

# Cardiovascular Health in Elderly

Arvind Mathur

---

Cardiovascular diseases are the primary cause of death in older adults, and among those without clinical disease, high levels of subclinical disease are associated with poor survival.<sup>1</sup> Vascular disease is so strongly age related that it has been proposed as a biomarker of ageing.<sup>2</sup> Studies of “healthy ageing” or “successful ageing” show that those who survive with intact health or function have low levels of modifiable risk factors for common chronic diseases, particularly risk factors for cardiovascular disease.<sup>3-6</sup> Risk factor modification can prevent cardiovascular events and mortality in older adults. *Successful ageing* means remaining free of major, life-threatening chronic diseases i.e. no prior diagnosis of cancer, an absence of cardiovascular disease (angina, myocardial infarction, cardiac revascularization procedure, congestive heart failure, stroke, transient ischemic attack, or claudication), no chronic obstructive pulmonary disease (chronic bronchitis, emphysema, or asthma), no reported difficulty with any activities of daily living (ADL), and a modified Mini-Mental State Examination score in the 80<sup>th</sup> percentile or higher, and having normal physical and cognitive functioning.<sup>7</sup>

Besides lipid levels, diabetes, sedentary lifestyle, and genetic factors, advancing age is an independent risk factor for coronary artery disease, hypertension, congestive heart failure, and stroke which are the quintessential cardiovascular diseases within our society. The prevalence of these diseases increases steeply with advancing age. Recent studies of subclinical cardiovascular disease suggest that some measures of the extent of vascular disease are associated with adverse health effects even in those with no symptoms of vascular disease or history of myocardial infarction, stroke, or other vascular events.<sup>8-9</sup> For example, patients with subclinical peripheral arterial disease have

mobility impairment even without claudication, those with white matter changes on brain magnetic resonance imaging but no history of stroke have demonstrably lower levels of physical performance, and those with subclinical disease appear to be more frail.<sup>10,11,12</sup> These data suggest that subclinical cardiovascular disease has adverse effects on health and function and, in addition to causing morbidity from heart disease directly, may contribute to disability and cognitive impairment.

There are several possible explanations for the dominant effect of age on the likelihood for occurrence of these cardiovascular diseases. Increasing age contributes to an increased exposure time to these other age dependent risk factors. In addition cardiovascular structure and function change with time due to “ageing process”, and this process alters the substrate on which specific pathophysiological disease mechanisms become superimposed. Accordingly the enhanced risk for older persons is due to age disease interaction. Age impacts the severity of disease manifestations for a given time at risk. Thus, age associated changes in cardiovascular structure and function become “partners” with pathophysiological disease mechanisms to determine the threshold, severity, and prognosis of cardiovascular disease occurrence in older persons. However, the true interactions are more complex and involve age, multiple risk factors, and genetics.<sup>13</sup>

To define why age (or an ageing process x exposure time interaction) is a major risk factor for cardiovascular diseases, the specific component of the risk associated with age is being looked for. Two complimentary approaches have evolved. On the one hand, epidemiologists are searching for novel measures of “sub clinical disease” (in addition to the more established risk factors that have already been well characterized) in large, unselected study cohorts composed of persons both with and without cardiovascular disease. In contrast, gerontologists are

---

Associate Professor of Medicine, In-charge Geriatric Clinic,  
M.D.M. Hospital, Dr. S.N. Medical College, Jodhpur

*Address for correspondence:*

II/1, M.D.M Hospital Campus, Shastri Nagar, Jodhpur  
email: mathur\_arvind@yahoo.com

attempting to develop quantitative information on cardiovascular structure and function in apparently healthy individuals to define and target the specific characteristics of ageing that render it such a major risk factor for cardiovascular disease, even in the absence of clinically apparent comorbidities.

## **Age associated changes in cardio vascular system**

### **Arterial changes**

Age associated changes in the arterial properties of healthy individuals have relevance to the steep age-dependent increase in vascular diseases. With age, arterial wall thickening and dilatation of large elastic arteries, endothelial dysfunction, arterial stiffening, and arterial pulse pressure widening are prominent changes.<sup>14</sup> Combinations of these processes occurring in varying degrees determine the overall vascular ageing profile of a given individual. Additional risk factors, including hypertension, smoking, dyslipidemia, diabetes, diet, and heretofore unidentified genetic factors, interact with vascular ageing to activate an atherosclerotic plaque. These traditional risk factors may themselves accelerate ageing of the vascular wall.

The aortic wall thickening that occurs with ageing consists mainly of intima media (IM) thickening which is independent of atherosclerosis.<sup>15</sup> There is marked heterogeneity in IM thickness among individuals of a given age. The increased IM thickness is an independent predictor of future cardiovascular events.

Age associated increase in IM thickening is accompanied by both luminal dilatation and a reduction in compliance or distensibility, with an increase in vessel wall stiffness. Pulse wave velocity (PWV), a relatively convenient, noninvasive index of vascular stiffening, increases with age both in men and women. The structural alterations in the vascular media include increased collagen, reduced elastin content, elastin fractures, and calcification. The cross-links, due to nonenzymatic glycation, which increase with age and markedly with diabetes contribute to age and disease related increase in large artery stiffening. However, the stiffness is governed not only by the structural changes within the matrix, as noted above, but also by endothelial regulation of vascular smooth muscle tone and of other aspects of vascular wall structure/function. Thus, there is evidence of a vicious cycle: altered

mechanical properties of the vessel wall influence the development of atherosclerosis and the latter, via endothelial cell dysfunction and other mechanisms, influence vascular stiffness.<sup>16</sup>

As large arteries become stiff, central systolic arterial pressure increases, diastolic arterial pressure decreases, and the pulse pressure increases for a given pattern of left ventricular ejection. The elevated levels of pulse pressure are associated with progression of IM thickening which in turn, is associated with widening of pulse pressure. Numerous studies have concurred that elevated pulse pressure is an independent risk factor for future cardiovascular events.<sup>17-20</sup> Thus, an increased PWV reflects three potential risk factors: increased systolic pressure, widened pulse pressure, and altered vascular wall properties. The elevated pulse wave velocity and reduced total systemic compliance assessed by stroke volume/pulse pressure, over and above blood pressure, are independent predictors of cardiovascular events. This suggests that altered structure/function of the stiff vessel wall, in addition to the associated increase in systolic arterial pressure and pulse pressure, is a risk factor for future vascular events.<sup>21</sup>

In addition to stroke volume, arterial pressure is determined by the interplay of peripheral resistance and central artery stiffness; the former raises both systolic and diastolic pressure to a similar degree, whereas the latter raises systolic but lowers diastolic pressure. Pulse pressure is a useful hemodynamic indicator of conduit artery vascular stiffness. Framingham investigators and others have reported an age dependent rise in average systolic blood pressure across all adult age groups. In contrast, average diastolic pressure was found to rise until 50 years of age, level off from ages 50 to 60, and decline thereafter. The age dependent changes in systolic, diastolic, and pulse pressure are consistent with the notion that in younger individuals, blood pressure is determined largely by peripheral vascular resistance, whereas in older individuals, it is determined to a greater extent by central conduit vessel stiffness.<sup>18-20</sup>

Owing to the decline in diastolic pressure in older men and women in whom systolic pressure is increasing, isolated systolic hypertension emerges as the most common form of hypertension in individuals over the age of 50.<sup>22</sup> Isolated systolic hypertension, even when

mild in severity (stage 1), is associated with an appreciable increase in cardiovascular disease risk.<sup>18</sup> Based on long-term follow up of middle-aged and older subjects, however, Framingham researchers have found pulse pressure to be a better predictor of coronary disease risk than the systolic or diastolic pressure.<sup>19</sup> When considered jointly with the systolic blood pressure in older subjects, diastolic blood pressure is inversely related to coronary risk. Consideration of the systolic and diastolic pressures jointly may be preferable to consideration of either value alone.<sup>18,19,22</sup>

Vascular ageing and vascular diseases are partners; each contributes specific components to what is presently referred to as "vascular disease." Thus, what clinical medicine and epidemiology now refer to as vascular disease should be regarded as the "vascular ageing-vascular disease interaction."<sup>13</sup> Ageing blood vessels provide the milieu in which vascular diseases can flourish. If vascular ageing is a risk factor for disease, then age-associated vascular changes represent a potential target for treatment and prevention.

### **Cardiac Changes**

There is a continuum of expression of cardiac structural and functional alterations that occurs with age in healthy humans, and these age-associated cardiac changes seem to have relevance to the steep increase in left ventricular hypertrophy, chronic heart failure, and atrial fibrillation seen with increasing age.<sup>23</sup>

### **Cardiac structure**

The left ventricular (LV) wall thickness increases progressively with age in both sexes. Cardiac myocyte enlargement and decrease in the estimated myocyte number was observed at autopsy in older hospitalized patients without apparent cardiovascular disease. An increase in the amount (focal increases) and a change in the physical properties of collagen (purportedly due to nonenzymatic cross-linking) also occur within the myocardium with ageing.

### **Left ventricular diastolic function**

The left ventricular early diastolic filling rate progressively slows after the age of 20 years, so that by 80 years the rate is reduced, on an average, up to 50%.<sup>24</sup> Structural (fibrous) changes within the left ventricular myocardium or residual myofilament calcium activation from the preceding systole are putative

mechanisms for a reduced early diastolic left ventricular filling rate. Despite the slowing of left ventricular filling early in diastole, more filling occurs in late diastole, due in part, to a more vigorous atrial contraction, which produces an exaggerated A wave. The augmented atrial contraction is accompanied by atrial hypertrophy and enlargement and on auscultation is manifested as a fourth heart sound (atrial gallop). Multiple regression analyses indicate that age is the major determinant of the E: A ratio; hence, the age-associated decrease in the doppler transmittal E: A ratio is identical in healthy Baltimore longitudinal study on ageing (BLSA) participant and in Framingham study participants.<sup>25, 26</sup>

Despite the age associated changes in the diastolic filling pattern in older healthy persons, their left ventricular end diastolic volume index (EDVI) (end diastolic volume normalized for body surface area) in the supine position is not compromised and does not substantially differ from their younger counterparts. There are altered responses of cardiac volumes to postural maneuvers associated with ageing.<sup>27</sup>

### **Left ventricular systolic function**

The LV ejection fraction (EF), the most commonly used clinical measure of LV systolic performance, is preserved during ageing. However, there is age-associated failure to augment EF with exercise due to a remarkable age associated deficit in the ability to reduce end-systolic volume index (ESVI); the acute ESV reserve at age 85 is only about one-fifth of that at age 20, and there is a similar age associated loss of EF reserve.

The net result of the age associated changes in EDV and ESV regulation during exercise is that the stroke volume index (SVI) is preserved in these older persons over a wide range of performance demand because of a greater use of the Frank Starling mechanism.

### **Heart rate and cardiac output**

In the supine position at rest, heart rate (HR) in healthy men does not change with age. With assumption of the seated position from the supine position, HR increases slightly, but significantly less in older than in younger men.<sup>27</sup> The maximum heart rate during exhaustive, dynamic exercise decreases with age, and the magnitude of this age-associated reduction in peak HR is about 30% between 20 and 85 years of age.. The reduction in HR response to exercise is the reason

why the maximum acute cardiac output reserve in healthy volunteers decreases, on average, by about 30% between ages 20 and 85 years. Healthy individuals at the older end of the age range can augment their cardiac index 2.5 fold, whereas those at the younger end of the age spectrum can increase their cardiac index 3.5 fold.

### **Left ventricular after load and vascular-ventricular load matching**

Cardiac afterload has two components, one of which is generated by the heart itself and the other generated by the vasculature. The cardiac component of afterload during exercise can be expected to increase slightly with age because the heart size increases in older persons throughout the cardiac cycle during exercise.<sup>28</sup> The vascular load on the heart has four components: conduit artery compliance characteristics, reflected pulse waves, inertance, and resistance. There is considerable evidence to indicate that each becomes altered during ageing and that at rest, the vascular load on the LV increases with age. Increased vascular loading on the heart is a likely cause of the increase in LV wall thickness associated with ageing.

There is optimal and efficient ejection of blood from the heart when ventricular and vascular loads are matched. It has been suggested that the precise cardiac and vascular load matching that is characteristic in younger persons is preserved at older ages, at least at rest, because the increased vascular stiffness in older persons at rest is matched by increased resting ventricular stiffness.<sup>29</sup>

### **Heart rhythm**

Beat-to-beat fluctuation of heart rate, commonly known as heart rate variability, declines steadily with age. Reduced heart rate variability is an indicator of cardiac autonomic regulation commonly found in older people and has been linked to increased risk for morbid and fatal outcomes.<sup>30</sup>

An increase in the prevalence and complexity of both supraventricular and ventricular arrhythmias e.g. isolated atrial premature beats, short bursts of paroxysmal supraventricular tachycardia, atrial fibrillation, and ventricular ectopy, occurs in otherwise healthy older patients. Neither the prevalence nor the complexity of these is a determinant of future coronary events.

### **Deficits in sympathetic modulation of heart rate and LV contractility**

All of the factors that have been identified to play a role in deficient cardiovascular regulation with ageing, including HR (and thus filling time), afterload (both cardiac and vascular), myocardial contractility, and redistribution of blood flow, exhibit a deficient sympathetic modulatory component.

Depending on the duration of the stress, enhanced or deficient neurotransmitter release might be a basis for apparent impairment of sympathetic cardiovascular regulation that occurs with ageing.

The age associated increase in neurotransmitter spillover into the circulation during acute stress implies a greater cellular receptor occupancy by these substances, which leads to desensitization of post-receptor signaling.

Potential age associated changes in the tissue levels or responses to growth factors, catecholamines, angiotensin II, endothelin, tumor growth factor  $\beta$  (TGF $\beta$ ), or fibroblast growth factor (FGF) that influence myocardial or vascular cells or their extracellular matrices may also have a role.<sup>14</sup>

Newman et al examined a composite measure of several noninvasive measures of the extent of cardiovascular disease including carotid doppler study, ankle-arm index (AAI), and electrocardiogram findings. The composite measure of any sub clinical disease was defined as presence of any of the following: common or internal carotid wall thickness above the 80th percentile of the distribution for all participants at baseline, maximum percent stenosis of the internal carotid artery greater than 25%, AAI of 0.9 or less, any major electrocardiogram abnormality, or Rose questionnaire findings that were positive for angina or claudication.<sup>12, 31</sup>

Men and women who reach old age in good health without disability or cognitive impairment vary widely in the likelihood of continuing to age successfully. The initial age at study entry was the strongest predictor of continued success, but even within age strata the number of expected years of success was strongly related to other factors, including the extent of subclinical vascular disease and health habits. Participants without sub clinical vascular disease continued to successfully age approximately 5 to 6 years longer than those with

sub clinical disease (women 6.5 years, men 5.6 years).<sup>31</sup>

The independent association of diabetes mellitus, smoking, and physical activity with fewer years of success is important because these risk factors are modifiable. Furthermore, it suggests that their adverse effects on maintaining health and function may be mediated by pathways other than vascular disease. A study in the Honolulu Heart Program cohort showed that smoking, diabetes mellitus, and hypertension are common factors underlying a composite outcome of successful ageing.<sup>4</sup> Smoking and physical activity were shown to dramatically affect active life expectancy in the MacArthur studies of successful ageing as well.<sup>32</sup> C-reactive protein level and increased physical activity were also associated with successful ageing. Higher CRP levels have previously been documented to predict mortality, cardiovascular disease, and its related events in older adults. Inflammation as measured by interleukin 6 has been shown to predict disability, while associations with CRP are less consistent. Factors that predict higher levels of disability include smoking and obesity, but the pathophysiologic basis of these associations is not well defined. Additional work is needed to examine these factors, as well as other factors such as insulin like growth factor, sex steroid hormones, and homocysteine levels that are hypothesized to affect the ageing process.<sup>33-37</sup>

There are increasing opportunities to identify genetic factors that might affect the age of onset of vascular disease. There is also a growing recognition that there may be protective genetic factors that result in delayed onset of vascular disease, even in the presence of modifiable risk factors such as smoking and cholesterol.<sup>38</sup> Family history also continues to predict vascular disease in late life, and its role is being examined in longevity and successful ageing.

Improving the quality of late life and maintaining intact health and function are important public health goals. Prevention of cardiovascular disease should be a major priority for the achievement of successful ageing. If the burden of vascular disease is shifted to later phase in life, the age related trajectory of decline would be attenuated by several years. Current treatments for cardiovascular risk factors, including smoking cessation, lipid lowering, blood pressure treatment, and avoidance of obesity through diet and exercise, are

underutilized. Recent recommendations for risk factor modification suggest that markers of the extent of subclinical vascular disease may be useful to target intervention in older adults, especially those without elevations in cardiovascular risk factors.<sup>39</sup>

Lifestyle interventions or pharmacotherapies to retard the rate of progression of subclinical disease might be considered before the clinical disease becomes manifested. Lifestyle changes are an effective approach to cardiovascular disease prevention. Patient education and counseling on nonpharmacologic preventive strategies like moderate exercise, proper diet, weight reduction and smoking cessation are useful.

With respect to lifestyle, the risk factor of lack of vigorous exercise increases dramatically with age in otherwise healthy persons. It is noteworthy that pulse pressure, PWV, and carotid augmentation index are lower and baroreceptor reflex function is improved in older persons who are physically conditioned compared with sedentary persons.<sup>40</sup> Exercise conditioning also improves endothelial function in older persons.

Preventive pharmacotherapies are efficacious and cost effective in the older population. Antiplatelet therapy, ACE inhibitors, beta blocker therapy, and cholesterol lowering drugs (statins) have shown to reduce cardiovascular disease risk. The cumulative impact of these four cardioprotective medication classes in post-MI patients is about 70%.<sup>41</sup>

Despite the demonstrated benefits of polypharmacy in the prevention of cardiovascular events, possible adverse effects and drug interactions must be considered whenever prescribing multiple agents in the older persons. Older patients often are receiving a number of drugs for the treatment of various conditions common with ageing. With concomitant medications use comes the increased potential for drug interactions and cumulative adverse effects. In elderly it is essential to evaluate complete medication profile, to eliminate any unwanted drugs and to use lowest effective dose of all agents to maintain effectiveness and promote safety and tolerability.

## References

1. Kuller LH, Shemanski L, Psaty PM, et al. Subclinical disease as an independent risk factor for cardiovascular disease. *Circulation* 1995; 92:720-726.

2. Grundy SM. Age as a risk factor: you are as old as your arteries. *Am J Cardio* 1999; 83:1455-1457.
3. Rowe JW, Kahn RL. Human aging: usual and successful. *Science* 1987; 237:143-149.
4. Reed DW, Foley DJ, White LR, et al. Predictors of healthy aging in men with high life expectancies. *Am J Public Health* 1998; 88:1463-1468.
5. Seeman TE, Charpentier PA, Berkman LF, et al. Predicting changes in physical performance in a high functioning elderly cohort: MacArthur studies of successful aging. *J Gerontol* 1994; 49: 97-108.
6. Burke GL, Arnold AM, Bild DE, et al. For the CHS Collaborative Research Group. Factors associated with healthy aging: the Cardiovascular Health Study. *J Am Geriatr Soc* 2001; 49:254-262.
7. Mittelmark MB, Psaty BM, Rautaharju PM, et al. Prevalence of cardiovascular diseases among older adults: the Cardiovascular Health Study. *Am J Epidemiol* 1993;137: 311-317.
8. O'Leary DH, Polak JF, Kronmal RA et al. For the Cardiovascular Health Study Collaborative Research Group. Carotid-artery intima and media thickness as a risk factor for myocardial infarction and stroke in older adults. *N Engl J Med* 1999;340: 14-22.
9. Kuller LH, Velentgas P, Barzilay J, et al. Diabetes mellitus: subclinical cardiovascular disease and risk of incident cardiovascular disease and all-cause mortality. *Arterioscler Thromb Vasc Biol* 2000;20:823-829.
10. McDermott MM, Ohlmler SM, Kiang L, et al. Gait alterations associated with walking impairment in people with peripheral arterial disease with and without intermittent claudication. *J Am Geriatr Soc* 2001;49:747-754.
11. Longstreth WT, Manolio TA, Arnold A, et al. For the Cardiovascular Health Study Collaborative Research Group. Clinical correlates of white matter findings on cranial magnetic resonance imaging of 3301 elderly people. *Stroke* 1996; 27:1274-1282.
12. Newman AB, Gottdiener JS, McBurnie MA, et al. For the Cardiovascular Health Study Research Group. Associations of subclinical cardiovascular disease with frailty. *J Gerontol A Biol Sci Med Sci* 2001;56:158-166.
13. Lakatta EG, Levy D. Arterial and cardiac aging: major shareholders in cardiovascular disease enterprises. Part I: aging arteries: a "set up" for vascular disease. *Circulation* 2003; 107: 139-146.
14. Lakatta EG. Cardiovascular regulatory mechanisms in advanced age. *Physiol Rev* 1993; 73: 413-465.
15. Virmani R, Avolio AP, Mergner WJ, et al. Effect of aging on aortic morphology in populations with high and low prevalence of hypertension and atherosclerosis: comparison between occidental and Chinese communities. *Am J Pathol* 1991; 139: 1119-1129.
16. Blacher J, Asmar R, Djane S, et al. Aortic pulse wave velocity as a marker of cardiovascular risk in hypertensive patients. *Hypertension* 1999; 33: 1111-1117.
17. Dart AM, Kingwell BA. Pulse pressure: a review of mechanisms and clinical relevance. *J Am Coll Cardiol* 2001; 37: 975-984.
18. Franklin SS, Larson MG, Khan SA, et al. Does the relation of blood pressure to coronary heart disease risk change with aging? The Framingham Heart Study. *Circulation* 2001; 103: 1245-1249.
19. Franklin SS, Khan SA, Wong ND, et al. Is pulse pressure useful in predicting risk for coronary heart disease? The Framingham Heart Study. *Circulation* 1999; 100: 354-360.
20. Franklin SS, Gustin IV W, Wong ND, et al. Hemodynamic patterns of age-related changes in blood pressure. The Framingham Heart Study. *Circulation* 1997; 96: 308-315.
21. Boutouyrie P, Tropeano AI, Asmar R, et al. Aortic stiffness is an independent predictor of primary coronary events in hypertensive patients: a longitudinal study. *Hypertension* 2002; 39: 10-15.
22. Sagie A, Larson MG, Levy D. The natural history of borderline isolated systolic hypertension. *N Engl J Med* 1993; 329: 1912-1917.
23. Lakatta EG, Levy D. Arterial and cardiac aging: major shareholders in cardiovascular disease enterprises. Part II: the aging heart in health: links to heart disease. *Circulation* 2003; 107: 346-354.
24. Schulman S, Lakatta EG, Fleg JL, et al. Age related decline in left ventricular filling at rest and exercise. *Am J Physiol* 1992; 263: 1932-1938.
25. Benjamin EJ, Levy D, Anderson KM, et al. Determinants of Doppler indexes of left ventricular diastolic function in normal subjects (the Framingham Heart Study). *Am J Cardiol* 1992; 70: 508-515.
26. Swinne CJ, Shapiro EP, Lima SD, et al. Age associated changes in left ventricular diastolic performance during isometric exercise in normal subjects. *Am J Cardiol* 1992; 69: 823-826
27. Rodeheffer RJ, Gerstenblith G, Beard E, et al. Postural changes in cardiac volumes in men in relation to adult age. *Exp Gerontol* 1986; 21: 367-378.
28. Fleg JL, O'Connor FC, Gerstenblith G, et al. Impact of age on the cardiovascular response to dynamic upright exercise in healthy men and women. *J Appl Physiol* 1995; 78: 890-900.
29. Chen CH, Nakayama M, Talbot M, et al. Verapamil acutely reduces ventricular-vascular stiffening and improves aerobic exercise performance in elderly individuals. *J Am Coll Cardiol* 1999; 33: 1602-1609.
30. Tsuji H, Larson MG, Venditti FJ, et al. Impact of reduced heart rate variability on risk for cardiac events. *Circulation* 1996; 94: 2850-2855.

31. Newman AB, Arnold AM, Naydeck BL et al. "Successful Aging"- Effect of subclinical cardiovascular disease. *Arch Intern Med* 2003; 163: 2315-2322.
32. Ferrucci L, Izmirlian G, Leveille S, et al. Smoking, physical activity, and active life expectancy. *Am J Epidemiol* 1999; 149:645-653
33. Harris TB, Ferrucci L, Tracy RP, et al. Associations of elevated interleukin-6 and C-reactive protein levels with mortality in the elderly. *Am J Med* 1999; 106:506-512.
34. Ridker PM, Glynn RJ, Hennekens C. C-reactive protein adds to the predictive value of total and HDL cholesterol in determining risk of first myocardial infarction. *Circulation* 1998; 97:2007-2011.
35. Ferrucci L, Harris TB, Guralnik JM, et al. Serum IL-6 level and the development of disability in older persons. *J Am Geriatr Soc* 1999; 47:639-646.
36. Walston J, McBurnie MA, Newman A, et al. Cardiovascular Health Study. Frailty and activation of the inflammation and coagulation systems with and without clinical comorbidities: results from the Cardiovascular Health Study. *Arch Intern Med* 2002; 162:2333-2341.
37. Pahor M, Kritchevsky S. Research hypotheses on muscle wasting, aging, loss of function and disability. *J Nutr Health Aging* 1998; 2:97-100.
38. Stein O, Thiery J, Stein Y. Is there a genetic basis for resistance to atherosclerosis? *Atherosclerosis* 2002; 160: 1-10.
39. Greenland P, Smith SC, Grundy SM. Improving coronary heart disease risk assessment in asymptomatic people: role of traditional risk factors and noninvasive cardiovascular tests. *Circulation* 2001; 104:1863-1867.
40. Vaitkevicius PV, Fleg JL, Engel JH, et al. Effects of age and aerobic capacity on arterial stiffness in healthy adults. *Circulation* 1993; 88: 1456-1462.
41. Pearson TA, Blair SN, Daniels SR et al. AHA guidelines for primary prevention of cardiovascular disease and stroke: 2002 Update. *Circulation* 2002; 106:338-391.

## Post Graduate Diploma in Geriatric Medicine

School of Health Sciences  
Indira Gandhi National Open University  
Maidan Garhi, New Delhi-110068, India  
[www.ignou.ac.in](http://www.ignou.ac.in)

This programme is a Diploma Programme of one year duration. It is aimed at MBBS doctors. This programme will equip the in-service doctors with knowledge and skills in the field of Geriatric Medicine and further enable them to deal with the special problems faced by the elderly.

The broad Objectives of the Programme are to:

- Upgrade the knowledge and skills for providing comprehensive health care to elderly
- Inculcate the inter-disciplinary approach for diagnosing and management of geriatric health problems
- Improve the clinical, social and communication skills by providing hands on training in medical colleges and district hospitals