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Management of Cardiovascular Diseases in the Elderly

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Introduction

Ageing of population is one of the major social and health burdens in most of the afferent countries. In particular, cardiovascular disease is prevalent in elderly, which contributes to major morbidity and mortality. In fact, most of the cardiac diseases are more common with increasing age, such as hypertension, ischaemic heart disease, congestive heart failure and cardiac arrhythmias. It is not uncommon for a general physician to see elderly patients with some form of cardiovascular disease in daily practice. In order to administer the most appropriate and specific therapy to these diseases in elderly patients, understanding the physiological changes of human senescence, as well as approach to investigation and management is important.

Cardiovascular Changes in Ageing

Ageing is associated with progressive and extensive changes in cardiovascular structures and function, which may affect the pattern and course and possibly outcome of cardiovascular diseases. There are structural changes occur in the myocardium, heart valves as well as vessels (Table 1). In the heart, myocardial mass increases with age at a rate of about 1 gram/year together with increases in left ventricular wall thickness caused by myocyte hypertrophy.[1,2] The left ventricular hypertrophy is explained by the increased in afterload secondary to raised vessel stiffness[3] as well as a response to myocyte loss due to apoptosis.[4] Besides, there is progressive accumulation of matrix tissue in the cardiac interstitium, namely collagen subtypes (in particular type I and III collagen fibrils)[5,6] and fibronectin.[7] These two factors summatively contribute to the progressive decrease in chamber compliance and subsequently development of diastolic dysfunction which is an important cause of heart failure in geriatric population.[8] Similarly, the vascular stiffness is increased due to the deposition of collagen and other connective tissue in the media and especially adventitia of the medium and large size arteries.[9] The increased impedence to flow also partially contributes to the development of left ventricular hypertrophy. Other structural changes include the degeneration of sinus node and decrease in number of conducting cells in the conduction system of the heart. The cardiac valves become thickened especially the left sided ones and the valvular circumference are also increased.[10] In the anterior mitral leaflet, nodular thickening of the free edges with lipid deposition occurs, whereas annular calcification, diffuse opacification of the leaflet, ballooning deformity and small scarring are found in the posterior leaflet.[11] The change in mitral leaflets is not uncommonly accompanied by mild degree of mitral regurgitation.[12] In the aortic valves, calcification and fenestrations develop with ageing.

Functionally, ageing is associated with reduced catecholamine responsiveness.[13] This limits the ability of the inotropic and chronotropic effect of the heart to β-adrengeric stimulation such as during stress and exercise. The heart rate at rest and during exercise is also decreased as well as the heart rate variability. However, the systolic function is preserved with normal left ventricular dimensions.[2] As the left ventricular compliance is decreased, diastolic pressure increases. This causes symptoms of exercise intolerance and development of diastolic heart failure which is much more common in elderly than young adults.[14]

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Table 1. Effect of ageing on the cardiovascular structure and function

- Increased myocardial mass and left ventricular (LV) wall thickness
- Increased deposition of extracellular matrix
- Decreased LV compliance
- Decreased early diastolic filling and increased late diastolic filling
- Decreased left ventricular cavity size
- Increased left atrial size
- Increased contraction and relaxation time with decreased heart rate (both at rest and during exercise)
- Decreased heart rate variability
- Decreased α-adrenergic receptors in LV and decreased responsiveness to β-adrenergic stimulation
- Increased valvular fibrosis and thickness
- Increased circumference of valves
- Mitral annular calcification
- Decreased compliance of large and medium-sized arteries
- Increased vessel wall thickness and connective tissue
- Degeneration of sino-atrial node cells
- Decreased number of conducting cells in the AV node

Congestive Heart Failure (CHF)

Epidemiology

Despite efforts in improving the management of various cardiovascular diseases such as hypertension and ischaemic heart disease, the incidence and prevalence of CHF remains increasing. One of the main reasons in this epidemiological change is the ageing of the population and improvement in treatment of medical diseases. Therefore cardiovascular diseases can last long enough to risk the occurrence of CHF. In fact, both the prevalence and incidence of CHF are highly age-dependent.[15] It is relatively uncommonly in young adults, and the prevalence increases about two-fold for each decade older. It is one of the commonest causes for hospital admission in elderly population.

One of the important terminology is to differentiate systolic from diastolic dysfunction as the cause of heart failure. Although both conditions are very similar in symptomatology, there are aetiological, prognostic and management differences. Diastolic heart failure refers to the increased resistance to filling with increased filling pressure in one or both cardiac ventricles.[16,17] Despite the wide variation in the reported prevalence, majority of studies reported that 40% of patients admitted for CHF are due to pure diastolic dysfunction.[18-20] Elderly age is a predictor of diastolic heart failure.[21] A multicentre study restricted to 623 middle-aged men with chronic heart failure found that the prevalence of normal systolic function was only 13.3%.[22] Wong et al found in a study of 99 consecutive patients admitted to hospital with heart failure, 41% of those >70 years had normal systolic function compared to only 6% of those <60 years.[8]

Aetiology

In general, the causes of CHF are similar in younger and older patients, namely ischaemic heart disease, hypertension, cardiomyopathies, cardiac arrhythmias as well as valvular heart diseases, especially left-sided valvular regurgitation or stenosis. In patients with ischaemic heart disease, patients survived from an initial myocardial infarction is a major cause of pump failure. Occasionally, patients with long-standing myocardial ischaemia and hibernation also contribute to systolic dysfunction, which can be improved after revascularisation.[23] Degenerative valvular heart disease is basically a condition of senescence, though in developing countries CHF complicated by chronic rheumatic heart disease is not uncommonly encountered. Common causes of CHF due to cardiac arrhythmia in relation to inadequate cardiac output in elderly population are atrial fibrillation, complete heart block and tachy-brady-arrhythmias. Other causes of

CHF include infective endocarditis, myocarditis, and high out-put failure as seen in chronic anaemia and thyrotoxicosis.

Diseases which prone to develop diastolic heart failure include hypertension,[24,25] myocardial ischaemia,[26] left ventricular hypertrophy (LVH) (e.g. hypertrophic cardiomyopathy,[27] pressure-overload, aortic stenosis, and salt-loading[28,29]), myocardial fibrosis[30] and myocardial disease (various causes of cardiomyopathies and myocardial restriction).[31] Systemic hypertension is also more common in heart failure with normal ejection fraction, which is at least twice commoner than those with impaired LV systolic function.[32] Abnormal left ventricular diastolic filling is a common and early finding in hypertension, even before any obvious LVH or change in systolic function develops.[33] The development of LVH is also common in ageing. A study found that LVH was present in 43% of those >70 years of age by echocardiography, compared with only 6% for those <30 years of age.[34] LVH is an adaptive response to the increased wall stress initially. However, this leads to impaired relaxation, and increased susceptibility to hypoxia-induced diastolic dysfunction[35] and increased in passive elasticity of the ventricular chamber. The result is an upward shift of diastolic pressure-volume relation, i.e., a higher diastolic pressure for any increases in left ventricular volume.[36] In myocardial ischaemia, reversible diastolic dysfunction occurs even in the context of normal LV systolic function as occurring in demand ischaemia.[37] This is caused by the accumulation of diastolic intracellular calcium[38] which impairs ventricular relaxation by slowing the actin-myosin dissociation and diastolic pressure is raised as a result.[39] Ageing itself is an independent cause of diastolic dysfunction. The declines in left ventricular diastolic function with increasing age is related to the increased chamber stiffness. This change can be explained by structural changes such as increased interstitial collagen content in the myocardium[40] as well as impaired active relaxation due to reduced calcium ion sequestration into the sarcoplasmic reticulum.[41] In addition, increases in left ventricular mass with ageing may further increase the passive stiffness of the chamber. Studies have shown that in normal subjects, ageing alters ventricular diastolic function independently of LVH or coronary artery disease with a reduced rate and duration of the rapid diastolic filling phase due to increased regional diastolic asynchrony.[42]

Diagnosis

Both systolic and diastolic heart failure share common clinical features including dyspnoea, which progress to symptoms of orthopnoea and paroxysmal nocturnal dyspnoea. Acute pulmonary oedema may also develop. Symptoms of right heart failure include ankle swelling, abdominal distension due to ascites, and upper abdominal discomfort due to liver congestion. For patients with diastolic heart failure, exercise intolerance is a dominant and early feature.[14] Pulmonary congestion may cause cough and wheezing. The presence of low cardiac output state results in lethargy and fatigability. In elderly patients confusion and rarely other mental manifestation can be the presenting feature. Symptoms of underlying aetiological factors should also be sought, such as angina, palpitation or long-standing history of hypertension.

Physical examination will confirm features of fluid overload, namely ankle oedema, raised jugular venous pressure, basal crepitation in the lung, ascites with congestive hepatomegaly, and not uncommonly some degree of pleural effusion. Cardiac asthma with rhonchi and wheeze in a restless and tachypnoeic patient can be the dominant features. Signs of sympathetic stimulation include tachycardia and cold extremities. The reduced cardiac output is manifested as hypotension and small pulse volume. Chyne-Stokes respiration, cardiac cachexia or anasarca may occur. Precordial examination may show signs of cardiomegaly, heart murmur and gallop rhythm. Physical signs in relation to aetiology of heart failure should also be sought, such as heart murmur of valvular lesions or hypertensive retionpathy.

Important investigations include electrocardiography, chest roentgenography and echocardiography. Relevant investigations to diagnose the cause of heart failure should be considered, such as exercise tolerance test and coronary angiography for suspected ischaemic heart disease. In the presence of LVH in electrocardiogram and relatively normal heart size on chest roentgenogram one should raise the suspicion of diastolic heart failure. However, echocardiography remains the cornerstone to identify the nature of heart failure as well as its possible aetiology.

Echocardiography

Echocardiography should be performed to all patients presented with CHF. M-mode and 2-D echocardiography is useful to assess the presence and severity of systolic dysfunction. With the combination of colour Doppler studies, causes which precipitate heart failure can be diagnosed such as left ventricular hypertrophy, regional wall motion abnormalities, myocardial infarction and aneurysm, valvular stenosis and regurgitation, mitral annular calcification, cardiomyopathies and pericardial disease. The assessment of diastolic function relies on the use of Doppler echocardiography. By using pulse Doppler studies, mitral flow velocity curve at diastole can be constructed. The commonly described pattern of diastolic dysfunction in diastolic heart failure is abnormal relaxation pattern, in which the early diastolic filling is decreased due to delayed relaxation, while atrial filling fraction and its filling velocity is increased.[43] This pattern is commonly seen in left ventricular hypertrophy and ischaemia.[44] The other end of the spectrum is restrictive filling pattern.[43] It signifies elevated left atrial and ventricular diastolic filling pressure and decreased compliance of the chambers. This pattern is more commonly seen in patients with accompanying left ventricular systolic dysfunction.[45] Occasionally patients can a "pseudonormal" pattern, with represent a transition from one end to the other in the spectrum of severity. This pattern has to be differentiated from normal flow pattern by interrogation of pulmonary venous flow profile.[46] On the interpretation of Doppler-derived diastolic indices, it is imperative to aware of physiological factors which may affect the measurement even in heart failure patients, such as age, gender, heart rate and respiration.

Treatment and Prognosis

General measures for the treatment of heart failure include a low dietary salt intake, oxygen therapy for the presence of hypoxia and the treatment of underlie cause, such as thrombolytic therapy for acute myocardial infarction and anti-arrhythmic agents for cardiac arrhythmias. The principle of treatment includes both decrease the preload and afterload. In general, the afterload should be decreased as much as possible by reducing the systemic resistance so that the workload of the heart can be optimally reduced. Agents useful for preload reduction include diuretics and venodilators such as nitrates. Arterial vasodilators as well as angiotensin converting enzyme inhibitor (ACEI) are agents of choice to decrease afterload. Loop diuretics are commonly used for acute fluid overload, and its dosage should be carefully monitored to avoid excess dehydration which is especially susceptible in elderly patients. There is also evidence of secondary neuroendocrine activation such as activation of renin-angiotensin system which might hasten the pathogenic process of myocardial structural damage.[48] For patients with mild heart failure, a thiazide rather than loop diuretic can be considered.

ACEI is both an arterial and venodilator, which reduces both preload and afterload. It also reduces the circulating and possibly tissue ACE level. It is known by its ability to lower morbidity and mortality of CHF in patients with left ventricular systolic dysfunction.[49] The exercise tolerance and quality of life is also improved. Its use should especially be considered in patients with accompanying diuretics intake or adopted a low salt diet in whom plasma renin is invariably activated. Recently, angiotensin II receptor antagonist (AIIA) is also shown to be effective in reducing mortality in elderly patients with heart failure due to systolic dysfunction.[50] Importantly, there is no significant deterioration of renal function in elderly population by either ACEI or AIIA.[50] On the other hand, renovascular disease is prevalent in elderly patients and these agents should be selected with caution.[51] Furthermore, one should aware of first-dose hypotensive effect of ACEI in elderly and give a lower initial dose since they have a lower renal clearance, which will be further exacerbated by the presence of heart failure.[52] Beta-blocker has been suggested to be a useful treatment more than twenty years but has not been accepted until recently that metoprolol and bisoprolol was shown to have survival benefit in heart failure in multi-centre trials (MERIT-HF and CIBIS-II).[53,54] In CIBIS-II trial there was a 32% reduction of mortality by bisoprolol when compared to placebo,[54] which was 34% in MERIT-HF trial.[53] Other vasodilating beta-blockers such as celiprolol and carvedilol were also shown to be at least equally effective in improve symptoms and exercise capacity.[55,56] These agents also improve left ventricular ejection fraction, but it is only evident after weeks to months, and an initial deterioration of ejection fraction should be alerted.[57] Therefore the drug should be started cautiously with an initially low dose with

close monitoring of hemodynamic status. Digoxin is particular helpful if CHF is accompanying by fast atrial fibrillation. It also possesses positive inotropic effect and sympathoinhibitory effect.[58] The benefit of cardiac glycoside in heart failure symptoms have been proven by large clinical studies.[59] A recent study also confirmed the survival benefit of amiodarone in heart failure, at lease in those with fast resting heart rate.[60] For patients with cardiogenic shock, inotropic support should be considered to improve the cardiac output. Cardiac transplantation is feasible for elderly patients with intractable heart failure. Although this choice is less likely considered for geriatric population, the short-term survival is proven to be comparable to younger subjects.[61] Other surgical measures include myocardial reduction surgery, and surgical treatment for underlying cause such as coronary artery bypass surgery for ischaemic heart disease and valvular replacement for valvular cause of heart failure. Another important aspect of non-medical therapy is the adoption of exercise training in CHF. Exercise training is useful to improve peak oxygen consumption, increase exercise capacity as well as improve quality of life.[62,63] The long-term effect of exercise training and the choice of optimal exercise protocol remain to be determined by randomised clinical trials.

For patients with diastolic heart failure as the sole cause of CHF, ACEI and diuretics are appropriate choices. In human studies, the use of ACEI has shown to be able to regress LVH as well as normalise left ventricular diastolic filling.[64] The use of diuretics in diastolic heart failure should be carefully monitored against the hydration status. Non-discriminatory use of diuretics may jeopardise the diastolic filling and compromise cardiac output since these patients are preload dependent in order to maintain a normal diastolic filling.[65] Other pharmacological agents which may be useful for diastolic heart failure include beta-blocker and calcium channel antagonist. The negative inotropic and/or chronotropic effect of these agents may allow more complete diastolic filling. However, treatment of diastolic dysfunction is still empirical and clinical trials are needed. The underlying cause for diastolic heart failure should be sought and treated such as control of hypertension and revascularisation procedure for ischaemia-induced diastolic dysfunction.

The prognosis for patients with diastolic heart failure is more favourable than those with systolic failure. The reported annual mortality rate is considerably variable, which varies from <5% to >15%.[66,67] The presence of ischaemic heart disease adversely affects the prognosis.[20] In contrast, patients with systolic failure had a much higher mortality.[68-70] In Framingham Study the 6-year mortality rate was 82% and 67% respectively for male and female gender.[15] The prognosis in old-old is particularly worse, and it was reported that the first year mortality was 63% in institutionalised elderly of 80 years or older after diagnosed to have heart failure.[71] In patients with diastolic heart failure, majority of patients are died of pump failure while in systolic failure, sudden cardiac death is also a major cause.[72,73] The presence of restrictive filling pattern in systolic dysfunction signify a worst prognosis, independent of age.[70,74] Another newer marker of mortality is the elevation of natriuretic peptides, especially brain natriuretic peptide.[75]

Ischaemic Heart Disease

Ischaemic heart disease is a common cardiovascular disease in the elderly, primarily because atherosclerosis is a time-dependent process. Although the incidence is lower in middle age women when compared to men, the loss of oestrogen protection render the disease equally prevalent in elderly population. Indeed, ischaemic heart disease is shown to be independently increased with age.[76] Studies found that angina occurs in approximately 10% of the population over 70 years of age.[77] The ageing of population especially in the afferent countries and the change in life style towards a higher cholesterol diet in the developing countries should alert clinicians the increase in incidence of ischaemic heart disease world-wide. On the other hand, the mortality of ischaemic heart disease has been declining in United States,[78] which is explained by the fall in incidence of acute myocardial infarction (AMI). There is an actual fall in death rate after AMI, as a result of improvement in medical therapy and monitoring in coronary care unit. With the advent of thrombolytic therapy and other supportive measures, the in-patient mortality of AMI decreased over 80% in the past three decades.

Clinical Features

There is a spectrum of severity of symptoms for

patient with ischaemic heart disease. This range from stable angina, unstable angina to acute myocardial infarction. Patients with stable angina typically present with exertional retrosternal, compressing chest tightness which radiates to the jaw and arm. Occasionally decubitus angina occur when the patient is lying down, which is related to the increase in venous return and hence ventricular chamber tension, rendering a higher oxygen demand for the greater stroke volume produced according to the Frank-Starling law. When there is angina of increasing frequency, severity or duration, or angina at rest, unstable angina is then assumed. Not uncommonly there are co-existing risk factors, which include cigarette smoking, hypertension, diabetes, hypercholesterolaemia and a positive family history. For acute myocardial infarction, elderly patients are less often presented with chest pain or discomfort, but rather, features of complications such as dyspnoea due to acute pulmonary congestion or even oedema,[79] or acute confusion, syncope or stroke.[80] Though a significant proportion of patients remain complain of chest pain, it may be perceived as less severe or atypical,[81] with less sweating and occasionally even silent myocardial infarction. Patients may also present with fatal arrhythmias which result in sudden cardiac death.

Diagnosis

The diagnosis of angina is largely clinical, though there may be accompanied evidence of ischaemic changes in ECG. However, in the elderly, it is not uncommon to have non-specific resting ST-segment and T-wave changes which make the correct interpretation of ischaemic changes difficult. Exercise tolerance test is indicated for the presence of anginal symptoms, though the diagnostic accuracy is lower for similar reasons. The sensitivity and specificity of the test is especially lowered by the presence of accompanying musculoskeletal disease which prevent patients to achieve the target heart rate. Therefore alternative methods can be considered in this age group such as dobutamine stress echocardiography or dipyridamole radionuclide scanning.

For the diagnosis of AMI, the usual diagnostic triad of chest pain, ST segment elevation at two or more consecutive leads in ECG and elevation of cardiac enzymes remain useful. However, because of the high prevalence of non-specific ST-segment and T-wave changes in the elderly population, as well as the higher incidence of bundle branch block, the diagnosis may be more difficult especially in the absence of typical chest pain.[81] Before the implementation of MB isoenzyme of creatine kinase, the use of creatine kinase alone to diagnose AMI in elderly has less value because of the decreased muscle mass in senescence.

Treatment and Prognosis

The aim of treatment for patients with angina is to decrease the frequency and severity of anginal attacks so as to improve exercise tolerance and quality of life of patients. General measures include patient education about risk factor modification, the prevention of precipitating factors of angina, and the proper way of using sublingual nitrate tablets or spray for symptomatic relief. Life style to be modified include smoking cessation, adopt a low cholesterol diet and weight reduction. In elderly, smoking remains a significant risk factor which is supported by the notion that current smokers have a higher mortality than ex-smokers and non-smokers.[82] The other risk factors for exacerbation of atherosclerosis should also be sought and treated such as medical therapy for co-existing hypertension and diabetes.

Medical therapy for angina in elderly age differs little from younger patients. The choice of anti-anginal drugs include nitrates, beta-blockers and calcium channel antagonist. Since elderly patients have less lean body mass and a hence a lower creatinine clearance, they are more susceptible to side effects of medications. Drugs should be prescribed with caution, especially the effect of bradycardia, heart block or precipitation of heart failure from combined beta-blocker and calcium channel antagonist. In addition, all these agents are able to induce hypotension, especially in combination therapy. It is advisable to start with a lower dose and titrate against symptoms and side effect. Although anti-anginal treatment is effective in decreasing symptoms, they have not been proven to be able to improve long-term prognosis.[83] Other adjunctive measures include the use of aspirin and lipid lowering agent for patients with hypercholesterolaemia. Peptic ulcer or gastritis is a relatively common problem in elderly patients taking aspirin, and other non-prostaglandin dependent inhibitors of platelets such as ticlopidine or clopidogrel should be considered.[84,85] Recently the use of HMG-Co A reductase inhibitors (statins) as primary and secondary prevention has been shown to reduce

myocardial infarction rate and cardiovascular mortality.[86,87] The safety and effectiveness of statin in elderly patients has been demonstrated.[88]

Revascularisation procedure such as percutaneous transluminal coronary angioplasty (PTCA) and coronary artery bypass surgery (CABG) can be considered in selected patients. The elderly patients represent a high risk group for both CABG and PTCA,[89-91] though others suggest that PTCA is a safer procedure in geriatric age group with a higher initial success rate than CABG.[92] However, provided that the overall functional status of the patient is satisfactory, elderly age should not be the sole reason for exclusion of aggressive treatment regimens. Not uncommonly, elderly patients are having more concomitant diseases which largely jeopardise their quality of life, such as chronic obstructive pulmonary disease and musculoskeletal problems. A study found that octogenarians had a higher frequency of CHF, angina, and previous MI.[93] Compared to young adults with IHD, elderly patients are more commonly suffered from multivessel or left mainstem disease.[94] Although procedure complications increase with age, critical complications such as MI, reocclusion, CABG and death were not different from younger age group while primary procedural success was similar in all strata of age.[93] On the other hand, as similar to medical therapy, revascularisation procedures have not been shown to improve survival in patients with stable angina, especially those with preserved left ventricular systolic function. Only in the subgroup with severe coronary disease and impaired left ventricular function that CABG was found to have a survival benefit.[95] Conversely, those patients with no concomitant illness and preserved left ventricular function have excellent prognosis after CABG. A study found that for patients >70 years the 5-year survival rate of multivessel angioplasty was the same as CABG after matched for left ventricular function, though the early morbidity and mortality was higher in the latter group.[96] Therefore revascularisation procedures should be considered for symptomatic control especially for those already on optimal anti-anginal drugs or unable to tolerate the side effects.

In acute myocardial infarction, close monitoring in coronary care unit and prompt treatment of complications such as arrhythmias and heart failure is the key for improvement of acute survival in the past few decades. Agents proven useful to decrease mortality include early thrombolytic therapy, aspirin, beta-blocker, and ACEI in those with significant left ventricular systolic dysfunction. Previously elderly age was a relative contraindication for thrombolytic therapy because of the increased risk of intracranial haemorrhage, especially the use of tissue plasminogen activator (tPA). In earlier studies, patients over 75 years age are excluded from thrombolytic therapy in most of the major clinical trials.[97] However, in ISIS-2 which included all ages for thrombolytic therapy found that even patients older than 70 years had a survival benefit of >30% when compared with aspirin therapy.[98] Further trials consistently showed a survival benefit in the older subgroup.[99] In GUSTO trial, elderly patients were associated with a higher 30-day mortality and were associated with higher bleeding complication and morbidities, though increasing age is confounded by angiographically more severe coronary artery disease and higher incidence of co-morbidities.[100] However, the use of accelerated t-PA remained to have survival benefit. It is prudent to start thrombolytic therapy early, as the shorter the door-to-needle time, the greater the survival benefit.[99] Aspirin has been proven valuable in post-myocardial infarction by randomised, large scale studies.[98] A 23% reduction in 5-week mortality was reported when compared with placebo.[98] Similarly, the use of beta-blocker was able to reduce the first week mortality by 15%, which is likely a result of decreasing acute cardiac rupture.[101] A reduction of mortality by beta-blockers was also demonstrated in elderly AMI patients.[102] Angiotensin converting enzyme inhibitors should also be initiated for patients with evidence of left ventricular systolic dysfunction, as these agents demonstrated a uniform reduction in long-term mortality in large clinical trials.[49,103] However, elderly patients are particularly prone to develop first-dose hypotension and therefore a smaller initial dose with close hemodynamic monitoring is advisable.[52] Other medication should be considered include adjunctive antithrombotic therapy such as intravenous heparin if accelerated t-PA is given, inotropic support for cardiogenic shock and anti-anginal medications for those with accompanying post-infarct angina. Anti-arrhythmic therapy should be prescribed for patients with secondary arrhythmias, though physicians should aware the negative inotropic property of most anti-arrhythmic agents. In elderly patients, the difference in

drug metabolism in elderly patients from younger adults may result in unusual side effects such as acute confusional state after lignocaine infusion. Medical therapy for optimal risk factor modification is important, and the use of statins has been shown to reduce subsequent cardiovascular mortality even for mild to moderate hypercholesterolaemia.[87] Revascularisation procedures are possible modality of treatment though it is less commonly performed for elderly than younger adults. However, for suitable candidates, PTCA or CABG has a role in post-AMI patients for symptomatic control. Age is a predictor of restenosis after primary angioplasty in AMI.[104]

Cardiac rehabilitation programme also plays an increasing role in improving the exercise capacity and the quality of life after AMI, even in elderly patients of older than 75 years.[105] Risk factor modification and education programmes can also be conducted in the rehabilitation programme. Exercise training can also improve oxygen consumption as well as ejection fraction. The long-term benefit of exercise after AMI in the post-thrombolytic era need to be evaluated by further clinical studies. The present data found that control of risk factors especially diabetes mellitus and enhancement of exercise capacity important targets to improve the prognosis for those recruited into the programme.[106]

Elderly patients with AMI were shown to have a significantly higher mortality than younger patients. In one study, increasing age is an independent risk factor of early and late mortality,[107] while other investigators found that in AMI patients older than 70 the mortality was at least 4 times higher than younger patients.[108] However, such a great difference may be partly confounded by the presence of more severe ischaemic heart disease as well as more concomitant medical illness. A recent report explained that the contemporary approach of more aggressive therapy of AMI increased its survival rendering more patients survived to the geriatric age with myocardial scarring, which result in the subsequent development of heart failure and associated mortality.[109]

Valvular Heart Disease

With the improvement in the medical treatment of various diseases worldwide, more individuals now live longer, rendering valvular heart diseases in the elderly become relatively prevalent. This is especially important in the developing countries where acute rheumatic fever is a prevalent cardiovascular disease in the past decades.

Mitral Stenosis

Mitral stenosis is less being encountered nowadays especially in the developed countries. Most of the patients presented in middle-age, though occasionally later in life. The main aetiology is rheumatic mitral stenosis, where accompanying mitral regurgitation is not uncommon. In elderly subjects, calcification of the mitral leaflets and commissures frequently occur and pliability of the valve is affected. Occasionally, mitral stenosis is caused by non-rheumatic mitral annular calcification.[110]

Clinical Features

The clinical features of mitral stenosis is not much different from younger adults. Patients can also be asymptomatic, or present with shortness of breath or even acute pulmonary oedema. Complications of mitral stenosis include pulmonary hypertension, acute pulmonary oedema, atrial fibrillation or thromboembolism. For patients with sinus rhythm, the severity of mitral stenosis and elderly age are predictors of thromboembolism.[111] Examination findings include tapping apex, a load first sound, mid-diastolic rumbling murmur which may only be heard in left decubitus position, an opening snap and signs of pulmonary hypertension. Other findings include a malar flush, atrial fibrillation and a low cardiac output state.

Diagnosis

In the assessment of mitral stenosis, electrocardiography will reveal evidence of p-mitrale, right ventricular hypertrophy, right axis deviation or atrial fibrillation. Chest roentgenography may show double atrial shadow, biatrial enlargement, enlargement of right ventricular silhouette and enlargement of pulmonary arteries. Acute pulmonary oedema in addition to pulmonary venous congestion may result from severe stenosis, which is especially precipitated by co-existing angina. Calcification of the mitral valve leaflets may be evident. Echocardiography with Doppler

studies will confirm the typical doming of the leaflets, and it is important to assess for the severity of stenosis, the presence of pulmonary hypertension, atrial enlargement, mitral regurgitation, and assessment of left ventricular function. The development of scoring system is helpful as an objective guideline of outcome after valvuloplasty, which include the assessment of valvular mobility, thickness, calcification and subvalvular involvement.[112] The use of pressure half-time is a rather accurate method to estimate mitral valve area, which has been shown to correlate well with Gorlin formula at cardiac catheterisation.[113] However, this method is not accurate in the presence of aortic regurgitation and immediately after mitral commissurotomy.[114] Transoesophageal echocardiography is particular helpful for the diagnosis of spontaneous echocardiographic contrast and atrial appendage thrombi which are predictors of subsequent thromboembolic complications.[115,116] Cardiac catheterisation should be performed if surgical treatment is contemplated in view of the high prevalence of ischaemic heart disease in geriatric patients.

Management

Management of mitral stenosis depends on the severity. Mild to moderate stenosis can be managed with medical therapy such as diuretics with regular echocardiography to monitor the progression of the disease. For severe cases, percutaneous transluminal mitral commissurotomy (PTMC) using Inove balloon is useful to relieve symptoms and delay or even obviates the need for valvular replacement.[117] This procedure now replaces close mitral valvotomy. However, in elderly patients, the result is less satisfactory than younger ones as they are associated with more severe mitral calcification and less pliable valves. Open mitral valvotomy can be considered in those with accompanying left atrial thrombus in which removal procedure can be performed in a single operation. Mitral valve replacement can be considered in those with non-pliable mitral leaflets, severe subvalvular involvement, or with significant accompanying mitral regurgitation. Tricuspid annuloplasty can be performed for patients with pulmonary hypertension with significant secondary tricuspid regurgitation. Patients with metallic valve replacement or atrial fibrillation need long-term anticoagulation therapy to prevent the development of thromboembolic complications. A recent study also found that in mitral stenosis the presence of spontaneous echo contrast is the major predictor of thromboembolic complication even a patient is in sinus rhythm, implicating the important of transesophageal echocardiography and the commencement of anticoagulation therapy in this disease group.[118]

Mitral Regurgitation

With the gradual decline in incidence of acute rheumatic fever and its sequel, myxomatous degeneration of mitral valve resulting in mitral regurgitation and mitral valve proplase become more prevalent than decades ago.[119] Other causes of mitral regurgitation include ischaemia-induced papillary muscle dysfunction, organic valvular disease and cardiomyopathy with secondary dilatation of the mitral annulus. Calcification of mitral annulus may be accompanied by some degree of mitral regurgitation. Causes of acute mitral regurgitation include rupture of chordae tendineae or torn leaflet secondary to infective endocarditis.

Clinical Features

Clinical features of mitral regurgitation depends on its underlie aetiology and severity. Mild mitral regurgitation is usually well tolerated by patients, while more severe disease will cause exercise intolerance due to pulmonary congestion. The elevation of diastolic filling pressure will result in diastolic dysfunction and even diastolic heart failure. Occasionally acute onset mitral regurgitation will result in acute pulmonary oedema. Patient may also present with complications such as atrial fibrillation or heart failure, and the latter is reported in up to 40% of patients.[120] Clinically the apical impulse is displaced with a holosystolic murmur on auscultation. The first heart sound can be muffled. Mitral valve prolapse can present with mid or late systolic murmur and a mid systolic click. Mitral regurgitation also at a greater risk of worsening in geriatric patients. In ischaemic papillary muscle dysfunction, patients can have mitral regurgitation due to dyschronised contraction of the papillary muscle base relative to other regions of the heart resulting in disturbance of valvular coaptation. Patient may present with angina or even symptoms of exercise intolerance.

Diagnosis

The diagnosis of mitral regurgitation nowadays rely largely on non-invasive measures such as echocardiography. Colour Doppler echocardiography is useful to assess the severity of regurgitation, as well as the degree of left ventricular and left atrial enlargement. The assessment of valvular pathology and systolic function is essential for deciding the necessity of operative management. Helmcke et al found that the ratio of the regurgitant jet area relative to the left atrial area correlated with angiographic severity.[121] The use of transoesophageal echocardiography is particularly helpful to assess valvular morphology and the presence of left atrial clot or spontaneous echocardiographic contrast as in case of mitral stenosis. In addition, the presence of reversal of systolic forward flow in the pulmonary vein by Doppler study also implicate severe mitral regurgitation. It is worth to note that some degree of mitral regurgitation detected by Doppler echocardiography is very common in the elderly which is found in up to half of normal subjects and therefore the finding should be interpreted with caution.[122] Cardiac catheterisation is also indicated especially for potential surgical candidates to define the severity of valvular regurgitation and to detect silent coronary artery disease in otherwise asymptomatic subjects.

Management

For those who have mild to moderate mitral regurgitation, medical therapy is preferred, which consists diuretics, angiotensin converting enzyme inhibitor or other vasodilators for afterload reduction. Complications such as atrial fibrillation and heart failure should be treated promptly. In view of the greater risk of operation in the elderly than younger patients,[123,124] surgery should be considered for symptomatic patients with severe disease before there is significant left ventricular dilatation and systolic dysfunction. In general, mitral valve reconstruction is the preferred approach than valvular replacement if technically feasible, which is reported to carry a lower surgical mortality.[125]

Mitral Annular Calcification

This is a very common condition in octogenarian patients, which is found to be more than half of the population, with female preponderance. Mitral annular calcification is associated with conditions such as aortic sclerosis, mitral valve prolapse, ischaemic heart disease, and systemic illness such as hypertension and chronic renal failure. It represents a degenerative process in the valves, which range from small calcified nodules to involve the entire valve cusps. Clinically mild to moderate mitral annular calcification are asymptomatic. In severe cases, it may present with mitral regurgitation due to the impaired ability of the mitral annular to reduce its circumference during systole and the decreased ability of coaptation, and displacement of mitral leaflets from their normal anatomic position. Other manifestations include heart block due to conduction bundle erosion by the calcification, mitral stenosis or embolic phenomenon.[126] The presence of mitral annular calcification increases operative difficulties for mitral valve repair with worsening operative risk.[127] Mild to moderate calcification in general does not require treatment, and only those with complications need to be treated accordingly.

Aortic Stenosis

Aortic stenosis is a very common form of valvular heart disease in the elderly. The most frequently encountered cause is senile calcific degeneration of the aortic valves, and hence the incidence rises with increasing age. At Mayo Clinic, it is reported that aortic stenosis comprised up to half of the cases referred for valvular operation in those who are 70 years or older. Other aetiologies include rheumatic heart disease,[128] congenital bicuspid aortic valve with subsequent calcification and congenital commissural fusion. In contrast, in senile aortic stenosis, typically the aortic leaflet is free of commissural fusion, but rather with nodular calcification within the aortic pockets.[129]

Clinical Features

Mild aortic stenosis is usually asymptomatic. Patients with severe disease may have features of exertional angina and syncopy. The physical signs for aortic stenosis can be misleading. In elderly subjects, murmur of aortic sclerosis without significant stenosis is frequently present. Conversely, the typical harsh ejection systolic murmur at the right second intercostal space that radiate to the carotid arteries may peak early

and the intensity of the aortic second sound may not be markedly reduced, leading to the underestimation of the severity.[130] In addition, a plateau pulse may not be obvious even in severe aortic stenosis. There may be accompanied signs of pressure overloading apical impulse due to LVH, and co-existing aortic regurgitation murmur. Occasionally patients may present with complication of aortic stenosis such as heart failure or infective endocarditis.

Diagnosis and Treatment

With the use of Doppler echocardiography, the need for invasive investigation is largely obviated except for those with severe disease in whom surgery is considered. Doppler echocardiography can accurately estimate transvalvular pressure gradient and aortic valve area. In addition, patients might have left ventricular hypertrophy in ECG and cardiomegaly in chest roentgenography. Because of the relatively high surgical mortality of aortic valve replacement in geriatric patients, patients who are asymptomatic even with severe aortic stenosis should probably be treated conservatively with close clinical monitoring.[131] However, those with symptoms of angina, dyspnoea and syncopy should be evaluated for considering surgery and CABG should coronary artery disease is detected by coronary angiography. Since the presence of left ventricular systolic dysfunction is the most important predictor of mortality in elderly undergoing aortic valvular replacement, surgery should therefore be performed before there is any significant cardiac decompensation.[132] Both open and close aortic valvotomy had disappointing results because of restenosis. Percutaneous balloon aortic valvuloplasty has acute success with improvement in leaflet mobility and orifice area, though the vascular complication rate is as high as 15%[133] and the long-term result is unsatisfactory with late death reported in nearly one-third of patients.[134] Therefore this procedure is used for palliation of symptoms for those who has poor surgical risk due to co-morbidities.

Aortic Regurgitation

Although severe aortic regurgitation is uncommon in the elderly, some degree of aortic regurgitation is prevalent.[135] In this age group, aortic root dilatation is the predominant cause. Other causes include chronic rheumatic heart disease, myxomatous degeneration, seropositive and seronegative arthritis. Although in most cases no underlying cause is found, aortic regurgitation is associated with hypertension in about 10% of patients, which is usually mild in severity.[136]

Clinical Features

Usually elderly patients are presented with chronic aortic regurgitation, though occasionally acute presentation can be due to infective endocarditis or associated with aortic dissection. The majority of patients with chronic disease are asymptomatic. Occasionally there are symptoms of palpitation, fatigue or angina. Examination will reveal a wide pulse pressure, displaced apical impulse, and a high pitch early diastolic decrescendo murmur over the sternal border. Severe aortic regurgitation may give rise to peripheral signs of hyperdynamic circulation such as Corrigan sign and pistol-shot femoral. A systolic murmur, long diastolic murmur, the presence of Austin-Flint murmur, or occurrence of third heart sound will signify significant aortic regurgitation.

Diagnosis and Treatment

The most useful investigation is probably the use of Doppler echocardiography. The visualisation of valvular anatomy allows the differentiation of aortic root dilatation from valvular cause of aortic regurgitation. Methods used to assess the severity include the reversal of thoracic aortic flow,[137] presystolic mitral valve closure or mitral regurgitation, a short aortic pressure half-time,[138] and a large ratio of regurgitant jet to left ventricular outflow tract width.[139] An integrated approach by a combination of various Doppler echocardiographic parameters has also been described recently.[140] The treatment of chronic aortic regurgitation is usually medical, which comprise the use of vasodilators to decrease the afterload of the heart such as hydralazine, calcium channel blocker and ACEI.[141] The use of ACEI has also been shown to regress LVH associated with volume overload.[142] The use of nifedipine has been shown to be able to improve cardiac output and reduces the need for aortic valve replacement.[143,144] Patients with mild to moderate aortic regurgitation usually have a good long-term prognosis, with a 10-year survival rate of about 90%.[145] Patients with severe aortic regurgitation should be closely monitored and aortic valve replacement considered shall

they develop symptoms of exercise intolerance or progressive left ventricular dilatation which herald the onset of left ventricular systolic dysfunction. It has been reported that elderly patients stand a higher risk of mortality for aortic valve replacement than younger subjects.[146]

Hypertension

Hypertension is more than a disease of ageing. The detrimental cardiovascular effect of high blood pressure such as myocardial infarction, heart failure and stroke is more prevalent in elderly hypertensives. In Framingham study the cardiovascular event risk is at least twice higher in those older than 65 year-old hypertensives than the younger subjects.[147] The prevalence of hypertension in the older population varies according to the definition. For a commonly used blood pressure cut-off of 160/95 mmHg, the prevalence is 30% in elderly men, and an additional 10-15% of total elderly population have isolated systolic hypertension.[148]

Clinical Features

In general the clinical features of hypertension in elderly are similar to young adults. However, one should aware of the existence of generalised atherosclerosis in elderly who may present with accompanying symptoms, such as acute pulmonary oedema in bilateral atherosclerotic renal artery stenosis who are previously asymptomatic. In addition, hypertensive complications are common presentation, such as congestive heart failure which can be due to diastolic dysfunction or pump failure,[25,149] and acute myocardial infarction. Since blood pressure in elderly is more labile than in the middle age, it should be measured repeatedly (at least in three different occasions) and in different postures (sitting and standing) in order to establish a baseline value for the diagnosis.[150] As secondary cause of hypertension is less likely, the investigation is usually directed towards the detection of cardiovascular complications caused by atherosclerosis such as carotid stenosis, ischaemic heart disease and renal artery stenosis when bruits are heard.

Management

In the management of hypertension, medical therapy for optimal blood pressure control remains the main goal. It is imperative to achieve a satisfactory blood pressure level as it is closely related to complication rate. A few clinical trials addressed the importance of drug treatment in elderly hypertensives. In Veterans Administration Study, medical treatment of patients aged 60 years or above significantly decreased cardiovascular events by 54% when compared to placebo.[151] Similarly, in Australian National Blood Pressure Study, treatment of patients aged 60 to 69 years reduced fatal and non-fatal cardiovascular events by 36%, although in significant.[152] Another trial which specifically conducted to assess the effect of antihypertensive therapy in 840 elderly found that the 5-year cardiac mortality was significantly decreased by 47% when compared with placebo, although the all-cause mortality was insignificantly reduced by 26%.[153] In another clinical trial comparing routine and intensified antihypertensive treatment, there was a 16% reduction in all-cause mortality in elderly age group.[154] Other investigators also reported a decrease in incidence of stroke.[155] The Hypertension Optimal Treatment addressed the medical treatment of hypertensive adults aged from 50 to 80 years that the optimal systolic and diastolic blood pressure for lowest cardiovascular mortality was 138.8 and 86.5mmHg respectively.[156]

The choice of antihypertensive agents in the elderly are not much different from the usual guideline, though one should aware of the higher vulnerability of side effects. Some patients may respond to monotherapy though it might be necessary to use combination of different agents for optimal blood pressure control as well as minimising the side effect of individual agent. Thiazide diuretics and β-blockers are commonly prescribed as first line agents.[157] For the former, potential risk of cardiac arrhythmia due to hypokalaemia should be alerted especially in those with co-existing LVH.[158] Regular monitoring of plasma electrolytes and adding a potassium-spearing diuretics is recommended. Although diuretics may adversely affect lipid profile, the effect is small and easily corrected with a diet low in cholesterol. The use of low dose diuretics (with potassium sparing) has been proven to reduce both stroke and myocardial infarction in the elderly.[159] Beta-blocker is advantageous in patients with accompanying ischaemic heart disease, though it should be avoided in those with peripheral vascular disease and chronic obstructive pulmonary disease. The theoretical disadvantage of decreased responsiveness and tolerability to β-blockers in relation to ageing has not been confirmed to have clinical significance.[160] The use

of newer agents for hypertensive control is gaining increasing popularity, namely ACEI, calcium channel blocker and AIIA. Despite having a lower plasma level of renin activity in the elderly than younger individuals, elderly patients respond equally well to these agents as evident by post-marketing surveillance studies.[161,162] The occurrence of side effect is also non age-dependent.[163] For the use of ACEI, long-acting ones are now in favour because of the higher trough to peak ratio, which should confer better blood pressure lowering effect. In the elderly, the use of ACEI may increase the susceptibility to orthostatic hypotension.[52] They are also having a higher risk of first-dose hypotension especially in the presence of risk factors such as renal impairment, hyponatraemia, low plasma volume or receiving high dose diuretic treatment. This can be alleviated by the use of longer-acting ACEIs.[164] In addition, the pre-existing or new occurrence of renal dysfunction should alert the possibility of renal artery stenosis. ACEI is preferred in patients with co-existing congestive heart failure, though it should be introduced even more cautiously with a lower initial dosage. The use of AIIA has been proven to be safe and effective in elderly hypertensives, and first-dose hypotension seems not a problem.[165] However, long term tolerability studies are needed. A large, prospective, randomised clinical trial is underway to compare ACEI and diuretic therapy in the reduction of cardiovascular events in elderly hypertensive subjects.[166] The use of calcium channel blocker in elderly is at least as effective as in younger hypertensive patients.[167] Compliance of medical treatment is a potential problem in the elderly and therefore medical regimens should be as simple as possible.

Atrial Fibrillation

Atrial fibrillation (AF) is the commonest cardiac arrhythmia in clinical practice. Its prevalence doubles with each advancing decade of age, from 0.5% at 50-59 years to nearly 9% at 80-89 years.[168] Also, the disease is more prevalent nowadays than 30 years ago when comparing patients of similar age groups,[168] and is present in 2% of an elderly population low in the incidence of coronary artery disease.[169] In the follow up of Framingham heart study, people who subsequently developed AF had increased risk of mortality, which is 1.5 and 1.9 fold in men and women respectively.[170] In contrary to the young adults in whom lone AF is more common, elderly patients develop AF usually secondary to cardiac disease, namely ischaemic heart disease, hypertension, sick sinus syndrome and various cardiomyopathies.[171] Not uncommonly, the occurrence of AF in the elderly is secondary to cardiac surgery, such as bypass surgery and valvular replacement, and non-cardiac conditions, such as pneumonia, thyrotoxicosis, major pulmonary embolism, diabetes and alcoholism. The Framingham heart study found that AF developed in 12% of population aged 28 to 62 years after 40 years of follow-up.[170]

From the duration and electrical behaviour of AF, it can be divided into recent onset and chronic ones. The chronic AF can be further classified into paroxysmal AF (converted spontaneously), persistent AF that required medical or electrical cardioversion, and permanent AF which refers to those either unable or have a low chance to be converted back to sinus rhythm by any means.

Clinical Features

Patients with AF usually have symptoms of palpitation, dyspnoea and impaired exercise tolerance.[172] The symptoms of decompensation is particularly obvious in advance age who depends more on the normal atrial kick to maintain an adequate diastolic filling. In acute onset and paroxysmal AF, symptoms may be less well tolerated than in chronic AF. The occurrence of acute AF in elderly can be a manifestation of underlie condition which should be investigated vigorously, such as thyrotoxicosis and pulmonary embolism. When there is a fast ventricular response, the risk of developing cardiac failure is increased, especially those with underlie structural cardiac disease such as old myocardial infarction and LVH. AF is also associated with tachycardia-induced cardiomyopathy and the irregular ventricular rate itself also predispose to the development of cardiac failure.[173,174] The other major complication of AF which resulting in morbidity and mortality is related to thromboembolism, especially stroke. The risk of stroke is increased by five- and seventeen-fold in non-rheumatic and rheumatic AF respectively. Age is another independent risk factor, and the stroke incidence in lone AF increases from 1.6% to 3% during the seventh to ninth decades of life.[175] Other risk factors for stroke

in AF include cardiac failure, symptomatic ischaemic heart disease, hypertension, diabetes, and importantly, previous stroke or transient ischaemic attack.[176] The occurrence of atrial thrombosis is likely related to atrial mechanical dysfunction (atrial stunning)[177,178] and possibly a hypercoagulable state due to activation of platelets or components of the coagulation cascade.[179,180]

Management

The management principles of AF include treatment of precipitating factors, restoration of sinus rhythm, rate control and anticoagulation prophylaxis. Restoration of sinus rhythm should be the primary goal since cardiac function (heart rate, diastolic filling and systolic function) can be normalised, while thromboembolic complication and side effect of anti-arrhythmic medication can be avoided. Cardioversion should be considered for every patients where possible. Synchronised electrical DC cardioversion is the choice for those with haemodynamic compromise. Apart from the conventionally used transthoracic external shock, internal defibrillation by transvenous catheter using low-energy shock is also performed by some centres. Pharmacological cardioversion is less effective than electrical cardioversion, though it obviates the need for sedation. The choice of drugs include class Ic (propaferone, flecanide), class III (amiodarone, sotalol, ibutilide) and class Ia agents (quinidine). For patients with impaired left ventricular function on echocardiography, amiodarone will be a better choice by virtue of its devoid of negative inotropic effect. For a AF duration of 48 hours or longer, anticoagulation should be started for 3 weeks before cardioversion, and at least for another 4 weeks after the procedure to minimise the risk of thromboembolism. However, atrial thrombi had been reported in acute AF of less than 3 days.[181] In the situation where emergency cardioversion is contemplated, intravenous heparin should be given to cover the procedure. Some centres might perform transoesophageal echocardiography to confirm the absence of atrial appendage thrombi before cardioversion in place of coumardin pre-treatment.[182] New development include the use of implantable device for internal fibrillation[183,184] and transvenous catheter ablation for focal atrial fibrillation.[185,186] Patients with persistent AF may also benefit from surgical procedures such as Corridor or Maze operation, which has been shown to be possible to restore sinus rhythm even in those with giant left atrium.[187] However, these procedures are associated with significant morbidity and mortality, and are at present used in a handful of patients in specialised centres.

The aim of rate control is to minimise symptom related to fast ventricular response for those suffering from permanent or paroxysmal AF. The potential difference of outcomes of this approach versus rhythm control is currently under investigation.[188] Pharmacological therapy of choice include β-blockers, calcium channel blockers in the phenylalkylamine (e.g. verapramil) and benzothiazepine (e.g. diltiazem) classes and digoxin. The latter agent has less effect in exercise-induced tachycardia, though its inotropic effect will favour the use in heart failure. More invasive therapy will be reserved for medically resistant AF. This includes the use of catheter-based atrioventricular modification or ablation with pacemaker implantation.[189,190]

Aspirin has been used as prophylaxis for thromboembolism for younger patients with lone AF. However, patients at higher risk of thromboembolism should be anticoagulated with warfarin, including elderly age itself. The benefit of warfarin in the prevention of stroke outweighs the risk of haemorrhage and should be considered in elderly unless contraindicated.[191] The use of warfarin has also been reported to reduce silent cerebral infarction in elderly patients with atrial fibrillation.[192] Indeed, it was found that anticoagulation therapy was underutilised in elderly and a study found a anticoagulation rate of only about 50% of eligible patients.[193] However, elderly might need more close monitoring of their INR in order to avoid over-anticoagulation and its associated higher haemorrhagic risk.[194] The fact that Chinese ethnics and elderly age require a lower maintenance dose also need to be entertained.[195]

Conclusion

Cardiovascular diseases are more common in the elderly population, primarily lots of these conditions are degenerative in nature, together with increases in risk factors of diseases. Management of these diseases in elderly can be different from younger patients, and special focus should be put onto the effect of ageing on

the pharmacokinetics and pharmacodynamis of medication, the overall medical and physical condition as well as co-existence of other organ dysfunction. On the other hand, standard therapy should not be limited because of age alone, which could involve invasive investigation and aggressive treatment modalities.

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