REVIEW OF LITERATURE

Best & Taylor’s statement defines homeostenosis: [1] “With the advancing age significant reduction in functional capacity occur in many different organ system.”

Mammalian gerontologists have defined aging in terms of gradual, insidious and progressive declines in structure and function (involving molecules, cells, tissues, organs and organism) that begin to unfold after the achievement of sexual maturity. [2]

The incidence, percentage values, the life expectancy, at birth and at 60 years have been mentioned for India and Internationally documented [3]

Western authorities [4] believe that, indeed there is scope for a great deal more work defining the hematological changes consequent of aging.

A number of Patho-Physiologic states have been stated having correlation to aging have been documented. [5-10]

Wintrobe [11] is of opinion that, although this field is neglected, from what is known, it seems unlikely that aging has effects on hematologic parameters, still however, fall in hemopoietic progenitors with age, chromosomal shortening, statistical fall in mean hemoglobin, during sixth through eighth decade, decrease in adaptive immunity, and significantly rising ESR, are mentioned.

Warell et al. [12] have stated that, several studies indicate pro coagulant and fibrino lytic activity changes and levels and diminishing coagulant factors. They have
noted, aged living in isolation have iron deficiency anemia and folate deficiency. Platelet count, serum erythropoietin and white cell count are not changed.

Wintrobe (Ed. John P. Greer et al. ) has quoted study of Larson et al. (2006),[13] Appelbaum et al.(2006 ),[14] Thieblemont and Coffier(2007),[15], NHANES III study ,(1988-1994)[16] and have mentioned decreasing survival chances in ALL, with increasing age, worse out come with AML in aged, increase in number of non Hodgkin Lymphoma with aging and increasing prevalence of anemia[WHO]defined , after 50 years of age respectively.

Bonow R.O. et al.[17] mention [In Braunwald’s ‘Heart Disease’ -2012] that, Cardiovascular disease is both most frequent diagnosis and the leading cause of death in both, man and women older than 65 years.

Lakatta E.G. et al. [18, 19, 20, and 21] have excellently elaborated hall marks of Cardio- vascular aging and relevant parameters, discussed in detail the cellular and molecular clues to heart and arterial aging. They have also correlated the aging at macroscopic and molecular levels. O’Rourke M and Hashimoto J. [22] have studied clinical perspectives of mechanical factors in arterial aging.

In Braunwald’s ‘Heart Disease’ , R.O.Bonow et al.(2012)[23] have summarized cardiovascular changes in aging , like, increase of intimal thickening, arterial stiffening, rise in pulse pressure, increase in pulse wave velocity, early central wave reflection, decrease endothelium mediated vaso-dilation, increase in L A size, appearance of premature complexes, decrease in maximal heart rate, diminished heart rate variability, prolonged conduction time, valvular sclerosis and calcification, rise in L V wall tension, prolonged myocardial contraction, longer end diastolic filling rate, decreased max. Cardiac output, RBB block, and appearance of ventricular premature
complexes, and have mentioned that any one or a set of combination can occur in aging.

Molecular mechanisms of aging and its evolution [26, 27, 28,] by ROI Free radicals, genetic faults and fractures, telomerase participation, IGF-1, m-TOR, and Histone deacetylase [Sirtuin] mediated cumulative burden of stress induced challenges, and progressively diminishing reparative genetic machinery, diminished inotropic response to aged myocardium, defects in Catechol mediated delivery of Calcium ions, have been described by authorities and their associates [24, 25, 26] in laudable depth.

Radio nuclide Scintigraphy, [27] and Echocardiography [28] have added clarity to aforesaid assessments and these investigative tools are used in documented evidences in cardiovascular responses in aging.

Alfred P. Fishman et al. [29] have demonstrated changes in shape of lung, larger airways, lung parenchyma, calcification/hypertrophy of mucus glands, flattening of alveoli, diminished elastic recoil/ left side shifting of P-V Curve, variation of PIMax and PEMax changes, [30,31] significant decrease in static compliance of chest wall and increase in FRC[32], diminished ventilatory response to hypoxia, [33], Non – linear PEP decrease[34], decrease in FEV1 and FVC,[35] increase in VD/VT ratio[36], decline in DLCO, [37], and ventilation to perfusion V/Q mismatch[38] is well documented.


In A] there is Lamin –A defect, in B]- WS ATP Dependant Helicase defect.