURES

Several changes in liver physiological features occur with aging. The size of the liver decreases after the age of 50 years, declining from roughly 2.5% of total body mass to a nadir of just more than 1.5%. Alterations in blood flow parallel this decrease. Interestingly, although the total number of hepatocytes in an aged liver is decreased, there is an increase in the mean cell volume, which some have interpreted as a cellular response to an increased
biological demand on the remaining cells. Despite this potential increased demand, most liver function test results remain normal in elderly persons, as do standard function test results, such as those for hepatic filtration, detoxification, ethanol elimination, and conjugation.\textsuperscript{68,77} Hepatic synthesis of several proteins, including clotting factors, can be reduced, although this does not impair baseline function. However, perhaps because of the larger anabolic burden placed on fewer hepatocytes, hepatic synthesis of these factors is unable to increase significantly beyond baseline when challenged.\textsuperscript{78}

No direct link between aging and major gallbladder function has been established, including absorption, mucosal physiological features, and contractile properties. Although there is an increase in the incidence of cholelithiasis in elderly persons (theoretically attributed to an increase in the ratio of lipid-cholesterol in bile), the underlying process by which this occurs has not been elucidated.\textsuperscript{68,79}

\section*{ALTERATIONS IN ENDOCRINE, IMMUNE, AND STRESS RESPONSES}

Age-related changes that occur in various endocrine functions have been well documented in the literature. Perhaps the best studied of these include postmenopausal changes, resulting in an estrogen-deficient state. Menopause is characterized by a cessation of menses and a decline in estrogen levels with a concomitant increase in luteinizing hormone and follicle-stimulating hormone levels.\textsuperscript{80} During the first decade after the initiation of menopause, women undergo rapid bone loss, most likely reflecting diminished estrogen levels. Thereafter, a slow phase of bone loss occurs, primarily from a loss of estrogen-mediated calcium homeostasis.\textsuperscript{81} The decrease in skeletal mass associated with menopause occurs in conjunction with normal age-related bone loss, further complicating the problems of osteoporosis and pathological bone fractures in elderly persons. In addition to its effects on bone, menopause removes estrogen's cardioprotective effect, increasing low-density lipoprotein level, decreasing high-density lipoprotein level, and increasing overall atherogenesis.\textsuperscript{82} Further effects of low estrogen levels experienced in menopause include changes in vasomotor symptoms, urogenital atrophy, increased mood swings, and loss of libido.\textsuperscript{83,84} Hormone therapy alleviates many of these estrogen-deficient symptoms, although well-known risks have been documented, including increased risks of venous thrombosis, stroke, and breast cancer.\textsuperscript{85-87}

In men, serum testosterone, estradiol, dehydroepiandrosterone, and dehydroepiandrosterone sulfate levels slowly decline with advancing age, while sex hormone–binding globulin, luteinizing hormone, and follicle-stimulating hormone levels increase.\textsuperscript{88} The decrease in sex hormone–binding globulin further decreases the level of free active testosterone. Low testosterone levels are associated with decreased libido, decreased hematocrit, muscle atrophy, osteoporosis, and possibly erectile dysfunction.\textsuperscript{89} In older men with testosterone-related hypogonadism or markedly low levels of testosterone, testosterone replacement has been shown to improve sexual drive, increase lean body mass, and possibly improve exercise-induced coronary ischemia.\textsuperscript{90} The benefits of testosterone supplementation for older men with normal to low-normal levels of testosterone without clinical signs of hypogonadism are less clear. Overall, the long-term effects of testosterone therapy are inconclusive and warrant further investigation.\textsuperscript{90-92}

In terms of pituitary function, an age-related decrease in growth hormone and its anabolic mediator, insulin-like growth factor 1, may be associated with the decreased lean body and bone mass and increased percentage body fat observed in aging persons.\textsuperscript{93-95} Rather than a defect in growth hormone release from the pituitary gland, changes in growth hormone–related regulatory hormones, such as growth hormone–releasing hormone and somatostatin, seem to mediate the age-related decrease in growth hormone.\textsuperscript{96} As for other pituitary hormones, baseline prolactin levels are similar as individuals age, although the increase in prolactin levels in response to mild surgical stress (eg, inguinal hernia repair) seems to be blunted in older individuals.\textsuperscript{97}

For nondiabetic individuals, progressive impairment of glucose tolerance occurs with advancing age, independent of obesity and sex.\textsuperscript{98} A previous investigation\textsuperscript{99} comparing the response of older (those in their 80s) with that of younger (those in their 20s) individuals to oral glucose tolerance tests reported a 45\% rate of impaired tolerance in older individuals. The pathogenesis of this age-related decline in glucose handling seems to result from increased insulin resistance rather than an impairment of insulin secretion.\textsuperscript{98} Possible explanations for this age-related insulin resistance include a decrease in fat-free mass, dietary and physical activity changes, neurohormonal changes, and a decreased capacity of the glucose uptake system.\textsuperscript{98,99} Overall, there is an age-related increase in fasting and postprandial glucose levels.\textsuperscript{83,100} As for enzymatic function of the pancreas, pancreatic lipase may be mildly decreased, possibly accounting for the slight impairment of fat absorption observed in elderly persons.\textsuperscript{98}

The anatomical features and function of the thyroid also undergo age-related changes. The thyroid gland in elderly persons is characterized by mild atrophy, increased fibrosis, and decreased size of the follicles.\textsuperscript{101} Functionally, there is less peripheral conversion of thyroxine to triiodothyronine, decreased uptake of iodine, and overall lower levels of thyroxine and free thyroxine.\textsuperscript{98} However, whether the age-related decline in thyroid function translates to clinical relevance is unclear. Similar to the thyroid, the neighboring parathyroid glands also undergo age-related changes. Several studies\textsuperscript{102} have demonstrated an increase in parathyroid hormone level with age and increased parathyroid hormone release in response to serum calcium compared with younger individuals. The increased baseline levels and augmented response of parathyroid hormone to stimuli have been implicated in osteoporosis and bone loss in elderly persons.\textsuperscript{103} In contrast to parathyroid hormone, calcitonin levels seem to decrease with age. Interestingly, adrenal function in advanced age leads to changes in the diurnal pattern of cortisol, which shifts earlier in the day and produces higher evening cortisol
CD18 expression) have been elevated after surgical stress though, are strongly associated with neuronal loss.115-117 Cerebral blood flow and cerebral oxygen consumption have been shown to decrease with age, particularly in areas with decreased gray and white matter.124-126 This increases the risk for cerebrovascular accidents with associated vascular disease. Vision, auditory function, and vibratortile sensation are blunted with age.121 The receptor cells of the retina, rods, and cones have been reported to undergo changes with aging.127 Stiffening of the tympanic membrane and sensory loss of the cochlea are a few examples of changes in the auditory system.129 Decreased mechanoreceptor density and sensitivity, and decreased peripheral nerve conductivity, have been shown in elderly patients with decreased vibratortile sensation.129 Loss of labyrinth hair cells, nerve fibers, and vestibular ganglion cells has been shown to affect the vestibular-ocular reflex in elderly persons.130 Decreased tactile and proprioceptive sensation has been reported in elderly persons.128,131,132 Kokmen et al132 reported decreased joint motion sensation in healthy aged individuals. They found a decrease in reflex time with age, but no difference in nerve fiber numbers.132

NEUROLOGICAL CHANGES

Traditional theories of normal neuronal loss with aging have been challenged.113 Histological studies114 of brain specimens from subjects without dementia or other cerebral pathological features have shown minimal neuronal loss with normal aging. Neurodegenerative processes (Alzheimer, Parkinson, and Huntington diseases), though, are strongly associated with neuronal loss.115-117 Cortical atrophy has been demonstrated to progress with age.118-120 A 6% to 11% loss of brain weight has been demonstrated in subjects older than 80 years.121 Coffey et al119 performed quantitative magnetic resonance imaging studies of the brain in subjects matched by Mini-Mental State Examination score and found statistical decreases in cerebral hemisphere volume, frontal region area, temporoparietal region area, and parieto-occipital region area with age. Increased lateral ventricle and third ventricle volumes in elderly persons were also shown in the same study.119 Cortical atrophy is said to precede neurodegeneration, if dementia has not already ensued.121 In patients with Alzheimer disease, Mouton et al122 have demonstrated a 20% to 25% greater cortical atrophy compared with age-matched control subjects. Interestingly, higher levels of education have been shown to protect against dementia.123 This concept is known as the reserve hypothesis.

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The autonomic neural responses are blunted in elderly persons. Collins et al133 found healthy elderly subjects to have significantly diminished beat-to-beat variation in response to postural change, decreased vasconstrictor response to cooling, and reduced baroreflex sensitivity. Peripheral vascular resistance is maintained with postural changes in healthy community-dwelling elderly individuals, despite blunting of the baroreflexes with age.134,135 Orthostatic intolerance is far more common in debilitated patients older than 70 years who are institutionalized for the long term.136 Pfeifer et al137 reported an age-related increase in cardiovascular sympathetic activity and a decrease in cardiac parasympathetic activity. Brodde and Michel138 attribute the diminished sympathetic tachycardia response and the diminished vagal bradycardia response of the aging heart to the reduced responsiveness of α-adrenergic and muscarinic receptors, respectively. Thermoregulatory mechanisms become less responsive with age, predisposing elderly persons to hypothermia in cold environments.83,139-141 The 2 primary responses to a cold challenge, vasoconstriction and shivering, are less effective.141,142 After a cold isotonic sodium chloride solution infusion in elderly patients, Frank et al135 reported a decreased maximum response intensity for vasoconstriction, total body oxygen consumption, and norepinephrine. In addition, decreased vasomotor responsiveness to norepinephrine and subjective sensory thermal perception were noted.143 Elderly persons have an increased threshold for pain.144 Adequate pain control is critical to minimize the risk for myocardial ischemia, tachycardia, hypertension, and pulmonary complications. In addition, effective analgesia encourages early patient mobilization, shortens hospital stays, and decreases medical costs.83 At the same time, caution must be taken not to overmedicate.
elderly persons. Overmedication with various agents may lead to hypoxia, hypercapnia, hypotension, or delirium. Agents with shorter elimination half-lives are recommended.145

In summary, cerebral atrophy lowers the threshold to neurodegeneration in patients. The most common postoperative neural complications are cerebrovascular accidents and delirium. Perioperative insults, such as hypotension, hypoxia, hypothermia, and malnutrition, pose a greater risk to the central nervous system of elderly persons compared with young persons. The blunted sympathetic and parasympathetic responses to stimuli result in more moderate responses to stress and typically longer recovery times to baseline functionality.

CONCLUSIONS

Elderly persons experience normal physiological changes associated with aging in all of their solid organ systems. Together, these changes lead to a diminished physiological reserve. Patients with advanced age have the highest mortality rate within the adult surgical population.3 A major risk factor for perioperative mortality is, therefore, considered to be advanced age.146 The effects of postoperative stresses are more detrimental to some organ systems than others. Myocardial infarctions have been reported as the leading cause of postoperative death among 80-year-old patients.4 Among patients older than 60 years, the incidence of postoperative myocardial infarction after noncardiac surgery is reported to be 0.1% to 0.15%, with a subsequent mortality of 50% to 83%.147 Mortality secondary to pulmonary complications is reported as between 0% and 0.6%, depending on risk factors.148,149 Pulmonary complications, including pneumonia, hypventilation, hypoxia, and atelectasis, are reported to occur in 2% to 10% of elderly patients.3 Cerebrovascular-related mortality in elderly patients undergoing urological procedures was reported to be 0.05%.3 Postoperative delirium varies between 5% and 61%, but only 1% of patients have persistent symptoms.130,131

The perioperative risk for mortality is heightened in the postoperative period.3 The postoperative period is physiologically most stressful, because this is when there are the greatest shifts in fluid, body temperature, adrenergic activity, and pulmonary function.132 Thorough preoperative assessment of organ function and reserve, intraoperative control of disease, close postoperative monitoring, and pain management are recommended to effectively decrease perioperative mortality.31

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