Epidemiology, Pathophysiology, and Etiology of Congestive Heart Failure in Older Adults

[Special Series]

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Abstract:

OBJECTIVES: To review the epidemiology, pathophysiology, and etiology of congestive heart failure (CHF) in older adults.

METHODS: Published reports relevant to the epidemiology, pathophysiology, and etiology of CHF were systematically reviewed. Studies involving older adults and more recent studies were emphasized.

RESULTS: More than 75% of patients with CHF in the United States are older than 65 years
of age, and CHF is the leading cause of hospitalization in older adults. CHF is also a major cause of chronic disability, and annual expenditures for CHF currently exceed $10 billion. In addition, both the incidence and prevalence of CHF are increasing, largely as a result of the aging of the population.

Older adults are predisposed to developing CHF as a result of age-related changes in the cardiovascular system and the high prevalence of hypertension, coronary artery disease, and valvular heart disease in this age group. Although the fundamental pathophysiology of CHF is similar in younger and older patients, older individuals are more prone to develop CHF in the setting of preserved left ventricular systolic function. This syndrome, referred to as diastolic heart failure, accounts for up to 50% of all cases of CHF in adults more than 65 years of age.

Coronary heart disease and hypertension are the most common etiologies of CHF in older adults, and they often coexist. Valvular heart disease, especially aortic stenosis and mitral regurgitation, are also common in older adults, whereas nonischemic dilated cardiomyopathy, hypertrophic cardiomyopathy, and restrictive cardiomyopathy occur less frequently.

CONCLUSIONS: Congestive heart failure is a major public health problem in the United States today as a result of its high and increasing prevalence in the older population as well as its substantial impact on healthcare costs and quality of life. There is an urgent need to develop more effective strategies for the prevention and treatment of CHF in older individuals.

Although the clinical syndrome of congestive heart failure (CHF) has been recognized by physicians for more than 2000 years, it has only been within the past 10 to 15 years that CHF has been identified as a major public health concern. A major factor contributing to the rise in interest in CHF is the high prevalence of this disorder in older adults. The purpose of this article, the first in a 3-part series, is to review the epidemiology, pathophysiology, and etiology of CHF in the older adult population.

EPIDEMIOLOGY

Despite progressive declines in age-adjusted mortality rates from coronary heart disease and hypertensive cardiovascular disease, both the incidence and prevalence of CHF are increasing, and it has been projected that these trends will continue well into the 21st century. Although several factors have contributed to the rise in CHF, principal among them is the progressive aging of the population. As discussed in more detail below, older adults are at increased risk for developing CHF as a result of age-related changes in cardiovascular structure and function in conjunction with a high prevalence of cardiovascular diseases, particularly coronary heart disease and hypertension. In addition, improved therapies for hypertension and ischemic heart disease are allowing patients with these disorders to survive (or avoid) other clinical manifestations of these diseases, only to develop CHF at a later time. Thus, patients who might have died from acute myocardial infarction (MI) 20 years ago are now surviving, but with residual left ventricular dysfunction that may contribute to CHF months or years later. Similarly, improved blood pressure control has led to a 60% decline in stroke mortality, yet these same patients remain at risk for developing CHF as a complication of hypertension and left ventricular hypertrophy. Finally, advances in the treatment of other chronic conditions, such as end-stage renal disease, have also contributed indirectly to the rising incidence and prevalence of CHF.

Congestive heart failure affects over 3 million Americans, and more than 400,000 new cases are diagnosed each year. Moreover, both the incidence and prevalence of CHF are strikingly age-dependent (Figure 1). Thus, whereas CHF is a relatively uncommon disorder in individuals
less than 45 years of age, the prevalence increases twofold for each decade thereafter, so that the prevalence in adults over 80 years of age approaches 10%. Similarly, mortality rates for CHF increase exponentially with advancing age in all major demographic subgroups of the U.S. population (Figure 2).7

<table>
<thead>
<tr>
<th>Age</th>
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<tr>
<td>55-64</td>
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<td>65-94</td>
<td>10</td>
<td>8</td>
</tr>
</tbody>
</table>

Figure 1. Incidence of heart failure by age and sex: 30-year followup from the Framingham Study. (Kannel WB, Belanger AJ. Am Heart J 1991;121:951-957, with permission).
Consistent with the high prevalence and substantial mortality associated with this disorder, CHF is currently the leading indication for hospital admission in adults older than age 65. In 1993, there were 875,000 hospitalizations in the US with a first-listed discharge diagnosis of CHF.8 Seventy-eight percent of these occurred in patients 65 years of age or older, and 50% occurred in individuals more than 75 years of age.8 As a result, CHF is not only the most common diagnosis-related group (DRG) in the Medicare population, it is also the most costly, with estimated annual inpatient expenditures in excess of $8 billion.9

Not surprisingly, out-patient resource utilization is also quite high in CHF patients. CHF accounts for more than 3 million physician office visits each year, and the annual cost of out-of-hospital care for these patients is approximately $3 billion.9 In addition, CHF is one of the most common chronic conditions resulting in impaired quality of life and loss of independence in older adults,10 and it is also a leading cause of death in the Medicare age group.11

In summary, CHF is an extremely common disorder in older adults, and it exacts a tremendous toll on society in terms of medical resource consumption and in-patient and out-patient expenditures. Moreover, CHF is a major source of chronic disability as well as mortality in the geriatric age group. Finally, and perhaps most alarmingly, the number of CHF cases older than age 65 is expected to double over the next 40 years as a result of the progressive aging of the population.

**PATHOPHYSIOLOGY**21
In a sense, CHF represents the prototypical disorder of cardiovascular aging, by virtue of the fact that the combination of age-related changes in the cardiovascular system in conjunction with a high prevalence of age-related cardiovascular diseases conspire to produce an exponential rise in CHF prevalence with advancing age.

Aging is associated with extensive and pervasive changes in cardiovascular structure and function (Table 1). However, in the absence of coexistent cardiovascular disease, resting cardiac function is well preserved even at very advanced age. Thus, the resting left ventricular ejection fraction, an index of left ventricular systolic performance, is unaffected by age in healthy individuals. Similarly, most studies indicate that resting cardiac output is either maintained or declines minimally with increasing age.
Gross anatomy
- Increased left ventricular wall thickness
- Decreased left ventricular cavity size
- Endocardial thickening and sclerosis
- Increased left atrial size
- Valvular fibrosis and sclerosis
- Increased epicardial fat

Histology
- Increased lipid and amyloid deposition
- Increased collagen degeneration and fibrosis
- Calcification of fibrous skeleton, valve rings, and coronary arteries
- Shrinkage of myocardial fibers with focal hypertrophy
- Decreased mitochondria, altered mitochondrial membranes
- Decreased nucleus: myofibril size ratio

Biochemical changes
- Decreased protein elasticity
- Numerous changes in enzyme content and activity affecting most metabolic pathways, but no change in myosin ATPase activity
- Decreased catechol synthesis, esp. norepinephrine
- Decreased acetylcholine synthesis

Conduction system
- Degeneration of sinus node pacemaker and transition cells
- Decreased number of conducting cells in the AV-node and HIS—Purkinje system
- Increased connective tissue, fat, and amyloid
- Increased calcification around conduction system

Vasculature
- Decreased distensibility of large and medium-sized arteries
- Aorta and muscular arteries become dilated, elongated, and tortuous
- Increased wall thickness
- Increased connective tissue and calcification

Autonomic nervous system
- Decreased responsiveness to β-adrenergic stimulation
- Increased circulating catecholamines, decreased tissue catecholamines
- Decreased α-adrenergic receptors in left ventricle
- Decreased cholinergic responsiveness
- Diminished response to Valsalva and baroreceptor
Table 1. Effects of Aging on the Cardiovascular System

From the clinical perspective, the changes associated with cardiovascular aging result in an impaired ability of the heart to respond to stress, whether that stress is physiologic (e.g., exercise) or pathologic (e.g., hypertension or myocardial ischemia). Four principle changes in the cardiovascular system contribute directly to the heart's attenuated capacity to augment cardiac output in response to stress. First, aging is associated with reduced responsiveness to \([\beta]-\)
adrenergic stimulation. The mechanism underlying this change has not been fully elucidated, but it is not caused by reduced circulatory catecholamine levels, decreased \([\beta]-\) receptor density on cardiac myocytes, or altered responsiveness to intracellular calcium. In any case, the diminished response to \([\beta]-\) adrenergic stimulation limits the heart's capacity to maximally increase heart rate and contractility in response to stress, and \([\beta]-\) mediated peripheral vasodilation is also compromised.

A second major effect of aging is increased vascular stiffness, which is related primarily to increased deposition of connective tissue and collagen in the media and adventitia of the large and medium sized arteries. Increased vascular stiffness results in an increased impedance to left ventricular ejection (i.e., increased afterload), and it also contributes to the increased propensity of older individuals to develop isolated systolic hypertension.

In addition to increased vascular stiffness, the heart itself becomes stiffer, or less compliant, at older age. Several factors contribute to the increased cardiac stiffness, including increased interstitial connective tissue content, compensatory myocyte hypertrophy in response to myocyte loss from apoptosis, and delayed myocyte relaxation attributable to impaired release of calcium from the contractile proteins and its subsequent reuptake by the sarcoplasmic reticulum and storage pool. The latter factor (i.e., altered calcium handling at the end of systole) leaves the heart in a state of "partial contraction" at the onset of diastole, and this significantly attenuates the peak early diastolic filling rate. In addition to these factors, increased vascular impedance results in compensatory left ventricular hypertrophy to overcome the increased resistance to ejection, and this hypertrophy further increases cardiac stiffness.

The changes in cardiac stiffness result in important alterations in diastolic filling and atrial function. Using Doppler echocardiographic techniques, it is possible to characterize the pattern of left ventricular filling by analyzing diastolic flow across the mitral valve. In healthy young persons, the transmural flow pattern is characterized by a large E-wave with a rapid upstroke representing rapid filling of the ventricle immediately following the opening of the mitral valve. This is followed by a period in which the rate of filling slows (the downslope of the E-wave), mid-diastolic diastasis (in which the left atrial and left ventricular pressures are essentially equal), and a second burst of flow at the end of diastole corresponding to atrial contraction (the A-wave, or atrial "kick"). Importantly, the majority of ventricular filling occurs in the first half of diastole in young individuals, with a relatively small contribution from atrial contraction.

In older persons, alterations in cardiac relaxation and stiffness result in characteristic changes in the pattern of diastolic filling. Early filling is impaired, and the upstroke of the E-wave is delayed. Similarly, the downslope of the E-wave is less steep as it takes a longer time to achieve diastasis. The left atrium, to compensate for increased resistance to ejection, enlarges and hypertrophies. This results in a more forceful left atrial contraction and an augmented A-wave. As a result of these changes, a greater proportion of filling occurs in the second half of diastole in older individuals, and as much as 30 to 40% of left ventricular end diastolic volume may be attributable to atrial contraction. Thus, older individuals become increasingly reliant on the atrial "kick" to maximize left ventricular filling.
A third pattern of diastolic filling, referred to as the restrictive pattern, occurs when the left ventricle's ability to accept blood becomes severely compromised. In this situation, very little flow occurs after the rapid filling phase in early diastole. This pattern is characterized by a tall, narrow E-wave with a rapid downslope as diastasis is achieved early in diastole. Little additional flow occurs during mid-diastole, and the A-wave is typically small, with an amplitude that is less than 50% of the E-wave. The restrictive pattern almost always indicates marked elevation of the left ventricular diastolic pressure and it is generally associated with a poor prognosis, especially in patients with concomitant systolic dysfunction.22,23 However, the restrictive pattern generally occurs in patients with advanced cardiac disease, and it rarely results from aging changes alone.

Age-related changes in diastolic filling have several important clinical implications. First, inability to distend the cardiac myocytes to an optimal length results in a failure of the Frank-Starling mechanism,24 one of the cardinal adaptive responses (along with sympathetic activation) for acutely increasing cardiac output. Second, impaired diastolic filling results in an increase in left ventricular diastolic pressure. This elevated diastolic pressure is transmitted back to the left atrium and "stretches" the left atrial myocytes. This, in turn, increases the likelihood of atrial ectopic beats and atrial arrhythmias, particularly atrial fibrillation. This explains, in part, the increase in prevalence of atrial fibrillation with advancing age.25 In addition, atrial fibrillation itself is a common precipitant of CHF in older adults for two reasons. First, the absence of a coordinated atrial contraction substantially compromises late diastolic filling because of loss of the atrial "kick". Second, the rapid, irregular ventricular rate associated with acute-onset atrial fibrillation shortens the diastolic filling period, which further attenuates ventricular filling. A third effect of altered diastolic filling is an increased propensity for older adults to develop diastolic CHF (i.e., CHC with normal left ventricular systolic function).26,27 As indicated above, increased myocardial stiffness tends to result in an increase in resting left atrial and ventricular diastolic pressures. Further increases in these pressures caused by ischemia or uncontrolled hypertension may lead to pulmonary congestion and edema. Moreover, individuals with impaired diastolic function are often "volume sensitive." That is, small increments in intravascular volume, as may occur with a dietary salt load or intravenous fluid administration, can not be accommodated by the noncompliant ventricle. As a result, the intraventricular pressure rises abruptly, and CHF ensues. Conversely, intravascular volume contraction, e.g., attributable to poor oral intake or overdiuresis, may cause a marked fall in intraventricular volume, which in turn leads to a fall in stroke volume and cardiac output.

The final major effect of cardiovascular aging is altered myocardial energy metabolism at the level of the mitochondria.12,13 Under resting conditions, older cardiac mitochondria are able to generate sufficient quantities of ATP to meet all of the heart's energy requirements. However, when stress causes an increase in ATP demands, the mitochondria are often unable to respond appropriately. Although the precise mechanism underlying this mitochondrial failure is unclear, the defect further contributes to the heart's inability to maintain normal metabolic function in response to stress.

To summarize, four major age-related changes in cardiovascular structure, function, and physiology combine to reduce cardiovascular reserve and greatly increase the risk of CHF in older adults. Recalling that cardiac output is determined by four primary factors (heart rate, preload, afterload, and contractile state), and recognizing that each of these factors is adversely affected by one or more of the four major effects of aging on the heart, and that superimposed upon these changes is the high prevalence of cardiac disease in older adults, it is indeed not surprising that the incidence and prevalence of CHF rise exponentially with advancing age.

Before leaving this section, it is important to note that aging is also associated with significant changes in other organ systems that impact directly or indirectly on the development and/or management of CHF. Aging is accompanied by a decline in glomerular filtration rate, which
averages 8 cc/min/decade. In addition, the aging kidney is less able to maintain intravascular volume and electrolyte homeostasis. The reduced capacity of the kidney to respond to intravascular volume overload or dietary sodium excess further increases the risk of CHF in older patients. In addition, older patients are less responsive to diuretics and more likely than younger patients to develop diuretic-induced electrolyte abnormalities, factors that may complicate the management of CHF in the older age group.

Aging is also associated with numerous changes in respiratory function that serve to diminish respiratory reserve. Some of these effects, such as V/Q mismatching and sleep-related breathing disorders, may contribute directly to the development of CHF by producing hypoxemia or pulmonary hypertension. Other changes reduce the capacity of the lungs to compensate for the failing heart by increasing tidal volume and minute ventilation, thereby contributing to the patient's sensation of dyspnea. In more severe cases of cardiac failure, such as pulmonary edema, acute respiratory failure may ensue, partly as a consequence of the inability of the lungs to maintain oxygenation and effective ventilation.

Age-related changes in nervous system function include an impaired thirst mechanism, may contribute to dehydration and intravascular volume contraction in patients treated with diuretics, and a reduced capacity of the central nervous system's autoregulatory mechanism to maintain cerebral perfusion in the face of changes in systemic arterial pressure. The latter effect may contribute to subtle changes in mental function in older patients treated with vasodilators for CHF. Aging is also associated with widespread changes in reflex responsiveness. For example, impaired responsiveness of the carotid baroreceptors to acute changes in blood pressure may cause orthostatic hypotension or syncope, and these effects may be further aggravated by many of the drugs used to treat CHF.

Finally, it is well recognized that aging is associated with significant changes in the pharmacokinetics and pharmacodynamics of almost all drugs. In addition, older patients tend to be at increased risk for both drug-drug and drug-disease interactions because of the high prevalence of comorbid conditions and the use of multiple pharmacologic agents. These factors often lead to alterations in drug efficacy and an increased side effect profile, and these effects must be taken into consideration when designing therapy for older CHF patients.

**ETIOLOGY**

In general, the etiology of CHF is similar in older and younger patients, but CHF in older individuals is more often multifactorial. As in younger patients, hypertension and coronary heart disease are the most common causes of CHF, accounting for more than 70% of cases. Hypertensive hypertrophic cardiomyopathy represents a more severe form of hypertensive heart disease, which is seen most commonly in older women and is often accompanied by heavy calcification of the mitral valve annulus. These patients often manifest severe diastolic dysfunction, and they may exhibit dynamic left ventricular outflow tract obstruction indistinguishable from that seen in classical hypertrophic cardiomyopathy.

Valvular heart disease is an increasingly common cause of CHF at older age. Calcific aortic stenosis is now the most common cause of aortic valve disease requiring surgical intervention, and aortic valve replacement is the second most common open heart procedure performed in patients more than 70 years of age (after coronary bypass grafting). Mitral regurgitation in older individuals may be caused by myxomatous degeneration of the mitral valve leaflets and chordae tendineae (mitral valve prolapse), mitral annular calcification, valvular vegetations, ischemic papillary muscle dysfunction, or altered ventricular geometry attributable to ischemic or nonischemic dilated cardiomyopathy. Importantly, mitral regurgitation may be acute (e.g., following acute myocardial infarction), subacute (e.g., endocarditis), or chronic (e.g.,
myxomatous degeneration), and the clinical manifestations may vary widely in each of these settings. In the US, rheumatic mitral stenosis is a less common cause of CHF in older adults, but it is still seen occasionally. Functional mitral stenosis attributable to severe mitral valve annulus calcification with encroachment upon the mitral valve orifice is an uncommon cause of CHF, but it is associated with a poor prognosis.39 Aortic insufficiency may be either acute (e.g., caused by endocarditis or type A aortic dissection) or chronic (e.g., annuloaortic ectasia or syphilitic aortitis), but it is a relatively infrequent cause of CHF in older adults. Finally, prosthetic valve dysfunction should be considered as a potential cause of CHF in any patient who has undergone previous valve replacement.

Cardiomyopathies are classified into three categories: dilated, hypertrophic, and restrictive. In older adults, ischemic heart disease with one or more previous myocardial infarctions is the most common cause of dilated cardiomyopathy. Nonischemic dilated cardiomyopathy is less common in older individuals than in younger ones; when present, it is most often either idiopathic in origin or attributable to chronic ethanol abuse.40 Less frequently, dilated cardiomyopathy may be caused by anthracycline toxicity or other causes. Classical hypertrophic cardiomyopathy, once thought to be rare in the geriatric age group, has been recognized increasingly in older adults since the advent of echocardiography.41 Similarly, restrictive cardiomyopathy, most commonly the result of amyloid deposition (so-called senile cardiac amyloid), is an occasional cause of CHF. In one autopsy series, cardiac amyloid deposition was thought to be clinically important in approximately 10% of individuals 90 years of age or older.42

Infecove endocarditis is an uncommon but important cause of CHF in older patients because it is one of the few causes of CHF for which curative pharmacological therapy is available. Endocarditis should be strongly suspected in any patient with persistent fever and either a prosthetic heart valve or pre-existing valvular lesion. It should also be considered in any patient with fever, recent dental work or other procedures, and a new or worsening heart murmur. It is important to recognize, however, that the clinical manifestations of endocarditis are often protean, and the absence of fever or a heart murmur does not exclude this diagnosis in older individuals.43

Myocarditis is a relatively rare cause of CHF in older adults. It may be infectious (e.g., post-viral) or noninfectious (e.g., due to sarcoid or collagen vascular disease). Pericardial effusion, for which there are numerous etiologies, occasionally presents with CHF symptomatology, including fatigue, exertional dyspnea, and edema. Constrictive pericarditis may be infectious (e.g., tuberculous) or noninfectious (e.g., post-radiation), but it is a rare cause of CHF in older patients.

High-output failure is an uncommon cause of CHF in older adults, but when present the diagnosis is often overlooked. Common causes of high-output failure include chronic anemia, hyperthyroidism, thiamine deficiency, and arteriovenous shunting (e.g., due to a dialysis fistula or arteriovenous malformations).

Finally, in some older patients with CHF, detailed investigation may not elicit any evidence of cardiovascular pathology. In cases with normal left ventricular systolic function, CHF may be attributed to age-related diastolic dysfunction.27 Although the Doppler-echocardiographic findings may suggest the presence of diastolic dysfunction, it must be emphasized that this remains a diagnosis of exclusion inasmuch as there are currently no specific criteria for establishing this diagnosis.

**PRECIPITATING FACTORS**41

In addition to determining the etiology of CHF, it is important to identify any coexisting factors that may have contributed to an acute or subacute CHF exacerbation (Table 2). The most
common precipitant in patients with pre-existing CHF is noncompliance with medications and/or diet, which may contribute to two-thirds of CHF exacerbations. In hospitalized patients, iatrogenic volume overload is also an important precipitant of CHF.

<table>
<thead>
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<tr>
<td>Medication noncompliance</td>
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<tr>
<td>Iatrogenic volume overload</td>
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<tr>
<td>Arrhythmias</td>
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<tr>
<td>Atrial fibrillation or flutter</td>
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<tr>
<td>Ventricular arrhythmias</td>
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<td>Bradyarrhythmias, esp. sick sinus syndrome</td>
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<tr>
<td>Fever</td>
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<tr>
<td>Infections, esp. pneumonia or sepsis</td>
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<td>Hyperthyroidism or hypothyroidism</td>
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<tr>
<td>Anemia</td>
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<tr>
<td>Renal insufficiency</td>
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<tr>
<td>Hypoxemia from chronic lung disease</td>
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<tr>
<td>Estrogen preparations</td>
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<tr>
<td>Antihypertensive agents (e.g., clonidine, minoxidil)</td>
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</table>

Table 2. Common Precipitants of Congestive Heart Failure in Older Adults
Among cardiac factors, myocardial ischemia or infarction and new-onset atrial fibrillation or flutter are the most common causes of an acute CHF episode. Other cardiac causes include ventricular arrhythmias, especially sustained ventricular tachycardia, and bradyarrhythmias, such as marked sinus bradycardia or advanced atrioventricular block. Sick sinus syndrome, which is common in older adults, is a frequent cause of bradyarrhythmias in this population.

As previously discussed, older patients have limited cardiovascular reserve and they are less able to compensate in the face of increased demands. As a result, CHF in older adults is often precipitated by acute or worsening noncardiac conditions. Patients with acute respiratory disorders, such as pneumonia, pulmonary embolism, or an exacerbation of chronic obstructive lung disease, are particularly prone to experience a deterioration in cardiac function. Other serious infections, such as sepsis or pyelonephritis, may also lead to a CHF exacerbation. In patients with hypertension, inadequate blood pressure control is a common cause of worsening CHF.44,46 Thyroid disease, anemia (e.g., due to gastrointestinal bleeding), and declining renal function may also contribute directly or indirectly to the development of CHF.

Finally, it is important to recognize that numerous drugs and medications may contribute to CHF exacerbations. Alcohol is a cardiac depressant, and it may also precipitate arrhythmias, especially atrial fibrillation. Beta blockers (incl. ophthalmologic agents) and calcium antagonists are used widely in older individuals with cardiovascular disease, but both classes of agents are negatively inotropic and may exacerbate CHF. Class Ia (e.g., quinidine, procainamide, disopyramide) and Ic (e.g., flecainide, moricizine, propafenone) antiarrhythmic agents also have significant myocardial depressant effects which may worsen CHF. Nonsteroidal anti-inflammatory drugs (NSAIDs), which are used widely by older adults, impair renal sodium and water excretion and may, therefore, contribute to intravascular volume overload.47 In addition, NSAIDs antagonize the effects of angiotensin-converting enzyme inhibitors, thereby limiting the efficacy of these agents.48 Corticosteroids and estrogen preparations may also cause fluid retention and an increase in total body water. The anti-hypertensive agent minoxidil also promotes fluid retention, and several other anti-hypertensive drugs (e.g., clonidine, guanethidine) may have unfavorable hemodynamic effects.

In summary, congestive heart failure is an exceedingly common and important clinical problem in older adults, in large part because of the complex interplay between age-related changes in the cardiovascular system, the high prevalence of cardiovascular and noncardiovascular diseases in the older population, and the widespread use of myriad drugs and other therapies that may adversely affect cardiovascular physiology. As the population of the US continues to age, CHF is likely to exact an increasing toll on our already stressed healthcare delivery system. In addition, the impact of CHF on quality of life and independence in the large number of older adults suffering from this disorder is staggering. There is, therefore, an urgent need to develop more effective strategies for the prevention and treatment of CHF, specifically focusing on the geriatric population. In the second and third articles in this series, the clinical features, diagnosis, and medical management of CHF in older persons will be reviewed.

ACKNOWLEDGMENT

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REFERENCES


http://gateway.ut.ovid.com/gwl/ovidweb.cgi


42. Waller BF, Roberts WC. Cardiovascular disease in the very elderly. Analysis of 40 necropsy patients aged 90 years or over. Am J Cardiol 1983;51:403-421. Bibliographic Links [Context Link]


Section Description:

CONGESTIVE HEART FAILURE IN OLDER ADULTS*

*This article is one in a series on congestive heart failure in older adults.

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