

of hematology so multiple myeloma is the disorders of plasma cells it is a plasma cell dyscrasia where the plasma cells will keep on secreting these light chain proteins so these light chain proteins will keep on depositing in various organs and responsible for so many complications reactive systemic amyloidosis AAA which is seen in mainly in chronic inflammatory conditions hemodialysis associated amyloidosis will have a beta microglobulin form so these are the different types of amyloidosis variables again CA a type of analysis family a minute Aryan fever again you see a type of amyloidosis family amyloid or topic neuropathy is where you see a ATR type of amyloid proteins system external amyloidosis again attr it will naturally peptide Lake proteins have seen in mainly in the heart also they are seen localized Nam analysis where only one are the to Arkansas get affected Inazuma this is for example a beta type of amulet will be deposited between the neurons and they are the one material responsible for the memory loss the medullary chasm of the earth is very peculiarly showed a portion of the a Cal Cal stands for calcitonin so AG care type of home already seen with the medullary carcinomas a lot of Langerhans in bankers also show a l a ppm type of amyloid isolated at al amyloidosis this is the one which you need to remember and that is the one which is responsible for suddenly cardiac death one of the important cause for sudden cardiac death is the portion of the amyloidosis so this you need to take the multiple sections from the heart of such a patients and if at all you see homogeneous material you have to stain with a special stain for amyloid then you have to confirm that it is a amyloid that is causing the arrhythmias and causing the death in these patients prioritises again there is a biggie later for form white prpsc that's a type of formula and that is seen with the prion disease so remember my friends there is a huge list of types of amyloidosis and there are so many conditions where amyloidosis are seen every year this particular chart is keep on you know modified every six months they will three months three most six months they will discover a new type of amyloid and they keep on adding so there are Amano projects who love to study and discover these different pathological proteins so what is the pathogenesis of this amyloid acid it is all because of the abnormal folding of the proteins so these misfolded proteins are very highly unstable in their configuration and they will self associate and such a misfolded proteins they will accumulate in the extrasolar matrix thereby resulting in the arc anomaly initial will be there we are gana McCauley but later on they results in the dysfunction of that particular arc on and finally it can desert even in the pressure atrophy of the cells so all those will be are genomically in initial event but later on if the patient surveys for long time then there can be

not allow even the HIV virus it does not allow any other viruses to enter inside the body the second component is difficult and excels when I use the word Rico static cells it is mainly there neutrophils and the macrophages the two important Fregosi attic cells are neutrophils and macrophages they are the calm part and parcel of the innate immune system the third is the in cases nothing but natural killer cells they are again a part and parcel of the innate immune system the fourth is the plasma proteins and even complement system so they are the for comfort components of the innate immune system as compared to that an optimal T is the one which develops after the microbes enter into the body and our body recognizes these microbes as antigens this sort of dignity is more powerful in combating the infections it consists of lymphocytes and their products their products means either antibodies of the cytokines so we classify the immune system as such into two things one is innate immunity which is also known as native or a natural immunity or dock immunity which is also known as the Quadra specific which is of two types humoral and cell mediated then use the word tomorrow which may be mediated by billion for sites which will themselves convert it into plasma cells so what is plasma cell it's nothing but modified billion for sites they are the one which will secrete the immunoglobulins are the antibodies gam Dane IgG IgM IgA ize so they are the antibodies different antibodies secreted by the class muscles cell mediated immunity mainly of all of T lymphocytes to subset of T lymphocytes cd4 and cd8 T lymphocytes so they will have attachment to the see molecules cd4 will classically belch to the MHC class 2 molecules whereas cd8 will classically bench to the MHC class 1 molecules so let us deal with all these components what is the role of the figure Sebek cells what are the different types of cells what is the role of in cases what is

accidents a lot of for traumatic factors the Verity of disorders like syndromes diabetes is a syndrome atherosclerosis the take the calorie intake all those factors will decide the aging process even more nutrition and obesity both will decide the aging process the psychological factors and even social health factors all those will decide the stress which endure have a lot of effect on the aging process what are the structural and biochemical changes that are seen at a molecular level functionally as the cells advances in its aging process there is reduced and reduced the phosphorylation oxidative phosphorylation by the mitochondria thereby resulting in decreased production of the ATP's decrease synthesis of the nucleic acids and even decrease synthesis of the enzymatic proteins decrease the capacity to uptake the nutrients and there will be decreased capacity to repair the DNA damage that has incurred during the aging process so the DNA damage is the one which occurs as the cell advances and the body is having that capacity to repair this DNA damage this particular mechanism or capacity it will be get hampered as the day's advances as the aging advances so morphologically we will say at electron microscopy level the ropes of a nucleus they will be irregularly folded and optimally folded you will see the mitochondria which are pleomorphic they vary in their size and shape they may show vacuous empty appearing vacuous decreased number of endoplasmic reticulum thereby decrease in the sious of the proteins distorted gorga apparatus again proteins whatever they are produced synthesized by endoplasmic reticulum they are not structurally stable they'll be distorted colgate practice so proteins of not tough structurally formed well and as the base advances there will be accumulation of a pigment very peculiar pigments openness lipofuscin which is also called as lipo chrome or a wear and tear pigment that

to avoid the aging process so that's about aging say astray espero is the history tomorrow is the mystery right today is the gift so that's why we have to live in today so that's percent"

Summary Points

- Aging is a progressive loss of structural and functional capacity of cells leading to cell death.
- Aging is influenced by genetic factors (60%) and environmental factors (40%).
- Environmental factors include accidents, traumatic factors, disorders like syndromes, diabetes, atherosclerosis, calorie intake, nutrition, obesity, psychological and social health factors.
- As cells age, there is reduced oxidative phosphorylation by mitochondria, decreased production of ATP, nucleic acids, and enzymatic proteins, decreased capacity to uptake nutrients and repair DNA damage.
- Morphological changes in cells include irregularly folded and optically folded nuclear ropes, pleomorphic mitochondria, decreased endoplasmic reticulum, accumulation of lipofuscin pigment and advanced glycation end-products (AGEs), accumulation of abnormally folded proteins, and defective organelles.

metaplasia there is a chance that it can undergo this prostate change and it can even become a premium rusty condition a normal protective mechanisms which are Lygia they are going to get lost for example we have a respiratory Soros satisfied ciliated respiratory epithelium in the you know this bronchial thing so in a continuous smoke asteroid smokers what will happen this particular epithelium will interpose promise metal is here so the normal functions the respiratory mechanism what will have a protective mechanism that is going to get last so metaplasia may can make that loss of normal protective mechanisms are may be last and persistent signals that results in metaplasia can predispose to the development of the neoplasia so in a matter procedure the cellular adaptation in which the indigenous cells are live replaced by the cells which are better suited for to tolerate the specific abnormal environment so example of first squamous metaplasia is classically in the end of cervix where you know at the ecto civics and endocervical junction the normal endocervical tall terminal ileum is replaced based common sense because of the continuous dilatation like what happens in a patient with a prolapse so this is an example of squamous metaplasia of the endo cervix similarly opposite to this that takes place in the use of a lower-end officer Figgis marble it is lying by stratified squamous epithelium but in a patient with the reflux esophagitis this stratified squamous epithelium is replaced by simple columnar gastric type of epithelium so that what happens in a patient is a G and E yes - it's official rip plus disease so that is so far as Barrett's esophagus Barrett's esophagus there is a classical example of a metaplasia here again it is a adoptive mechanism where lower end of use of figures is replaced by gastric type of epithelium but remember it may be a premium plastic it can predispose to the development of the