Review of Literature
History: Mergery Warren established geriatric medicine in 1930s. Her message was the need for assessment and rehabilitation of elderly disabled people (Warren MV, 1943).

In 1947, British Medical Association set up a working group for care of elderly and infirm and made recommendation of twenty one members, four were active in the new specialty of geriatrics. They noted that all elderly men attended institution or hospital for chronic sick (Brocklehurst JC, Dall JC, 1998).

In 1972, the Royal College of Physicians of London set up a working party whose membership consisted of thirteen geriatricians and some college officers (Brocklehurst JC Dall JC, 1998).

In 1976, total working party membership of one hundred and seventy six consultant geriatricians was established (Brocklehurst JC, Dall JC, 1998).

Aging is a natural process. In the words of Seneca: "Old age is an incurable disease". In another statement, by Sir James Sterling Ross, he said; "You do not heal old age. You protect it; you promote it; you extend it" (Park K, 1997).

In the beginning of the last century about twelve million Indians were aged sixty years or more. The number doubled to twenty four million in the next sixty years. Since then there has been a large increase in the number of the elderly, to about fifty six million in 1991. The approximate number of elderly was seventy million by 2001 and will be one hundred and seventy seven million by 2025. Forty five percent of the elderly Indians have chronic diseases or some or the other kind of disability. The number increases to ninety five percent among those who seek health screening. The common diseases among the ambulatory elderly are hypertension, cataract, osteoarthritis, chronic obstructive pulmonary disease, ischaemic heart disease, diabetes, benign...
prostatic hypertrophy, dyspepsia, irritable bowel syndrome and depression which account for eighty five percent of the burden of ill health. The very old people usually suffer from stroke, dementia, osteoporosis, heart failure and frailty and physical dependence. The commonest causes of death in the Indian elderly are bronchitis, pneumonia, ischaemic heart disease, stroke, cancer and TB. As per WHO projection, in the next twenty years, over three fourth of the deaths in the developing world will be due to non communicable diseases exceeding communicable diseases and injuries (Dey AB, 2001).

The age at which patients become "elderly" is difficult to define because aging is a continuous process and the boundaries between the middle and the old age are blurred. Ideally, the term "elderly" should be marked with distinct changes in physiology, but the rate of physiological aging varies. It may not advance parallel to chronological changes, so that one can be physiologically young but chronologically old and vice versa (Davies Ioan, 1998, Park K, 1997).

Ageing is characterized by a gradual loss of function in many organ systems, unrelated to pathological condition (Braunwald Eugene, 2001; Cheitlin Melvin D, Zypes Douglas P, 2001). From a physiologic standpoint human aging is characterized by progressive constriction of the homeostatic reserve of every organ system. This is referred to as "homeostenosis". It is usually evident by the third decade and is gradual and progressive. The decline of each organ system occurs independently of changes in the other organ systems. It is influenced by diet, environment, personal habits and genetic factors (Resnik Neil M, 2001). According to Masoro EJ (1998) the deterioration of the physiologic systems that starts during young adulthood is caused by many damaging processes and agents that organisms encounter during life. The repair systems during post maturational life are not able to fully eliminate the damage. This accumulation of damage leads to progressive functional inadequacy of the physiologic systems. In another statement, the process of human aging involves individuals developing, changing and adapting throughout their life span. Their physical bodies grow or develop, then after a long plateau in adult maturity, decline in some degree and then more sharply in late old age (Johnson Malcolm L, 1998). According to Kirkwood TBL (1998) aging is a

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progressive, generalized impairment of function, resulting in a loss of adaptive response to stress and a growing risk of age related disease. The overall effect of these changes is summed up in the increase in the probability of dying.

MECHANISMS OF AGING:

There are several mechanisms of cellular aging. The ones most commonly described include changes in structure of chromosomes. Other theories proposed include presence of DNA cross linking, increased frequency of single strand breaks, decrease in DNA methylation and loss of DNA telomeric sequences (Resnick Neil M, 2001). At the protein level, the primary structure does not change, but the posttranslational changes like deamidation, oxidation, and cross-linking and non-enzymatic glycation increase (Davies loan, 1998). The various organelles show changes with aging.

MITOCHONDRIA: A number of changes occur in the mitochondria in its ultrastructure. There is alteration in mitochondrial cristae and formation of myelin like whorls of membrane in degenerating organelles. Maximal respiratory capacity decreases slowly. The oxidative phosphorylation remains unchanged (Davies loan, 1998).

RIBOSOMES: Ribosomes are an important cellular component. The number of rRNA genes available for transcription varies at different stages of life cycle. With the advancing age there is a loss of rRNA genes. But they are not responsible for changes in the levels of protein synthesis. So, in all Ribosomes doesn't change markedly with age. However changes in tRNA complements is seen with aging (Davies loan, 1998).

LYSOSOMES AND LIPOFUSCIN: Lipofuscin is also known as aging pigment granule. It emits a yellow green to orange fluorescence when excited by UV light. It is very irregular in shape and varies with the cell type. It was thought that lipofuscin produces intracellular malfunction and that it is an indicator of age associated cellular damage. It causes aging by free radical reactions and lipid peroxidation. Lysosomes on the other hand cause aging by leakage of lysosomal enzymes from damaged organelle resulting in an altered intracellular

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chemistry. It causes damage to the membranes, changes the confirmation of cellular proteins, and changes the RNA, affecting the translation of proteins. It also damages the DNA, causing deletion and mutations (Davies loan, 1998).

CELL MEMBRANE: It is very important for the cellular functioning. It regulates the passage of metabolites and ions in and out of the cell and at the subcellular level also. It also has components of structural elements such as respiratory assemblies of mitochondria and other membrane bound enzymes and receptors. With aging there is alteration in its lipid composition, membrane bound enzymes and hormone receptors (Davies loan, 1998).

THEORIES OF AGING:

The following theories have been put forward to explain the aging process -

a. Somatic mutation theory: Accumulation of a sufficient level of mutations in somatic cells (Martin GM et al, 1985)

b. Immune theory: In the elderly there is diminished antibody production reflecting vulnerability to infections. There is also a decline in immune regulation leading to an increase in autoimmune diseases (Walford RL 1969).

c. Genetic theory: There is loss of important genetic material during DNA repair and also there is impaired DNA repair. Specific genes encode genetically determined life span (Martin GM et al, 1996).

d. Neuroendocrine theory: This theory states that neural loss occurs in selective areas of brain such as cerelues and substantia nigra, which is a part of the aging process. There is also decreased secretion of growth hormone with age. This impairs protein synthesis and cell division, which are governed by growth hormone (Sharma OP, 2001).

e. Free radical theory: Free radicals are highly reactive and cause oxidative alteration in collagen elastin and DNA leading to changes of mitochondrial membrane and fibrosis of arterioles and

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capillaries secondary to vessel injury (Gutteridge JMC, 1994, Bernstein H, Gensler HL, 1993).

f. Cell aging theory: Adult tissues respond to injury or physiologic demand usually with enlargement of pre-existing cells (i.e. hypertrophy) rather than cell division (leading to hyperplasia). The telomerase causes shortening of chromosomes and apoptosis is seen in nervous system during the diseases of elderly such as Alzheimer’s disease and Parkinson and also in aging prostate. There is also wear and tear to important organs by continuous functioning (Strehler BL, 1962).

g. Other theories:
(a) Deprivation and deficiency of important nutrients and oxygen (Rothstein M, 1982).
(b) Non enzymatic glycosylation of proteins (Harding JJ, Beswick HT et al, 1989)
(c) Accumulation of stress over life time with its resultant effect (Adelman R, Saul RL et al, 1988).

Evolutionary basis of aging: Currently, aging is being attributed to the evolution of species. It is probable that aging occurs through the process of natural selection. Survival after the reproductive era is not beneficial to the propagation of species because it leads to overcrowding and competition for resources for survival. Aging is beneficial in the weeding out of species not engaged in active reproduction if it survives predatory elimination, accidents, environmental hazards and disease. Thus aging is not physiological but natural phenomenon mediated by genes (Kirkwood TBL and Rose MR, 1991).

However, the biological changes are clearer than the mechanisms that mediate them. In fact, although the senescent phenotype appears to be ubiquitous, biologists disagree about whether senescence even exists beyond zoos and civilized societies, and whether it occurs at all in many theories of aging as investigators. As a group, the theories can be divided into two broad categories, based on whether they attribute aging to a genetic programme or to

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progressive and random damage to homeostatic systems of the genetic, theories "pleiotropic antagonism" is the only viable one compatible with the limited evolutionary selection pressure to reproduction. The 'random damage' theories are based on the possibility that the balance between ongoing damage and repair is disrupted. The theories differ in the emphasis placed on increased damage (e.g. by free radicals, oxidation or glycation) versus deficient repair and about the mechanisms that might mediate each. Although such theories are attractive, it remains unclear whether the described abnormalities are the cause or the result of senescence (Rusting RL, 1992; Masaro EJ (ed), 1995; Vijg J, Wei JY, 1995).

To date, the only intervention known to delay aging is caloric restriction. Its salutary effect has been documented in multiple species. The mechanism is still not determined, but it is specific to caloric restriction rather than to reduction in any dietary compartment (e.g. fat intake) or supplements with vitamin or anti-oxidants (Weindruch R, 1996; Masaro EJ, 1993).

Age at which person is designated elderly:

Considering the age at which a person should be designated as elderly, there is a lot of controversy, both in case of male and female.

(1) According to Resnick Neil M, 2001, the age at which a person should be designated as elderly is 65 years and the introduction of his chapter on geriatric medicine in Harrison’s principles of Internal Medicine, 15th edition, goes like this:

'Of all the people who have ever lived to age 65, more than half are now alive. This statistic has important demographic and economic implications, and its impact on medical care is also substantial' (Resnick, Neil M, 2001).

(2) According to Freedman, Michael L, 1987, by the year 2030, it is estimated that 22% of all Americans will be more than 65 years old and that 75% of an average internist's practice will consist of those elderly persons. (Freedman, Michael L, 1987).

(3) According to Venkoba Rao A, 1998, during the last 3 decades, life expectancy in India has increased to 62 years and today there are around 70
million people over the age of 60 years. They are classifiable into young old (60-70 years), old-old (70-80 years) and the oldest old (80 years and above) (Venkoba Rao A, 1998).

(4) Grundy, Emily, 1998, in Brocklehurst's textbook of Geriatrics and Gerontology, states that primary population aging is a consequence of long term downward trends in fertility. As a consequence, the developed western world have 10% or more of their population aged 65 years or more (Grundy, Emily, 1998).

(5) In Harrison's Principle of Internal Medicine, 14th edition, Resnick Neil M states that 'The onset of a new disease in the elderly (usually defined as over age 75-80 years) generally affects the organ system made most vulnerable by prior physiologic and pathologic changes' (Resnick, Neil M, 1997).

(6) According to WHO Draft 1980, all persons aged 60 years or more will be designated as elderly (WHO Draft, 1980).

For our present study, we have selected persons aged 60 years and above as elderly persons and have included them in our study.

SELECTED AGE RELATED CHANGES:

Many changes in the various organ systems of the humans are observed, as they grow old. These are usually the normal physiological changes associated with aging but may very well be confused with a disease process. So it is very important to develop an understanding of the normal physiologic processes in the elderly and being able to differentiate it with the pathological changes.

Some of the physiological age related changes are listed below according to the various organ systems involved:

Cardiovascular system:

Cardiovascular system is the major system to show changes with old age, which needs gross attention and management. There is decrease in myocardial myocytes number and increase in volume, decline in SA node discharge, disturbed atrioventricular (AV) conduction, rigid and narrowed blood vessels,

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Respiratory system:

It is the next most important system to show changes with aging. Its changes are more often than not related to changes in the cardiovascular system. The changes include decrease in the number of glandular epithelial cells in the large airway and consequent decline in the production of protective mucous, loss of supportive tissues and dilatation of smaller airways and air space, reduction in alveolar surface for gas exchange, decline in respiratory measurement strength and stiffening of thoracic cage, decrease in cough reflex and in ciliary action in the lungs, decline in vital capacity and ventilatory response to hypoxia and hypercapnia (Dey AB, 2001; Resnick Neil M, 2001; Conolly Martin J, 1998).

Gastrointestinal system:

The changes in the gastrointestinal system are multivariate and involve each of its components in one way or the other.

- **Oral cavity:** Atrophy of mucous membrane, loss of teeth, weakness of muscles of mastication, decrease in number of taste buds, reduction in secretion of saliva.

- **Oesophagus:** Defective swallowing mechanism due to weak oesophageal peristalsis, weak pharyngeal muscles and disturbed coordination between pharyngeal muscles and oesophageal sphincter.

- **Stomach:** Delay in gastric emptying, reduction in gastric acid secretion.

- **Small intestine:** Reduction in absorption of multiple nutrients in small intestine.

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• Normal gall bladder, pancreatic and colonic functions.

• Liver: Reduction in liver volume, blood flow and perfusion, decline in regenerative capacity of hepatocytes, impairment of metabolism and detoxification of toxins, hormones and drugs.


Endocrine system:

This is a very dynamic system showing changes in every stage of life with considerable changes accumulating till the man becomes old. The changes in this system include decline in hypothalamic responsiveness to changes in internal milieu and releasing hormone, decline in the circadian rhythm, decline in circulating growth hormone level, reduction in secretion and metabolism of the thyroid hormones, impaired glucose tolerance due to impaired insulin secretion and glucose utilization, normal basal and maximal levels of glucocorticoids and mineralocorticoids, menopause in women, decline in the level of testosterone in men (Dey AB, 2001; Resnick Neil M, 2001; Davies Joan, 1998, Belchetz Paul E, Hammond Peter, 1998; Miller Myron, 1998; Sinclair Alan J, Croxson Simon CM, 1998).

Musculoskeletal system:

By and large this the largest system in the body and this too doesn’t escape from the affects of aging.

Muscles:

1. Loss of muscle strength due to irreversible loss of motor units and muscle fibers, deposition of fat and connective tissues.

2. Failure to achieve maximal activation of muscles due to loss of motivation, reflex inhibition, disuse and detraining.

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Bones:

1. Loss of bone mass (minerals, proteins and tissues). It is more common among women.

Joint and periarticular soft tissues:

1. Breakdown of articular cartilage.

2. Decrease in tensile stiffness, fatigue resistance and strength of the joints.

3. Stiffness of periarticular soft tissues (inter-vertebral discs, ligaments, tendons, joint cap scales)

4. Thickening and sclerosis of subchondral bone.

(Dey AB, 2001; Resnick Neil M, 2001; Bruce Stuart, 1998; Dieppe Paul, Tobias Jonathan, 1998)

Urinary system:

The changes seen in the urinary system with aging are as follows-reduction in the number of nephrons and kidney size, distortion of micro architecture of the nephron, reduction in renal blood flow, decline in GFR, reduction in the responsiveness of the kidney to ADH and sodium loss in conserving water and salt, decrease in bladder capacity, increase in the size of prostate in men, reduction in the muscle tone of pelvis in women required to prevent involuntary passage of urine (Dey AB, 2001; Resnick Neil M, 2001; Jassal Vanita et al, 1998; George NJR, 1998).

Central nervous system:

This the main system controlling all the other systems of the body and shows the following major changes with advancing age. These include reduction in brain size, enlarged ventricles, loss of neurons and accumulation of end products of metabolism (e.g. amyloid), reduction in efficiency of neural

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transmission, reduction in brain catecholamine synthesis, decrease in brain dopaminergic synthesis (Parkinsonism), decrease in the righting reflexes, reduction in stage four sleeps, cognitive impairment (Dey AB, 2001; Resnick Neil M, 2001; Mann Davil Michael Andrew, 1998; Mutch William J, Inglis Fraser G, 1998).

Sensory system:

The changes seen in this system are as follows:

Skin:

- Epidermis: Thinning of epidermis, loss of moisture, decrease in skin melanocyte and langerhans cells, appearance of small hypopigmented spots and decline in immunity.

- Dermis: Decreased production of dermal fibroblast and wrinkling, decline in vascularity.

- Decline in sweat and sebaceous gland secretions.

- Graying of hair due to loss of melanin.

- Slow growth of nails.

Eye:

- Lax eyelids, rotated lid margins, reduced lacrimal gland secretion, dry eyes.

- Clouding of cornea, arcus senilis.

- Distortion of the anterior aspect of uveal tract and chronic close angle glaucoma.

- Rigid lens with loss of accommodation; denaturation of lens proteins and cataract.

- Degeneration of retina.

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Ears:

- There is loss of hair cells and ganglion cells decrease in the organ of corti. Blood supply to cochlea decreases. There is also a decline in the number of sensory nerve fibres from the sense organ.

Taste and smell:

- With aging the number of taste buds decreases and the remaining buds have a higher threshold for stimulation to activate them.

(Dey AB, 2001; Resnick Neil M, 2001; Brodie Scott E, 1998; Weinstein Barbara E, 1998)

CARDIOVASCULAR CHANGES (STRUCTURAL AND FUNCTIONAL) WITH AGING

Age related changes in the cardiovascular system, overt and occult cardiovascular disease and decreased physical activity affect cardiovascular function in the elderly persons (Aronow Wilbert S, 1998).

The definition of whether and how advancing age affects cardiovascular structure and function is affected by factors other than age, i.e. life style e.g. physical fitness, nutritional status and specific pathophysiological entities, like diseases (Lakatta Edward G, 1993).

In older people cardiovascular disease plays a significant role and is the most common cause of morbidity and mortality. Heart failure is the most common diagnostic related group in the Medicare population 65 years and above. In Framingham heart study, 44% of men and 28% of women aged 75 to 84 years had cardiovascular disease. In the age group of 85-94 years, the prevalence increased to 48% in men and 43% in women (Cheitlin Melvin D, Zypes Douglas P, 2001).

On the other hand, effects of some types of disease on cardiovascular function in humans may not be recognized, particularly occult coronary artery disease. Of all the male individuals who are 60 years plus who die, 60% have 75-100%
narrowing of at least one major coronary artery and only 15-20% of them manifest its symptoms (Lakatta Edward G, 1993).

Also, because the cardiovascular system at rest functions at only a fraction of its capacity, the signs of age associated differences in its function between the young and the old become manifest, in particular during stress, e.g. during acute exercise. But, in older individuals, the non-cardiovascular factors often limit the amount of physical work that can be performed (Lakatta Edward G, 1993).

The cardiac output is regulated by multiple mechanisms like the heart rate, preload (quantity of blood that fills the heart before excitation), after load (the mechanical load encountered following the onset of contraction), contractile or inotropic state and coronary flow. All these factors are highly interdependent (Lakatta Edward G, 1993).

**CARDIAC STRUCTURE:**

Autopsy data from numerous hearts, showed that in subjects aged 30-90 years, the heart increases in mass by an average of 1 gm/year in men and 1.5 gm/year in women. In both sexes the interventricular septal thickness increases more with aging than does the left ventricular free wall thickness. LV mass may decrease in the very old (80-100 years of age) perhaps because of an extremely sedentary life style leading to a regression of cardiac mass. During the life span, the effect of age on heart size and mass can be assessed by chest X-ray, echocardiography and gated blood pool scans (Lakatta Edward G, 1993).

In a study by Gardin Julius M et al, 1979 a significant increase was seen in ventricular septal wall (20%) and left ventricular free wall thickness (18%) and left ventricular mass (15%) when the oldest group (over 70 years) was compared with the youngest group (21-30 years) (Gardin JM et al, 1979).

An increase in the heart mass with aging is due mostly to an increase in the average myocyte size. In some older individuals in whom LV mass decreases

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with age, there is usually a decrease in the myocyte number seen concurrently with an increase in myocyte enlargement (Aronow Wilber S, 1998)

Collagen in the myocardium also shows an increase in the amount and change in its physical proportion. Myocardial lipofuscin increases with age. Also, some form of amyloid protein can be found in the heart of about half of the aged over 70 years (Hwang Y T et al, 1993).

To summarize, aging between 20 and 80 years appears to be associated with a modest increase in left ventricular wall thickness, due mainly to an increase in the size of cardiac myocytes. The increase in the heart mass with aging is increased by coexisting disease like coronary artery disease or hypertension and is also influenced by life style to a great extent.

ARterial Structure:


Vascular thickness can be quantified in terms of its elastic modulus. In autopsy samples a circumferential static elastic modulus, derived by exerting circumferential strain, i.e. by increasing the volume in an isolated aortic segment at 100 mmHg, increased from 10kg/cm² at 20 years of age to 42.5 kg/cm² at the age of 85 years (Lakatta Edward J, 1993).

Pressure strain moduli of aorta and pulmonary artery can be measured during cardiac catheterization.

The arterial stiffening due to aging is also found to be associated with an increase in the pulse wave velocity (Avolio AP, Fa Quan D, Wel Qiang L et al, 1985; Aronow Wilbert S, 1998, Lakatta Edward J, 1993).

The age associated increase in arterial stiffness is a result of a diffuse process that occurs in the vessel wall. There is an increase in the chondroitin sulphate and heparin sulphate whereas a decrease in cyaluronate and chondroitin content. The stiffening is mainly because of loss of elastin and an increase in

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the amount of collagen. A change in the distribution of unstretched collagen is also seen with age. Aging is also associated with a decrease in the coiling and twisting of molecular chains and a reduction in the effective chain length (Lakatta Edward G, 1993).

Arterial stiffness is also determined by vascular smooth muscle (VSM) contractile tone, which is controlled by neurohumoral factors like catecholamines and angiotensin (Lakatta Edward G, 1993).

All these factors affecting the arteries in the elderly causes an increase in the arterial diameter and wall thickness. The mean systolic internal radius of ascending aorta increases by 9% per decade over the age of 20-60 years (Lakatta Edward G, 1993). Up to the age of 60 years the aortic buffering capacity is not markedly decreased by the increased aortic wall stiffness because the concomitant increase in aortic volume accommodates a given volume with less change in its radius. Thus the volume elasticity shows no change up to the age of 60 years but after that it decreases markedly. The aorta stiffens with age; less diastolic recoil occurs and results in a decreased aortic contribution to forward flow (Lakatta Edward G, 1993).

Peripheral vessels are also affected by aging. The increase in the wall thickness is more in them as compared to the increase in diameter (Lakatta Edward G, 1993). Changes in the pressure pulse contour with age are observed which includes a large secondary systolic wave and disappearance of the diastolic pressure wave. The duration of the dicrotic wave also decreases with age. All these features are primarily due to early reflected, pulse waves. And in turn this is responsible for increase in systolic and pulse pressures with aging (Lakatta Edward G, 1993). There is also an increase in the sodium chloride dependence of arterial pressure with age (Lakatta Edward G, 1993).

To summarize, arteries increase in diameter and wall thickness with aging and these changes are associated with an increase in arterial wall stiffness and a reduction in volume elasticity. Diet and physical conditioning modify the extent to which these changes occur with aging.

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VENTRICULAR VASCULAR INTERACTIONS:

Arterial and Cardiac Changes that occur with aging in normotensives:

In a study by Nicholas Wilmes W et al, 1985 effects of age on the interrelation between the physical properties of the arterial tree and left ventricular performance were studied. They concluded that there was no change in the heart rate and end diastolic aortic pressure with age. But the aortic systolic, mean and pulse pressures and aortic radius increased. Late systolic pressure exceeded early systolic pressure. Peripheral resistance increased over 37% over the age range of 20 to 60 years whereas impedance increased 137%. All these indicated an increase in the aortic wall stiffness and decrease in the cross section of peripheral vascular bed, increased pulse wave velocity and wave

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reflection. This increase on the vascular load causes decrease in stroke volume and cardiac output and development of mild LVH and prolonged relaxation with advancing age (Nicholas Wilmes W et al, 1985).

CARDIAC FILLING (DIASTOLIC PROPERTIES):

Early LV filling begins as ventricular pressure decreases below that in the atrium and continues during the cardiac diastole with a further evolution of atrioventricular pressure gradient (Chatterjee CC, 1992).

The stiffness of the ventricular myocardium is a determinant of the ventricular pressure and thus of the AV pressure gradient. The time course of isovolumic myocardial relaxation between aortic valve closure and mitral valve opening increases by 40% with age in both sexes. The peak rate at which the LV fills with blood during early diastole is reduced by 50% between the ages of 20 and 80 years. But the LV end diastolic volume (EDV) is not reduced in healthy old persons. In fact, it increases with age in men. The atrial contraction contributes to a great extent to ventricular filling with the advancing age. This leads to left atrial enlargement and is also the basis of an audible fourth heart sound in most healthy older individuals (Spirito Paolo, Maron Barry J, 1988; Lakatta Edward G, 1993).

CARDIAC SYSTOLE:

Myocardial contractile properties: The index of the myocardial contractility is the trajectory of end systolic volume versus mean arterial pressure, derived from a series of pressure volume loops measured over a range of cardiac volumes. It is not reduced at rest with age in healthy persons (Lakatta Edward G, 1993).

Cardiac volumes and ejection fraction: Overall systolic function of the heart is best judged from the ejection fraction.

(End diastolic volume - End systolic volume)/End diastolic volume
The blood volumes do not change with age. The end diastolic volume index (EDVI) at rest is increased in elderly (Lakatta Edward G, 1993). There is also an increase in myocyte size (Aronow Wilbert S, 1998) and LV thickness (Lakatta Edward G 1993; Aronow Wilbert S, 1998; Gardin Julius M et al, 1979). This decreases the LV wall tension, which could occur due to increase in LV EDVI and peak LV systolic pressure with aging. The systolic volume index (SVI) has been found to be elevated owing to increase in the EDVI with age. But in women neither the EDVI nor the SVI increases with age. The ejection fraction at rest is not altered with aging in both healthy men and women (Lakatta Edward G, 1993).

Heart rate and rhythm: With advancing age there is an increase in elastic and collagenous tissue in all parts of the conduction system and fat accumulates around the SA node. By the beginning of 60 years there is a great decrease in the number of pacemaker cells in the SA node and by the age of 75 years only 10% cells are remaining (Aronow Wilbert S, 1998; Cheitlin Melvin D, Zypes Douglas P, 2000; Dey AB, 2001; Davies MJ, Pomerance Ariela, 1972). Calcification occurs in the aortic and mitral annuli, central fibrous body summit of the interventricular septum. AV node, AV bundle, bifurcation and proximal left and right bundle branches are affected due to idiopathic heart block, increase in PR interval and increased frequency of supraventricular and ventricular premature beats (Lakatta Edward G, 1993).

No change in the heart rate is seen in supine position however a decrease in the basal heart rate is observed in both men and women in sitting position with advancing age (Lakatta Edward G, 1993).

The respiratory variation in the heart rate, which is also determined by autonomic tone, is decreased with advancing age (Lakatta Edward G, 1993, Faulkner James M, 1980).

Plasma levels of norepinephrine and epinephrine at rest is increased with age (Lakatta Edward G, 1993).

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Cardiac Output: The studies on change in the resting cardiac output have shown variable results. Cardiac output is influenced by the basal metabolic rate and body composition. In older hypertensive men, i.e. in whom arterial pressure exceeds 140/90mmHg, cardiac index (CI) at rest is less than in younger hypertensive men and is associated with a decrease in SVI. However the resting CI decreases with age in women, due to absence of cardiac dilatation at end diastole and increase in SVI (Lakatta Edward G, 1993).

Systolic brachial arterial pressure increases with age at rest in both sexes; mean arterial pressure increases, mildly with age due to increase in systolic pressure. PVR at rest is not affected by age in healthy men but increases with age in women (Aronow Wilbert S, 1998).

In individuals with hypertension, the same vascular and cardiac changes are observed as in aged who are normotensive. The similarities between aging and hypertension are so striking that aging is referred to as "muted hypertension" or hypertension can be referred to as "accelerated aging" (Lakatta Edward G, 1993).

However, some changes occur with aging in hypertensives that are not seen in normotensives. In the former, the PVR increases with aging whereas in the latter it remains normal. Also in older hypertensive men, resting SVI and CI are not maintained at levels measured in younger hypertensive men (Lakatta Edward G, 1993; Brandfonbrener M et al, 1955).

A decrease in the B-adrenergic stimulation of both the heart and vasculature occurs with aging and in hypertension and is associated with the myocardial changes of decreased heart rate at rest and during stress (Lakatta Edward G, 1993; Aronow Wilbert S, 1998)

The various electrocardiograms were interpreted with the following principle guidelines.

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DETERMINATION OF RATE:

(A) Ventricular rate: The interval between 2R waves is measured and corrected for rate per minute by dividing into 60.

(B) Atrial rate: If sinus rhythm with normal AV conduction is present, the atrial rate is the same as the ventricular rate. If the rhythm is not normal sinus and regular atrial activity is evident, the PP interval is measured and the atrial rate per minute is determined by dividing into 60.

DETERMINATION OF RHYTHM:

- Sinus rhythm: Regular and equal atrial and ventricular rates; P waves have normal morphology, the PR interval is normal.
  - Sinus bradycardia: Sinus rhythm, rate less than 60/min.
  - Sinus tachycardia: Sinus rhythm, rate greater than 100/min.
  - Sinus arrhythmia: Sinus rhythm with cyclic variations in rate usually related to respiration; the rate increased with inspiration and slows with expiration.

- Atrioventricular (AV) block:
  - First degree: Sinus rhythm, PR interval greater than 0.21sec.
  - Second degree: At regular or irregular intervals a P wave is not followed by a QRS complex. The PP interval is constant.

  - Type I (Wenckebach) - In a cyclical manner, there is progressive lengthening of the PR interval until a P wave is not followed by a QRS complex.
• **Type II:** In a regular or an irregular sequence, a P wave is not followed by a QRS complex. The PR interval for the conducted P waves is constant.

• **Third degree:** Complete dissociation between atrial and ventricular rhythm. The atrial rhythm may be normal sinus or any atrial arrhythmia. Ventricular depolarization (QRS complex) is initiated by a subsidiary pacemaker located in the AV junction, resulting in a regular QRS rhythm having a rate of 50-60/min. With normal appearing QRS complexes, or in Purkinje ventricular tissue, resulting in a regular QRS rhythm having a rate of 30-40/min with broad slurred or notched QRS complexes.

(C) Atrial arrhythmias:

(i) **Pauses in sinus rate:**

(a) **Sinoatrial (SA) block:** Episodes of bradycardia during which P waves are not present. The reappearance of the sinus P wave occurred at a multiple (e.g. x2, x3) of the basic sinus rate.

(b) **SA arrest:** Episodes of bradycardia during which P waves are not present. If sinus rhythm resumes, it does so at a PP interval which is not a multiple of the basic sinus rate. If sinus rhythm doesn’t resume, the ventricles will be depolarized from a subsidiary pacemaker in the AV junction (normal appearing junctional escape complexes) or Purkinje-ventricular tissue (broad ventricular escape complexes).

(c) **SA Wenckebach:** In a cyclical manner, there is a progressive shortening of the PP interval until a P wave fails to appear. The PR interval is constant.

(ii) **Atrial premature(ectopic complexes):** A P wave occurs prematurely relative to the basic PP interval. It may be followed by a normal appearing or aberrant QRS-T complex, or it may fail to be conducted ("blocked" APC), and will not be followed by a QRS complex. The PR interval of the premature P
wave may be the same as, longer than, or shorter than the PR interval of the sinus conducted complexes.

(a) High atrial focus of origin: the axis of the P wave is normal (upright in I and aVF).

(b) Low atrial focus of origin: The P wave is inverted in leads II, III, aVF. The PR interval usually shorter than that of the sinus P-QRST complexes. These complexes might be indistinguishable from AV junctional complexes with retrograde conduction to the atria.

(c) Wandering atrial pacemaker: The P waves have varying morphology and the PP and PR intervals vary. The rate is less than 110/min.

(iii) Atrial tachycardia:

a) Automatic (ectopic) atrial tachycardia: The PP interval is constant and the atrial rate is 160-220/min. The P wave axis is normal. The AV conduction rates can be 1.5 or higher. The usual AV rates in atrial tachycardia with block is 2:1.

b) Multiple atrial tachycardia: The P waves vary in morphology and axis. The PP and PR intervals vary, thus the ventricular rate varies too. The atrial rate exceeds 110/min. P waves may be blocked and thus not followed by QRS complexes. The QRS complex may be aberrant.

(iv) Atrial flutter: Regular atrial activity at a rate of 260-320 is present. The P waves have characteristic "Saw tooth" appearance in leads II, III and aVF. AV block is almost always present. The AV ratio is usually fixed (e.g. 2:1, 4:1), resulting in a regular ventricular rate, but variation is possible, resulting in an irregular ventricular rate. Type I (Wenckerbach) block of some flutter impulses could occur, producing a "regularly irregular" ventricular rate. The QRS complexes may be aberrant.
(v) **Atrial fibrillation:** Atrial activity is totally irregular, resulting in an undulation of the baseline of the ECG and no isoelectric interval. The ventricular rhythm is grossly irregular. The QRS complexes may be aberrant if the ventricular rate is rapid. If the ventricular rhythm is regular, complete AV dissociation is present. Normal appearing QRS complexes indicated an AV junctional focus of origin; the rate is usually 50-100/min. Wide, slurred or noticed QRS complexes indicate a Purkinje ventricular focus of origin; the rate is less than 50/min.

(D) **AV junctional rhythms:**

(i) **Junctional ectopic complexes:** A QRS complex occurs prematurely relative to the basic RR interval. It may be unassociated with a P wave, dissociated from a sinus P wave or associated with a retrograde P wave, which can precede or follow it.

(ii) **Junctional rhythm:** A regular QRS rhythm of a rate of 50-100/min. P wave may not be seen, when they are seen, they are inverted in leads II, III and aVF. The PR interval is short. The rhythm may represent an escape rhythm or an accelerated one; escape rhythms are generally slow whereas accelerated rhythms are generally normal or rapid.

(iii) **Junctional tachycardia:** Similar to junctional rhythm, but the rate is 120-250/min. The tachycardia may be automatic or reentrant.

(E) **Ventricular rhythm:**

(i) **Ventricular premature (ectopic) complexes:** A wide, notched, or slurred QRS complex not preceded by a premature P wave, occurs prematurely relative to the PR interval.

   a) **Uniform complex:** All ventricular premature complexes have a similar morphology in a given electrocardiographic lead.

   b) **Multiform:** The ventricular ectopic complexes vary in morphology and axis in a given electrocardiographic lead.

"Study of Electrocardiographic Changes in Elderly Persons"
c) "R-on-T": A ventricular premature complex, which occurs on the peak or demonstrate of the preceding T wave.

(ii) Ventricular tachycardia: A run of 3 or more consecutive ventricular ectopic complexes. The rate is usually 100-200/min. and may be slightly irregular. No P waves are seen, but occasionally independently occurring regular P waves can be identified. Fusion QRS complexes can occur which has a configuration intermediate between the ventricular complex and the sinus generated QRS complex.

(iii) "Idioventricular" rhythm: A regular or slightly irregular ventricular rhythm at a rate of 30-40/min. The atrial rhythm is dissociated from it.

(iv) Accelerated ventricular rhythm: A ventricular rhythm whose rate is 60-120/min.

(v) Ventricular fibrillation: A very rapid and irregular ventricular rhythm having no distinct morphology on the electrocardiographic recording (VF arrest).

(vi) Ventricular asystole: Absence of QRS complexes for seconds to minutes.

(F) Ventricular pre-excitation:

(i) Wolff-Parkinson-White (WPW): The P wave has normal morphology and axis. The PR interval is short. An initial slurring of the upstroke of the R wave (delta wave) or of the down stroke of a Q wave is present which prolongs the QRS interval. Concomitant ST depression and T wave inversion are often present.

(ii) Short PR interval syndromes: The P waves are normal, the PR interval is short and the QRS complexes are normal. The cause of the short PR interval may be an intra-AV nodal bypass tract, an anatomically short AV node, or an atrio-His connection which bypasses the AV node.
DETERMINATION OF THE P-QRST MORPHOLOGY:

(A) Hypertrophy patterns:

(i) **Left atrial hypertrophy**: The P waves are broad and notched in several frontal plane leads and in V4-V6. They are diaphasic in lead V1, with a terminal portion that is negative (at least 1mm) and broad (at least 0.04 sec).

(ii) **Right atrial hypertrophy**: The P waves are tall (over 2.5mm) and peaked in leads II, III and aVF.

(iii) **Left ventricular hypertrophy**:

a) Voltage criteria (valid only over age 35):

   I. RI and S III greater than 26mm.

   II. R aVL greater than 11mm.

   III. RV5 or RV6 greater than 26mm.

   IV. SV1 and RV5 (or RV6) greater than 35mm.

b) ST depression and T wave inversion is present in those leads with high QRS voltage.

c) The frontal plane QRS axis is often superior to -30 degree.

d) Slight to moderate prolongation of the QRS interval (0.10-0.12 sec) may be present.

(iv) **Right ventricular hypertrophy**:

   a) Right axis deviation is present, with the mean frontal plane QRS axis greater than +90 degree in adults over 40 years of age.

   b) Prominent anterior forces (RV1 greater than 5mm or r:s ratio greater than 1 in lead V1 or both). Right ventricular hypertrophy acquired later

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in life (e.g., pulmonale in adults over about age 60) may show small or absent 'r' waves in V1-3.

c) ST depression and inverted T waves in leads V1-3.

d) The presence of criteria for left or right atrial hypertrophy aids in the electrocardiographic diagnosis of right ventricular hypertrophy.

e) Bi-ventricular hypertrophy: Voltage criteria for left ventricular hypertrophy with ST depression and T wave inversion in leads V5-6, plus a frontal plane QRS axis of +90 degree or greater.

(B) Intraventricular conduction delays:

(i) Bundle branch block patterns:

(a) Right bundle branch block patterns:

(i) The QRS interval is 0.12 sec or greater.

(ii) Wide, slurred S waves are present in leads I and V5-6.

(iii) Wide, slurred rR' waves are seen in leads V1-2 (V3).

(b) Left bundle branch block pattern:

(i) The QRS interval is 0.12 sec or greater.

(ii) No q wave is seen in leads I and V5-6.

(iii) The QRS complexes are wide, notched on slurred.

(iv) The QRS complexes are upright on leads I and V5-6.

(v) ST depression and T wave inversion are present in leads I and V5-6.

(ii) Fascicular conduction delays:

(a) Left anterior fascicular block:

(i) The frontal plane QRS axis is superior to -30 degree.

(ii) There is a prominent R wave in lead I.

(iii) The R:S ratio in lead II is less than 1.

"Study of Electrocardiographic Changes in Elderly Persons"
(b) Left posterior fascicular block:

(i) The frontal plane QRS axis is greater than +110 degree.

(ii) A qR pattern is present in leads II, III, aVF.

(iii) Fascicular block pattern:

a) RBBB with LAHB: All criteria for RBBB, plus a frontal plane QRS axis superior to -30 degree.

b) RBBB with LPHB:

I. All criteria for RBBB plus a frontal plane QRS axis greater than +110 degree.

II. Absence of clinical evidence of right ventricular hypertrophy or lateral wall infarction.

c) RBBB with 1 degree AV block.

d) LBBB plus 1-degree AV block.

e) Alternating right and left bundle branch block in single or serial ECGs.

(iv) Trifascicular block:

a) RBBB

b) Left anterior (or posterior) fascicular block.

c) First degree block.

(C) Myocardial ischaemia:

It is recognized by transient, reversible ST segment and T wave abnormalities, which may be non-transmural or transmural.

"Study of Electrocardiographic Changes in Elderly Persons"
Nontransmural ischaemia is characterized by:

I. ST segment depression of 1mm or more in one or more ECG leads.

II. Downsloping, horizontal or slowly upsloping ST segment. Slowly upsloping ST segment are defined as 2mm of ST depression 80msec after the J point.

Transmural myocardial ischaemia is characterized by ST segment elevation.

(D) Myocardial infarction: It is characterized by:

I. ST segment elevation (minutes to 1-2 wks)

II. Tall ("giant", "hyperacute") upright T waves. These are often present in those ECG leads showing the ST segment elevation, and are usually no longer present at 24 hrs.

III. T wave inversion (days to years)

IV. Q wave (or QS complex) (hours to years). The pathologic Q wave was:

   a) 0.04 sec or more in duration.

   b) 25% of the R wave height in a given lead (except leads III, aVL and V1)

V. The infarct location was established by applying these criteria to specific lead groups:

   a) Anterior infarction: leads V2-6.

   b) Inferior infarction: leads II and avF.

   c) Lateral infarction: leads I, aVL and V6.
d) Posterior infarction: V1-2 (reciprocal ST depression was seen in the anterior precordial leads; an abnormal Q wave was recorded only from the posterior surface of the heart).

(E) Pericarditis:

I. Initially, ST segment elevation is present in all leads except aVR and V1-2. Depression of PR segment might occur.

II. Electrical alternans, which can involve the P waves, QRS complexes, and T-U waves. It is not a specific finding for pericardial effusion, nor is a very frequent finding; but when it occurs malignant pericardial effusion is usually present. Myocardial disease and acute myocardial ischaemia may also produce electrical alternans. Occasionally, a rapid reentrant supraventricular tachycardia may show QRS alternans; this is of no clinical significance.

(F) Myocarditis: The wide variety of ECG abnormalities resulting from myocarditis includes:

I. Those associated with pericarditis.

II. AV or intraventricular conduction delays.

III. Ventricular (usually left) hypertrophy.

IV. Ventricular arrhythmias

V. Non specific ST-T wave changes.

(G) Electrolyte abnormalities:

(i) Hyperkalemia:

(a) Narrow based, peaked T waves. The T waves may or may not be tall, and full amplitude is not a criterion for diagnosis.

(b) AV conduction delays.

(c) Low voltage and eventual disappearance of P waves
(d) Widening of QRS interval
(e) Sine wave, QRS-T pattern
(f) Ventricular tachycardia
(g) Ventricular fibrillation

(ii) Hyperkalemia:
(a) ST depression
(b) Lowering of T wave amplitude
(c) Prolongation of ventricular repolarization with large, upright U waves.
(d) "Giant" U waves

(iii) Hypercalcemia: marked shortening of QT interval is seen. The ST segment, as a distinct isoelectric period, is eliminated and the T wave begins immediately at the end of the QRS.

(iv) Hypocalcemia: Prolongation of the QT interval due to lengthening of the ST segment occurs.

The results of various studies done on the elderly persons and their ECG changes have shown results, which are summarized as follows:

MAJOR Q OR QS PATTERN/ST CHANGES:

It is suggestive of existence of ischaemic heart disease (Schamroth Leo, 1990) Silent ischaemia is quite common with the increasing age. About one third of the asymptomatic hypertensive elderly patients have myocardial infarction discovered accidentally on ECG. In these patients an ECG taken during the time of discomfort show ST segment elevation or depression or marked T wave inversion, which reverses after the discomfort, abates (Schamroth Leo, 1990; Cheitlin Melvin D, Zypes Douglas P, 2001)

In a study by Fleg Jerome L et al, the prevalence of exercise induced silent ischaemia, which was defined by concordant ST segment depression and a
Thallium perfusion defect increased more than seven fold from 2% in the fifth and sixth decade to 15% in the ninth decade. Over a mean follow up period of 4.6 years, cardiac events done in 9.8% of subjects and consisted of twenty cases of new angina pectoris with ST segment elevation, thirteen myocardial infarctions showing QS pattern and seven deaths. These events occurred in 7% of individuals with both negative Th and ECG changes; 8% of those with either test positive; and 48% of those in whom both tests were positive. Thus in an asymptomatic population the presence of exercise induced silent myocardial infarction increases progressively with age. And ECG, especially stress ECG in these patients is an important predictor of the extent of the disease (Fleg Jerome L, Gerstenblith Gary et al, 1990).

Myocardial infarction is an extreme of the coronary artery disease in the elderly. Non Q wave myocardial infarction is more common presentation of acute myocardial infarction in the elderly (Antman Elliot M, Braunwald Engene, 2001). Age is a powerful independent predictor of short-term and long-term mortality of patients with an acute myocardial infarction. In patients admitted with a first ST segment elevation myocardial infarction and treated with thrombolytic therapy, in hospital mortality increases exponentially as a function of age from 1.9%, among patient 40 years and younger to 31.9% among patients older than 80 years (Lakatta Edward G, Schulman Steven P, Gerstenblith Gary, 2001).

Table no.3
Table showing accuracy of ECG, Th and concordant positive ECG and 201Th results for predicting coronary event

<table>
<thead>
<tr>
<th>Descriptor</th>
<th>ECG result</th>
<th>Th result</th>
<th>Concordant positive ECG and Th</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensitivity</td>
<td>40</td>
<td>30</td>
<td>28</td>
</tr>
<tr>
<td>Specificity</td>
<td>86</td>
<td>88</td>
<td>97</td>
</tr>
<tr>
<td>Positive predictive value</td>
<td>24</td>
<td>22</td>
<td>48</td>
</tr>
<tr>
<td>Negative predictive value</td>
<td>93</td>
<td>92</td>
<td>92</td>
</tr>
</tbody>
</table>

(Fleg Jerome L, Gerstenblith Gary, 1996)

"Study of Electrocardiographic Changes in Elderly Persons"
Thus in the study by Fleg Jerome L et al, 1996 ST segment depression was found to be increasing in percentage from 2 to 15 in a span between fifth and the ninth decade. According to Lakatta Edward G et al, 2001 incidence of myocardial infarction too showed an upward trend with age with a value of 1.9% in patients 40 years and younger to 31.9% in patients older than 80 years.

NON-SPECIFIC ST SEGMENT/T WAVES CHANGES:


T wave inversion is most commonly seen in myocardial ischaemia or infarction (Schamroth L, 1990; Wagner GS, 1998). The other causes can be left or right ventricular overload with strain pattern (Goldschlager Nora, Goldman MJ 1989, Schamroth L, 1990), apical hypertrophic cardiomyopathy (Goldberger AL, 1999; Wyne Joshua, Braunwald Eugene, 2001), secondary T wave changes as in bundle branch blocks, Wolff Parkinson white pattern (Schamroth L, 1990) ventricular ectopics or paced beats (Morvis David M, Goldberger Ary L, 2000, Goldschlager Nora, Goldman MJ, 1990), cerebrovascular accident

ABNORMAL AXIS DEVIATION/RIGHT OR LEFT VENTRICULAR HYPERTROPHY PATTERN:

The important factors that predict deterioration of cardiac function include low vital opacity, sinus tachycardia and ECG evidence of LVH. ECG-LVH heralded the occurrence of serious cardiovascular disease of all varieties, with particular higher risk ratios for heart failure. ECG-LVH characterized by increased voltage unaccompanied by repolarization abnormality carried a lesser risk of failure than that associated with evidence of strain. Also, it is likely that ECG versions reflect both ischemic myocardial damage and anatomic hypertrophy. When both are present the risk is substantially higher than when either is present alone (Kannel WB, Belanger AJ, 1991)

According to Framingham Heart Study also ECG-LVH is an independent and effective predictor of cardiovascular risk for the elderly (Kannel WB, Belanger AJ, 1991).

Table no.4

<table>
<thead>
<tr>
<th>Age</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>55-64</td>
<td>10.1</td>
<td>4.1</td>
</tr>
<tr>
<td>65-74</td>
<td>7.1</td>
<td>9.6</td>
</tr>
</tbody>
</table>

"Study of Electrocardiographic Changes in Elderly Persons"
In another study of ECG patterns of LVH or LVH strain in standard limb leads was studied. The following data was obtained from the study:

**Frequency of abnormalities in unipolar limb and precordial leads in LVH:**

- Total no. of cases: 147
- Total no. of cases with normal or borderline standard leads: 34
- Abnormal RST-T finding: 136
- Lead I: 69
- Lead I and/or III: 20
- Lead V4 to V6: 111
- Lead aVL: 88
- Lead aVR: 38
- Lead aVF: 40

**Abnormal voltage:**

- R1+S3 ≥25mm: 26
- RV6/RV5≥26mm: 29
- RaVL ≥11mm: 33
- RV6+SV1≥35mm: 48

**Delayed onset of intrinsic deflection**

(delayed ventricular activation time) ≥0.06sec in V5 or V6: 52

(Sokolow M, Lyon I, 1949)

In the valvular heart diseases, affecting the elderly, the aortic valve is the most commonly affected and the disease is usually degenerative in nature and also because they are subjected to greater pressure and trauma (Braunwald Eugene, 2001; Channer KS, 1998).

"Study of Electrocardiographic Changes in Elderly Persons"
In a study performed by Aronow Wilbert S and Kronzon Itzhak, 1991 prevalence and severity of valvular AS by Doppler echocardiography and its association with electrocardiographic LVH was studied.

The following data was obtained:

**Table no. 5**

<table>
<thead>
<tr>
<th>Severity of AS</th>
<th>No. Of patients</th>
<th>LVH By echo</th>
<th>LVH By ECG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>74</td>
<td>55</td>
<td>8</td>
</tr>
<tr>
<td>Moderate</td>
<td>49</td>
<td>47</td>
<td>15</td>
</tr>
<tr>
<td>Severe</td>
<td>19</td>
<td>19</td>
<td>11</td>
</tr>
</tbody>
</table>

(Aronow Wilbert S, Kronzon Itzhak, 1991)

In another study of severe AS in patients over 65 years of age, 21 patients were studied. Various ECG changes were observed in these patients out of whom maximum number showed LVH (11 patients). 7 patients showed AF, 1 patient had LBBB and RBBB each and transient heart block was seen in 3 patients (Roberts William C et al, 1970).

Thus LVH in elderly has been reported to be 4.1% to 10.1%. Kannel WB, Belanger AJ, 1991, reported it to be 10.1% in men between 55 to 64 years and 4.1% in the women of the same age groups. Different writers had different findings because of the variation in selection of patients, underlying heart disease and the criteria for defining LVH. On and all LVH can result due to anatomical hypertrophy or myocardial damage, but it is difficult to differentiate this from ECG.

"Study of Electrocardiographic Changes in Elderly Persons"
CONDUCTION DEFECTS/RHYTHM ABNORMALITIES:

Rhythm abnormalities are found to be so frequent in hospitalized elderly patients that it can be said that certain arrhythmias should be regarded as normal findings in them (Camm AJ et al, 1980).

A number of studies have been conducted to study the frequency and affects of various arrhythmias in the elderly person. Ambulatory electrocardiography has been found to demonstrate about five times as many arrhythmias as a resting ECG (Martin Anthony, 1998).

In a study by Jerome L. Fleg et al, 1990, over 98 healthy active subjects between the age of 60 to 85 years, 28% subjects had abnormalities in their standard twelve lead electrocardiogram.

Table no.6

<table>
<thead>
<tr>
<th></th>
<th>No. Of subjects</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAHB</td>
<td>14</td>
<td>14.28</td>
</tr>
<tr>
<td>1 degree AV block</td>
<td>6</td>
<td>6.12</td>
</tr>
<tr>
<td>Supraventricular arrhythmias</td>
<td>11</td>
<td>11.22</td>
</tr>
<tr>
<td>Isolated VPCs</td>
<td>7</td>
<td>7.14</td>
</tr>
</tbody>
</table>

In the same patients a 24 hrs ambulatory ECG monitoring gave the following results.

"Study of Electrocardiographic Changes in Elderly Persons"
Table no. 7
Table showing supraventricular and ventricular arrhythmias observed in 98 healthy elderly subjects on 24hr ambulatory ECG monitoring

<table>
<thead>
<tr>
<th></th>
<th>No. of subjects</th>
<th>% of total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Supraventricular</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any</td>
<td>86</td>
<td>88</td>
</tr>
<tr>
<td>Isolated ectopic beats</td>
<td>86</td>
<td>88</td>
</tr>
<tr>
<td>≥ 30 beats in any hr</td>
<td>22</td>
<td>22</td>
</tr>
<tr>
<td>≥ 100 betas in 24 hr</td>
<td>25</td>
<td>26</td>
</tr>
<tr>
<td>Benign slow atrial tachycardia</td>
<td>27</td>
<td>28</td>
</tr>
<tr>
<td>Paroxysmal atrial tachycardia</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Accelerated junctional rhythm</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><strong>Ventricular</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any</td>
<td>78</td>
<td>80</td>
</tr>
<tr>
<td>Isolated ectopic beats</td>
<td>76</td>
<td>78</td>
</tr>
<tr>
<td>≥ in any hr</td>
<td>37</td>
<td>38</td>
</tr>
<tr>
<td>≥ 30 in any hr</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>≥ 60 in any hr</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>≥ 100 in 24 hrs</td>
<td>17</td>
<td>17</td>
</tr>
<tr>
<td>Multiform</td>
<td>34</td>
<td>35</td>
</tr>
<tr>
<td>Ventricular couplets</td>
<td>11</td>
<td>11</td>
</tr>
<tr>
<td>VT</td>
<td>14</td>
<td>4</td>
</tr>
<tr>
<td>R on T</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

In another study by Manyari DE, et al atrial and ventricular arrhythmias were studied in asymptomatic active elderly subjects. Atrial arrhythmias were present in 74% of the subjects whereas 64% of the subjects had ventricular arrhythmias.
Table no. 8
Table showing arrhythmias observed at different age groups in 86 asymptomatic elderly persons

<table>
<thead>
<tr>
<th>Arrhythmia</th>
<th>All subjects (%)</th>
<th>60-69 years (%)</th>
<th>70-75 years (%)</th>
<th>75 years (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Atrial premature depolarization</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;100/24 hrs</td>
<td>24</td>
<td>16</td>
<td>36</td>
<td>24</td>
</tr>
<tr>
<td>&gt;100/24 hrs</td>
<td>13</td>
<td>16</td>
<td>11</td>
<td>10</td>
</tr>
<tr>
<td>Couplets</td>
<td>3</td>
<td>3</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>Benign slow atrial tachycardia</td>
<td>30</td>
<td>27</td>
<td>36</td>
<td>29</td>
</tr>
<tr>
<td>Paroxysmal atrial fibrillation</td>
<td>3</td>
<td>5</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td><strong>Ventricular premature depolarization</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;50/24 hrs</td>
<td>35</td>
<td>46</td>
<td>29</td>
<td>24</td>
</tr>
<tr>
<td>&gt;50/24 hrs</td>
<td>9</td>
<td>3</td>
<td>14</td>
<td>5</td>
</tr>
<tr>
<td>Multiform</td>
<td>10</td>
<td>3</td>
<td>14</td>
<td>19</td>
</tr>
<tr>
<td>R on T phenomenon</td>
<td>3</td>
<td>0</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>Couplets</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>V Tachycardia</td>
<td>2</td>
<td>3</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Accelerated idioventricular rhythm</td>
<td>1</td>
<td>0</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td><strong>AV block</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First degree</td>
<td>7</td>
<td>3</td>
<td>4</td>
<td>19</td>
</tr>
<tr>
<td>Second degree type I</td>
<td>1</td>
<td>0</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Intraventricular conduction defect</td>
<td>5</td>
<td>3</td>
<td>0</td>
<td>14</td>
</tr>
</tbody>
</table>

(Manyani DE et al. 1990)
A. John Camm et al (1990) conducted a similar study on active elderly subjects and got the following results.

Table no. 9
Table showing the spectrum of cardiac arrhythmias detected by 24 hr DCG monitoring in 106 elderly subjects

<table>
<thead>
<tr>
<th>Arrhythmia</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial fibrillation</td>
<td>10</td>
</tr>
<tr>
<td>Established AF</td>
<td>8</td>
</tr>
<tr>
<td>Paroxysmal AF</td>
<td>3</td>
</tr>
<tr>
<td>Rapid AF</td>
<td>1</td>
</tr>
<tr>
<td>Slow atrial fibrillation</td>
<td>2</td>
</tr>
<tr>
<td>Supraventricular arrhythmias</td>
<td>27</td>
</tr>
<tr>
<td>APCs</td>
<td>21</td>
</tr>
<tr>
<td>Junctional premature beats</td>
<td>3</td>
</tr>
<tr>
<td>Atrial tachycardia</td>
<td>1</td>
</tr>
<tr>
<td>Junctional tachycardia</td>
<td>2</td>
</tr>
<tr>
<td>Junctional escape beats</td>
<td>4</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>0</td>
</tr>
<tr>
<td>Ventricular arrhythmias</td>
<td>69</td>
</tr>
<tr>
<td>Isolated VPCs (&gt;10 min/hr)</td>
<td>42</td>
</tr>
<tr>
<td>Moderate VPCs (10-100/hr)</td>
<td>14</td>
</tr>
<tr>
<td>Frequent VPCs (&gt;100/hr)</td>
<td>12</td>
</tr>
<tr>
<td>Multiform VPCs</td>
<td>22</td>
</tr>
<tr>
<td>Paired VPCs</td>
<td>4</td>
</tr>
<tr>
<td>Ventricular bigeminy</td>
<td>5</td>
</tr>
<tr>
<td>VT</td>
<td>4</td>
</tr>
</tbody>
</table>

"Study of Electrocardiographic Changes in Elderly Persons"
Results of single 12 lead ECG where -

Table no. 10

Table showing the prevalence of arrhythmias detected by 12-lead ECG in 106 elderly subjects

<table>
<thead>
<tr>
<th>Arrhythmia</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>68%</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>9%</td>
</tr>
<tr>
<td>Supraventricular arrhythmias</td>
<td>15%</td>
</tr>
<tr>
<td>Major ventricular arrhythmias</td>
<td>15%</td>
</tr>
<tr>
<td>Complete heart block</td>
<td>0</td>
</tr>
</tbody>
</table>

(Camm AJ et al, 1980)

Thus from the above studies it was concluded that both atrial and ventricular arrhythmias are common among elderly persons.

ATRIAL:

Atrial fibrillation: It is the most common and important arrhythmia in the elderly. Jerome L. Fleg et al, 1990 had reported an incidence of AF in 11% of their subjects whereas Manyari DE et al, 1990 reported it in 3% and Camm AJ et al, 1980 found it in 10% of his subjects.

In Framingham heart study also 2325 men and 2866 women between 30 and 62 years of age, studied biennially over 27 years showed the development of chronic atrial fibrillation which increased in frequency sharply with age. They found that 49 men and 49 women developed AF. It usually followed the development of overt cardiovascular disease. Only 18 men and 12 women had chronic AF in absence of cardiovascular disease (Kannel WB, Abbott RD et al, 1982).

Atrial premature beats: They occur with great frequency in elderly and their frequency increases with advancing age. Jerome L. Fleg et al, 1990 reported it in as high as 88% of his subjects. Manyari DE et al, 1990 reported it in 40% of this subjects. Camm AJ et al, 1980 reported it in 21% of his subjects.
Multifocal atrial tachycardia: It is uncommon in asymptomatic elderly persons and is usually manifested in ill old people. Fleg Jerome L et al, 1990 reported an incidence of 28% and 13% of benign slow atrial tachycardia and paroxysmal atrial tachycardia respectively. Manyari DE et al, 1990 reported it in 30% and 3% subjects respectively. Camm AJ et al, 1980 reported atrial tachycardia in just 1% of his subjects.

VENTRICULAR:

Ventricular ectopic beats: It occurs very commonly in the elderly. Its incidence was found to be as high as 80% in the study of Fleg JL et al, 1990. Manyari DE et al, 1990 reported it in 54% of his subjects whereas Camm AJ et al, 1980 reported it in 94% of his subjects.

Ventricular tachycardia: It is the next major kind of arrhythmia. Its incidence has been reported to be around 2% (Manyari DE et al, 1990) to 4% (Fleg JL et al, 1990 Camm AJ et al, 1980).

Atrioventricular block and intraventricular conduction abnormalities: In the study by Manyari DE et al, 1990 first degree AV block was detected in 7% of subjects and second degree AV block in 1% of subjects. Widening of QRS was seen in 5% of subjects with presence of RBBB in 3 subjects and LBBB in 1 subject.

In another study, as mentioned earlier on patients of severe aortic stenosis, over 65 years of age LBBB and RBBB were reported in 4.9% each and transient heart block was seen in 14.2% of subjects. (Roberts William C et al, 1970).

Table no. 11

Table showing prevalence of arrhythmias in two different studies of asymptomatic elderly persons

<table>
<thead>
<tr>
<th></th>
<th>Fleg JL</th>
<th>Camm AJ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>62%</td>
<td>68%</td>
</tr>
<tr>
<td>Supraventricular arrhythmia</td>
<td>11%</td>
<td>15%</td>
</tr>
<tr>
<td>Ventricular arrhythmia</td>
<td>7%</td>
<td>15%</td>
</tr>
<tr>
<td>AV block and intraventricular conduction defects</td>
<td>20%</td>
<td>0</td>
</tr>
</tbody>
</table>

"Study of Electrocardiographic Changes in Elderly Persons"
RATE DISTURBANCES:

As the age advances there is a decline in the automaticity of the SA node. It has been shown that there is a significant fall in the percentage of muscles in the older age range associated with a corresponding increase in the percentage of fibrous tissue (Davies MJ, Pomerance Ariela, 1972).

Studies have shown that this decline in the discharge rate with advancing age is hardly significant (Kostis John B, Moreyra Abd E, Alnendo, Manuel T, 1982; Landowne M, Brandonbrener M, Sheck NW, 1955).

The results various studies are as follows:

Table no. 12

<table>
<thead>
<tr>
<th></th>
<th>Fleg JL</th>
<th>Manyari DE</th>
<th>Camm AJ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus rhythm</td>
<td>100%</td>
<td>-</td>
<td>93%</td>
</tr>
<tr>
<td>Sinus tachycardia</td>
<td>77%</td>
<td>86%</td>
<td>14%</td>
</tr>
<tr>
<td>Sinus bradycardia</td>
<td>89%</td>
<td>73%</td>
<td>10%</td>
</tr>
<tr>
<td>Sinus pause</td>
<td>2%</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Sinus arrhythmia</td>
<td></td>
<td></td>
<td>14%</td>
</tr>
<tr>
<td>Mild</td>
<td></td>
<td></td>
<td>43%</td>
</tr>
<tr>
<td>Moderate</td>
<td></td>
<td></td>
<td>47%</td>
</tr>
<tr>
<td>Marked</td>
<td></td>
<td></td>
<td>10%</td>
</tr>
</tbody>
</table>


Thus from the above study results, it can be summarized that sinus rhythm prevails in most of the people with advancing age; 100% according to Fleg JL et al, 1982 and 93% according to Camm AJ et al, 1980. Sinus tachycardia was found in 77% of cases by Fleg JL et al, 1982, 86% by Manyari DE et al, 1990 and 14% by Camm AJ et al, 1980. Sinus bradycardia was found in 89%, 73%, and 10% of elderly in the studies by Fleg JL et al, 1982, Manyari DE et al, 1990 and Camm AJ et al, 1980 respectively. Sinus pause was reported in only 2% cases by Fleg JL et al, 1982. Sinus arrhythmia was reported in as high as 47% in the study by Manyari DE et al, 1990.

"Study of Electrocardiographic Changes in Elderly Persons"
With this detailed interpretation and analysis guide for each ECG, various conclusions regarding the underlying diseases in the elderly can be made. It helps us to know the various hidden disorders and also to study in detail the known disorders in this group. The ECG changes are varied with varied forms of heart diseases and other systemic diseases. For study we have classified the various cardiovascular diseases into -

1. Heart failure
2. Hypertension
3. Coronary artery diseases
4. Arrhythmias
5. Valvular heart diseases
6. Cardiomyopathy

**HEART FAILURE:** It is the leading first listed diagnosis among hospitalized older adults. In evaluating patients with heart failure, it is important to identify not only the underlying, but also the precipitating cause. The various precipitating causes could be infection, anaemia, thyrotoxicosis and arrhythmia rheumatic, viral and other forms of myocarditis, infective endocarditis, physical, dietary, fluid, environmental and emotional excesses, systemic hypertension, myocardial infarction, pulmonary embolism (*Braunwald Eugene, 2001*).

In a study, heart failure was found to be highly prevalent, affecting 1% of person in their fifties and rising progressively with age to afflict 10% of persons in their eighties. The annual incidence also increased with age, from about 0.2% in persons 45-54 years to 4% in men 88 to 94 years with the incidence approximately doubling with each decade of age. Women lag behind men in incidences at all ages. Male preponderance was higher because of a higher rate of coronary heart disease. Heart failure, once manifest was highly lethal, with 37% of men and 33% of women dying within 2 years of diagnosis. The 6 years mortality rate was 82% in men and 67% in women, which corresponds to a death rate four fold to eight fold greater than that of the general population of the same age. Sudden death accounted for 28% cardiovascular deaths in men and 14% in women with heart failure.

"Study of Electrocardiographic Changes in Elderly Persons"
Significance of ECG:

Factors, which included deteriorating cardiac function, included low vital capacity, sinus tachycardia and ECG evidence of LVH. Besides ECG-LVH, other ECG abnormalities like intraventricular conduction disturbance and non-specific repolarisation abnormality is associated with substantial risk, which heralded the occurrence of serious cardiovascular disease of all varieties, with higher risk ratios for heart failure. ECG-LVH characterized by increased voltage unaccompanied by repolarization abnormality carried a lesser risk of failure than that associated with evidence of strain. Also, it is likely that ECG version reflects both ischaemic myocardial damage and anatomic hypertrophy. When both are present, the risk is substantially higher than when either is present alone (Kannel WB, Belanger AJ, 1991).

HYPERTENSION: It is the commonest health problem in the old age. Essential hypertension is the commonest cause of high blood pressure in old age. A large number of elderly hypertensives have isolated systolic hypertension. It has a greater predictability for stroke, ischaemic heart disease, chronic heart failure, renal failure and mortality than diastolic blood pressure in old age (William Gordon H, 2001).

In fact hypertension is not considered as a disease but as a risk factor for the complications it causes. Cardiovascular mortality has been shown in the Framingham study to be about three times as high in hypertensive elderly people compared with those with normal blood pressure. Two main mechanisms have been suggested for this high level of morbidity and mortality. Firstly, there is accelerated development of atheroma of the coronary arteries, resulting in ischaemic heart disease, secondly, left ventricular hypertrophy occurs due to the increase in left ventricular workload. LVH is related to the severity and duration of the hypertension and serves as a major prognostic marker. These two factors work together - like LVH makes the myocardium more susceptible to ischaemic injury in association with coronary atheroma (Scott Andrew K, 1998).

Blood pressures greater than 140mmHg systolic and diastolic less than 90mmHg are quite prominent in the elderly. The diagnosis of hypertension should be

"Study of Electrocardiographic Changes in Elderly Persons"
made only after the blood pressure is found to be elevated on three separate occasions. In the elderly there are several problems in obscuring a correct blood pressure using the sphygmomanometer cuff. The non-complicated arteries cause greater changes in the stroke volume, which results on wide variations in systolic blood pressure. Another problem, which is termed as "pseudohypertension" is caused by a calcified brachial artery, which is not easily compressed by sphygmomanometer cuff. Falsely low systolic blood pressure can also occur because of the auscultatory gap in 20% of the elderly (Chelitlin Melvin D, Zypes Douglas P, 2001)

The magnitude of the problem of hypertension is such that it is considered an "iceberg disease". A rule of halves has been described to tell the magnitude of this problem, according to which about half the hypertension subjects in the general population of most developed countries were aware of the condition, only about half of those aware of the problem were being treated and only about half of those treated were considered adequately treated. The prevalence of hypertension is as high as 25% in some developed countries. In India, the data has been suggested by two well planned studies conducted by WHO in Rohtak, which shows a prevalence of hypertension as 59.9/1000 males and 69.9/1000 females in urban areas and 35.5 and 35.9 per 1000 in males and females respectively in rural population (Park K, 1997).

Table no.13
Table showing death rate per 1,00,000 populations from hypertensive disease in selected countries.

<table>
<thead>
<tr>
<th>Country</th>
<th>Total</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>USA (1980)</td>
<td>14.4</td>
<td>12.6</td>
<td>16.0</td>
</tr>
<tr>
<td>Japan (1982)</td>
<td>11.6</td>
<td>9.5</td>
<td>13.6</td>
</tr>
<tr>
<td>Eng and Wales (1982)</td>
<td>10.4</td>
<td>9.4</td>
<td>11.3</td>
</tr>
<tr>
<td>France (1981)</td>
<td>9.9</td>
<td>7.7</td>
<td>12.1</td>
</tr>
<tr>
<td>New Zealand (1981)</td>
<td>9.2</td>
<td>8.3</td>
<td>10.1</td>
</tr>
<tr>
<td>Scotland (1983)</td>
<td>8.3</td>
<td>7.0</td>
<td>9.6</td>
</tr>
<tr>
<td>Canada (1982)</td>
<td>6.1</td>
<td>5.0</td>
<td>7.2</td>
</tr>
<tr>
<td>Netherlands (1982)</td>
<td>4.8</td>
<td>3.9</td>
<td>5.7</td>
</tr>
</tbody>
</table>

(Park K, 1997)

"Study of Electrocardiographic Changes in Elderly Persons"
Significance of ECG:

ECG is an important investigation in these patients. In the SHEP study, Baseline ECG abnormalities were detected in as high as 61% of the subjects. A relative risk of non-fatal myocardial infarction plus coronary death for the subgroup of people free of baseline ECG abnormalities for active treatment compared with placebo was calculated. It was found to be 0.83 and for those with baseline ECG abnormalities was 0.69. Thus ECG provides important information regarding the risk of various complications possible in elderly hypertensives. (JAMA, 1991)

Thus as large is the magnitude of the problem of hypertension, its treatment is equally important. Nine major trials with 15,599 patients older than 59 years, death rates in the control group varied between 2.7% and 77.2%. Stroke and coronary mortality increased with the severity of illness. With treatment there was an approximate reduction of 12% in all causes of mortality. There was a 36% reduction in stroke mortality and a 25% reduction in coronary heart disease mortality. Coronary morbidity was reduced by 15% and stroke morbidity was reduced by 35% (Insua JT, Sacks HG, Lav TS et al, 1994).

Thus lowering the systolic blood pressure decreases cardiovascular and total mortality as well as hypertensive complications of heart failure, renal failure and stroke. The goal of treatment of hypertension in elderly patients should be a lowering of systolic blood pressure to less than 140mmHg. The Swedish Trial in old patients with hypertension (STOP-H), subjects were put on atenolol, metoprolol, pindolol or hydrochlorothiazide and amiloride and compared with a placebo. A 28% decrease in fatal myocardial infarction and a 67% decrease in sudden death and 47% decrease in stroke resulted (Cheitlin Melvin D, Zypes Douglas P, 2001).

CORONARY ARTERY DISEASE: In general, advancing age is associated with increasingly severe, diffuse atherosclerosis and with damage to the left ventricle. Therefore, almost all clinical manifestations of ischaemic heart disease have a higher mortality rate and a worse outcome in the older population. Also, their clinical assessment is limited by the co-existence of

"Study of Electrocardiographic Changes in Elderly Persons"
other diseases that make interpretation of symptoms difficult (Chetlin Melvin D, Zypes Douglas P, 2001). Thus, in elderly, a high clinical index of suspicion plus the use of objective parameters such as stress test results are important in assessing and diagnosing ischaemic heart disease. Treadmill testing is also useful to detect silent ischaemia, which occurs with increasing frequency in the elderly and is a strong risk factor for the development of future symptomatic cardiac disease (Lakatta Edward G, Schulman Steven P, Gerstenblith Gary, 2001, Gerstenblith Gary, 1998).

As the population grows older there is an increase in the prevalence and annual incidence of development of CAD. There is also male propensity observed, with men developing CAD at a younger age than women. (37% men aged 64-74 years as compared to 22% women of the serve age group). The prevalence of CAD in the Framingham cohort aged 75-84 years was 44% in men and 28% in women. In 85-94 years age group it was 48% in men and 43% in women (Chetlin Melvin D, Zypes Douglas P, 2001).

In Baltimore longitudinal study of aging population, the prevalence of exercise induced silent ischaemia which was defined by both electrocardiographic and thallium scintigraphic criteria increased from 2% in the fifth decade to 15% in the ninth decade (Gerstenblith Gary, 1998).

The investigators in the Mayo clinic did an autopsy study and reported that 72% of men and 54% of women 70 years on older had 75% or greater stenosis of at least one of the major coronary arteries (Gerstenblith Gary, 1998)

In India the prevalence was found to be 65.4 and 47.8 per 1000 males and females respectively in urban areas. In rural areas the prevalence was 22.8 and 17.3 per 1000 males and females respectively (Park K, 1997)

The main risk factors attributed to CHD include modifiable and non-modifiable factors. The non-modifiable factors are age, sex, family history and genetic factors. The modifiable risk factors are hypertension, cigarette smoking, hyperlipidemia, diabetes, obesity, sedentary habits and stress (Gerstenblith Gary, 1998; Park K, 1997; Selwyn Andrew P, Chetlin Melvin D, Zypes Douglas P, 1998)
CONDUCTION DEFECTS: Cardiac arrhythmias occur with increasing frequency with advancing age both in apparently healthy individuals and in those with cardiopulmonary disease. Two principle mechanisms have been suggested for this, first is increased automaticity in cardiac cellular tissue and the other is the presence of re-entry phenomenon (Martin Anthony, 1998).

The major symptoms produced by these defects include shortness of breath, angina, palpitation and dizziness or fainting. AF may be complicated by thromboembolism giving rise to strokes or peripheral emboli (Martin Anthony, 1998).

The diagnosis of these arrhythmias can be made in asymptomatic elderly people by clinical examination and a 12-lead ECG. But this can be at times confusing and an ambulatory electrocardiography or dynamic electrocardiography (DCG) is a better option (Camm AJ et al, 1980).

Various studies have shown that a healthy population of elderly subjects shows a substantial prevalence of supraventricular and ventricular ectopic beats, both isolated and complex. High degree AV block, profound sinus bradycardia, abnormal sinus pauses and sinus arrest are rare in normal elderly subjects. (Fleg Jerome L et al, 1982; Manyari Dante E et al, 1990; Camm John et al, 1980).

VALVULAR HEART DISEASES: Most of the causes of valvular heart disease in the young also affect the old, but the majority of valvular heart disease in the elderly is degenerative. All the values may be affected, but the mitral and aortic valves are more commonly affected because they are subjected to greater pressure and trauma (Braunwald Engene, 2001; Channer KS, 1998).

Valvular dysfunction in the elderly is not as well tolerated as in young. The functional adaptations are limited by age related degeneration a cardiac muscle and an increased incidence of associated disease. As has been described earlier, the left ventricle hypertrophies with age and increases in weight. This is associated with a decrease in compliance. Ventricular myocardium is less responsive to catecholamines, and exercise induced changes in heart rate are
also blunted. The cardiac conduction tissue also degenerates, and a consequent bradyarrhythmias associated either with sinus or atrioventricular node dysfunction increases with age. Atrial and ventricular extrasystoles and tachycardias are more common in the elderly. Arterial compliance also decreases, causing a rapid upstroke in the carotid pulse and higher peak systolic pressures (Channer KS, 1998). These changes and interactions are important since they influence the presentation, history and prognosis of valvular heart disease.

Of all the valvular heart diseases, aortic valve disease is the most common in the elderly resulting due to calcific deposits in it and leading to stenosis (Aronow Wilbert S, Kronzon Itzhak, 1990).

In a study of 781 patients, congestive heart failure, syncope angina pectoris was present in 24% with mild AS and in 61% with moderate AS and 89% with severe AS. An aortic systolic ejection murmur was heard in all (100%) patients with severe AS and moderate AS and in 95% patients with mild AS. LVH was the most important ECG finding in these patients (Aronow Wilbert S, Kronzon Itzhak, 1990).

In another study, comparison certain clinical and anatomic features of subjects below 65 years and those above 65 years was done. Striking differences were observed between the two age groups. In 90% elderly cause of valvular lesions was simple degeneration. The systemic arterial pulse had a normal rate of rise and a normal or increased pulse pressure and the dominant sex was the females. Whereas in the younger age group 45% subjects had tricuspid valve and the cause of the lesion was found most commonly to be congenital malformation and it was more common in the males (Roberts William C et al, 1987).

ECG in these elderly patients revealed LVH in 11 patients, AF in complete LBBB in 1, complete RBBB in 1 and transient complete heart block in 3 (Roberts William C et al, 1987).

"Study of Electrocardiographic Changes in Elderly Persons"
CARDIOMYOPATHY: Both dilated (DCM) and hypertrophic (HCM) cardiomyopathy occur in elderly. Most of the DCM is of unknown etiology (Chetlin Melvin D, Zypes Douglas P, 2001).

Amyloid cardiomyopathy: It is noted primarily in the older people (Chetlin Melvin D, Zypes Douglas P, 2001). In a report by Hwang Yue-Ting et al, 1993 the major cardiac manifestations of amyloid cardiomyopathy consists of congestive heart failure, cardiomegaly or a variety of rhythm and conduction disturbances. An abnormal ECG was noted in 40 of 57 patients. They found low voltage QRS (<15mm in I, II and III) in 63%, a "myocardial infarction pattern" in 83%, an abnormal QRS axis in 73%, arrhythmia in 73%, various degrees of heart blockage in 45% and complete bundle branch block in 18% (Hwang Yue Ting, Tsong Chuen-Den et al, 1993).

Hypertrophic cardiomyopathy: It is another important disease that is being diagnosed increasingly in elderly patients. Compared with younger patients, the elderly have milder hypertension and fewer have a history of syncope. ECG shows LVH and widespread deep, broad Q waves that suggest an old myocardial infarction. Many patients have arrhythmia both atrial (SVT) and ventricular (VT) during ambulatory (Holter) monitoring (Wyne Joshua, Braunwald Eugene, 2001).

SOCIAL ISSUES RELATED TO AGING AND HEALTH:

Paucity: Especially in Asian and African countries, the main functions of the family include providing shelter and food, child rearing, sexual gratification and procreation, socialization and religious initiations and care of the sick, the dependent and the elderly.

The traditional family dynamics according to an ancient Sanskrit saying highlight this for the sake of the family - sacrifice the community and for the sake of the soul sacrifice the whole world. This was the advice tendered by Vidura to Dhritrashtra at the time of Duryodhan’s birth (Rao Venkoba AJ, 1999).
An analysis of the type of family by the task force project on elderly funded by Indian Council of Medical Research in Madurai revealed that only 38% lived in a joint family, while 52% belonged to an extended family and 10% to a nuclear family. The attitude of family members towards the elderly varied from neutral in 10%, being rejected, unwanted and just tolerated in 38% while 62% were loved and respected but had no control over the other members. It was found that in a small percentage of families, the elderly were happily integrated and had control over the family (Rao Venkoba AJ, 1999).

Aging is associated with several social problems namely isolation, poverty apparent reduction in family support, inadequate housing, impairment of cognitive functioning, mental illness, widowhood, loss bereavement. Limited options of living arrangements towards end of life and so on. However, with industrialization, urbanization and disintegration of traditional joint family. The elderly in India are likely to face even more social problems (Sharma OP, 1999).

Disability and dependence in elderly:

Office of population census and surveys (OPC) survey, evidenced that majority of people with hearing or visual impairment, mental health problem and mobility restriction were beyond the retirement age.

Major survey conducted by OPCS was in 1980 and it included private household and private communal establishments. The survey studied, those who were deemed to be disabled and were assessed on a range of dimensions to identify the nature and degree of disability. This allowed researches to classify respondents on a scale of severity from 1 to 10. For e.g. some one with a score of 1 had hearing impairment in one ear. A patient with a score of 6 had a stroke or heart condition, which limited his/her mobility and severely restricted self-care whereas a patient with a score of 10 was totally dependent on others, advanced dementia, immobility or multiple chronic illnesses (Johnson Malcolm L, 1999)

"Study of Electrocardiographic Changes in Elderly Persons"
Chronic illnesses in elderly:

Bond and Carstairs in their survey of elderly people in Scotland, identified heart condition in 19%, arthritis and related condition affecting the back in 10-15%, as the most widely reported illness that restricted functional capacity. This in turn has consequences for the performance of activities of daily living, where bending and stooping are the ingredients in the process. The ability to cope with every day tasks of personal hygiene and domestic task is critical for the maintenance of an independent existence. That is why ADLS has received much attention in social surveys (Ramsey MJ, 1995).