Effect of Aging on Cardiac Function Plus Monitoring and Support

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Martin D. Avery, R. Shayn Martin, and Michael C. Chang

Key Points

- Elderly surgical patients are common and often require evaluation and support of the cardiovascular system.
- Aging significantly impacts ventricular and vascular anatomy resulting in altered cardiac functionality.
- Physiological changes of aging include a blunted baroreflex and altered beta-adrenergic responsiveness resulting in a decreased dependence on chronotropy and an increased reliance on stroke volume in response to stress.
- Monitoring of the elderly cardiovascular system is valuable and can be achieved in a number of noninvasive and invasive methods.
- Management of shock in the elderly benefits from an understanding of the needs of the specific patient and a recognition of the risks/benefits of each cardiovascular medication.

Introduction

As the world's population continues to grow, advanced age has become an increasingly important risk factor influencing morbidity and mortality. It is estimated that in the United States alone, approximately 20 % of the population will be over the age of 65 by the year 2030 [1–3]. As the fastest-growing population, specialized attention to the physiology of these aging patients is paramount to the successful treatment of the elderly. Specifically, cardiovascular disease remains the most prevalent and influential comorbidity affecting outcomes in the elderly surgical patient. Half of all heart failure cases in the United States are older than 75, and 90 % of heart failure deaths occur in adults older than 65 [4]. Heart failure is also the leading cause for hospitalization in Medicare beneficiaries. With advances in the care of chronic diseases and longer life expectancy, familiarity with the effects of aging and how to treat elderly patients in all fields of medicine is required to successfully treat this population. The unique physiology of the aging cardiovascular system as well as the impact of these changes during the stress of surgery is outlined in Table 2.1. Understanding these changes and their implications to the treatment of the elderly patient will improve care and outcomes in this population.

Effect of Aging on the Right Ventricle

The right ventricle is connected in series to the left ventricle and is therefore obligated to pump the same stroke volume. As the cardiovascular system ages, this relationship is not always maintained, and right heart flow may not always equal left heart flow. Radio-nucleotide studies and echocardiography have demonstrated impairment in both systolic and diastolic right ventricular function. The mechanism for this reduction is believed to be secondary to a gradual age-related increase in pulmonary arterial vascular resistance, clinically evident by increased pulmonary artery systolic pressures [3]. Using M-mode echocardiography in combination with Doppler technology, right ventricular impairment is demonstrated by observing a reduction of tricuspid annular plane systolic excursion. The tricuspid annular plane systolic excursion (TAPSE) estimates the longitudinal contractile properties of the right ventricle. These modalities demonstrate a significant reduction in TAPSE in otherwise healthy subjects as they age. Pulsed tissue-derived measurements of right ventricular systolic function have confirmed these findings agreeing with findings of older studies demonstrating reduced systolic function on echocardiography.

M.D. Avery, MD • R.S. Martin, MD (✉) • M.C. Chang, MD
Department of Surgery, Wake Forest School of Medicine, Medical Center Boulevard, Winston-Salem, NC 27157, USA
e-mail: mavery@wakehealth.edu; romartin@wakehealth.edu; mchang@wakehealth.edu

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Table 2.1 Summary of the effect of aging on the cardiovascular system

Cardiovascular element	Alteration in the elderly
Right ventricle	Reduced systolic function Reduced diastolic function
Left ventricle	Left ventricular hypertrophy Dependence on atrial contribution Age-related impaired contractility and relaxation
Vascular structures	Increased arterial stiffness Systolic hypertension
Cardiac output	Preserved resting cardiac output Preserved ejection fraction
Changes in physiology	Blunted baroreceptor reflex Decreased adrenergic responsiveness
Response to stress	Decreased reliance on heart rate Increased cardiac output due to increased stroke volume

Inefficient rotational motions and non-longitudinal muscular movement contribute to the age-related decrease in right heart systolic function.

The aging process also affects right heart diastolic function. Diastolic functional properties can be characterized by determining right atrial pressure (RAP), tricuspid inflow velocity (E), myocardial early diastolic velocity (Ea), and atrial peak velocity (Aa) [5, 6]. Age is significantly correlated with progressive increases in Aa and decreases in Ea. Additionally, there is a negative relationship between the Ea/Aa ratio and increasing age, indicating less filling velocities in the ventricle despite higher atrial velocities [5, 6]. In the same way that systolic functional decline is attributed to increasing stiffness of the pulmonary vasculature, diastolic functional changes are attributed to increased right heart afterload [5].

Effect of Aging on the Left Ventricle

Years of ongoing stress on the heart result in changes in cardiac function related to increased workload. As aging blood vessels stiffen leading to elevated systolic blood pressure, the left ventricle (LV) changes in response. The heart is required to perform greater amounts of stroke work (stroke volume times blood pressure) in the presence of sustained elevations in systolic pressure resulting in LV wall thickening in elderly patients [7]. Structural changes observed on cardiac MRI demonstrate a significant increase in myocardial thickness as a result of increased cardiomyocyte size. The overall shape of the heart also changes from an elliptical to spheroid shape with asymmetric increase in the intraventricular septum as opposed to free wall hypertrophy [8, 9].

Resting diastolic filling rates decline with age as evidenced by studies utilizing M-mode echocardiography and gated blood pool scans. Diastolic filling of the ventricles occurs in passive and active phases. As individuals age, the heart fills more slowly, and the bulk of ventricular filling shifts to late diastole, with less passive filling. As a result, ventricular filling during diastole becomes more dependent on the active phase. Atrial enlargement is observed as atrial contraction contributes more and more to ventricular filling [8].

Studies using cardiac MRI have investigated LV structure and function. These studies have demonstrated the development of LV hypertrophy and fibrosis leading to diastolic dysfunction and heart failure with preserved systolic function. Indices of cardiac function (such as ejection fraction and ejection velocity) were preserved despite reductions in both LVEDV and LVESV [10]. It appears that modest hypertrophic changes in the left ventricular wall are adaptive to preserve cardiac function at rest. However, exercise capacity is reduced. Fibrotic cardiac remodeling plays an important role in the development of diastolic heart failure with age, and adaptive changes to maintain cardiac output play a pivotal role in senescent cardiac function [9, 11]. These adaptations of the aging LV to maintain cardiac output include prolonged contraction, atrial enlargement, and increased contribution to LV filling [12].

Effect of Aging on Vascular Structures

Increasing arterial stiffness is the predominant change that occurs within the cardiovascular system in the setting of advanced age. The degree of arterial stiffness is proportionally greater in the diseased cardiovascular system. Potential energy released during the cardiac cycle stretches elastin fibers in the arteries and subsequently transmits this energy smoothly downstream to the muscular arterioles and capillary beds [13]. The aging process causes elastin to become depleted and replaced with increased amounts of non-distensible collagen and calcium [14].

The depletion of elastin and replacement with calcium and collagen results in systolic hypertension syndrome that is characterized by an increase in systolic pressures with a lowering or maintenance of the diastolic pressure level resulting in a widened pulse pressure [15]. These changes in the walls of vascular structures predispose to non-laminar and turbulent blood flow, which increases tensile and shear forces on the vessel wall resulting in progressive injury. To compensate for arterial stiffening, cardiac changes result in increased blood velocity to overcome the increased afterload of the stiffened central arterial tree [16].

Vascular changes that occur due to aging result in compromised diastolic filling and subsequently the ability of elderly patients to tolerate the stress of injury or surgery. Central arterial elasticity decreases with age and is paralleled by increased pulse wave velocity occurring in the forward and backward (reflected) direction. Based on the intrinsic compliance of their vessels, young patients have pulse wave

reflections occurring in diastole that augment coronary perfusion and ameliorate tensile shear forces of pulsatile blood flow [16]. Blood flow in less compliant vessels has enhanced shear due to turbulent flow and does not augment diastolic filling of coronary vessels that are already at risk due to atherosclerosis. A widened pulse pressure is the manifestation of stiffened central arteries due to a cardiac impulse transmitted downstream with greater force, causing reflected waves to return at end or peak systole [16].

Effect of Aging on Cardiac Output

With healthy aging the overall resting systolic function does not change. Cardiac imaging utilizing both echocardiography and radio-nucleotide studies has confirmed the preservation of systolic function [11]. The maintenance of myocardial performance was felt to be due to increases in left ventricular thickness, a prolongation of contraction times, an enlargement of the atria, and an increase in the contribution of the atrium to left ventricular filling [17]. With the development of cardiac MRI has come an advanced understanding of the performance of the heart in the elderly. It is now recognized that although older myocytes do increase in size, there is an overall myocyte depletion that is associated with increased collagen deposition and nonenzymatic cross-linking [18]. While the older ventricle increases in overall mass, it does not increase in functional mass, as evidenced by increasing left ventricular mass to volume ratios and associated declines in LVEDV in relation to left ventricular mass. Although the resting EF is preserved, absolute stroke volume does not remain comparable [19]. While both LVEDV and LVESV decrease with age, the decrease in LVEDV is proportionally greater than the decrease in LVESV, which leads to an overall age-related decline in resting stroke volume [19]. It was previously felt that the preservation of EF meant that elderly patient could respond to stress similar to their younger counterparts. Though the preservation of the net systolic function remains unaltered, with exercise the effects of aging are more evident. The reduction of cardiac reserve is a result of multiple factors including increased vascular afterload, arterial-ventricular mismatch, reduced contractility, impaired autonomic regulation, and physical conditioning.

Effect of Aging on the Beta-Adrenergic Response

The response of the cardiovascular system to surgical stress relies greatly on adrenergic stimulation. Exercise and stressors stimulate sympathetic output to increase heart rate, augment contractility and relaxation, and decrease afterload. Unfortunately, one of the consequences of the normal aging process is a decreased responsiveness to beta-adrenergic stimulation. Maximal heart rate (HRmax) decreases in the setting of aging and is responsible for decreases in aerobic work capacity. Decreases in HRmax are independent of gender, regular exercise, and other factors [6, 20]. This attenuation of heart rate responsiveness contributes significantly to an age-related reduction in maximal cardiac output and therefore determines aerobic exercise capacity [20].

The decrease in chronotropic responsiveness (HRmax) to exercise seen throughout the normal aging process remains poorly understood [20]. Proposed mechanisms for the decreased cardiovascular response to adrenergic stimulation are alterations to the conduction pathways as well as decreased receptor expression. With generalized increase in collagenous tissue and fibrosis of the cardiac myocytes, changes in the cardiac conduction system develop. Variable degrees of fibrosis and calcification of the cardiac skeleton can impact AV nodal conduction as well as the development of atrioventricular conduction block. Fat accumulation around the SA node is also observed with aging. This may cause partial or total separation of the SA node from the surrounding atrial tissue and may lead to decreased intrinsic heart rate. However, SA node dysfunction is not always identified in the setting of myocardial remodeling and instead may indicate a molecular change in the pacemaker cells [21]. The number of pacemaker cells also significantly declines with advanced age further decreasing the cardiovascular response to adrenergic stimulation [8]. Other observations have shown reductions in calcium channel proteins, which may lead to decreased sinus node depolarization reserve and thus suppression of action potential formation and propagation [22].

Another mechanism proposed for the decreased adrenergic responsiveness is a decrease in cardiac adrenergic receptor density. Elevated adrenergic neurotransmitter levels have been observed in the elderly and appear to be a compensatory response to decreased receptor expression and deficient NE uptake at nerve endings [8]. With prolonged adrenergic expression and deficient uptake, neurotransmitter depletion can contribute further to the blunted cardiac response and LV systolic performance seen with exercise and stress in the elderly.

Effect of Aging on the Baroreflex Response

In the normally functioning cardiovascular system, the baroreflex serves as an efficient component of a complex feedback loop that maintains adequate cardiovascular function. The effect of aging on the baroreflex has been studied by relating pulse interval to changes in systolic blood pressure after phenylephrine injection. This work revealed a linear relationship between pulse interval and change in systolic

blood pressure as well as a distinct decrease in the baroreceptor reflex sensitivity in the elderly [23, 24]. Others have found that an age-related decline in baroreflex sensitivity is independent of systolic blood pressure and systemic adrenergic levels [25]. Decreased baroreceptor reflex sensitivity was also demonstrated in a study of healthy volunteers examining cardiac response to angiotensin II (ANG II) infusions. The elderly, unlike younger patients, do not exhibit decreases in heart rate when blood pressure is increased via ANG II infusion [24, 26].

Effect of Surgery on the Geriatric Cardiovascular System

Much of what we know about the response of the aging cardiovascular system to surgery has been elucidated from a body of work evaluating the impact of exercise. Surgery results in a substantial amount of physical and metabolic stress on the body due to blood loss, the inflammatory response, and the effects of anesthesia. The effects of stress vary greatly depending on the age of the patient and the presence of associated comorbidities. Exercise provides a controlled stress state that allows some understanding of the effects of surgery on the elderly.

The normal response to exercise and presumably to surgical stress consists of an increase in cardiac output to meet the elevated metabolic needs of the body. Initially, it was believed that the elderly demonstrated a depressed cardiac output. Subsequent studies that excluded patients with coronary artery and myocardial disease showed a more appropriate increase in cardiac output although the mechanism appears to be different than in the young [27, 28]. Older patients cannot increase cardiac output with the typical increases in heart rate secondary to decreases in HRint and B-adrenergic responsiveness. The elderly optimize the Frank-Starling mechanism by increasing their end-diastolic volume and stroke volume during exercise, thereby increasing cardiac output without substantially increasing heart rate. While the elderly are able to augment stroke volume during exercise, the increase in ejection fraction is less than that observed in younger counterparts secondary to a decreased ability to reduce end-systolic volume. This physiologic response is similar to that which is seen in young patients administered with exogenous beta-blockade and then stressed with increasing exercise loads.

Surgery and injury are frequently associated with hypovolemia secondary to blood loss and capillary leak commonly leading to cardiovascular compromise. Free water loss, chronic poor oral intake, pharmacologic vasodilation (home medications), and decreased plasma oncotic pressure (poor nutrition) also commonly lead to further intravascular volume depletion. Given the dependence on the Frank-Starling modulation of cardiac output rather than chronotropy, the elderly patient is particularly sensitive to preload reductions. While Shannon and colleagues showed that elderly patients mount a blood pressure increase and slight HR increase similar to younger patients during tilt tests, this response is negatively affected by hypovolemia [29]. When the same test is performed after preload reduction with diuretics, elderly patients sustain a symptomatic fall in blood pressure due to an inability to mount a tachycardic response in contrast to younger patients who exhibit an appropriate increase in both heart rate and blood pressure [29].

The body of literature evaluating the effects of exercise on the aging cardiovascular system has demonstrated the ability to maintain cardiac output in response to the stress of surgery [27, 28]. The mechanism appears to depend upon stroke volume by increasing end-diastolic volumes and contractility rather than through the augmentation of heart rate [31]. Newer technology including cardiac MRI and pulsed tissue Doppler echocardiography in 2D and 3D has shown that while elderly patients can mount a cardiac output response to stress, this is of lesser magnitude than in their younger counterparts due to decreased cardiac reserve [12, 30, 31]. It is clear that elderly patients generate increased cardiac performance in the face of stress; however, the magnitude of this response is attenuated and less robust than that of younger counterparts.

Effect of Comorbidities on Cardiovascular Function: Atrial Fibrillation

Elderly patients rely heavily on prolonged contraction times and increased atrial contribution for adequate left ventricular filling. Atrial fibrillation (AF) is particularly problematic because atrial arrhythmias result in inconsistent and often inadequate ventricular filling due to limited contraction and decreased filling time. Age-related increases in left (and right) atrial size in older patients are a risk factor for the development of AF [32]. Additional age-related risk factors include inflammatory cytokines, local and systemic stress, altered calcium handling, and electrical remodeling on a chronic basis [33]. In the acute setting, pulsatile mechanical atrial stretch and inflammatory cytokines (from surgery, injury, or sepsis) contribute to arrhythmogenesis [33]. Numerous cytokines may contribute to the development of AF including interleukin-6, interleukin-8, and hsCRP [33]. These same cytokines are present in high levels in the serum of injured patients and can be used to predict progression to multiple organ failure in the injured patient [34]. Surgical patients are exposed to other risk factors including large-volume resuscitation causing atrial stretch, increased endogenous catecholamine release, rapid fluid and electrolyte shifts, hypoxia, and hypercarbia [35, 36]. Another common

risk factor is withdrawal from chronic beta-blockade in the elderly following surgical procedures.

Atrial fibrillation significantly impacts elderly surgical patients and frequently complicates the postoperative course. Chronic AF should be managed with the main goal being control of heart rate as this results in more optimal long-term outcomes [37]. Maintenance management of AF usually consists of beta-blocker, calcium channel blocker, or antiarrhythmia medications such as amiodarone. These should be continued through the perioperative period as much as possible although this can be challenging in the setting of hemodynamic compromise and limited gastrointestinal function. In the setting of significant surgery or severe injury, acute AF is common and results in prolonged hospital length of stay. There is no superior treatment regimen, and therapy is usually tailored to meet the unique patient care needs present at the time of diagnosis. Trauma patients have been found to benefit from beta-blockade due to the commonly high levels of catecholamines present at the time of injury [38]. For postoperative patients, beta-blockade and calcium channel blockade are the most common and efficacious approaches in the presence of adequate perfusion. Patients with hemodynamic compromise at the time of AF onset may require synchronized cardioversion or the initiation of antiarrhythmics such as amiodarone. Often, therapy for acute AF is only needed during the perioperative period of time and can be discontinued as the body heals and the cytokine environment returns to normal. Nevertheless, AF should be diagnosed and managed expeditiously in the elderly due to greater expected reductions in cardiac output secondary to loss of atrial kick and need for longer diastolic filling times.

Effect of Comorbidities on Cardiovascular Function: Ischemic Heart Disease

Surgical patients are at significant risk for acute myocardial ischemia given the associated endogenous catecholamine release, systemic inflammation, and increased myocardial oxygen demand. Additionally, hyperdynamic blood flow during resuscitation and its associated turbulent and non-laminar blood flow increase vessel wall shear forces. This increased shear may cause the rupture of coronary atherosclerotic plaques and predispose to myocardial infarction (MI) [39]. The risk of MI is compounded in the elderly in whom arterial pulse wave indices do not support diastolic filling of coronary vessels and arterial stiffening only exacerbates conditions of turbulent arterial blood flow. Elderly patients are also at greater risk due to preexisting coronary artery and intrinsic cardiac disease. Perioperative MI represents an important disease entity to address as it is associated with worse outcomes especially in the aged [40].

The elderly are the most at risk to experience an MI after surgery, are the most likely to suffer poor MI-related outcomes, and subsequently are the most likely to benefit from intervention. Due to atypical symptomatology and presentation, MI is difficult to diagnose in the critically ill elderly patients. A high index of suspicion and liberal use of diagnostic modalities such as ECG and serial troponin measurements are required to identify acute myocardial ischemia. Myocardial ischemia should be considered in the setting of unexplained vital sign decompensation after hemorrhage and hypovolemia are ruled out. Echocardiogram may be valuable to identify wall motion abnormalities in the face of non-diagnostic troponin elevation [41]. Cardiology consultation should be obtained liberally in the setting of acute coronary syndrome as the patient may be a candidate for reperfusion with coronary intervention.

Effect of Comorbidities on Cardiovascular Function: Heart Failure with Preserved Ejection Fraction

Heart failure (HF) with preserved EF is defined as heart failure with an ejection fraction equal to or greater than 50 % and represents up to 40 % of patients with heart failure [42]. This clinical condition is important because patients will appear normal when at rest and this resting EF is often erroneously used in these patients as a surrogate for achievable cardiac performance under stress. Several exercise studies of patients with HF with preserved EF demonstrated an inability to adequately increase LV systolic elastance. Further, these patients demonstrate lower peripheral resistance, increase heart rate, and reductions in ventricular-arterial coupling that result in an intolerance of submaximal and maximal exercise workloads [42]. Patients with HF express maladaptive inotropic, lusitropic, chronotropic, and vasodilatory responses to the physical stress of exercise and are believed to have similar inadequate responses to the physical stress of surgery.

Monitoring the Aging Cardiovascular System

Due to the significant anatomic and physiologic limitations described above, the elderly cardiovascular system often requires multiple monitoring techniques to provide the necessary support during the perioperative period of time. The elderly do not have the same reserve as the young surgical patient and therefore require more exact maintenance of preload, contractility, and afterload to ensure adequate cardiac performance. The initial question that must be answered for any surgical patient should always be, “Is the patient in shock and underperfused?” The answer to this question is provided

by clinical examination (mental status, peripheral pulse quality, skin temperature, urine output) as well as laboratory studies. Comorbidities often present in the elderly may mimic clinical findings such as dementia/delirium, chronic peripheral vascular disease, and chronic renal insufficiency. Laboratory findings such as lactic acid and base deficit remain valuable indicators of global hypoperfusion. At a minimum, an arterial catheter is required in any patient in shock with marginal hemodynamics to provide continuous assessment of systolic and mean arterial blood pressure. More invasive monitoring techniques are often necessary to determine the intravascular volume status as well as cardiac and vascular abnormalities that may be present. It is important to recognize that the value of any monitoring technique is highly dependent on the ability to correctly interpret the results of the device to make therapeutic decisions. The decision to implement a more advanced monitoring device should simply occur when the clinician can no longer confidently describe the patient's intravascular volume status.

Modalities that provide the ability to monitor intravascular volume status as well as intrinsic cardiac function include arterial pulse waveform/indicator dilution analysis, pulmonary artery catheters (PACs), and echocardiography. A central venous catheter measuring central venous pressure (CVP) provides the most basic assessment of intravascular volume status. Unfortunately, CVP is limited greatly by any factor that affects intrathoracic pressure such as mechanical ventilation. Therefore, CVP can be valuable when low, but a normal to elevated level does not rule out intravascular volume depletion. Systems that analyze arterial pulse waveforms attempt to determine the variability to stroke volume based on changes in the arterial waveform. Increased stroke volume variability can be used as an indicator of decreased intravascular volume that may benefit from volume expansion. Similarly, indicator dilution techniques utilize the peripheral arterial catheter by measuring the dilution of an injected substance from the time of venous injection to arterial sampling. This technique is capable of providing an indication of cardiac flow (CF) although this and arterial pulse waveform analysis methods depend upon peripheral arterial blood flow and may be limited in the setting of peripheral arterial disease. A PAC provides pressure measurements from the pulmonary vasculature which better correlate with left heart filling although can still be affected by mechanical ventilation. Newer generation PACs provide calculated measurements of end-diastolic and end-systolic volumes as well as blood flow and right heart ejection fraction that may be of greater assistance in optimizing intravascular volume status and cardiac performance. Despite studies challenging the value of PACs, when used correctly these devices continue to provide valuable information used to guide resuscitation especially in the elderly patient. More recently, besides echocardiography used for resuscitative purposes has become more popular due to the ability to directly image the left heart. Both transthoracic and transesophageal methods are now available that allow the surgeon to visualize the left heart frequently without the challenges of obtaining formal echocardiography. These resuscitative echocardiographic techniques provide the ability to visualize and measure left heart filling as well as the associated ejection fraction. Repeat imaging after volume expansion or inotrope/vasopressor manipulation allows the clinician to immediately determine the impact of the recent interventions on improving cardiovascular function. The main challenge has been the education required to have adequate numbers of clinicians capable of obtaining the necessary images and the associated interpretations.

Support of the Aging Cardiovascular System

The failing elderly cardiovascular system requires a careful determination of the cause of the inadequate perfusion. Most importantly, each patient should receive what he or she needs to correct the cardiovascular abnormality that is present. Unlike young patients, the elderly will often be intolerant to under- or overcorrection again due to the lack of reserve and the unique physiology that occurs with aging. Most surgical patients, whether young or old, require expansion of intravascular volume to optimize cardiac function. The elderly are more sensitive to hypovolemia, as described above, and therefore an early decision to institute invasive monitoring may be required to ensure the proper amount of volume expansion. Given that over-resuscitation may be deleterious to the elderly surgical patient, this monitoring provides an opportunity to administer the appropriate amount of fluid. In the absence of portal hypertension physiology, crystalloid remains the most efficacious, safe, and inexpensive choice of fluid for volume expansion. Despite theoretical benefits, colloid has yet to demonstrate superior outcome results when compared to crystalloid alone.

When optimization of volume status fails to correct global hypoperfusion, correction of contractility and/or afterload with the initiation or modification of inotropes/vasopressors is required. The choice of agent to use remains a point of discussion throughout the surgical community. There is very little data supporting one agent over another, and the clinical scenario is likely more important than having standard medications of choice. Nevertheless, each of the agents has unique attributes that make them more or less appealing given the circumstances. Patients that have isolated contractility problems may benefit from dobutamine due to the specific myocardial stimulation that is provided via $beta_1$ stimulation. Unfortunately, dobutamine can cause profound tachycardia that in the elderly may not be tolerated due to the significant increase in myocardial oxygen demand and the risk of

precipitating atrial fibrillation. While not as effective, milrinone provides enhanced myocardial contractile function without the effects on heart rate. Further, milrinone is appealing in the elderly due to its non-adrenergic mechanism of action given the presence of poor adrenergic responsiveness.

In the setting of decreased afterload, norepinephrine and vasopressin are commonly used to support hemodynamic function. Norepinephrine provides strong afterload augmentation via alpha-adrenergic stimulation while offering minimal beta stimulation on the heart. For this reason, norepinephrine is the most common vasopressor used in the setting of septic shock when there is a marked reduction in afterload. The result is an increase in blood pressure without affecting heart rate or contractility. Similarly, vasopressin is a non-catecholamine hormone which results in significant vasoconstriction and can improve blood pressure. Vasopressin does not function through adrenergic stimulation and therefore is appealing in the elderly patient who has blunted adrenergic responsiveness. Both norepinephrine and vasopressin benefit from increasing blood pressure without significant increases in heart rate, thus making them less arrhythmogenic. Nevertheless, it is important in the elderly, who have limited contractile reserve, to avoid implementing unopposed afterload augmentation that might result in myocardial decompensation and decreased cardiac performance.

When monitoring of the cardiovascular system reveals findings consistent with reduced contractility and afterload, epinephrine may be a valuable agent to select to provide support. Epinephrine stimulates both alpha- and beta-adrenergic receptors resulting in enhanced contractility and vasoconstriction. The result is increased cardiac output and blood pressure as well as improved coronary blood flow. With that said, tachycardia is common resulting in increased myocardial oxygen demand and a greater likelihood of AF. Nevertheless, epinephrine is a valuable agent to employ but should be selected carefully and the results monitored closely.

Summary

The elderly surgical patient presents challenges related to anatomic and physiological changes as well as preexisting conditions. Altered cardiovascular function is common in the elderly surgical patient resulting in an inability to adequately perfuse the organs of the body. The elderly cardiovascular system exhibits a blunted response to adrenergic stimulation due to reductions in intrinsic heart rate and beta-adrenergic responsiveness. The ability to increase cardiac output during surgery relies more on increased ventricular filling and stroke volume than on increases in heart rate and EF. The elderly also demonstrate decreases in baroreceptor reflexes that make them more sensitive to hypovolemia and hemorrhage. Atrial dysrhythmias are common and must be rapidly diagnosed and treated due to a reliance on atrial contributions for adequate ventricular filling and cardiac output. Monitoring of cardiovascular function may be more beneficial than in the young due to the importance of achieving optimal volume status. When inotrope/vasopressor agents are necessary, careful selection based on the specific needs of the patient is important given the unique risks and benefits of each medication. Frequent reassessment after initiating or modifying cardiovascular medications is required to ensure achieving improved perfusion but to also monitor for untoward complications.

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