OBESITY
AN EPIDEMIC OF MODERN ERA

-Dr. VIPIN KUMAR

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OBESITY:

AN EPIDEMIC OF MODERN ERA

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PREFACE

Obesity is an epidemic of present century as 600 million adults & 100 million of childerns were obese. In year 2013 the American Medical Association classified obesity as a disease. At present time according to WHO approximately 30% of world population not performing sufficient physical exercise. People worldwide having less active life style. Obesity is a common medical condition in which excessive body fat has accumulated in the body, beyond a limit it have many adverse effect on our heath. Three factors are primarily responsible for the obesity, these are excessive food intake, lack of physical activity & genetic susceptibility. Sedentary life style is a main culprit for obesity. While among the other rare causes are endocrines disturbences, genes, medications & psychological disorders.

Obesity is responsible for increasing mortality & morbidity due to its severe & life threatening complications such as Diabetes mallitus-2, hypertension & Ischaemic Heart Disease (IHD) are the prominent. While the others complications are Sleep apnoea, Acute Respiratory Death Syndrome (ARDS), Insomnia, Gall stone, Osteoarthritis, Impotency, Depression & Varicose veins.

Mortality due to obesity is preventable. Among the preventable aspects positive changes in life style i.e. diet & exercise. There are limited role of medicines too in the decrease of appetite & fat absorption from intestine. The aim of this introductory book to evaluate the common & less common causes of obesity& much more focus on the preventive aspects of the disease since “Prevention is better than cure”. Since Medicines have a limited role in the treatment of this disease. According to our ancient books of Indian medicine Exercise is the only & best tool to reduce the obesity. World population can get rid of obesity& its complications by exercising better & healthy life style.

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Obesity is a common metabolic disorder now a day's worldwide. It is one of the oldest disorder documented in medical science. It is widely described in modern system of medicine. Its a metabolic disease & related with life style.

World Health Organisation declared Obesity as the greatest health Problem of the present century. According to WHO, approximately 20-40 % of adult population & 10-20% of children are obese (park & park - S.P.M. 18 th edition, 2005) Obesity now became a Pandemic affecting almost all the continents. Obesity & Dyslipidaemia are receiving much attention in medical field due to increasing Morbidity & mortality in our society. There are so many complications of Obesity, some main are Diabetes mellitus, Hypertension, Coronary Artery Disease (CAD) , Impotency & Cancers. The mortality due to association of Obesity &diabetes increasing very fast in society. The death due to diabetes & its complications stand 4th in this table.

Genetic Predispotion as well saturated , high calorie over diet along with Decreased energy expenditure have been regarded as the primary aetiological factors.

ETYMOLOGY OF OBESITY

The term obesity is derived from Latin word Obesitas, which mean Stout, fat or Plump. Esus is the past participle of edere (to eat), ob (over) added to it (online Etymology Dictionary, obesity, 2010).

Obese is derived from Latin word Obesus, Past participle of 'Obedere' to eat all over.

Ob (Over) + Edere (Eat)

DEFINITION OF OBESITY

A condition in which there is an excessive amount of fat is known as obesity.

It may be defined as an excess of body fat that poses a health risk. Obesity is a state of excess adipose tissue mass.

Obesity may be defined as an abnormal growth of the adipose tissue due to an enlargement of fat cell (hypertrophic Obesity) or an increase in cell number (hyperplasic Obesity) or a combination both.

OVERWEIGHT

An individual is said to be overweight if its weight is greater than average weight for his age, height & sex. Overweight is generally due to obesity but it may arise due to increase muscle mass, other factors such as fluid retention. In this way, very muscular individuals may be overweight by arbitarary standards without having increased adiposity. Obesity is therefore more effectively defined by assessing its linkage to morbidity or mortality.

10 % increase weight above normal is considered as overweight & over 30 % excess of body weight is considered to be obese. Body Mass Index (BMI) is a measure of body fat in relation to height. It is calculated by dividing the body weight (kg) by the square of height in meters . BMI > 30 is most commonly used as indicator of obesity in both the sexes.

The lipids are stored in the form of triglycerides in adipocytes or the fat cells which constitutes the Adipose tissue. The adipose tissue along with adipocytes also contains a stromal or vascular compartment in which preadipocytes reside. The adipose mass increases by enlargement of adipose cell through lipid deposition as well as an increase in no. of adipocytes. Apart from storing fat, an adipocytes releases numerous molecules in regulated manner. These includes the energy balance regulating harmone Leptin, cytokines such Tumor Necrosis Factor (TNF), Compliment factor such as Factor D (also known as Adipsin), Prothombotic agents such as Plasminogen Activator Inhibitor & a component of the blood pressure regulating system Angiotensinogen. These factors play an important role in physiology of lipid Haemostasis, Insulin sensitivity, blood pressure management & coagulation & are likely to contribute to obesity related pathologies.

EPIDEMIOLOGY OF OBESITY

WHO in 1997 recognised obesity as a global epidemic. In the ancient time prevalence of obesity was rare. In the 20th century it became common. In 2008, WHO estimated that approximately 500 million adults (10 %) were obese with higher rates in women than men. In year 2015 600 million adults (12%) & 100 million childern were obese. 40 % of population in United State were obese in year 2015 .The rate obesity also increases with age at least upto 50 or 60 years old.

In India urbançation & modernçation has been associated with obesity. In northern India obesity was most prevalent in urban population (male - 5.5%, female - 12.6%), followed by the urban slums (male - 1.9%, female - 7.2%). Obesity rates were the lowest in rural population (male - 1.6%, female - 3.8%) . Socioeconomic class also had an effect on the rate of obesity. In women of high socioeconomic class rate were 10.4% while in lower socioeconomic class rate were only 0.9%. Due to Urbanisation & increasing wealth concerns Obesity epidemics growing very fast in India. In India Overweight (female) - 47.5%, while (male) - 32% & Obese(female) - 14%, male - 03%.Abdominal Adiposity(female) - 35%, male - 49%.Approximately 01 Billion Population Overweight World wide&>350 million Obese.2.5 Million Obesity related deaths/ yr.Childhood Obesity also increasing very fast in Delhi 28% children are overweight/ obese in the 14 - 18 yr. age group.

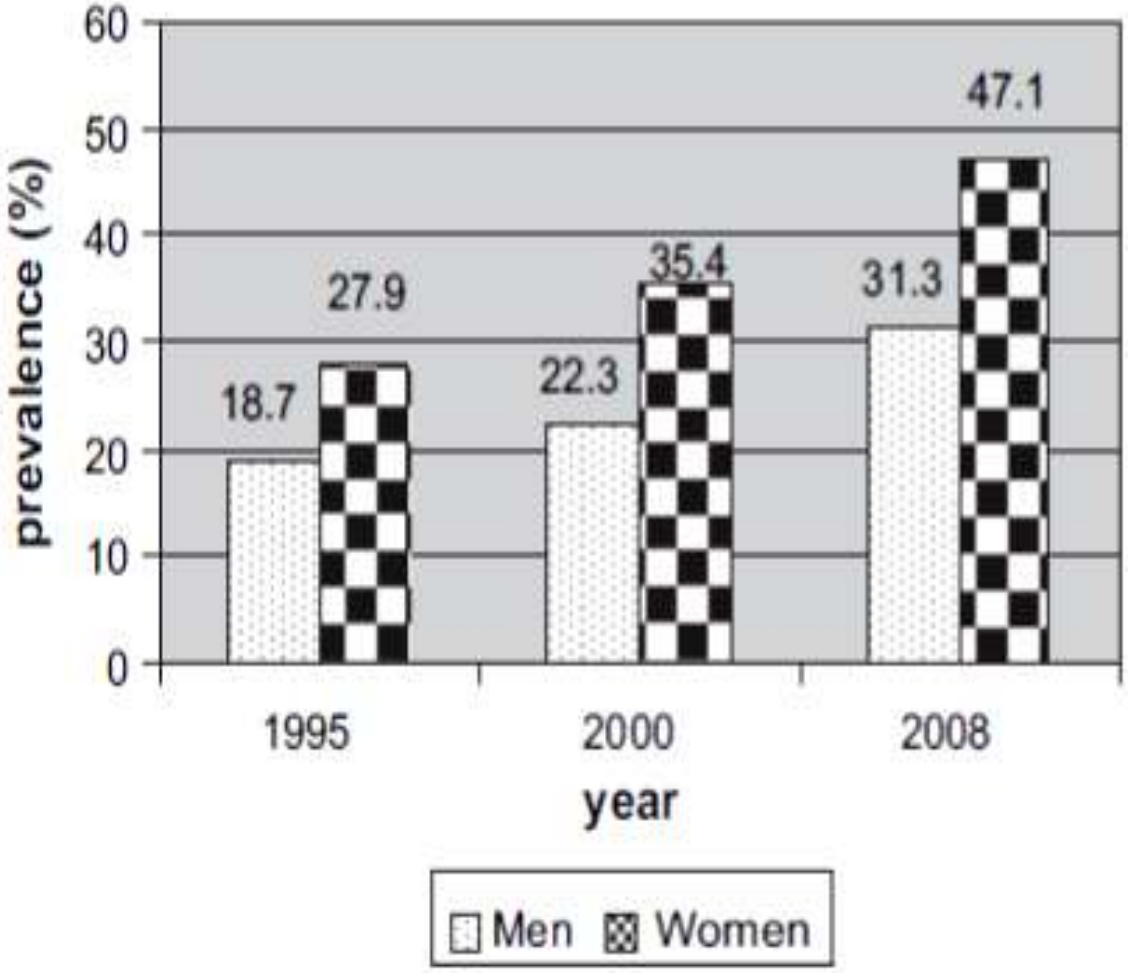


Figure 1: Trends in the prevalence of being overweight among men and women

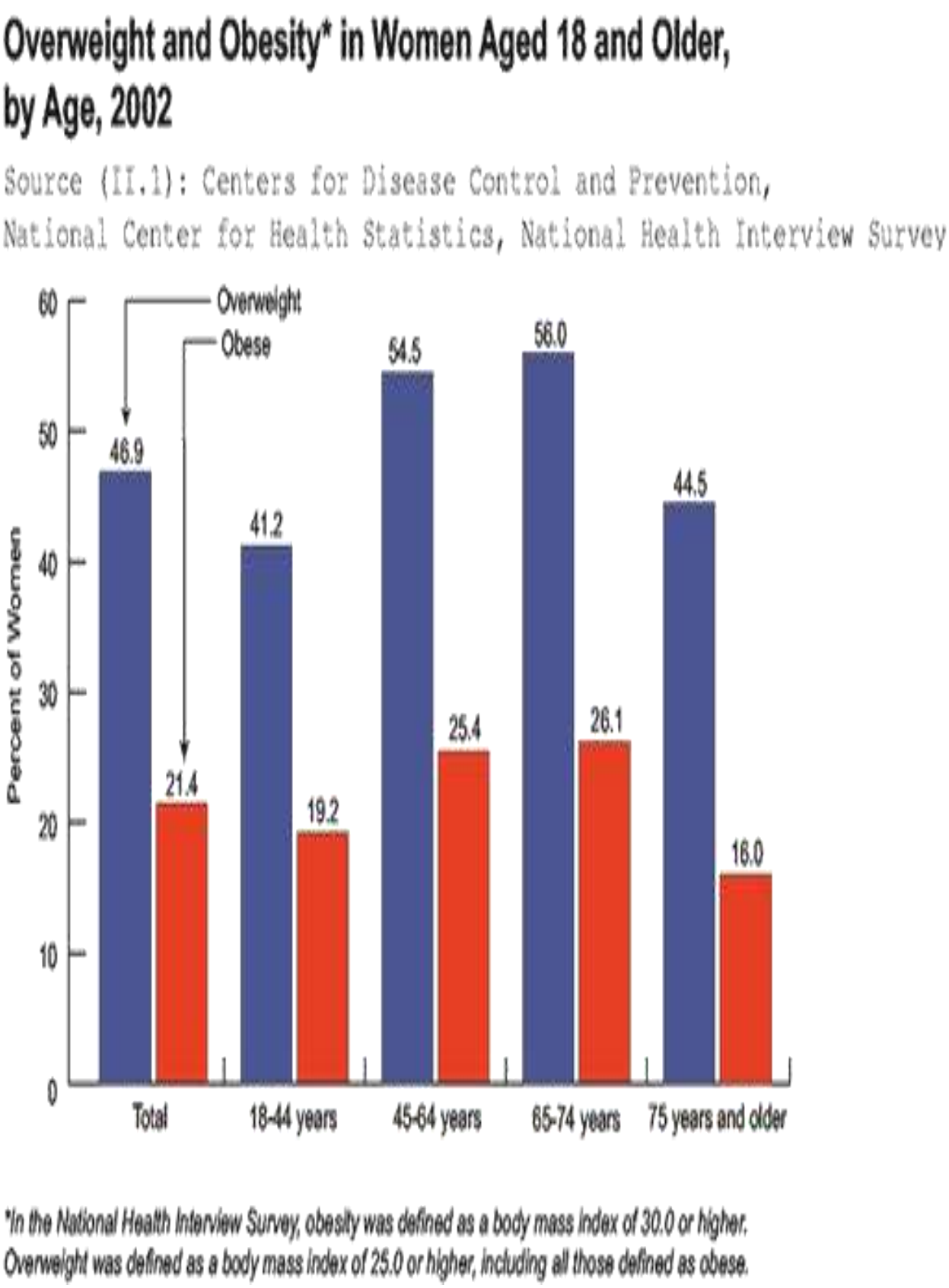


Fig. No. 2- Prevelence Of Overweight & Obesity in women

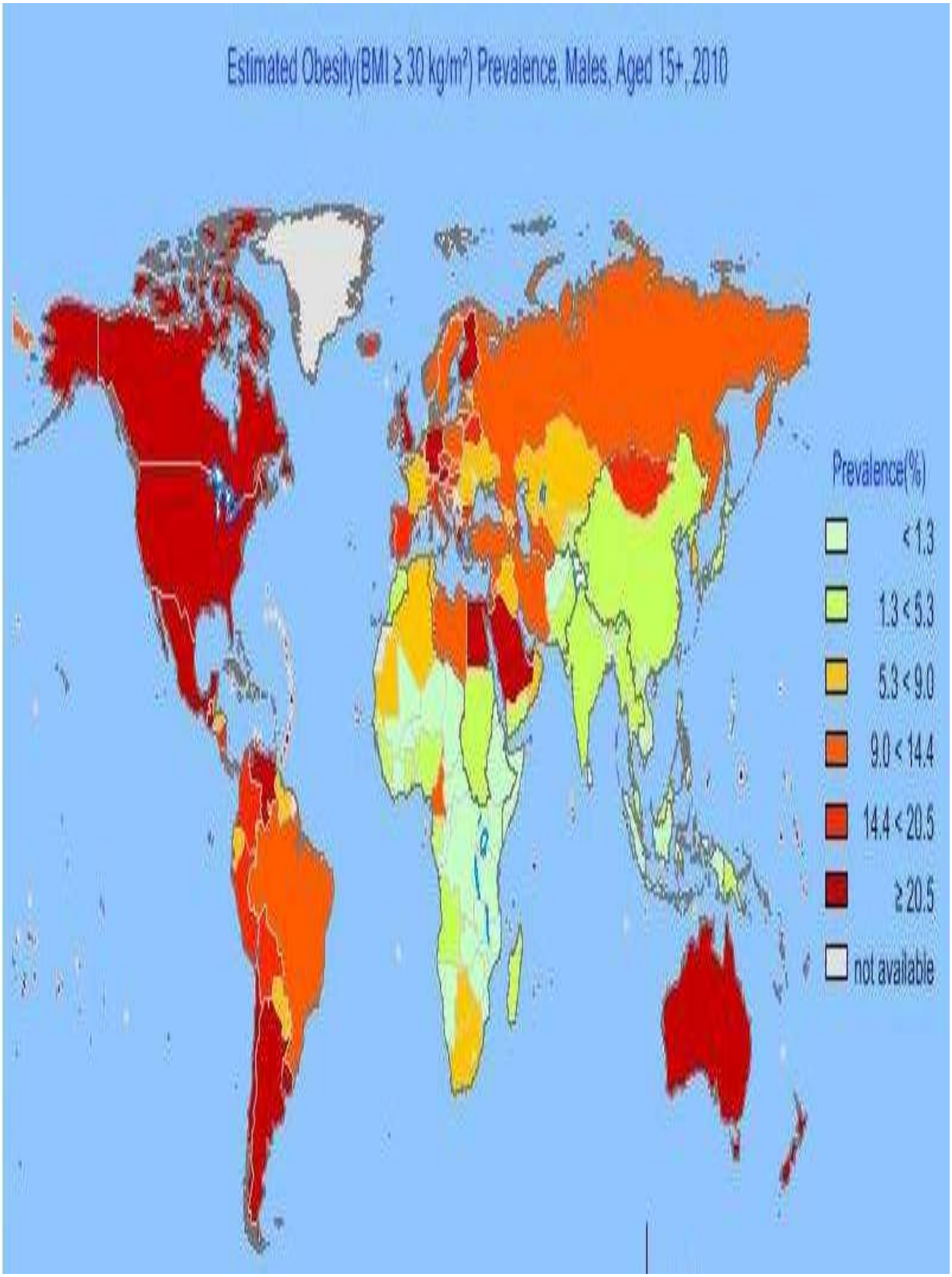


Fig. No. 3- Showing prevalence of obesity worldwide.

AETIOLOGY

The most common cause of obesity is excess calorie intake & Physical inactivity. It is a heterogeneous group of disorders. Patho- physiology of obesity seems simple in excess high calorie nutrients intake relative to the low level of energy expenditure. However due to complexity of the neuroendocrine & metabolic systems that regulate energy intake, storage & expenditure, it has been difficult to quantitate all the relevant parameters (food intake & energy expenditure) over time in human subjects.

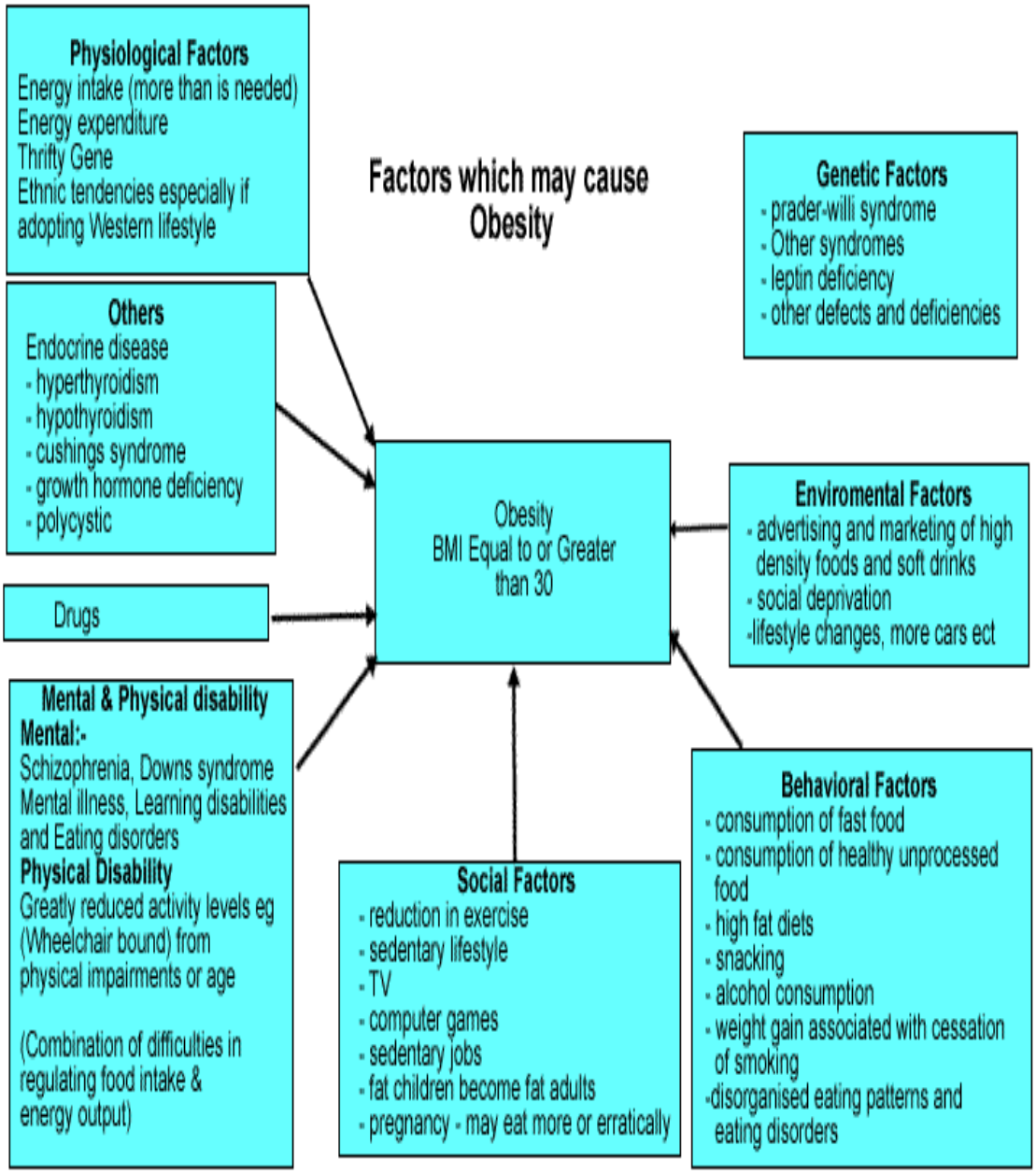


Fig. No. 4- Showing Factors Causing Obesity

1. Genetic Predisposition:

Obesity is commonly seen in families but inheritance is usually not Mendelian, however it is difficult to distinguish the role of genes & environmental factors. Adoptees usually resemble there biologic rather adoptive parents, providing strong support for genetic influences. Twin studies have shown a close correlation between the weight of identical twins even when they are rared in dissimilar environments.

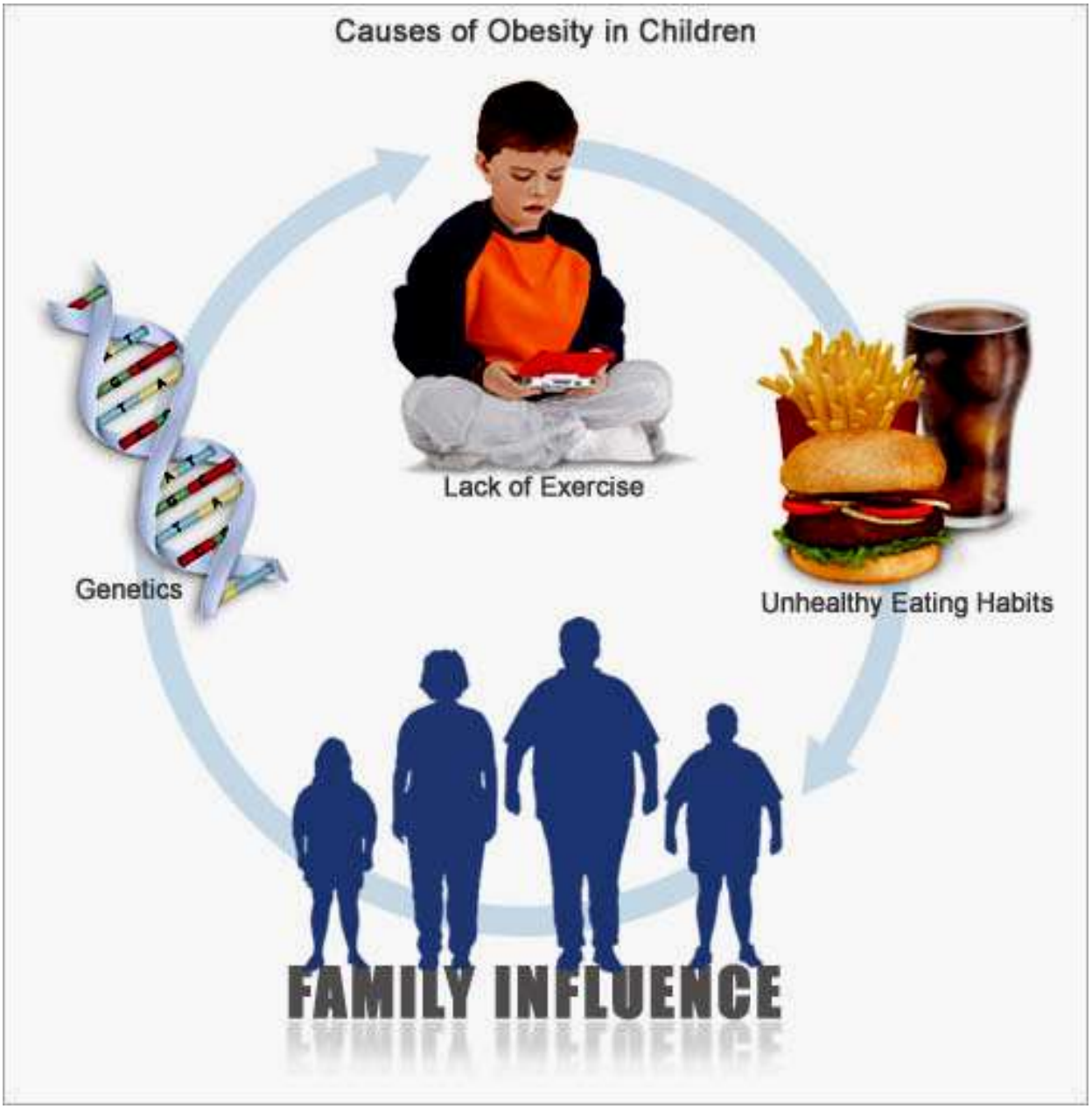


Fig.No. 5- Showing family Influence on obesity

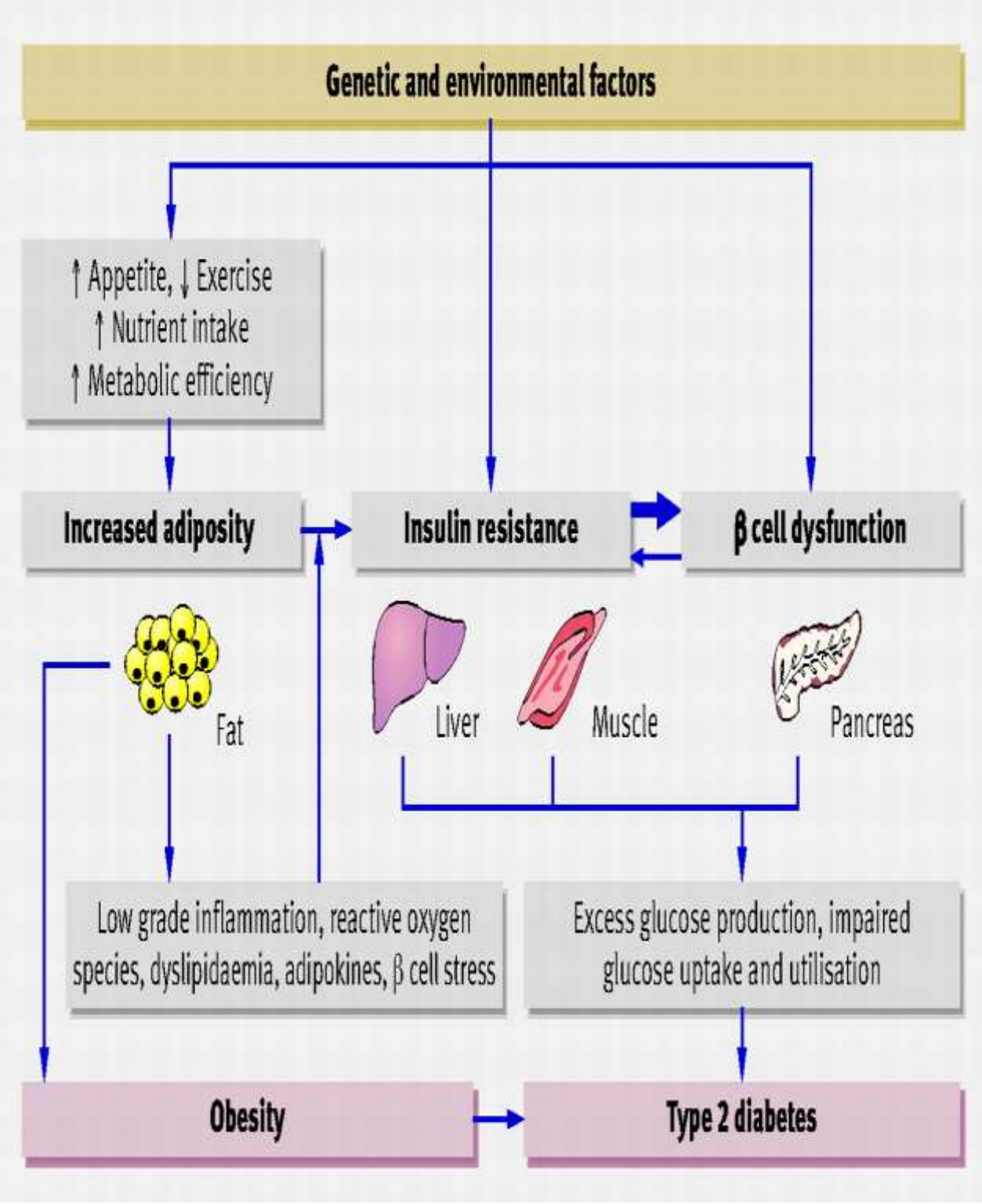


Fig. No. 6- Showing Genetic & Environmental Effects On Obesity

2. Socioeconomic & Environmental Factors :

Environmental & cultural factors strongly influence prevalence of obesity as evidenced by the fact that famine prevents obesity in even the most obesity prone individuals. Recent rapidincrease in the prevalence of obesity in developing countries due to change in gene pool.

Cultural factors are also responsible for obesity because these relate to availability & composition of diet & also to changes in the level of physical activity.

Ethnic groups in many industrialçed countries appear to be specially susceptible to the development of obesity & its complications. This may be due to a genetic predisposition to obesity that only become apparent when such groups are exposed to a more affluent life style.

3. Reduced Energy Expenditure:

Energy expenditure includes the following four components:

1. Basal Metabolic Rate

2. Energy cost of metabolçing & storing food.

3. The thermal effect of exercise

4. Adaptive thermo genesis, which varies in response to chronic calorie intake. BMR accounts for about 70% of daily energy expenditure whereas active physical activity contributes 5-10%.

The excess calories are stored in white adipose tissue & if this net positive calorie balance is prolonged, it result in obesity.

Adaptive thermo genesis depends upon the concentration of the Brown Adipose Tissue (BAT), which is rich in mitochondria & can store large quantities of triglycerides in the form of multiple droplets in the cytoplasm. It can release energy as heat through oxidation of triglycerides. BAT deficiency in rodents causes obesity & diabetes while stimulation of BAT with a specific adrenergic agonist (a 3 agonist) protects against diabetes & obesity. BAT exists in human specially neonates, but its physiological role is not yet established.

One newly described component of thermogenesis called Non Exercise Activity Thermo genesis(NEAT), has been linked to obesity. It is the thermo genesis that accompanies physical activities other than volitional exercise, such as the activities of daily living, fidgeting spontaneous muscles contraction & maintaining posture. NEAT accounts for about 2/3rd of increased daily energy expenditure induced by overfeeding, the wide variation in fat storage seen in overfed individuals is predicted by the degree to which NEAT is induced, the molecular basis for NEAT & its regulation are unknown.

4. Eating Habits:

Eating habits (e.g. eating in between the meals, excess intake of sweets, saturated fatty foods) are established very early in the life. The composition of diet, the periodicity of food taking & the amount of energy taken all are directly related with obesity.

A diet containing more energy than needed may lead to prolonged postprandial hyperlipidaemia and to deposition of triglyceride in the adipose tissue resulting in obesity.Consumption of sweeten drinks, fast food believed to be contributing to rising treands of obesity.

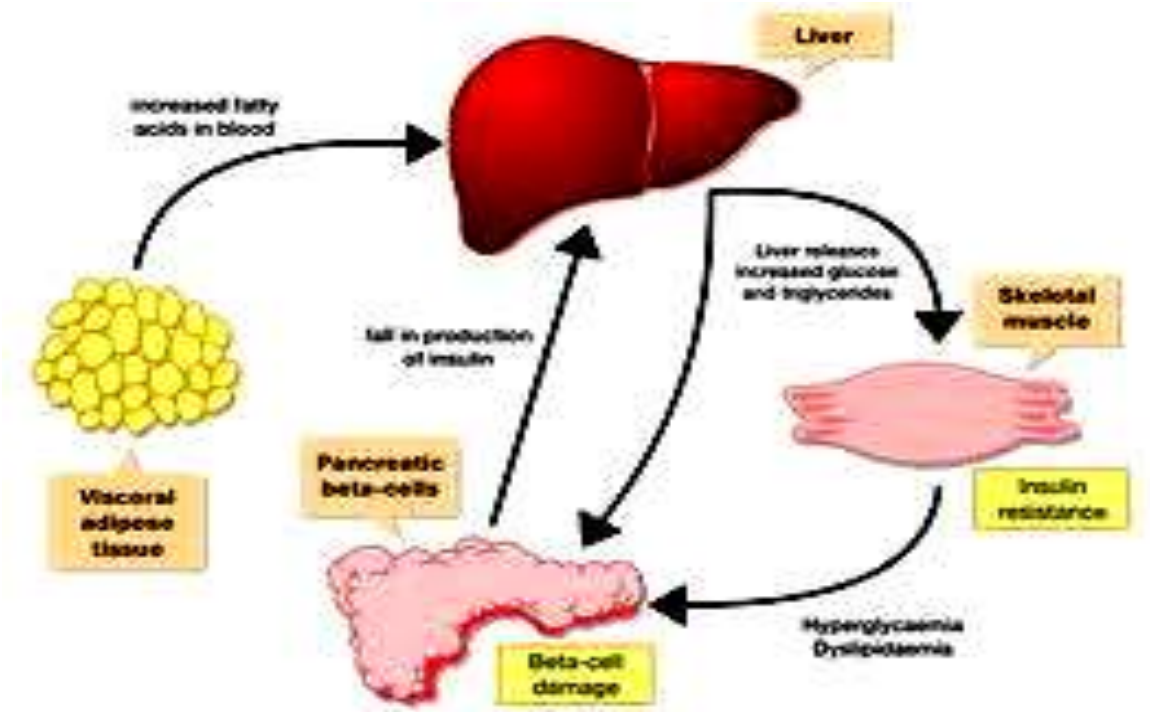


Fig. No. 7 A & B – Showing Faulty Food habit & Its Metabolic Effects

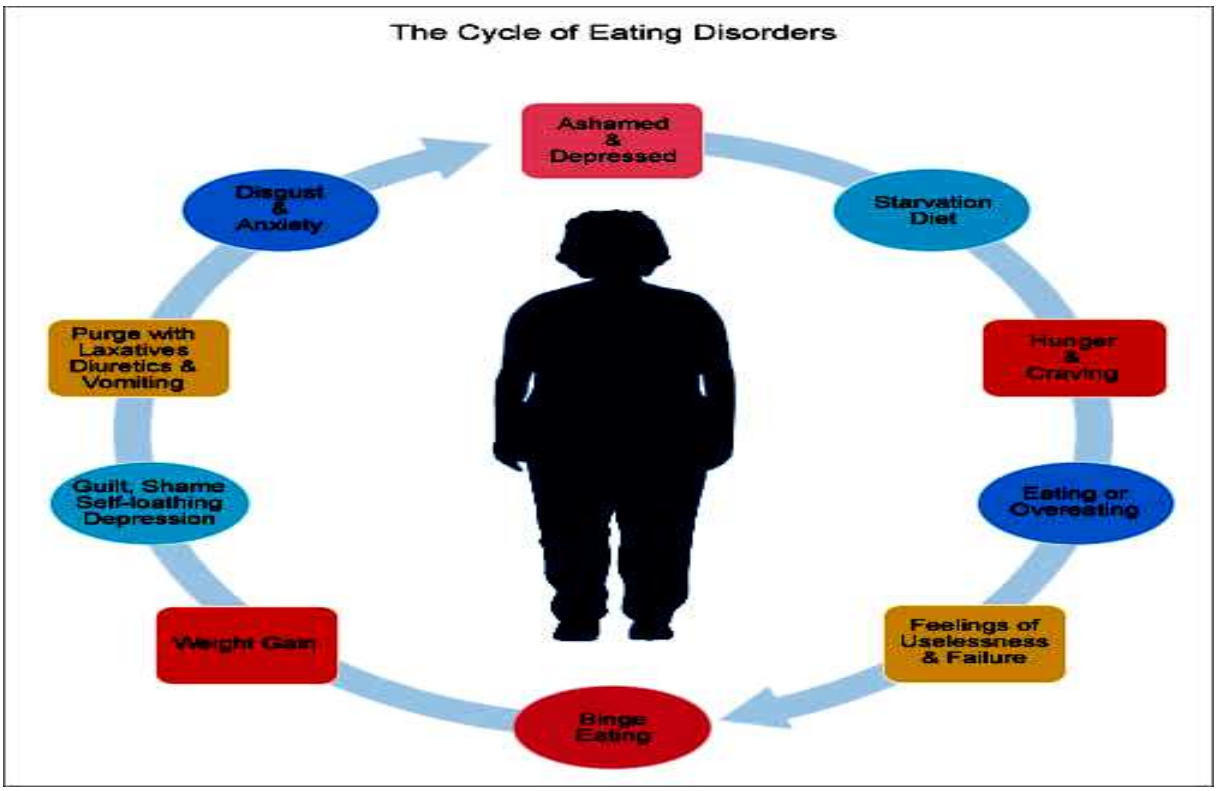


Fig. No. 8- Showing Cycle Of Faulty Eating habits

5. Sedentary Life style:

A sedentary life style plays a significant role in obesity. World widethere has been a large shift towards less demanding work currently at least 60% of the world's population gets insufficient exercise. This is primarily due to increasing use of mechanical transportation and a greater prevalence of labor saving technology in the home. In children, thereappear to be declines in the level of physical activity due to less walking & physical education. A 2008 meta analysisfound 63 out of 73 studies (86%) showed an increased rate of childhood obesity with increased media exposure, with rates increasing proportionally to time spent watching television.

Fig.No. 9 A & B- Showing Sedentary Life Style

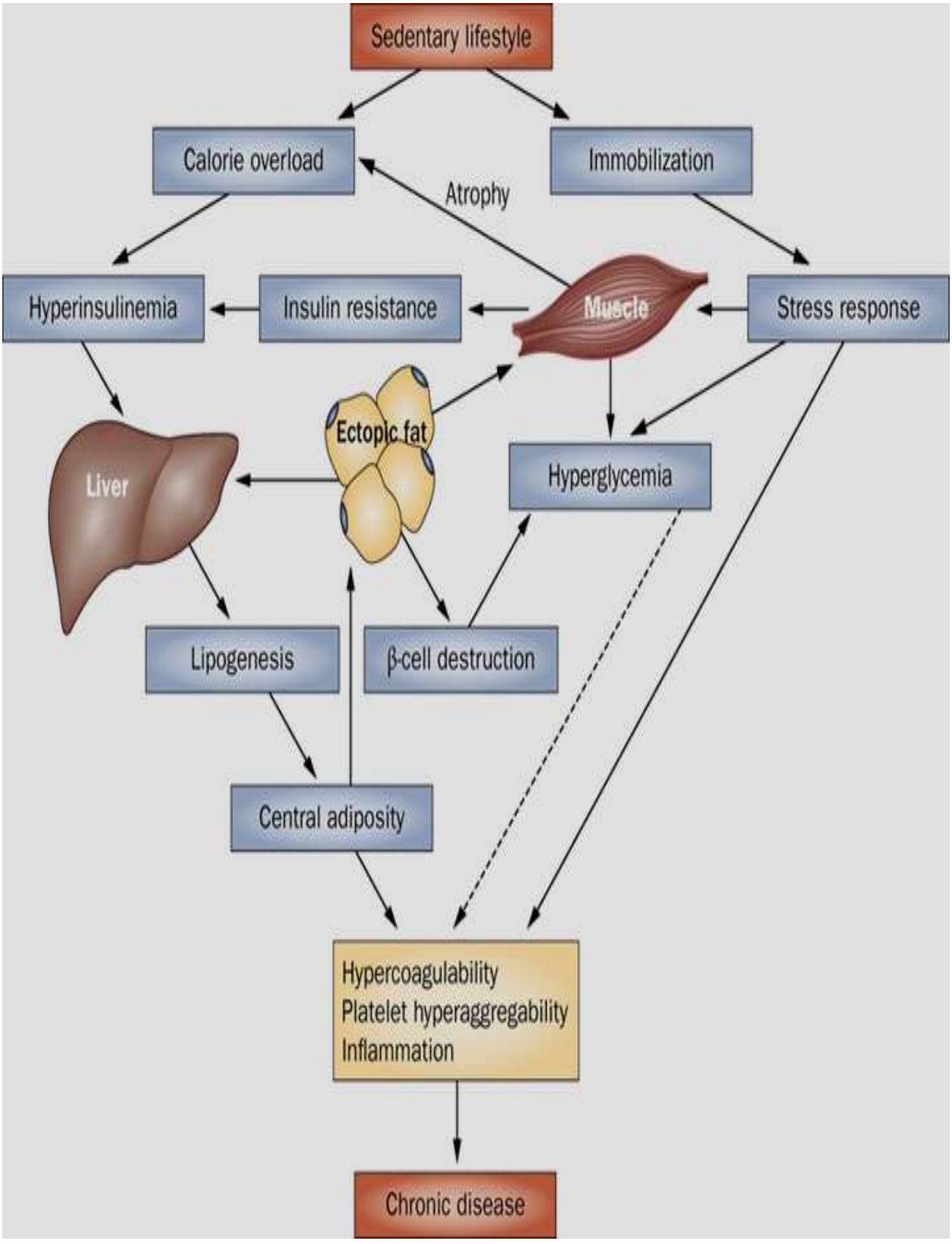


Fig. No. 10- Showing effects of sedentary life style on differents parts Causing obesity

6. Infectious Agents:

The role of infectious agents on the metabolism is still not much clear. Gut flora has been shown to differ between lean & obese persons. In some studies it is indication that Gut flora can affect the metabolism. This apperant alteration of the metabolic potential is believe to confer a greater capacity to harvest energy contributing to obesity. Wheather these differences are the direct cause or the result of obesity has yet to be determined unequivocally.

An association between viruses & obesity has been found in humans & several different animal species. The amount that these associations may have contributed to the rising rate of obesity is yet to be determined.

7. Other causes:

A. Disorders of Hypothalamus:

Weight regulation centre is situated in the hypothalamus of brain. Increase level of serotonin in the hypothalamus increase satiety while neither increased levels of norepinephrine decrease hunger. Hypothalamic dysfunction of systems controlling satiety (Ventro-medial nucleus), hunger (ventro-lateral) & energy expenditure through obesity of varying.

Fig. No. 11-Showing Obesity due to Cushing Syndrome

B. Hypothyroidism:

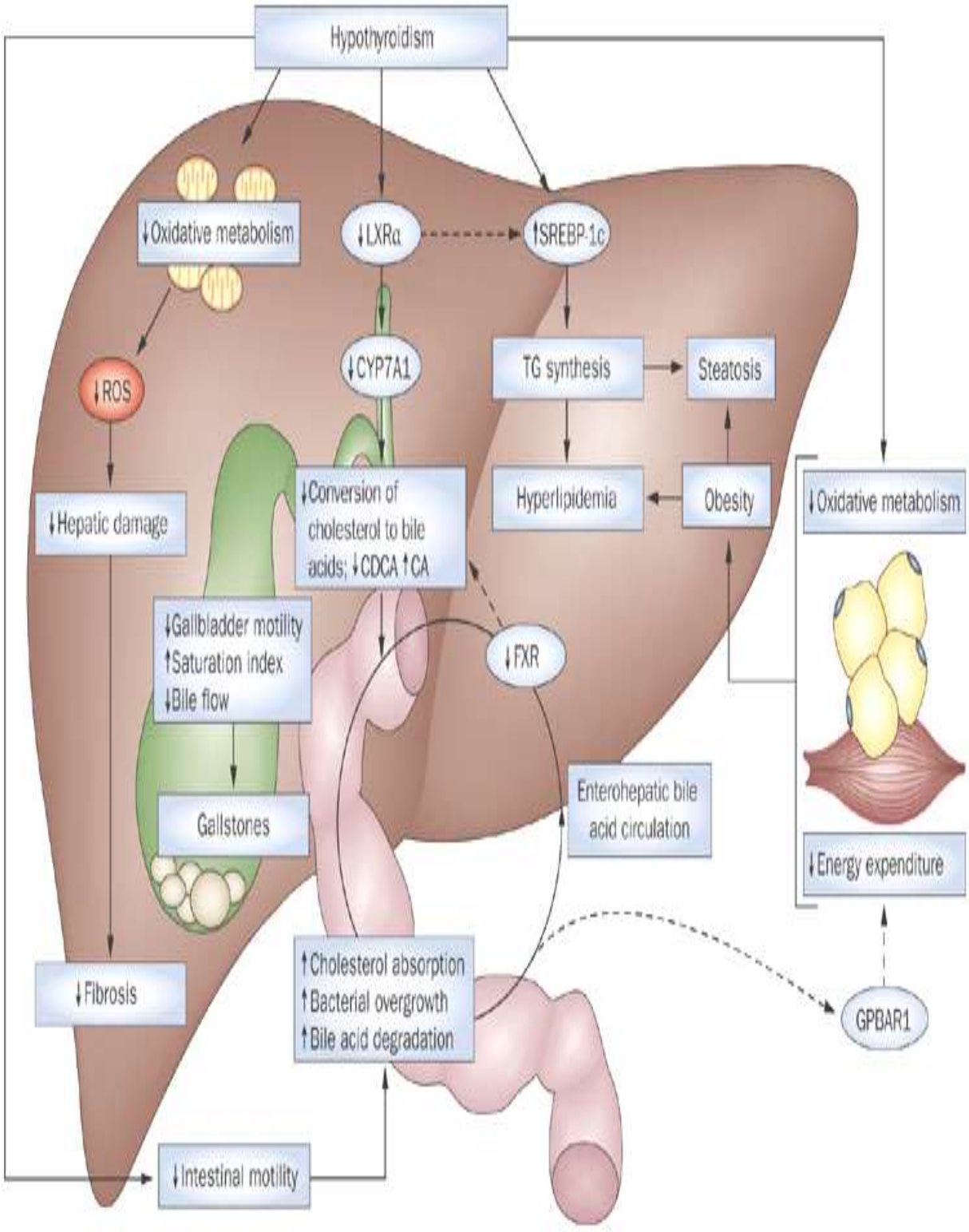


Fig. No. 12- Showing adverse effects of Hypothyroidism

Much of the weight gain that occurs in hypothyroidism is due to myxodema but it can cause obesity due to decreased calorie needs & hyperphagia to combat hypoglycemia symptoms.

C. Insulinoma: Patients with insulinoma often gain weight as a result of overeating to avoid hypoglycemia symptoms. The increase the substrate plus high insulin level promotes energy storage in fat.

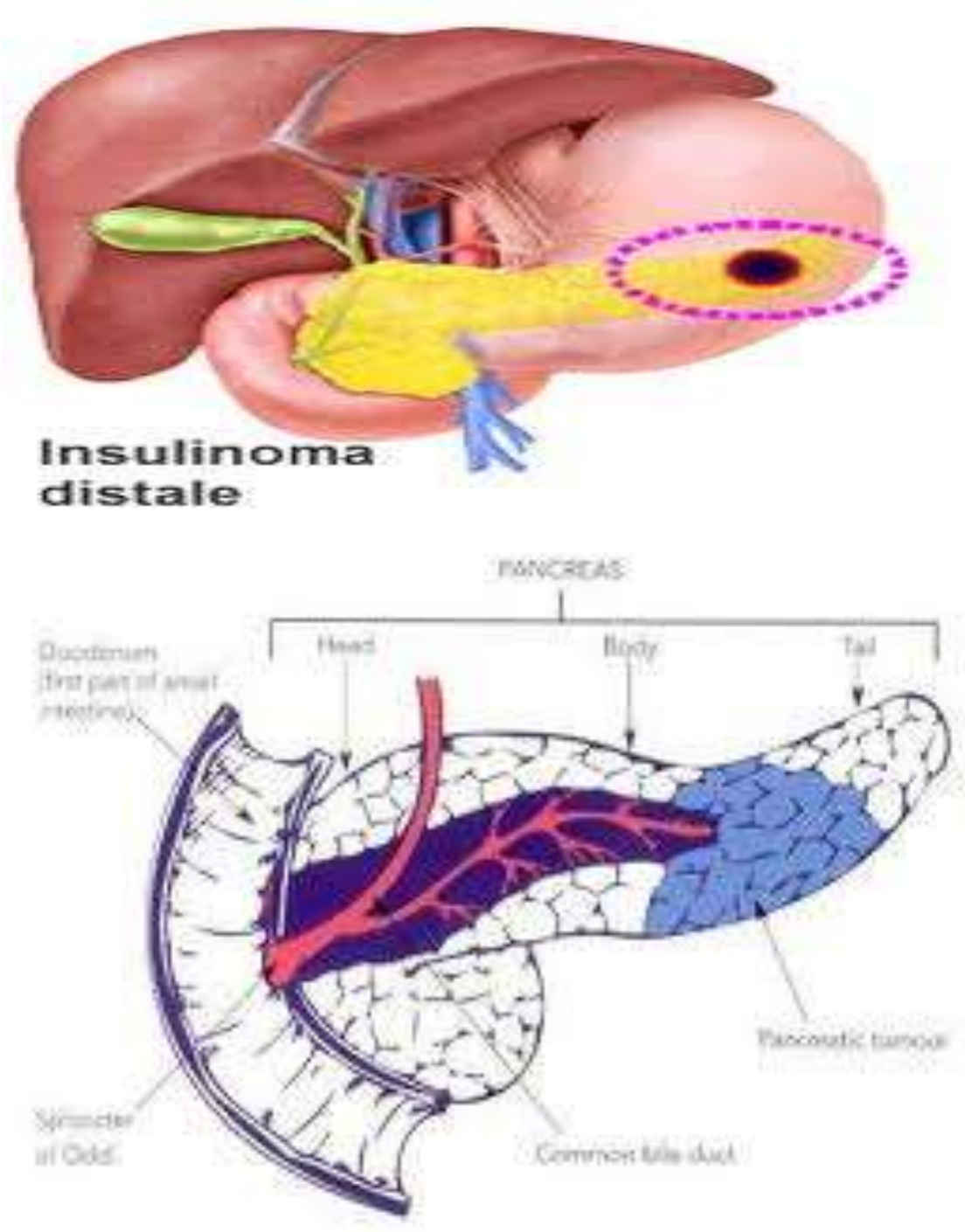


Fig. No. 13- Representation of Insulinoma Causing Obesity

D. Drug Induced Obesity :

Drugs such as Cyproheptadine, Phenothiazines, Tricyclic antidepressants, Lithium, Insulin, Sulphonylureas, Thiazolidiones, Phenytoin , valproate & Steroids can lead to weight gain.

Estrogen replacement therapy& Estrogen containing contraceptive pills have been found to cause weight gain.

E. Alcohol:

A recent review of studies concluded that the relationship between alcohol consumption & weight gain was generally positive for male & negative for female.

PATHOPHYSIOLOGY OF OBESITY:

Obesity can result from increased energy intake, decreased energy expenditure or both things. There is increased interest in the concept of a body weight 'set point'. This idea is supported by physiologic mechanisms centered on a sensing in adipose tissue that reflects fat stores, and a receptor or 'adipostat' that is in the hypothalamic centre. When fat stores are depleted, the adipostat signal is low & the hypothalamus responds by a stimulating hunger & decreasing energy expenditure to conserve energy. Conversely when fat stores are abundant the signal is increased, and the hypothalamus responds by decreasing hunger & increasing energy expenditure. The recent discovery of the obesity gene, and its product leptin, provides a molecular basis for the physiological concepts.

Since discovery of leptin in 1994, many other hormonal mechanisms involved in the development & maintenance of obesity have been elucidated. That participate in the regulation of appetite & food intake, storage patterns of adipose tissue & development of insulin resistence. Since leptin's discovery, ghrelin, insulin, orexin, peptide yy (pyy)-3-36, cholecystokinin, adiponectin, as well as many other mediators have been studied. The adipokines are mediators produced by adipose tissue, their action is thought to modify many obesity related diseases.

Leptin and ghrelin are considered to be complimentary in their influence on appetite. Leptin is produced by adipose tissue to signal fat storage reserves in the body and mediates long term appetitive controls (i.e. to eat more when fat storages are low and less when fat storages are high). Ghrelin is produced by the stomach & modulates short – term appetitive control (i.e. to eat when stomach is empty and to stop when stomach is stretched). Although administration of leptin may be effective in a small subset of obese individuals who are leptin deficient, most obese are thought to be leptin resistant and have been found to have high levels of leptin. The resistence explains in past why administration of leptin has not been effective in suppressing appetite in most obese people.

Although leptin and ghrelin are produced peripherally, they control appetite through their actions on the central nervous system. In particular, they and other appetite related hormones act on the hypothalamus which is a region of the brain that regulates food intake and energy expenditure. There are several circuits within the hypothalamus that contributes to its role in integrating appetite, the melanocortin pathway being the best understood. The circuit begins with an area of the hypothalamus, the arcuate nucleus, that has outputs to the lateral hypothalamus (LH) and ventromedial hypothalamus (VMH), the brains feeding and satiety centers respectively.

There are two distinct groups of neurons in the arcuate nucleus. The first group co express neuropeptide y (NPY) and agouti- related peptide (AgRP) and has stimulatory inputs to the lateral hypothalamus (LH) and inhibitory inputs to the VMH. The second group of neurons co expresses pro-opiomelanocortin(POMC) andcocain- and amphetamine-regulated transcript (CART) and has stimulatory inputs to the VMH and inhibitory inputs to the LH. As a result NPY/AgRP neurons stimulate feeding and inhibitory satiety, while POMC/CART neurons stimulate satiety and inhibit feeding. Leptin in part regulates both groups of arcuate nucleus neurons. Leptin inhibits the NPY/AgRP group while stimulating the POMC/CART group. Therefore, a deficiency in leptin signaling, either via leptin deficiency or leptinresistence, leads to overfeeding, and may account for some genetic and acquired forms of obesity.

Certain enzymatic activities are also responsible for development of obesity.

1. ATPase Activity:

This enzyme is present in all the cells in the body and helps to burn 15-40% of all calories at reast. The obese individuals generally have 20-25% less ATPase than person of normal weight on same diet.

2. LPL Activity:

Increased lipoprotein Plasma lipase (LPL) activity proceeds the development of hyperphagia and there after obesity; because the activity of LPL in adipose tissue can potentiate hunger by alerting the availability of circulating metabolites.

CLASSIFICATION OF OBESITY:

Obesity can be classified into various types on the basis of Etiology, Histopathology, Fat distribution, BMI, Onset, Waist- hip ratio & psychological factors as per description given below:

1. **On the basis of cause**

   A. Exogenous- due to overeating

   B. Endogenous-due to endocrine disorder

2. **On the basis of Histopathology**

   A. Hypertrophic Obesity

   B. Hyperplastic Obesity

3. **On the basis of distribution of fat**

   A. Generalised

   B. Truncal

   C. Superior

   D. Inferior

4. **On the basis of severity according to BMI**

   A. Mild

B. Moderate

C. Severe

5. On the basis of onset

A. Incideuous onset

B. Rapid onset

6. On the basis of Waist to hip ratio

A. Android obesity

B. Gynoid obesity

7. Psychological classification

A. Constitutional obesity

B. Developmental obesity

C. Reactive obesity

Exogenous Obesity

This type of obesity results from excess calorie intake coupled with physical inactivity.

Endogenous Obesity

In this type of obesity there is defect in endocrine functions at the level of hypothalamus & pituitary or adrenal cortex or due to defective metabolism. It may or may not be associated with excess calorie consumption.

Hyperplastic Obesity

There is increase in both adipose cell number & size and increase calorie consumption can be traced to early childhood when the adipose tissue proliferates resulting in too much adiposity with fat in reasonably moderate quantity. In this type of Obesity, adipose cell shrink after weight reduction but hypercellularity remain constant.

Fig. No. 14- Showing Hyperplastic Obesity

Hypertrophic Obesity

This type of obesity occurs after the age of 20 years & is characterised by hypertrophy with minimal increase in cell number in response to over consumption of food &decreased physical activity after maturity. Fat deposition occurs mainly over trunk.

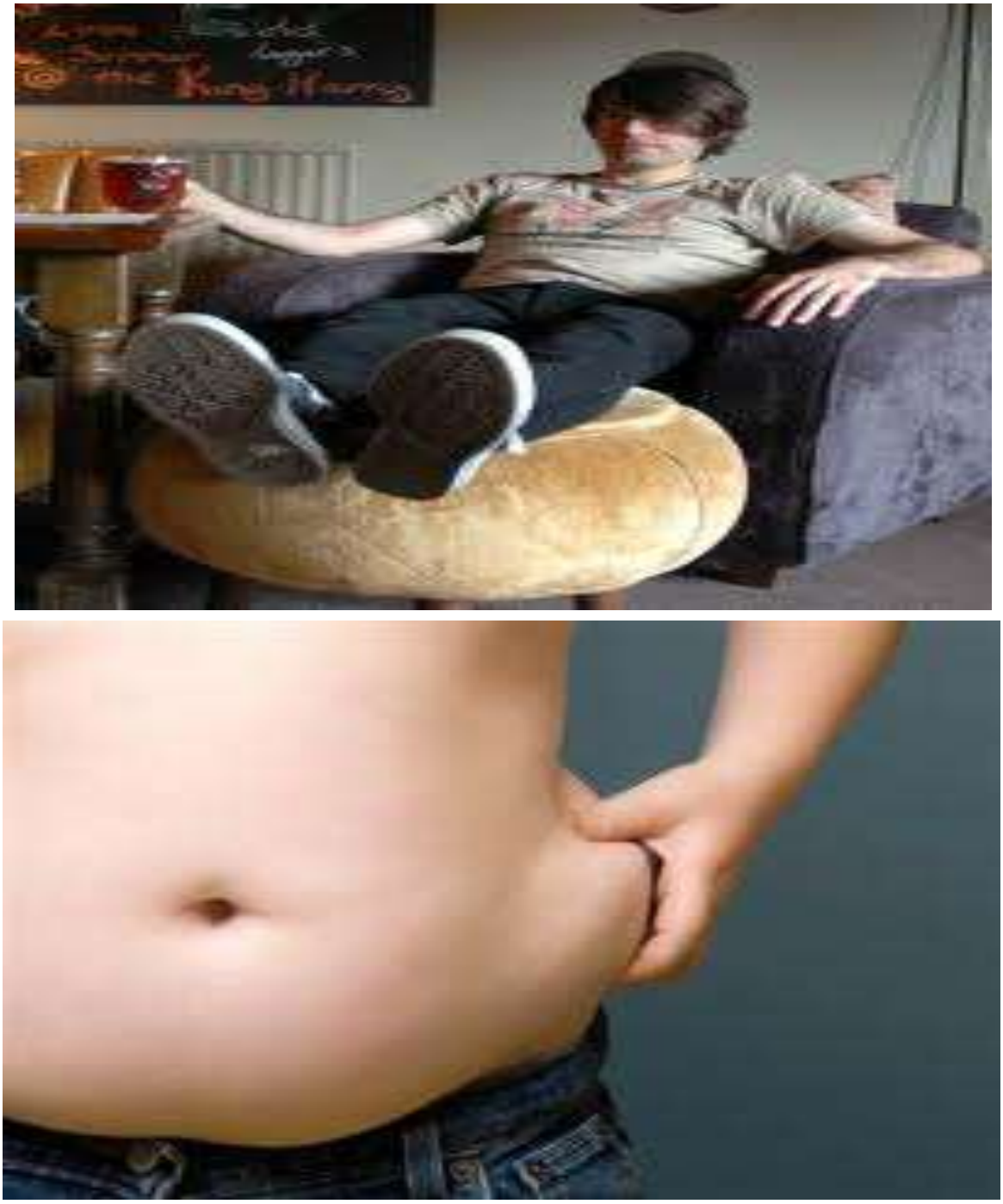


Fig.No. 15- representing Hypertrophic Obesity

Android Obesity (Apple shaped obesity)

In this type, the obesity is maximum on the upper half of the body, secondarily on the abdomen, epiploon & the mesentry. It is also called Central or Abdominal

obesity or male pattern obesity. The face is often congested & flushed & is associated with decreased physical activity & intake of alcohol. Android obesity is associated with an increasd risk of metabolic complications such as Coronary Heart Disease, Diabetes mellitus, Dyslipidaemia & hypertension.

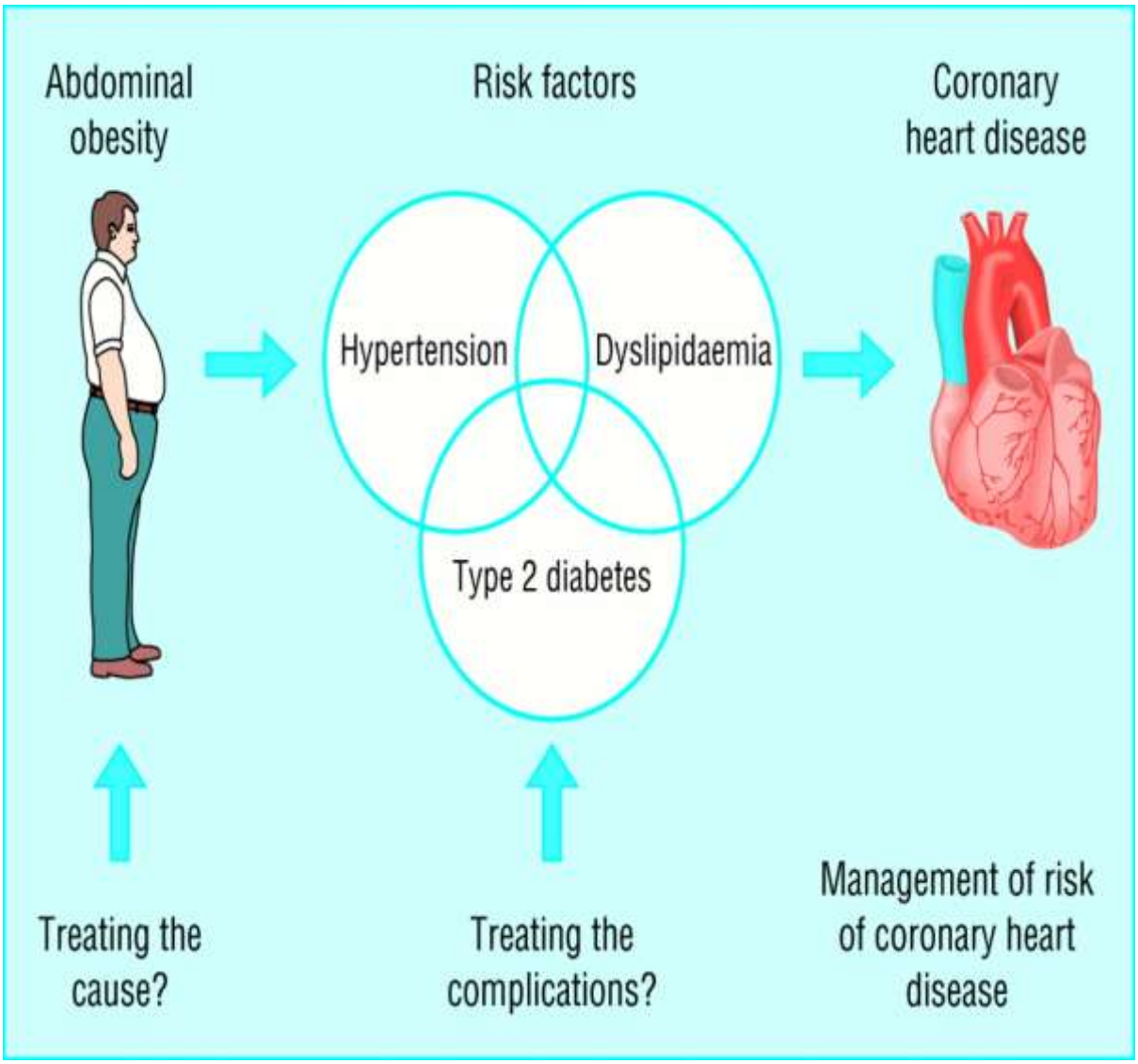


Fig. No. 16- Tripoid Complications of Android Obesity (abdominal)

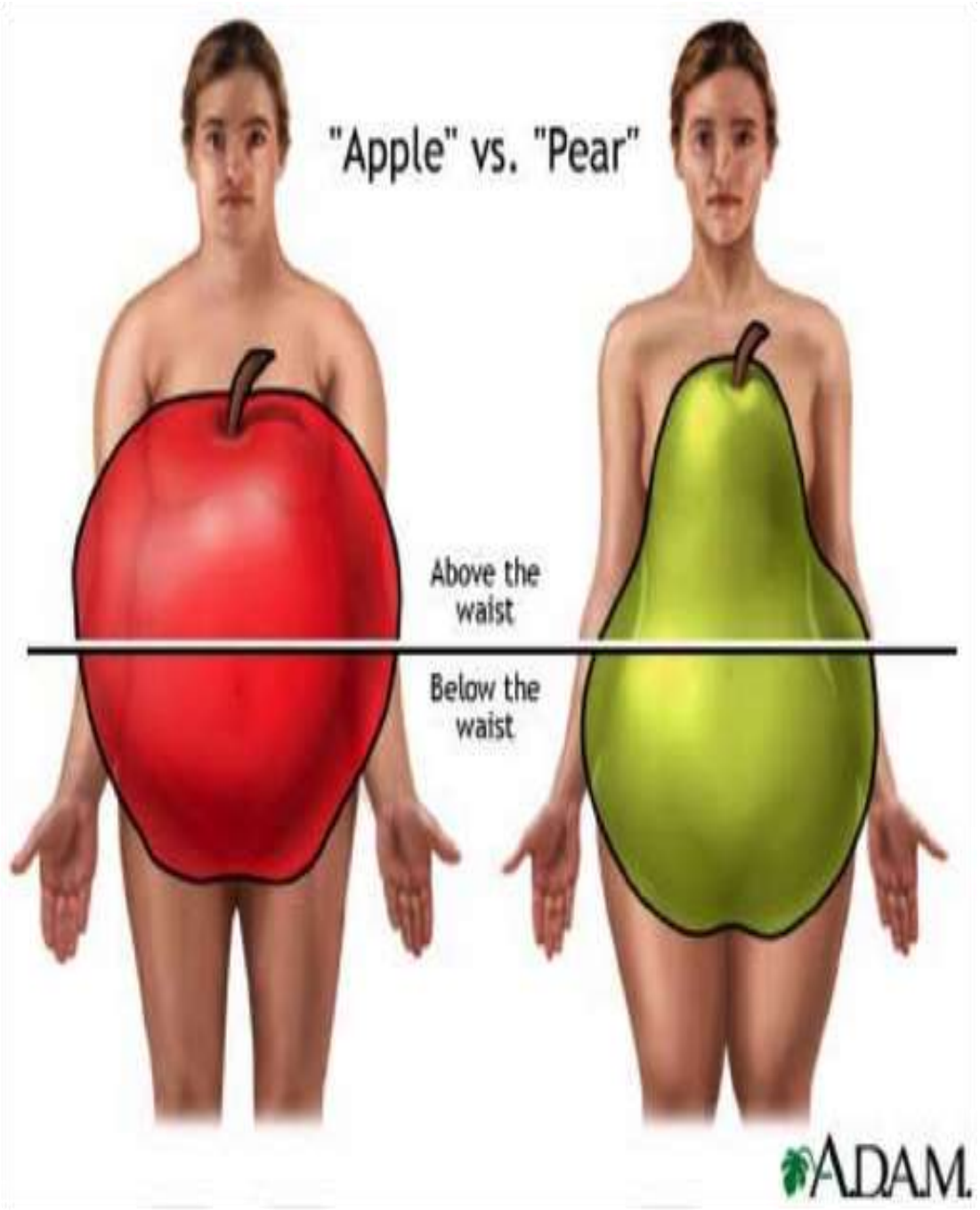


Fig. No.17- Representation Of Apple vs Pear Obesity

Gynoid Obesity

In this type fat accumulates on the Hips & Buttocks, below the waist or glutifemoral region (Pear shaped) in response to excess calorie intake. The sufferer is more prone to development of mechanical disorders such as varicose vein & osteoarthritis.

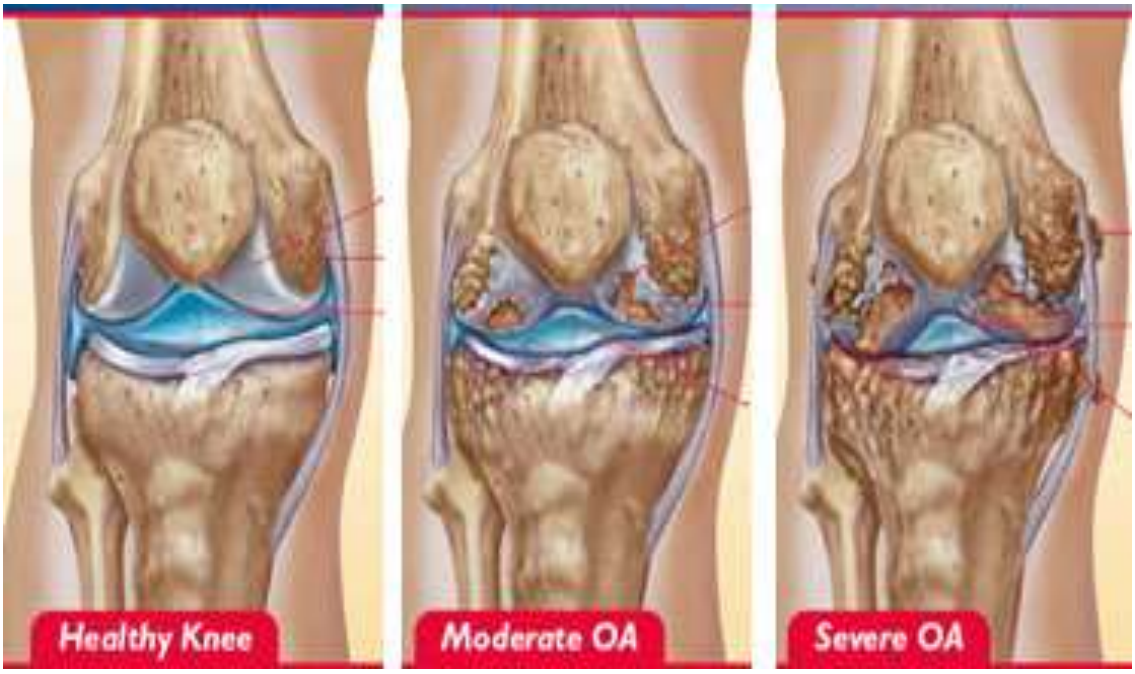


Fig. No. 18-Osteoarthritic Changes A complication Of Gynoid Obesity

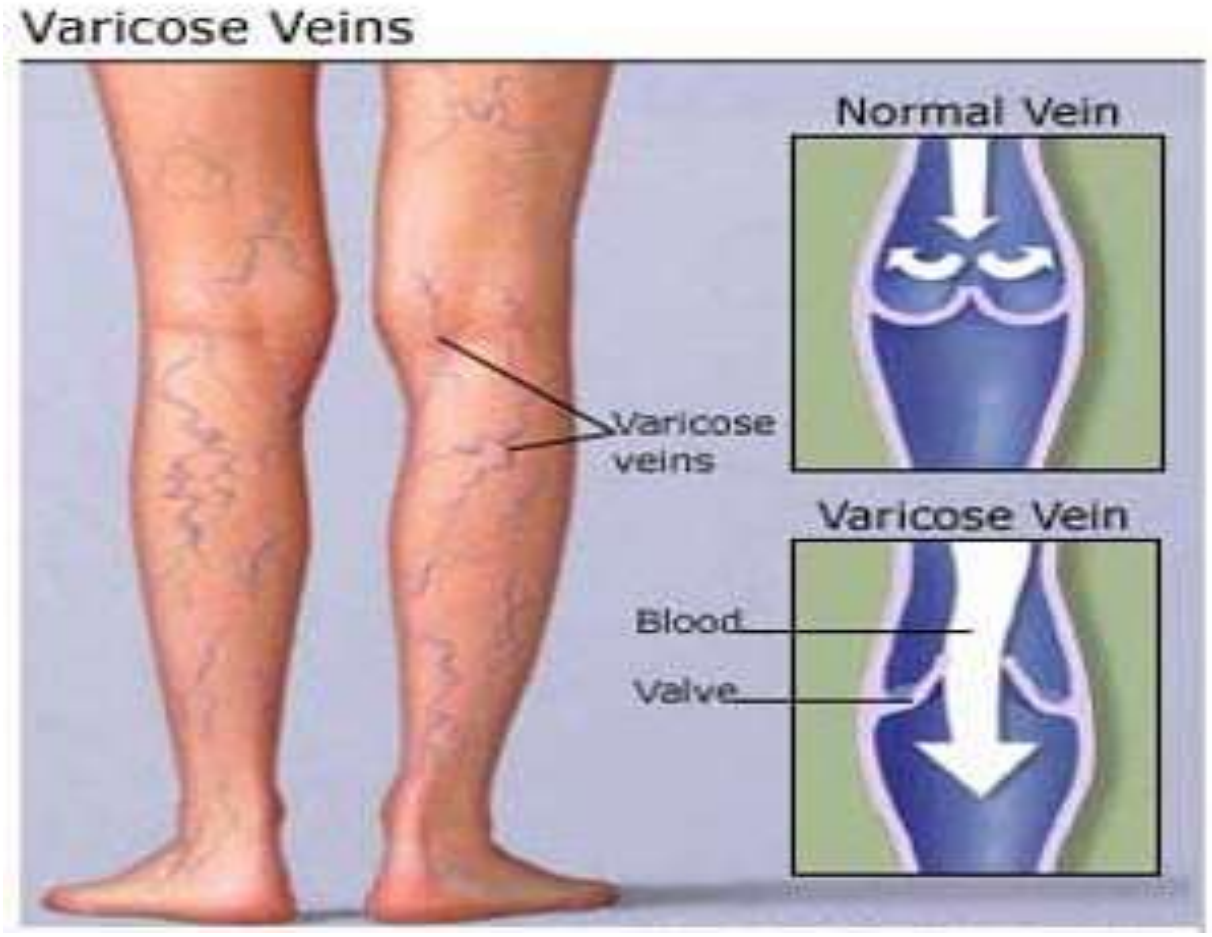


Fig.No. 19-Representation Of Varicose Veins Complication Of Gynoid Obesity

Constitutional Obesity

It is thought to due to Genetic Physiological causes & is usually associated with normal personality development. Women are more sufferer than men.

Reactive Obesity

Anoxious person may develop this type of obesity due to overeating with much reduction in physical activity under the effect of tension emotional upset.

Fig. No. 20. A Presentation of Stressfull beaviour

Fig.No.20. B Anoxious Behaviour a responsible for obesity

Developmental Obesity

It is gift of modern era life style begins in childhood with prominent psychological effect. The teenagers generally have a such type of family in which both parents are working & trea them as a object.

Fig. No. 21- Frustrated Child, frustration is one of cause of obesity

ASSESMENT OF OBESITY:

Obesity is a state of exess adipose tissue mass. Hence obesity can be measured on the basis of measurement of total amount of fat in the body. The overall composition of the body can be expressed in tterms of

A. The active mass (muscles, heart, liver etc.)

B. The fatty mass

C. The extracellular fluid (blood, lymph etc.)

D. The connective tissues (skin, bones etc.)

In obesity there is increase in the fatty mass at the expense of other parts of the body. The water content of the body is never increased in case of obesity.

Obesity can easily be identified at first sight but an accurate assesment requires various measurements & reference standards. The most widely used critaria are as follows.

1. Body Weight :

Though body weight is not an accurate measure of excess fat, however it is widely used index. It is conventional to accept +2 SD (standard daviations) from the median weight for height as a cut of point for obesity.

1. **Body Mass Index (BMI) or Quetelet's Index**

Body mas Index is a measure of body fat relative to height. It is calculated by the following formulae

$$BMI = \frac{\text{weight (lb)} * 703}{\text{height}^2\ (\text{in}^2)}$$

OR

$$BMI = \frac{\text{weight (kg)}}{\text{height}^2\ (\text{m}^2)} \quad \text{(metric)}$$

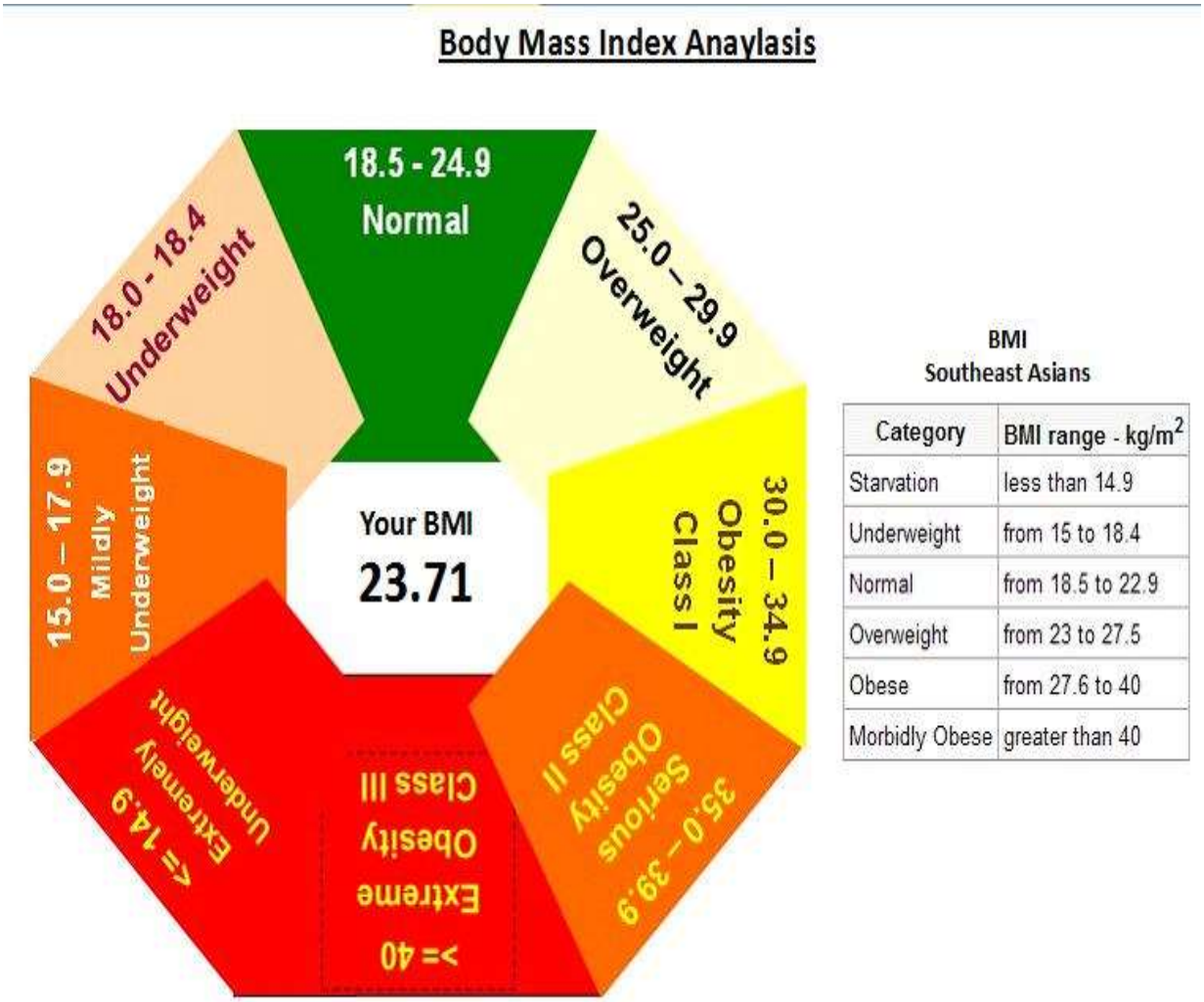
Although relative weight and BMI corelate with the degree of adiposity, excess weight may be in the form of lean tissue or fat tissue. Therefore even heavy muscle atheletes would be considered obese by this method.

In childern, a healthy weight varies with age & sex. Obesity in childern & adolscents is define not as absolute number, but in relation to a historical normal group, such that obesity is a BMI greater than 95%, recently BMJ published new standards for childern based on data from six countries. These standards were developed by a working group of world experts of the International Obesity Task Force (IOTF0), a committee of the international Association for the study of Obesity. Age & sex specific BMI cut of points for defining overweight & obesity have been derived by identifying percentiles in childern analogus to adult BMI 25 & 30 respectively. (S. Gupte, Paediatrics-17, vol-1)

According to World Health Organisation (WHO) guidelines,published in the year 2000 as BMI of 25-29.9 is classified as overweight. Normal BMI for mid point of height & frames among both men & women range from 18.5-24.9. Though women have more body fat at a given BMI, a BMI of 30 or >30 is classified as obese in both sexes. BMI >/= 35 kg/m^2 –severe obesity, BMI 35-40 kg/m^2 –experiences obesity related health problems, BMI 40-44.9 kg/m^2 –morbid obesity, BMI >/= 45 kg/m^2 – super obesity.

In Asian subcontinent, morbidity & mortality occurs at a lower BMI, therefore cut of points for overweight & obesity are lower amongest Indians. The Japanese have defined obesity as any BMI greater than 25 while Chinese use a BMI of greater than 28.

Fig.No.22 .A



BMI
Southeast Asians

Category	BMI range - kg/m^2
Starvation	less than 14.9
Underweight	from 15 to 18.4
Normal	from 18.5 to 22.9
Overweight	from 23 to 27.5
Obese	from 27.6 to 40
Morbidly Obese	greater than 40

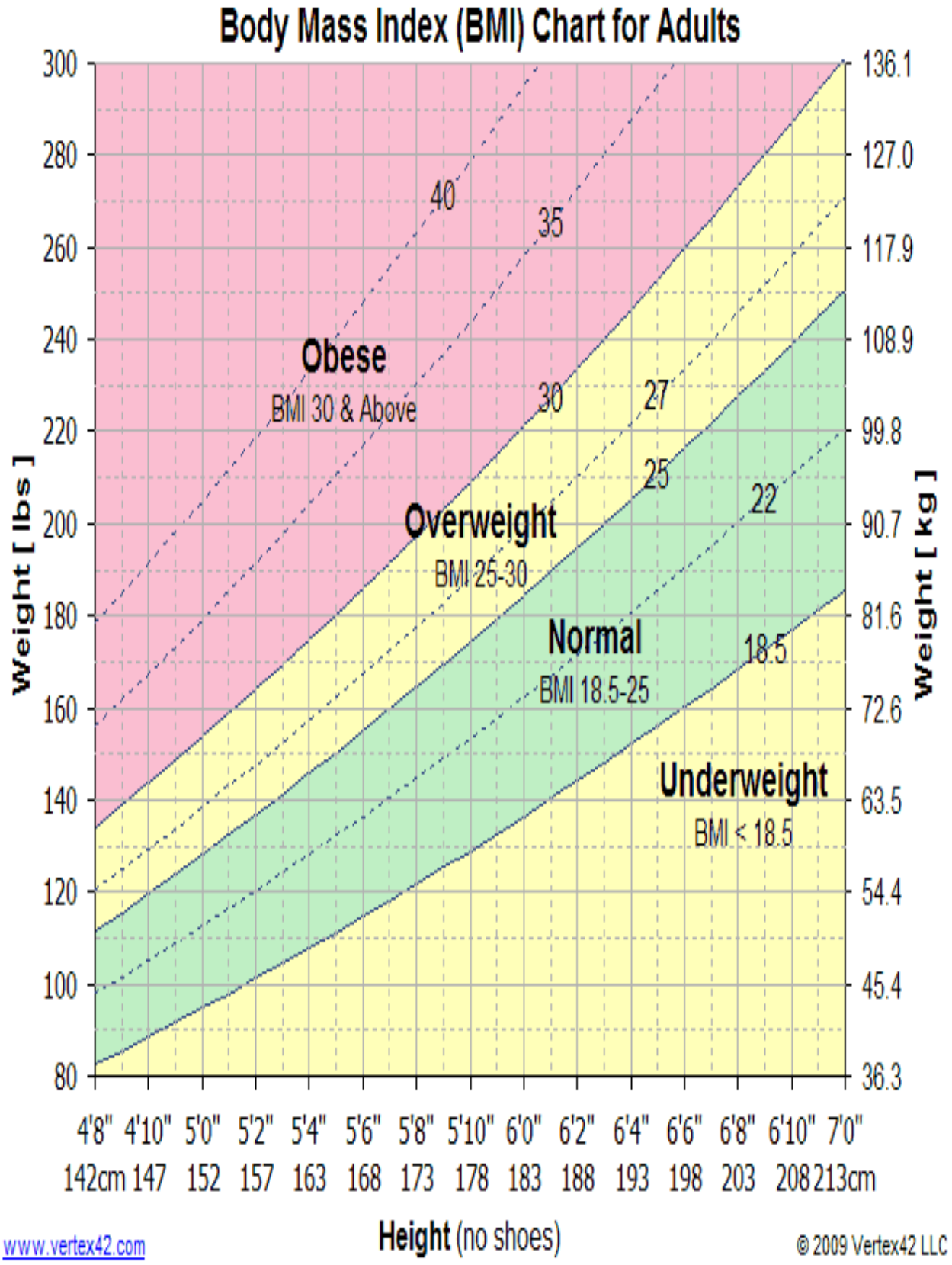


Fig. No. 22. A & B- Representation Of BMI

The drawback of BMI method is that it can classify verry muscular or verry short individuals as obese. Therefore a more important tool to measure obesity is Waist to Hip ratio.

Waist To Hip Ratio:

Waist circumference is the minimum circumference between the costal margin & iliac crest which is measured in the horçontal plain with the person standing. Hip circumference is the maximum circumference in the horçontal plain, measured over the buttocks. The ratio of former to the latter provides an index of the proportion of intra-abdominal fat. Waist to Hip ratio is calculated as -

WHR=Measurement at waist (narrowest point on relaxed stomach)/Measurement at hip (measured at fullest point)

Both in men & women, high waist/ Hip ratio (WHR) is considered a risk factor for Ischaemic heart disease, stroke, hypertension & death.

In men the risk of the disease increases when the WHR rises above 1.0 & in women when it rises above 0.8. In men there is increase risk if waist circumference is 94c.m. & substantial risk if it is 102 cm or more. For women, the figures are 80cm & 88cm or more respectively.

Persons with abdominal obesity (android obesity) are at greater risk for cardiovascular complications than those with gluteal obesity (gynoid obesity)

Skin Folds Thickness

Most of the adipose tissue is normally situated in a subcutaneus layer, the thickness of which can be estimated by measuring skin folds. The method is inexpensive, but requires a skilled observer. This is measured by using skin folds calipers called Herpanden skin calipers & lange calipers. Common sites for measurements are mid triceps, biceps, sub scapular & suprailiac regions. Two readings at the selected site are made by cleanching the tissue by using left thumb & index fingers while taking care that muscle is not lifted. Simultaneously & then average reading recorded. Unfortunately standards for subcutaneus fat donot exist for comparison. Further in extreme obesity, measurements may be impossible because in verry obese people skin folds would not fit between the jaws of measuring caliper. The main drawback of skin fold measurements is their poor repeatability.

Mid Arm/ Mid Thigh Circumferences

These serve as a general index of nutritional status reflecting both calorie adequacy & musscle mass.

Clinical Evaluation

It includes medical history & physical examination. It provides an overall impression of nutritional status & can reveal specific signs of malnutrition & overnutrition when they exist.

Laboratory Assesment

1. Total body water & Lean body mass can be determined from dilutional techniques provide indirect estimation of fat component of the body.

Body fat = Body weight -- lean body mass

Lean body mass = Body water /0.72.

Total body water is determined by using Antipyrene or deuterium oxide as tracer. The techniques involved are relatively complex & cannot be used for routine clinical purposes.

1. Fat nutrition assesment by cholesterol & triglycerides concentration in blood.

2. USG & Radiographic techniques can be used to estimate fat content of the body. Soft tissues X-ray can be usefull in assessing the thickness of subcutaneus fat. Since fat is less dense than musscles & other components of lean body mass, fat is more opaque on X-ray.

COMPLICATIONS OF OBESITY:

Fig. No. 23- Representation Of Consequences & Complications Of Obesity

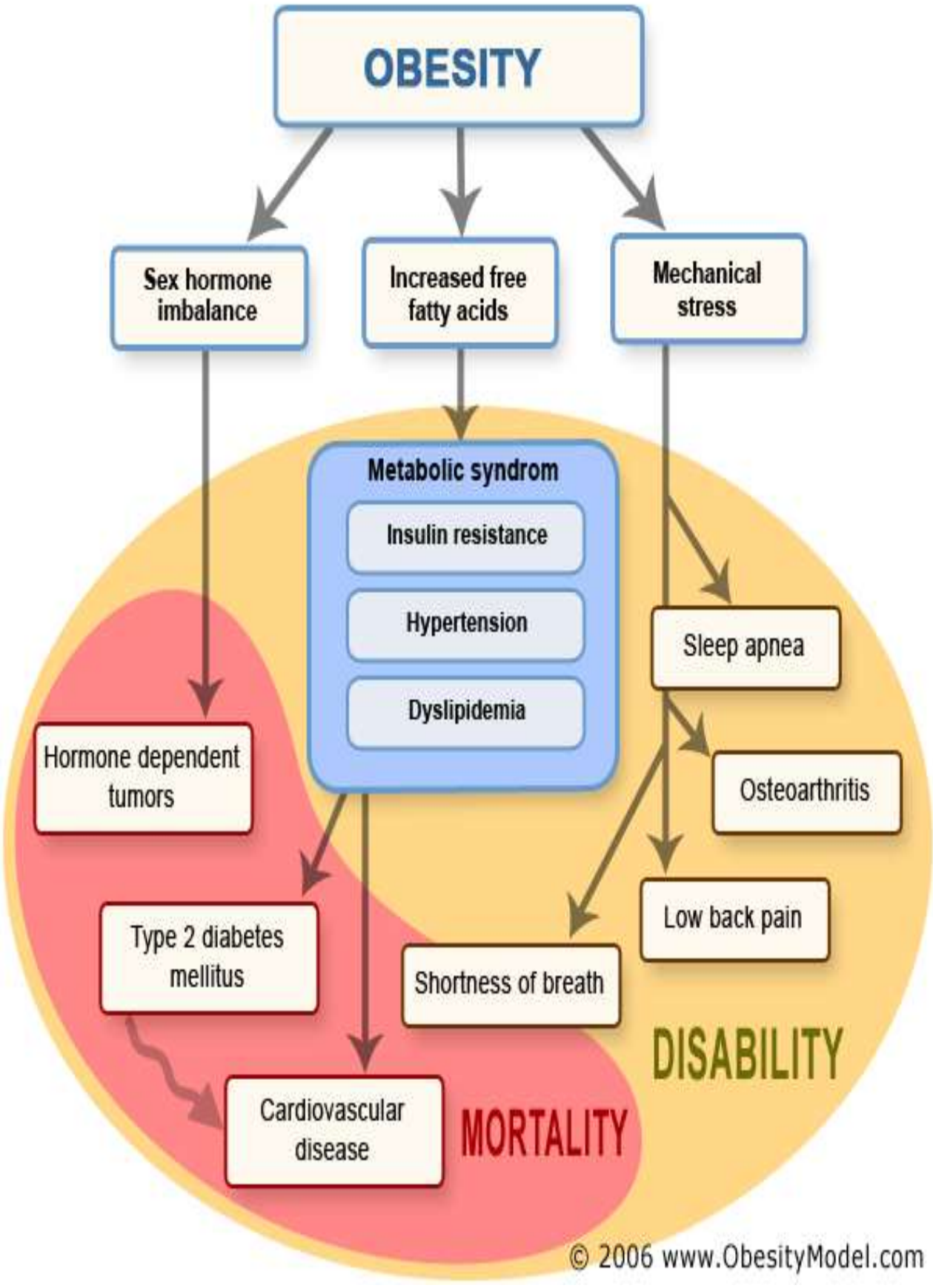


Fig. No. 24- Showing Complications Of Obesity

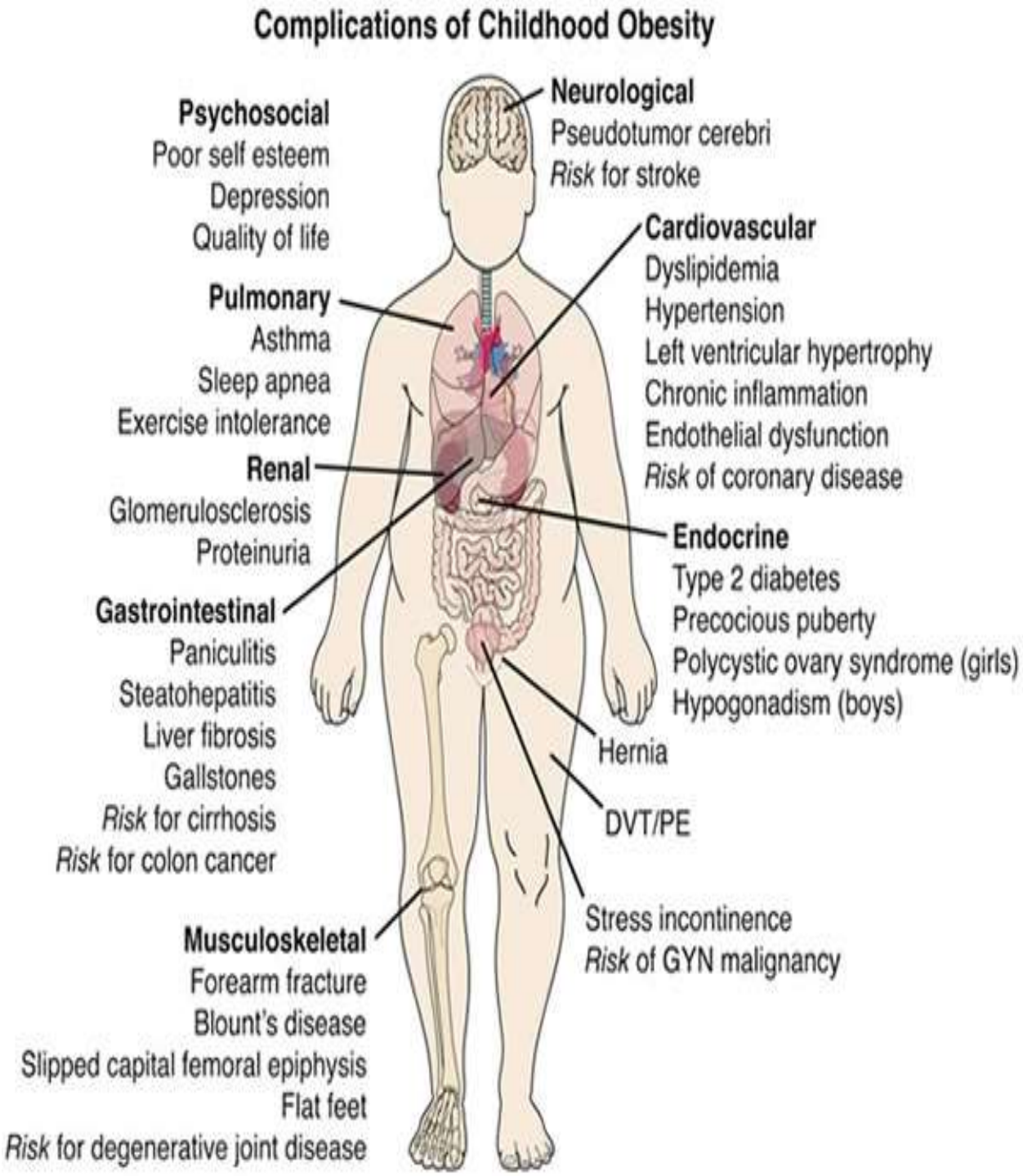


Fig. No. 25- Showing Complications Of Childhood Obesity

Obesity has numerous adverse effect on health. The relation in between obesity & mortality from diabetes, hypertension & coronary artery disease is well established

Morbidity of obese persons muc h more than ideal body weight (approximately 200 %) as much as twelve fold in mortality. Mortality rate rises as obesity increases, perticularly when obesity is asociated with excess intra abdominal fat. Obesity is associated with pathological as well as metabolic sequeles along with a number of many other complications.

1. Pathological Sequele

There is an abnormal growth of the adipose tissues due to an enlargement of hypertrophy or hyperplacia of fat cells or a combination of both. Increased adipose tissues are deposited subcutaneously around almost all the internal organs throughout omentum & in intramusular spaces.

2. Metabolic sequele

Obesity predisposes to carbohydrates intolerence by increasing insulin resistence . Both hyperinsulinaemia & insulin resistence increase with weight gain, it is particularly common with android obesity. There are three probable mechanisms proposed for it.

1. Insulin itself, by inducing receptor down regulation

2. Free fatty acids are known to increase & capable of impairing insulin action.

3. The cytokinin TNF-? Which is produced by adipocytes , over expressed in obese adipocytes & capable of inhibiting insulin action.

All these conditions contribute to complications of diabetes mellitus.

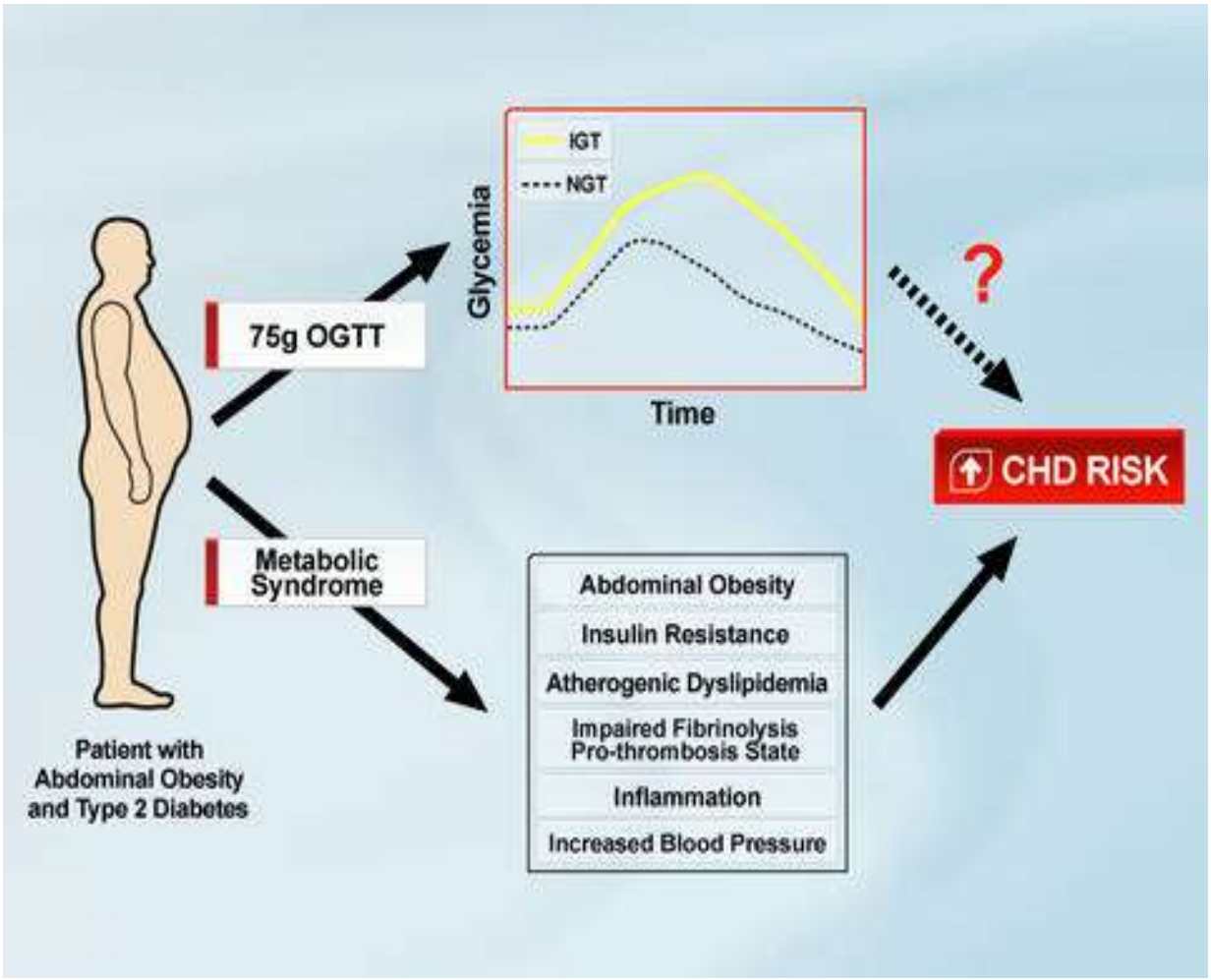


Fig. No. 26- Showing the Metabolic syndrome Of Obesity ending in Coronary Heart Disease (CHD)

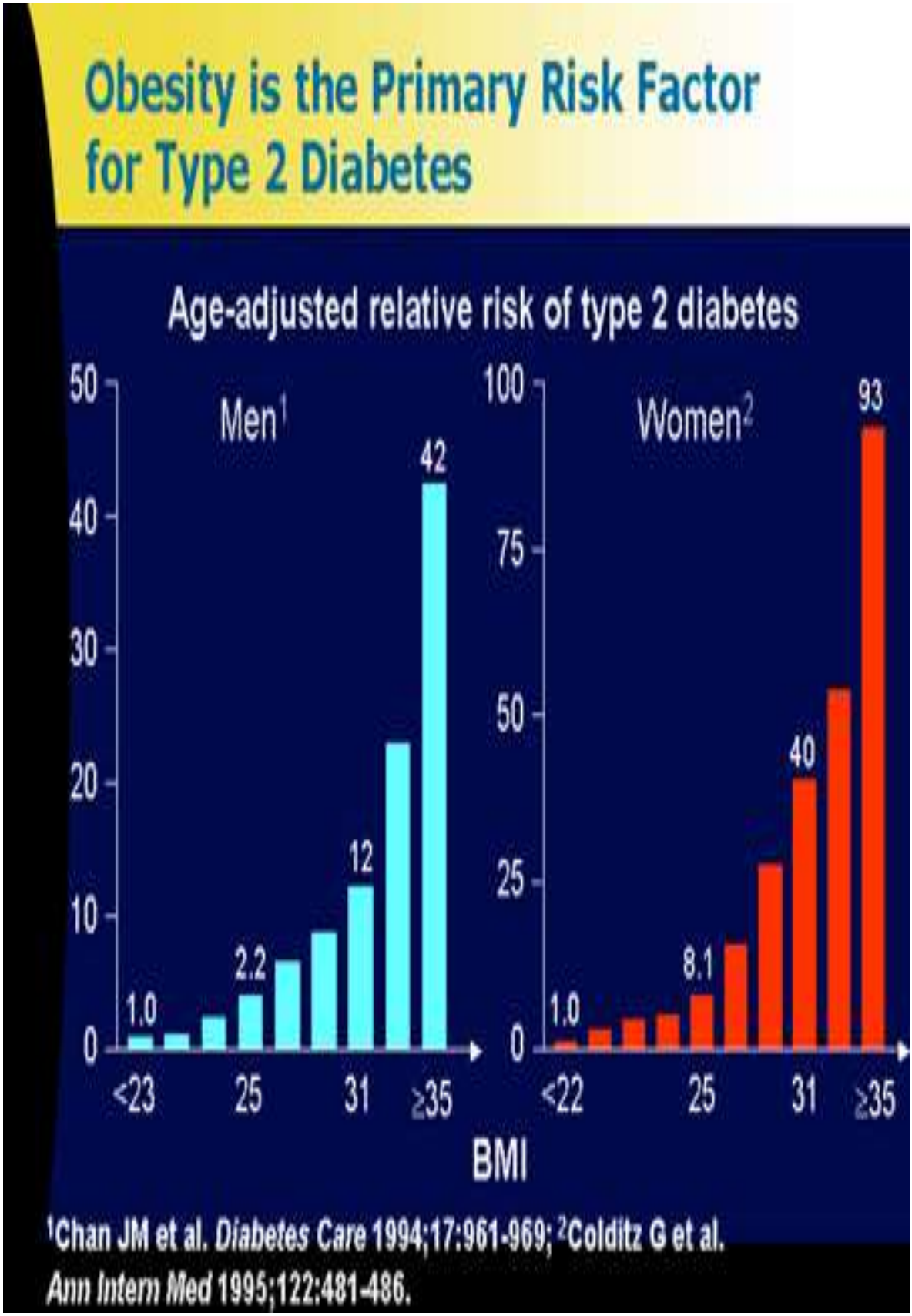


Fig. No. 27- Showing Incidences Of DM-2 due to Obesity

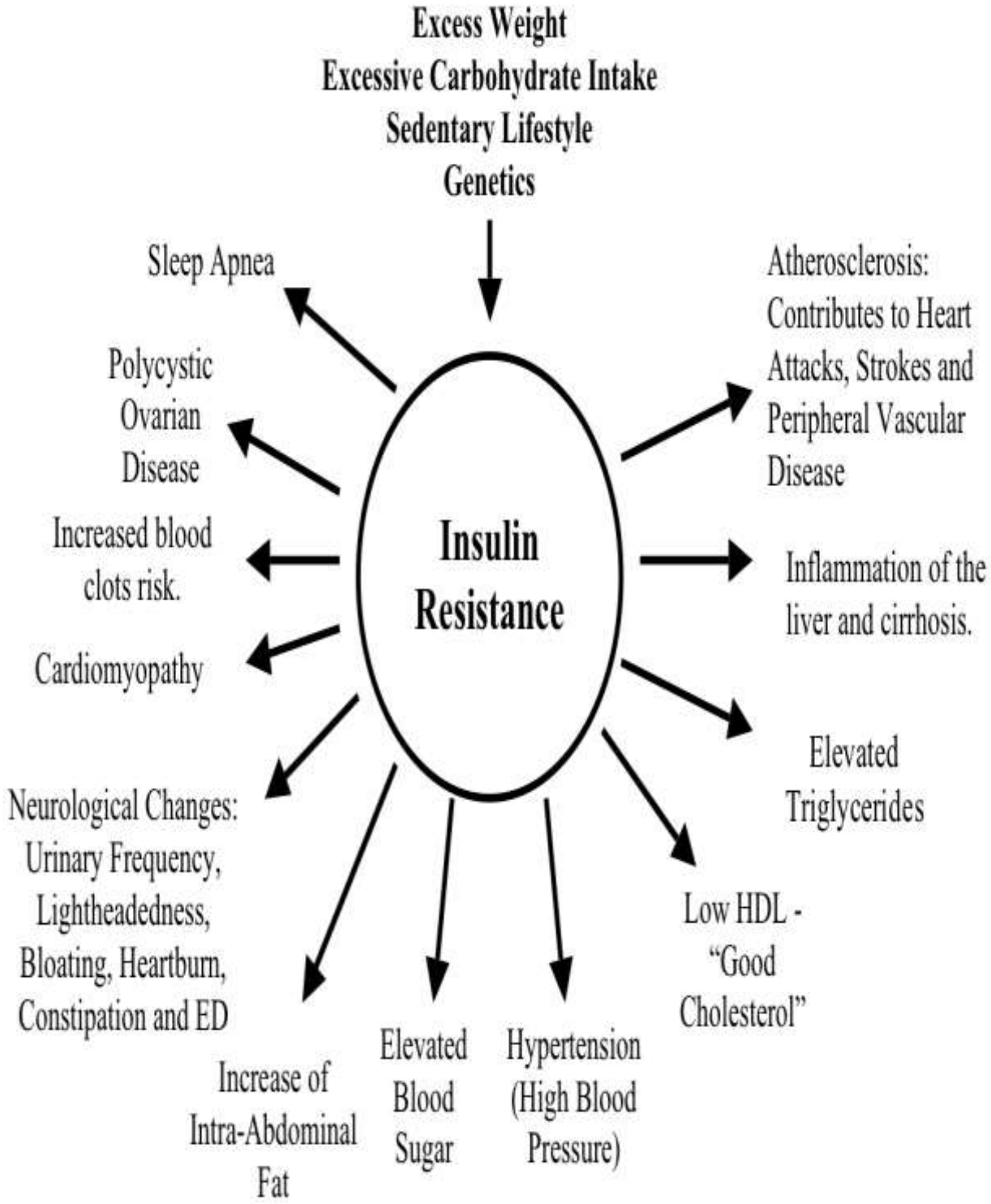


Fig. No. 28- Showing Insulin Resistence & its adverse Effect on the body

3. Cardiovascular diseases - The Framingham study reaveled that Obesity was an independent risk factor for the so many circulatory changes such as increase in

pulmonary & systemic blood volume & increase in stroke volume and cardiac output. This increased work load on heart leads to dilatation & hypertrophy which predisposis to Lt. ventricular hypertrophy (LVH) & if leave untreated then can converted in to Congestive Cardiac Failure (CHF)**.** The effect of obesity on deaths due to cardiac complications in asian women may be seen at BMI as low as 25. The waist/hip may be the best predictor of these risks. When the additional effects of hypertension & glucose intolerence associated with obesity are included the adverse impact of obesity is even more evident. Obesity specially abdominal obesity, is associated with an atherogenic lipid profile, with increase low density lipoprotein, cholesterol, Very low density lipoprotein & triglyceride, & decreased high density lipoprotein.

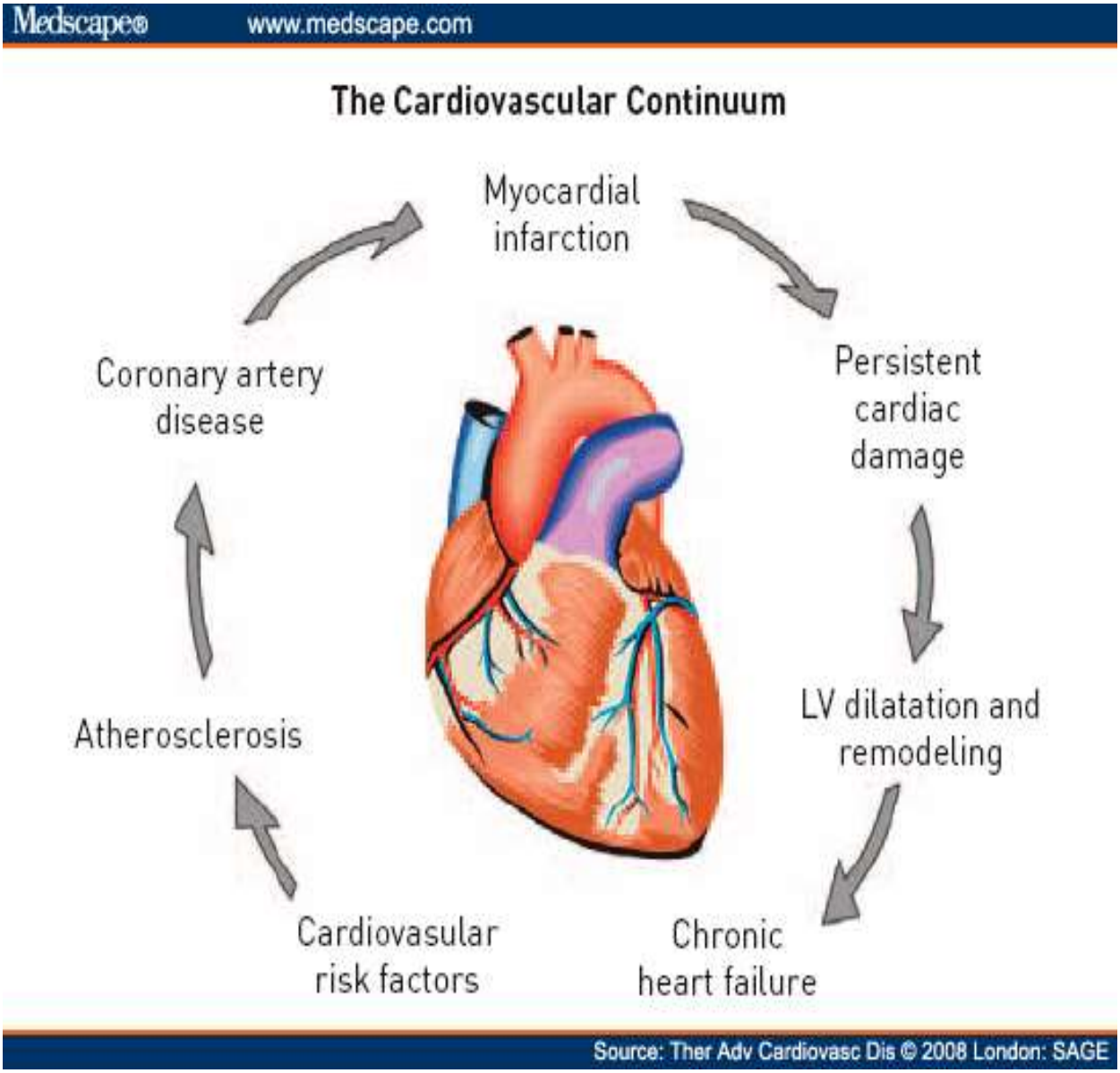


Fig. No. 29-Cycle of Cardiovascular disease

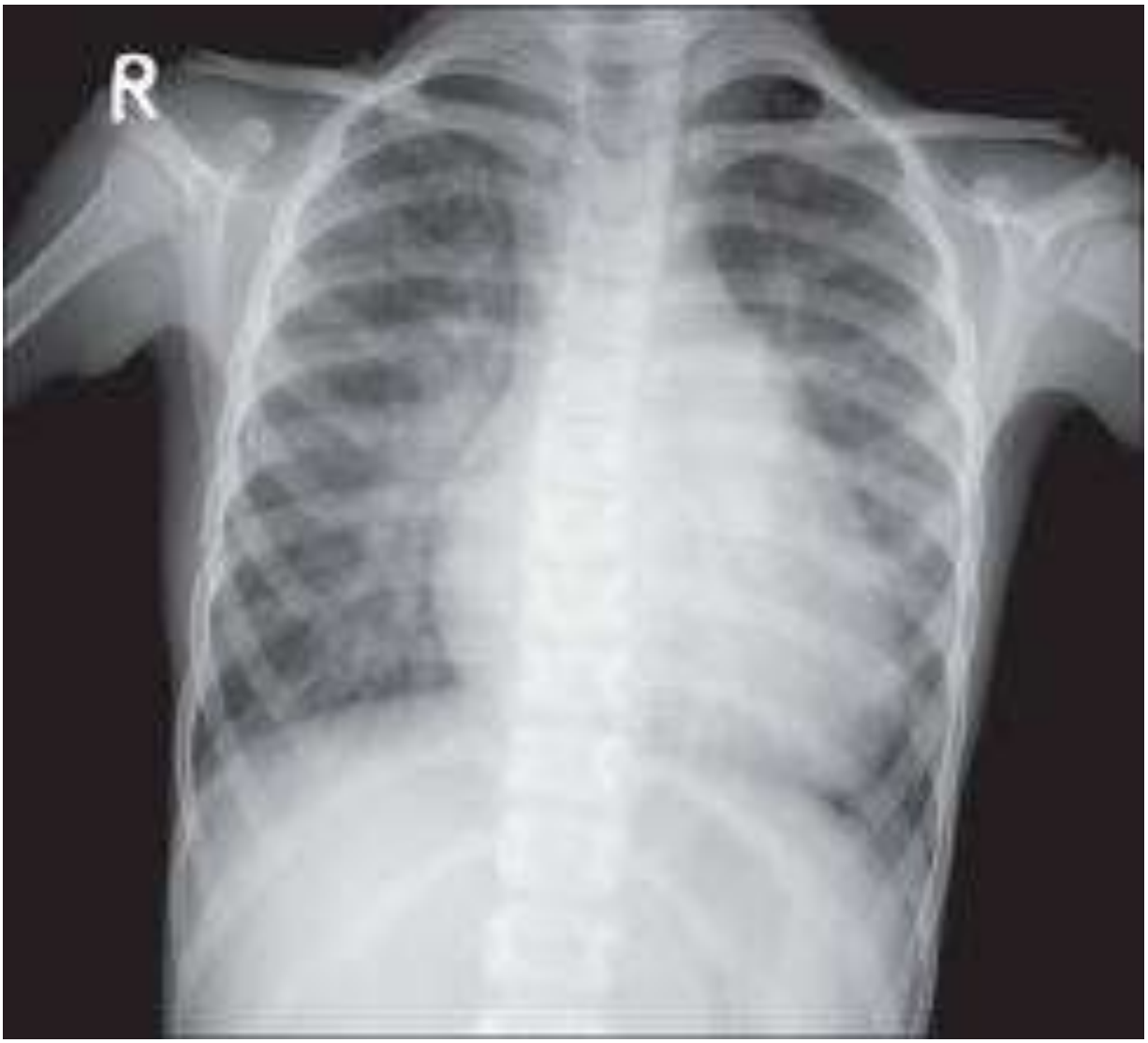


Fig. No.30- X-ray Showing Cardiomegaly (Lt. ventricular hypertrophy)

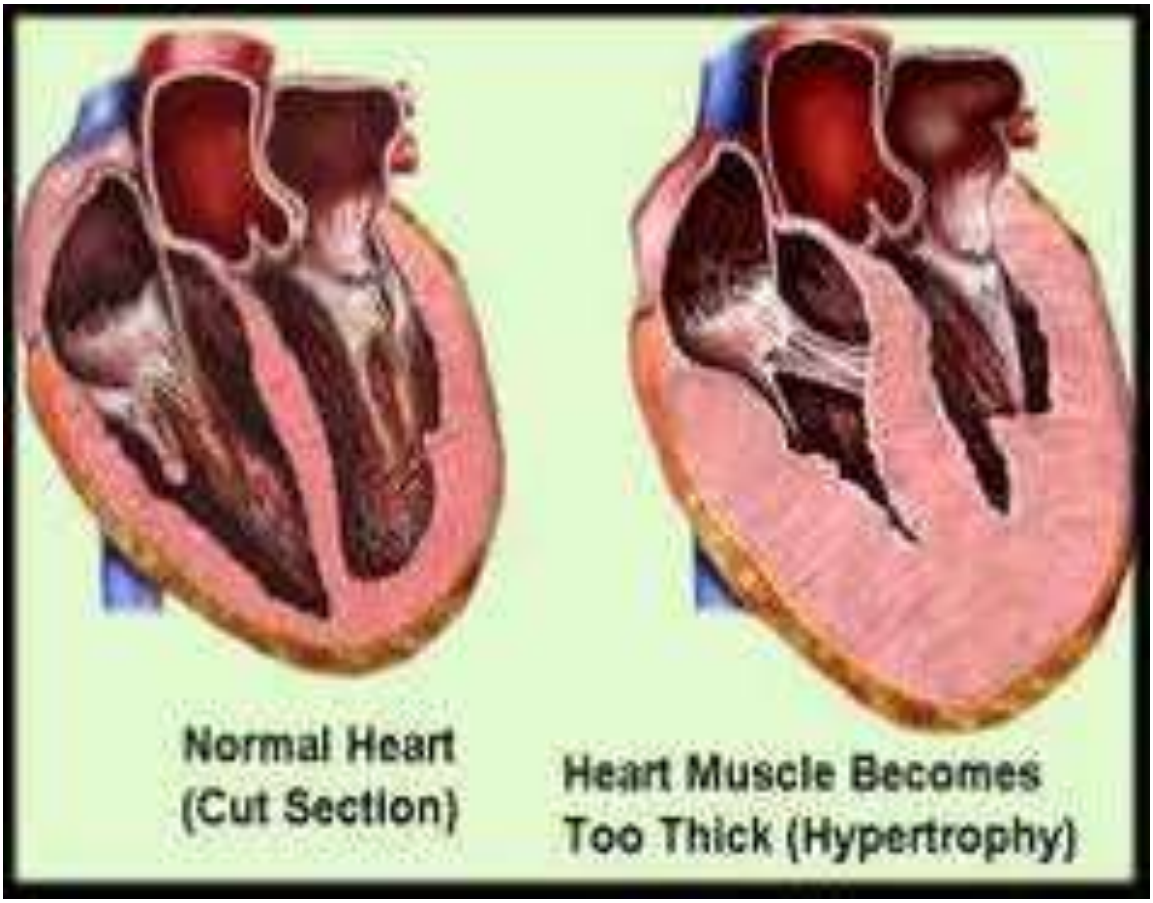


Fig. No. 31-Cut sections of normal & Hypertrophy of Lt. ventricle

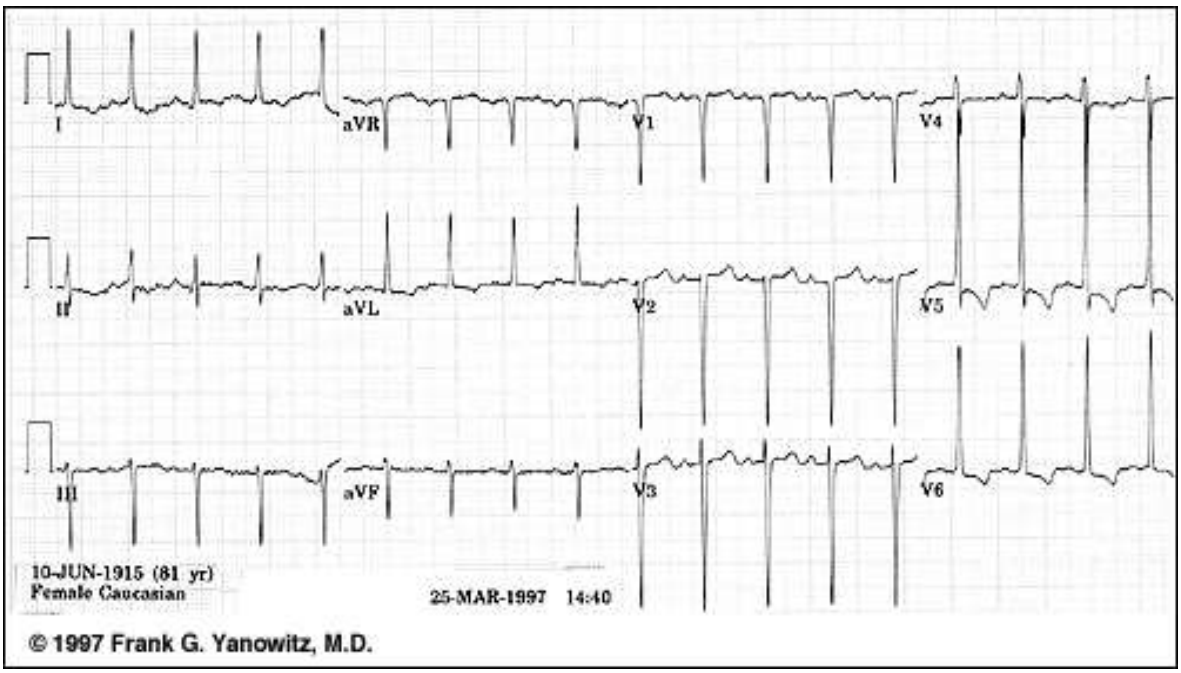


Fig. No. 32- Showing E.C.G. of LVH (deep s wave in V1& V2 leads, increased R wave in leads V5 & V6) also ischaemic changes

1. **Hypertension**

Obesity induced hypertensive have twelve time risk of CAD than non obese. Obesity induced hypertension is associated with increased peripheral resistence & cardiac out put, increased symphathetic nervous activity, increased salt sensitivity & insulin mediated salt retenton & often responds to moderate weightloss.

2.Pulmonary Disease

Severe obesity may be associated with Obstructive sleep apnoea & Pickwick's syndrome or obesity hypoventilation syndrome. Which is characterised by somnolence, obesity & hypoventilation.

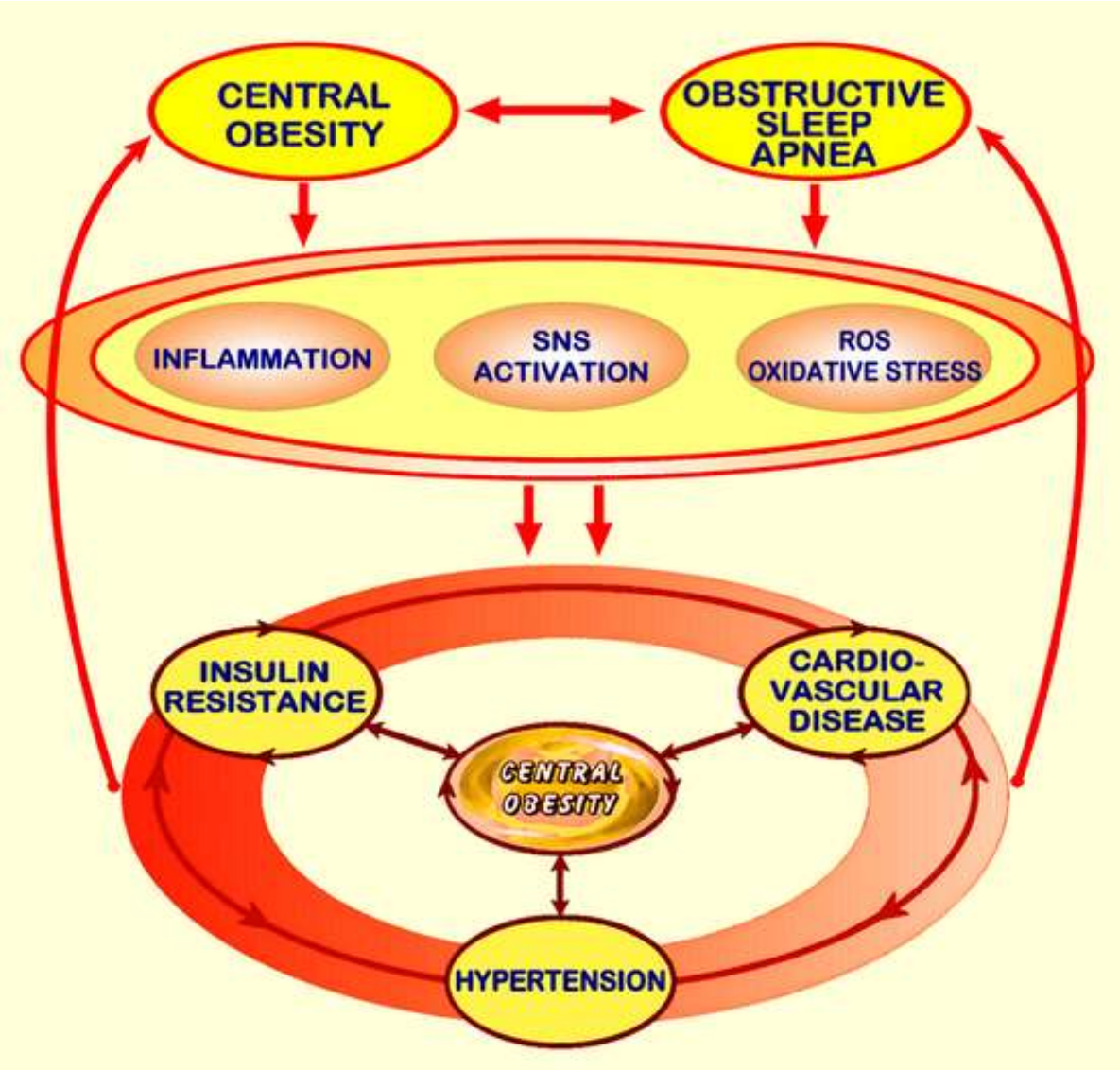


Fig. No. 33- Showing pulmonary complications

3.Cholelithiasis

Obesity is associated with increased biliary secreation of cholesterol, super saturation of bile & a higher incidences of gall stone particularly cholesterol gall stone.

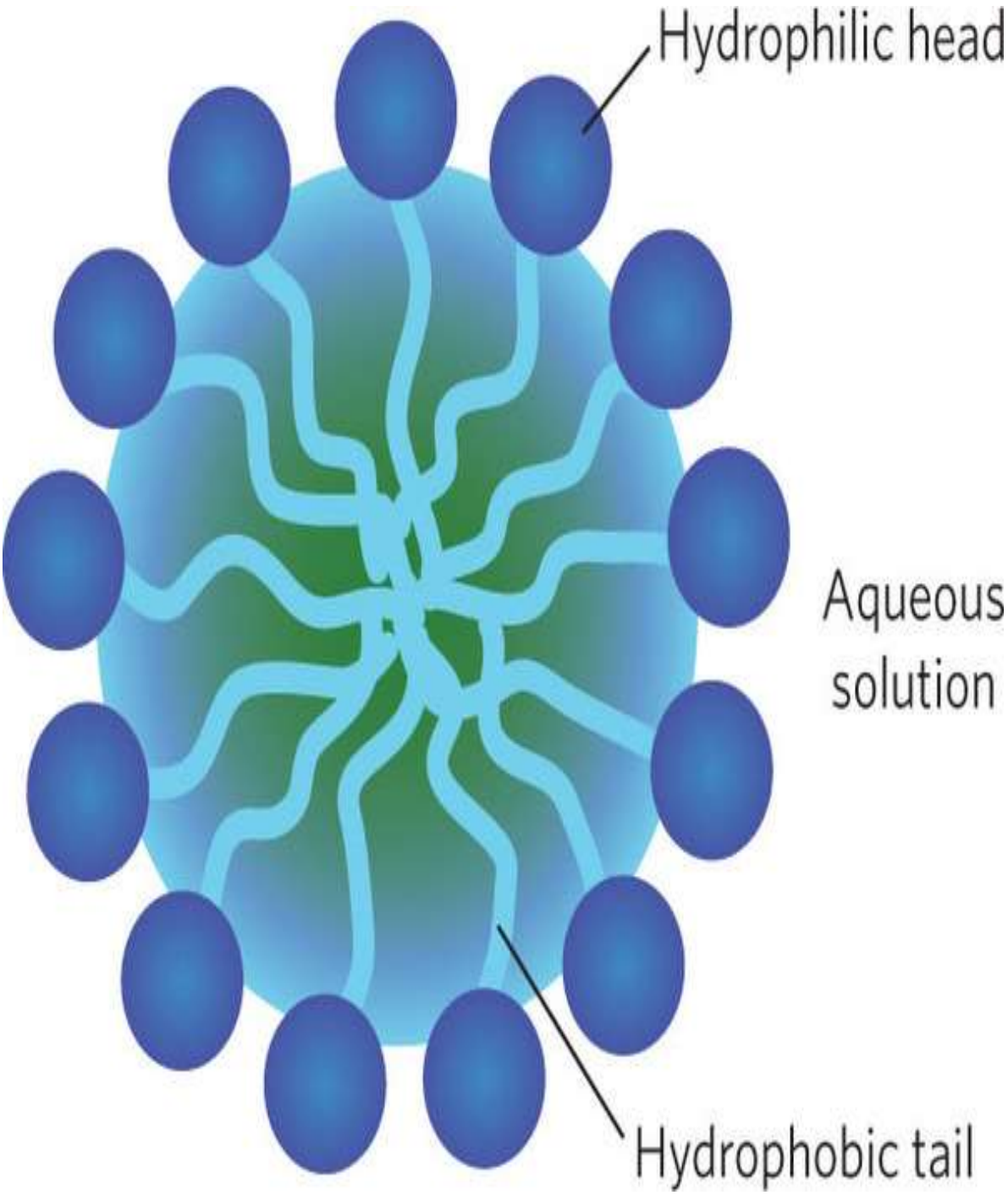


Fig. No. 34-Micle formation

4. Dyslipidaemia High intake of fatty diet is associated with dyslipidaemia however all obese may not be dyslipidaemic. The increase in serum triglycerides in obesity is related to an increase in the hepatic production of VLDL due to an increased flux of non esterified fatty acids out of adipose tissues. This process is enhanced in android type of obesity & may also be due to decreased sensitivity to insulin, resulting in increased insulin production which stimulates liver to produce excess endogenous VLDL by enhencing lipogenesis & lipoprotein packaging & by preventing triglycerides breakdown. Hepatic VLDL production is frequentely greater in obese persons even in absence of hypertriglycerides. This is due to compensatory increase in rate of removal of TG from circulatory TG rich lipoprotein s due to increased LPL activity. Obesity is also accompanied with with an increase in cholesterol synthesis thus serum cholesterol levels. It cause type IV hyperlipoproteinmia or IIa hyperlipoprotaenmia but may excerabate other forms. Obese persons with typehyperlipoprotaeinmia or pre existing LPL impairment may develop marked type.

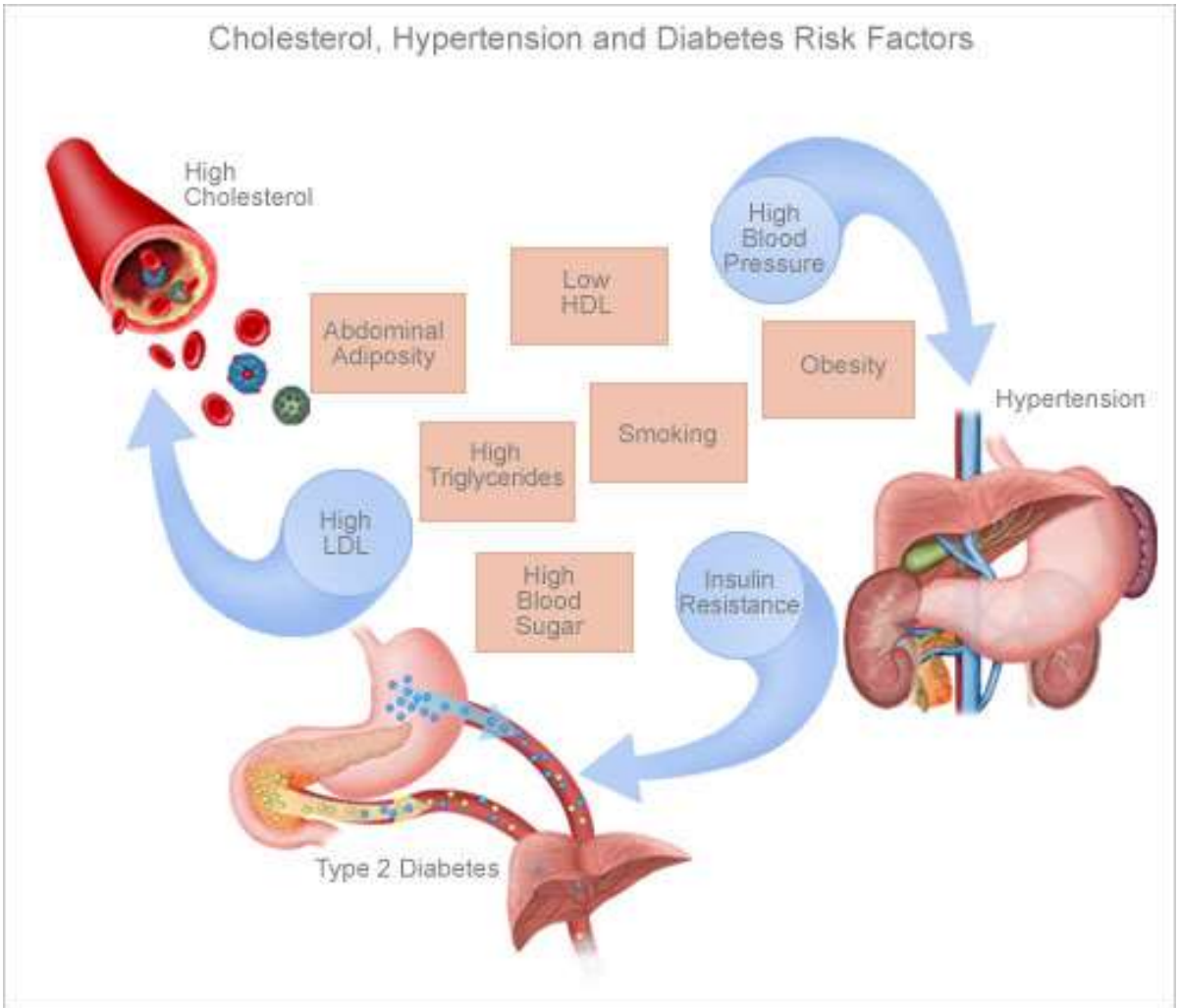


Fig. No.35-Interreled Complications of Dyslipidaemia & DM-2

5. Bone, Joints & Cutaneous diseases

Obesity is associated with an increased risk of osteoarthritis due to trauma of added weight bearing. Prevelenve of gout is also increased. skin menifestations includes acanthosis nigricans menifested by darkening & thickening of skin folds on the neck, elbows & dorsal interphalangeal spaces. Acanthosis reflects the severity of insulin resistence & improve with weight loss. Friability of skin may increase at skin folds with increased sweating & fungal & bacterial infections. Finally venous stasis is increased in the obese persons.

6. Cancers

Obesity in males is associated with higher mortality from cancer of colon, rectum & prostate; & in females it is associated with mortality from cancer of gall bladder, bile ducts, breast , endometrium, cervix & ovaries. Due to increased rates of conversion of androstenedione to estron in adipose tissue of obese individuals.

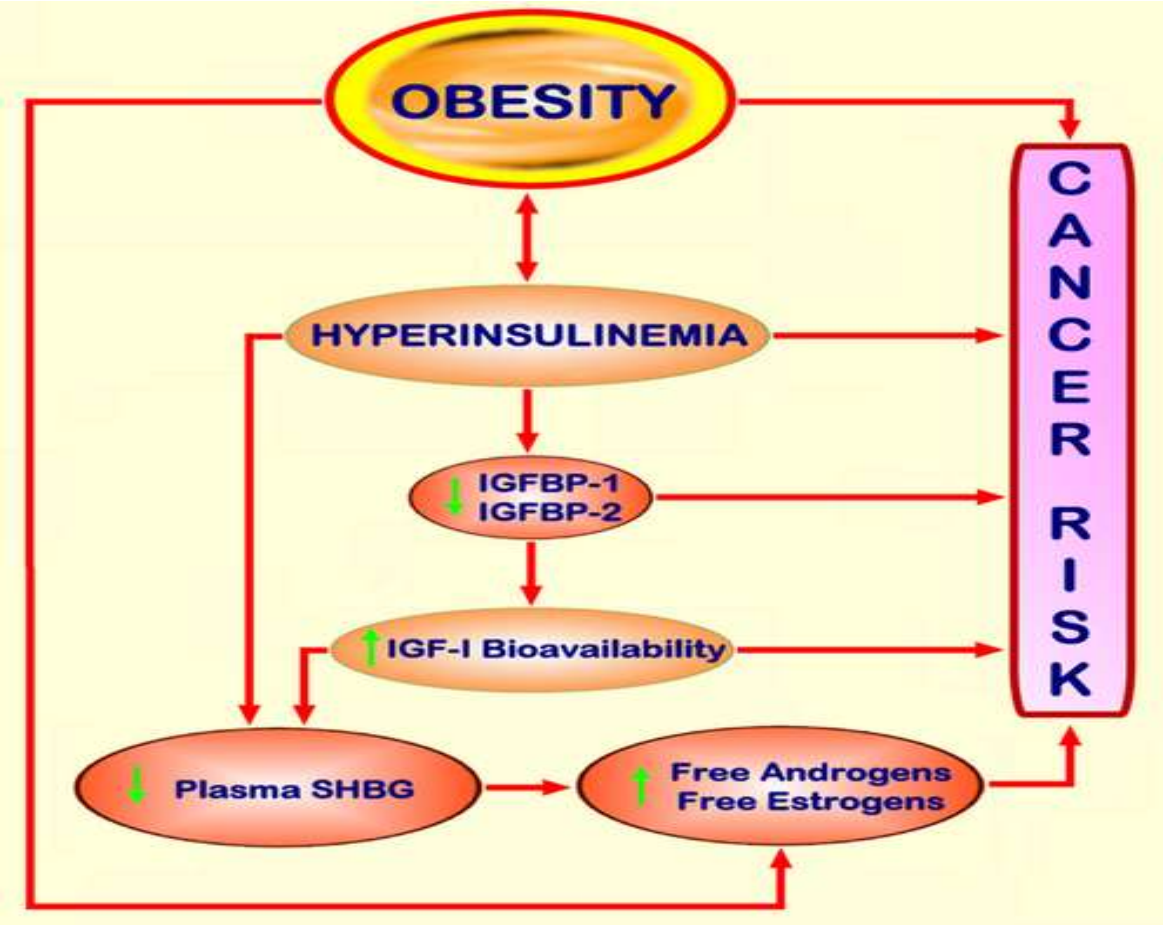


Fig. No. 36-Risk of Cancers due to Hyperinsulinemia

7. Reproductive Disorders

Male hypogonandism is associated with increased adipose tissue. In men >160% of ideal body weight, plasma testosteron & sex hormone binding globulin are often reduced & estrogen levels are increased. Gynacomastia may be seen. In females, suffering from obesity common conditions are increased androgen production, decreased sex hormone binding globulin & increased perpheral conversion of androgen to esterogen 40% of women with poly cystic ovarian syndrome (PCOS) are obese.

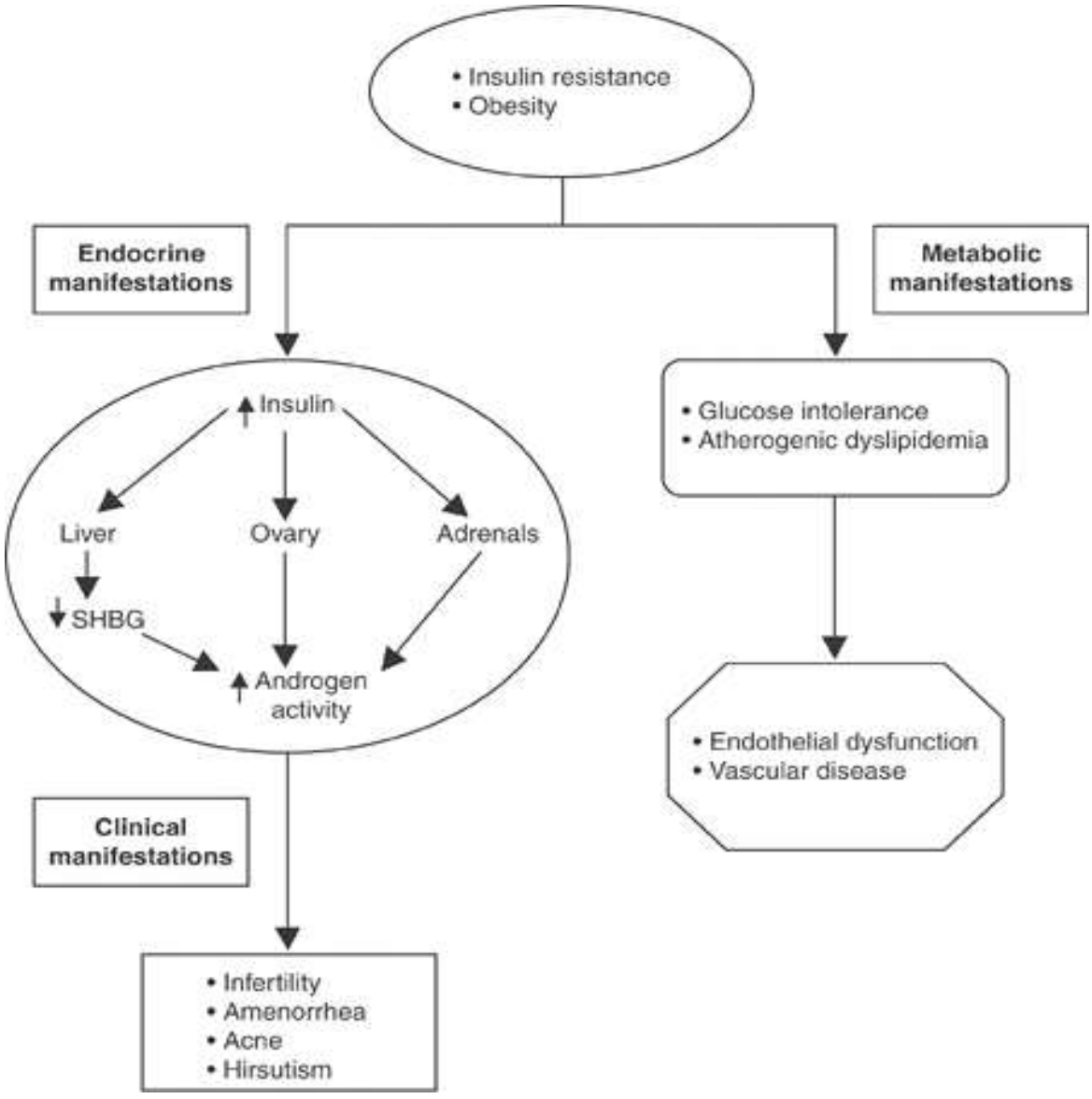


Fig. No. 37- Showing Effect of Obesity on Reproductie System

8. Psychological sequele

Obese patients, particularly young females have poor self esteem & may suffer from chronic depression. Anoxious & emotionally disturbed patients may suffer from eating disorders & its psychological menifestations.

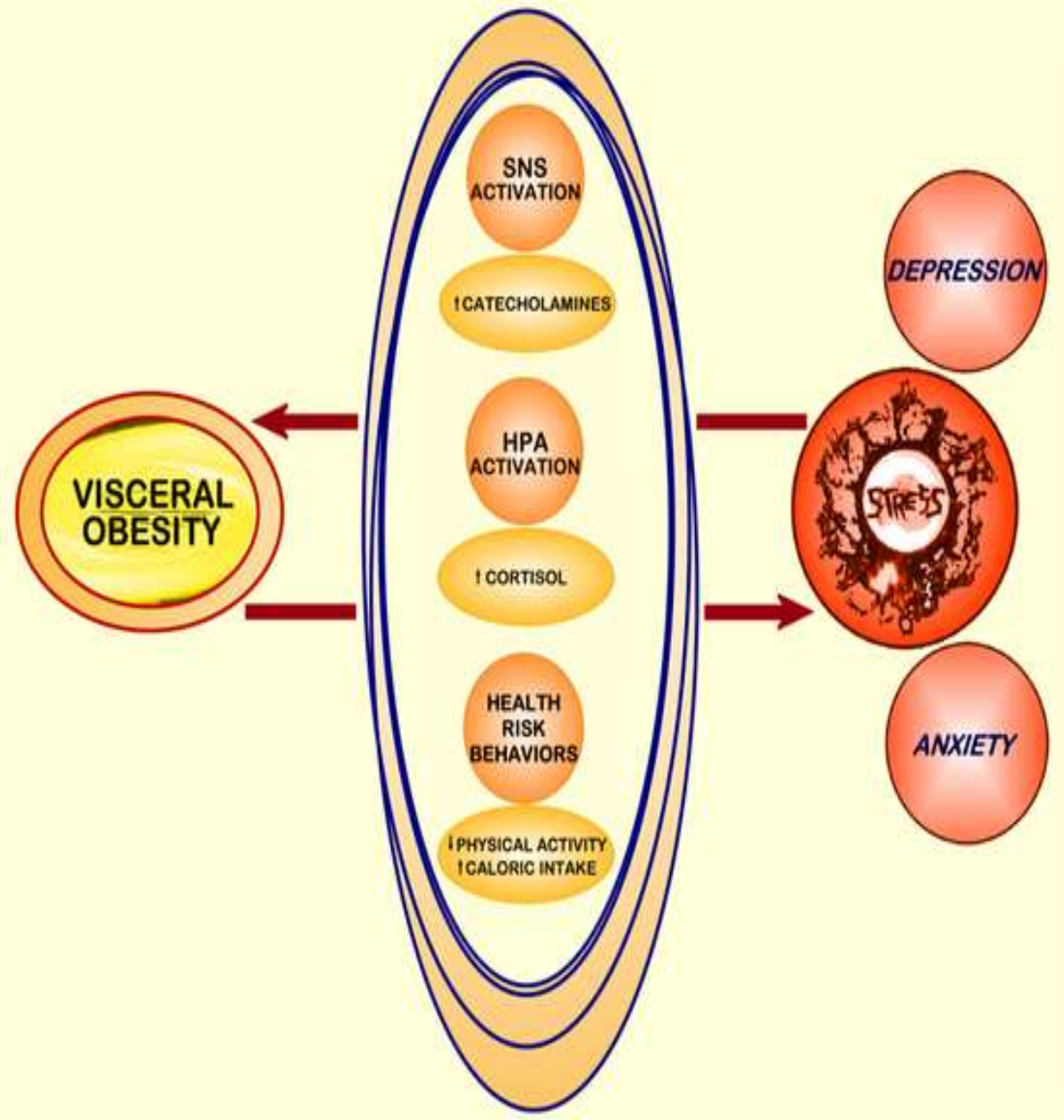


Fig. No.38- Interrelationship in between Obesity & Stress

MANAGEMENT:

Obesity is a chronic metabolic disorder. Progressively attain of normal body weight without producing unwanted treatment induced side effects & morbidity,

rarely achieved in medicinal treatment. Short term weight loss is beneficial but long term weight loss is not common. Treatment goals should be guided by the health risk of obesity. The physician should always consider the possibilities to identified the cause of obesity, such as hypothyroidism, hypercortisolism, male hypogonadism, insulinoma, any central nervous system disease that effects hypothalamic function, however these causes are least common. The ultimate cause of obesity is energy imbalance in between intake & output which leads to deposition of the extra calorie in the form of fat leading to weight gain. Hence weight reduction can be achieved by energy intake or by increasing output or by combination of both. Successful management of obesity is defined as the sustained attainment of normal body weight without producing unacceptable treatment induced morbidity. The management of obesity has following aspects.

1. Dietary modification

2. Behaviour modification

3. Exercise

4. Drug therapy

5. Surgery

1. Dietary modification

In the Obesity management cornerstone is to reduced calorie intake. The fundamental rule is sustained reduction of energy intake below that of energy expenditure. The diet should provide 500 - 1000 kcal/day less then the maintanance energy requirements in order to achive weight loss. Middle aged obese house wife needs 800 - 1000 kcal /day whereas an obese man engaged in active physical work, need 1500 - 2000 kcal /day. The diet must provide the essential nutrients. It is desirable to restrict intake of saturated fat , to reduced the risk of atherosclerosis & ultimately CAD. The food that can be consumed without restriction s are fruits, green & leafy vegetables & whole grain cereals. Since these are rich source of micronutrients & non starchy polysaccharides. The low calorie diet i.e. 800-1500

kcal/day is more fruitful in weight reduction. Diet less then 800 kcal/day have been found to be no more effective than low calorie diet in reducing weight.

A simple way to calculate diet for an individual is to allow 15-20 calories/k.g. of ideal weight. Normal diet should contain protein 50gm, carbohydrate 100gm, fat 20gm, minerals like iron, calcium & salts, suppliments of vita A, C & fluid 2 liters.

2. Behaviour Modification - Behaviour Modification The patient is advised for monitoring the eating, and rewards are given to modify the bad/ maladaptive habits. Patient may benefit from counselling offered in a stable group for a longer time even after weight loss. In behaviour modification the patient is made aware of what & how much they consume. Initially in the education process, careful food intake chart are kept. These charts are analysed & nutrient densities of food should disscussed. New modes of eating are suggested, including no eating in between the meals, eating always at table, eating always three times per day, watching the food portions eaten, not doing other activities while eating & eating slowly with concentration. Patients may benefit from coun celling offered in a stable group setting or extended periods of time, including after weight loss.

3. Exercise

Exercise is an important component of the treatment of obesity. Exercise helps in expenditure of energy. In many cases the only exercise is the sole therapy . advantages of exercise are as follows decrease body fat, obesity, hypertension, triglycerides, cholesterol, Low Density Lipid (LDL), Very Low Density Lipid(VLDL) & increase High Density Lipid (HDL) & insulin sensitivity. Even if exercise had no such marked effect it would be valuable in the obese individual for its effect on cardiovascular tone & blood pressure. Since many obese individuals have not perform exercise regularly & may have cardiovascular risk factors, it should be introduced gradually & under medical supervision. Increased energy expenditure is the most obvious mechanism for an effect of exercise. Scientific researches show that regular exercise for 10 minutes at a stretch 4-5 times/day is as beneficial as exercise for 40-50 minutes at a time. Exercise in the morning suggested for keeping metabolism higher all the day. Aerobic activities are most often reccomnded for weight management programmes owing to the increased number of calories expended during activity. Patient shouldbe

emphasised to start with light exercise , gradually increase it & then maintain it regularly. In short jogging, brisk walking, swimming, badminton, aerobic exercise i.e. exercise in open air much better for weight reduction. Regular exercise also improves general health&enables patients to control their diet.

Fig. 39. A. Jogging

Fig. No. 39. B. BriskWalking/ Jogging

Fig. No. 39. C Swimming

Fig. No. 39.D playing Badminton

Fig. No. 39 A-D - Showing Different Exercise to check the Obesity

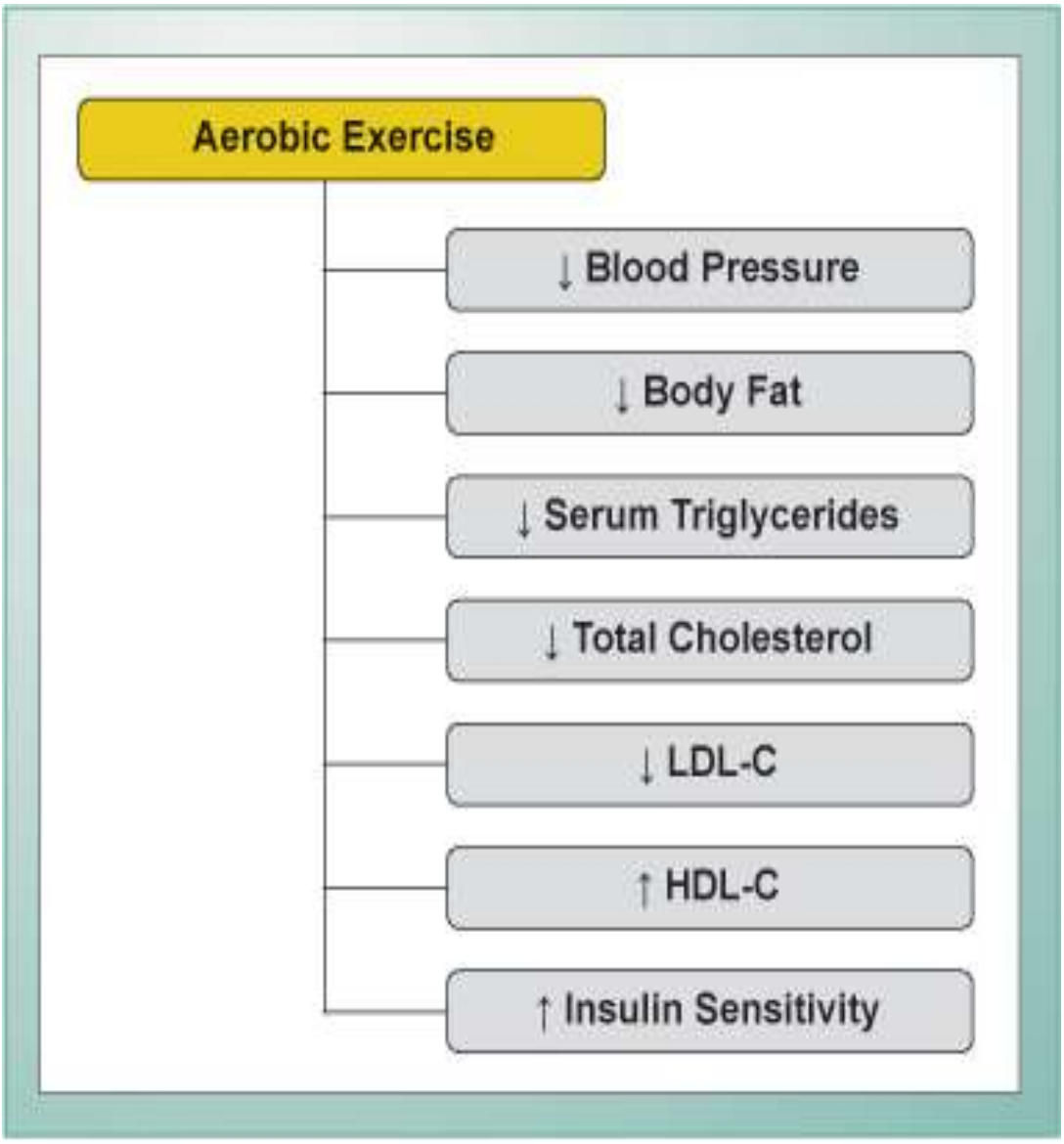


Fig. 2 - Desirable benefits from aerobic training.

Fig. no. 40-Showing Benefits of Aerobic Exercise

4. Drug therapy

Diet is the Cornerstone of obesity treatment is reduced calorie intake. The chief goal is the sustained reduction of energy intake. Drugs treatment of obesity is less effective.

Sibutramine is a novel central reuptake inhibitor of both norepinephrine & serotonin. Using once daily dose over 24 weeks it produced a 7% weight loss. It lowers cholestrol & triglyceride level & exhibited similar clinical efficacy to fenfluramine. Sibutramine increase pulse & blood pressure in some patients & long term safety is not established.

Orlistaris an inhibitor of intestinal lipase that cause modest weight loss due to drug induced fat malabsorption. The mechanism appears to involve inhibition of appetite

Drug treatment of obesity not much effective. Despite short term benefits, medication induced weight loss is often associated with reboun d weight gain after the stop of drugs, side effects from medications & potential for drug abuse. hence, drug therapy, should be given as an adjunct in careful selected patients. International guidelines advocate the use 7 drugs therapy in BMI >23 in presence of co morbid condition (Hypertension, Dyslipidaemia) or for those with BMI >25.

Anti- Obesity Drugs

There are following general classes of anti obesity drugs:

1**. Catechoaminergic medicines:**

These includes Amphitamine like agents which act at the level of hypothalamus & reduced appetite.

2. **Serotonin release Promotores Or Serotonin reuptake Inhibitors:**

A. Sibutramine- It blocks uptake of both Serotonin & norepinephrine in the central nervous system. It suppresses Appetite & appears to stimulate thermogenesis. Daily once dose 10 mg upto 24 weeks, it produced a 7 % wt. loss in a double blind placebo controlled trial. Side effects include dry mouth, constipation, anorexia , insomnia & diziness.

B. Fenfluramine: It is having tranquillçing effect on hypothalamus & reduced food seeking behaviour as well as decreased quqntity of food consumed at any meal with mild elevation of BMR.

Side effects are depression on sudden withdrawal, lethargy, drowsiness, dry mouth, diarrhoea, loss of libido.

Dexfenfluramine is more active analogue with higher efficacy & lower side effects.

3. Drugs inhibiting Fat absorption:

Orlistat works on the GIT. By inhibiting intestinal lipase, it reduces fat absorption. Orlistat may result in dirrhoea & flatus & cramping and perhaps also reduced absorption of fat soluble vitamines. In randomçed trials with upto 2 years of follow up orlistat resulting in 2-4 kg more wt. loss than placebo. The recommnded dose is 120 mg three times a day with meals.

4. Thyroid hormone suppliments:

It causees loss of lean body mass & raises the risk of hyperthyroid state & is hence useful only in hypothyroid state.

5. Other potential drugs for obesity:

A. Recombinant human leptin is in clinlcal trials. In the rare cases of leptin deficiency caused by murtation of leptin gene, it has been tried & has been found highly effective.

B. ß$_3$-Adrenergic receptor agonists may provide a new treatment approach for the obesity. These drugs stimulating thermogenesis in brown adipose tissue, They also stimulate lipolysis in white adipose tissue. This drug also reduced insulin resistance & lower blood glucose by a mechanism that is not yet defined. Recombinant human leptin is also in clinical trials. In the rare case of leptin deficiency caused by mutations of the leptin gene, the administration of recombinant leptin is high effective. Primary reports suggest that the response to leptin is limited or absent in common causes of obesity, which are associated with hyperleptinemia & leptin resistance. New drugs are also being developed based on insights into central pathways that regulate body weight.

C. Antagonist for NPY receptors & agonist for melanocortin-4 receptors are also being clinical tried.

5. Surgical Treatment:

Morbid obesity 100% above ideal body weight, is estimated to increase mortality by as much as twelvefold in men between 25-34 years of age & six fold between 35 & 45 years of age. Deaths from cardiovascular disease, diabetes & complications have been recorded. In response to in effective treatment using diet, exercise, drugs, surgical procedures have been tried. The potential benefits of surgery include major weight

loss & improvement in hypertte3nsion, diabetes, sleep apnea, cardiac heart failure, angina, hyperlipidaemia & venous disease. Many different approaches have been used often without adequate assessment of efficacy & complications. Jejuno illeal bypass surgery has largely been avoided due to complications, which have included electrolyte disturbences, nephrolithiasis, gall stone, gastric ulcer, arthritis & hepatic dysfunction, with cirrhosis occurring in as many as7% of patients. Two procedures in common use today are the vertical banded gastroplasty & the Roux-en-Y gastric bypass.

Following the National Institute of health Consensus conference on Gastrointestinal surgery for severe obesity, it was recommended that suitable patients for selected using the following critaria

1. The presence of 45kg (100lb) or 100% above ideal body weight, or one or more severe medical conditions related to refractory obesity,
2. Repeated failure of the other therapeutic approaches,
3. At eligible weight for 3-5 years,
4. 4 capability to tolerating surgery,
5. Absence of alcoholism, other addictions or major psychopathology &
6. Prior clearance by a psychiatrist. It is recommended that an appropriataley experienced surgeon work together with nutritionists & other support personel, evaluation & follow-up programmes should be monitored closely & wisely.

Surgical meathods for Obesity fall into 3 categories:

A. Excision of Fat

This one is not much practical surgery & the obese individual is exposed to the risk of anaesthesia, surgery & poor wound healing.

B. Surgery to cause Malabsorption

This includes jejunoilleal bypass surgery where the upper jejunum is divided, the distal end is closed & the proximal end is anastomosed either end to side or end to end to the distal ileum.

C. Operation to restrict Food intake-

Both gastric bypass & gastroplasty are gastric reduction operations in which a new stomach is surgically constucted from the upper part of the normal stomach. It produce a sense of fullness & helps to stop eating.

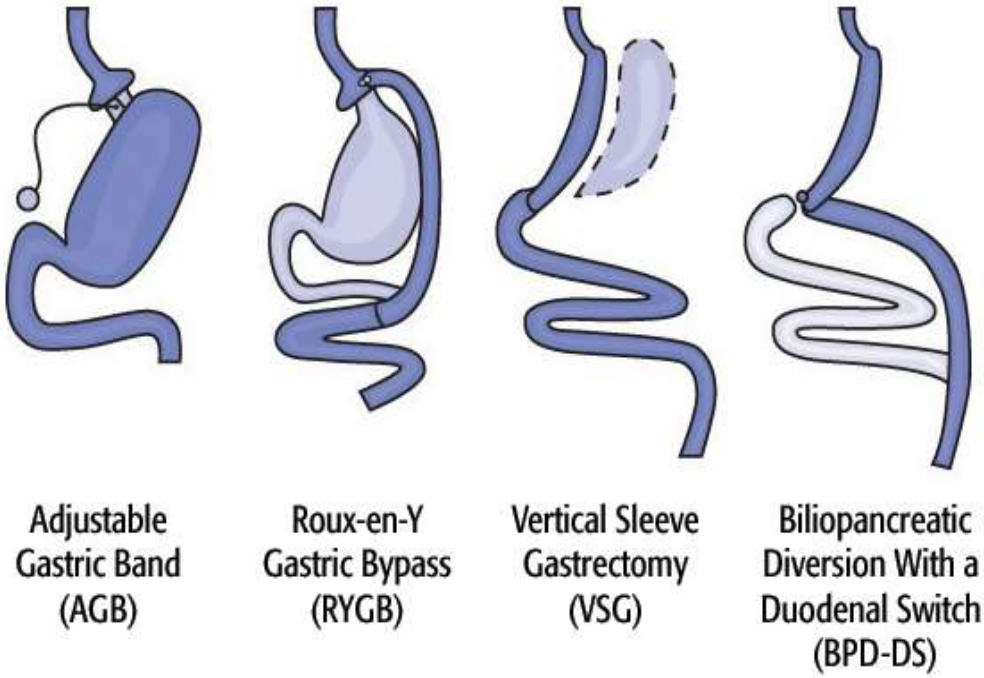


Fig. No. 41- Different Surgical Procedures For the treatment of Obesity

Table no. 12.

Table. Complications of Bariatric Surgery

Early Complications (Within 30 Days of Surgery)	Late Complications (More Than 30 Days After Surgery)
Bowel obstruction	Anastomotic stricture
Deep venous thrombosis	Bowel obstruction
Gastrointestinal or intra-abdominal bleeding	Cholelithiasis
Leaks	Dehiscence/fistulization
Pulmonary embolism	Gastrointestinal or abdominal bleeding
Wound infection	Incisional hernia
	Marginal ulceration
	Nutritional deficiencies

DYSLIPIDAEMIA

Dyslipidaemia is abnormal an abnormal amount of lipids (Colesterol, triglycerides etc) in the serum. The term dyslipidaemia is used to expressed all the defects of lipid metabolism, their rate of synthesis or clearence from plasma. In developed countries, most dyslipidaemics are hyperlipidaemics, i.e. increased in lipids in the serum, often due to faulty diet & life style. Obesity & dyslipidaemia are

interrelated conditions with obesity ultimately leading to dyslipidaemia & together they may lead to chronic morbid conditions such as Diabetes mellitus, CAD, Arteriosclerosis & hypertension.

Basic Concepts of Lipid/ Fat

Lipids are groups of orgenic substances of fatty nature, insoluble in water but soluble in fat solvents. They include fats, sterols, waxes & phospholipids etc. Fats are solid at 20 °C, they are called oils if they are liquid at that tempereture. Fats yield fatty acids & glycerol on hydrolysis. Lipids are classified as:

1**. Simple lipids:** Easters of fatty acids with alcohols.

A. Neutral fats : Triglycerides, esters of fatty acids with glycerol

B. Waxes: Cholesterol & its esters

2. Compound lipids: They are esters of fatty acids with alcohols & containing other groups:

A. Phospholipids: esters containing phosphoric acid & a nitrogenous base e.g. lecithin, cephalin.

B. Glycolipids: Esters containing a carbohydrate & a nitrogenous base e.g. cerebrocides.

C. Sulpholipids: Esters containing sulphuric acid

D. Lipoproteins: Lipids attached with proteins

3. Derived lipids : These are derivatives obtained by the hydrolysis of 1 & 2 molecules & which still possess the physical properties of lipids which are derived as :

a. Fatty acids: saturated & unsaturated

b. Steroids

c. Fat soluble vitamins

The human body can synthesçe triglycerides & cholesterol endogenously. Most of the body fat (99% of adipose tissue) is in the form of triglycerides. In normal human person, adipose tissue constitutes 10-15 % of body weight.

The lipids in the body physiologically form following two components:

1. Elements constant or structral lipids: The value of these lipids remain constant even under extreme starvation. It is chiefly composed of phospholipids, cytoplasm & cell membrane of all organs are composed of element constant.

2. Elements Variable: It is composed mainly of neutral fat. It presents mainly in the depot fat or adipose tissue. The depot fat is not static but it is in a continuous state of change due to its continuous synthesis & breakdown in the body.

Depot fats

Fat in the body presents in two forms i.e. blood fat & depot fat. The major part of body fat remains stored in the so called fat depots.

Distribution of fat in the body tissues:

a. Subcutaneous tissue - 50%

b. Peripheral tissue- 15%

c. Mesentery- 20%

d. Omentum- 10%

e. Intra muscular connective tissue-05%

Composition of Depot fats:

It is mainly composed of mixed triglycerides, traces of lecithin & cholesterol, as well as a little amount of poly unsaturated fatty acids.

Sources of depot fats: fat depot is derived from the following sources:

a. Food fat- It is the chief source.

b. Carbohydrate- Carbohydrate of food may be easily converted into fat which are more saturated & have a higher melting point.

c. **Proteins-** Recent evidences have shown that carbohydrates derived from proteins may be converted into fat & pyridoxine in collaboration of thiamine; catalyses the formation of fat from protein.

Functions of Depot Fat:

Depot fat provides protection against injury, helps in regulation of body tempreture & storage of energy (1100 cal/kg body wt.)

Plasma Lipids

Plasma lipids mainly composed of:

1. Cholesterol & its esters
2. Triglycerides
3. Phospholipids
4. Non-esterified fatty acids

1. Cholesterol:

It is an unsaturated type of steroid, distributed in brain & suprarenal gland, in largest amount & in muscles according to the degree of activities. Approximately 60-70 % cholesterol is transported by LDL, 20-30% by HDL & 5-10% by VLDL.

2. Triglycerides: They are esters of free fatty acids & glycerol. They are divided into two groups

a. Simple : All three fatty acids are same.

b. Mixed : All three fatty acids are different.

Triglycerides are transported mainly as chylomicrones & VLDL but in minor amounts as LDL & HDL also.

1. **Phospholipids**: It is compound type of lipid, containing phosphoric acid with nitrogen base & constitutes 20 % LDL & 30% HDL.
2. **Non-esterified fatty acids:** They are very small fraction in plasma.

Plasma Lipoproteins

Lipoproteins are classified on the basis of density, which is determined by the amounts of triglyceride (Makes them less dense) & apoproteins (which makes them denser). There are four types of lipoproteins:

A. Chylomicrons

B. LDL

C. VLDL

D. HDL

The least dence particles, known as chylomicrons, are normally present in the blood, only after fat containing foods have been eaten. The densest & smallest particles consists mainly of apoprotein 7 cholesterol & is called High Density Lipoproteins (HDL). Some what less dense are the Low density Lipoproteins (LDL). Least dense are the large, Verry Low- Density Lipoproteins (VLDL), consisting mainly of triglycerides. In fasting blood most of the cholesterol is carried on LDL particles and is therefore reffered to as LDL cholesterol; most of the triglycerides is found in VLDL particles.

Lipid Transport & Metabolism

Most of the fat in the diet except short chain fatty acids are absorbed from the intestine into the lymph. Most triglycerides are split into monoglycerides & fatty acids during the process of digestion. While passing through intestinal mucosa they are resynthesçed into new molecules of triglycerides that enter into lymph as minute disperse droplets called chylomicrons having sçe 0.08 -0.06 mm. Most of the cholesterol, small amounts of phospholipid synthesçed by intestinal mucosa also enter the chylomicrons. Thus chylomicrons are maimly made up of triglycerides but they also contains about 9% phospholipids, 3% cholesterol & 01% apoproteins. Then the chylomicrons are transported to the thoracic duct & empatied into the venous blood. Half an hour after the meals, the chylomicron concentration in plasma may rise to 1-2%.

The Fat of Chylomicrons is removed in two different ways:

A. Hydrolysis of Chylomicron Triglycerides by Lipoprotein lipase: By the action of enzyme lipoprotein lipase, the major part of triglyceride in chylomicron is hydrolysed in to glycerol & fatty acids. Further this glycerol is metabolçed in the same way of glucose, while fatty acids are transported to various cells of the body for resynthesis of triglyceride.

C. Abosorption of whole triglyceride: Chylomicrons are removed from blood by transportation through capillary wall, directly into liver cell, where it is used for energy.

AETIOLOGY OF DYSLIPIDAEMIA

Dyslipidaemia is a disorder of lipoprotein metabolism, which can include both the overproduction & deficiency of lipoprotein. Dyslipidaemia can manifest as the elevation of serum cholesterol, triglycerides or both. It can also be manifasted by the elevation of 'bad' low- density lipoprotein (LDL) cholesterol & the decreased of 'good' high density lipoprotein (HDL) cholesterol in the blood. Most cases of dyslipidaemia are instances of hyperlipidaemia, which is caused by an elevation of lipids in the serum. Hyperlipidaemia often results from dietary & life style choices.. however, the causes of dyslipidaemia can be genetic or secondary.

Primary Dyslipidaemia:

Primary causes of dyslipidaemia are single or multiple gene mutations that lead to overproduction of triglycerides & LDL cholesterol or the overproduction or excessive clearence of HDL cholesterol. The majority of cases dyslipidaemia in adults are not caused by primary disorders. The new classification, clinical features & prevelence of primary dyslipidaemia are given in the table as per AACE lipid guidelines, 2002.

Table No. 13 :Features of Major Genetic Lipoprotein Disorders:

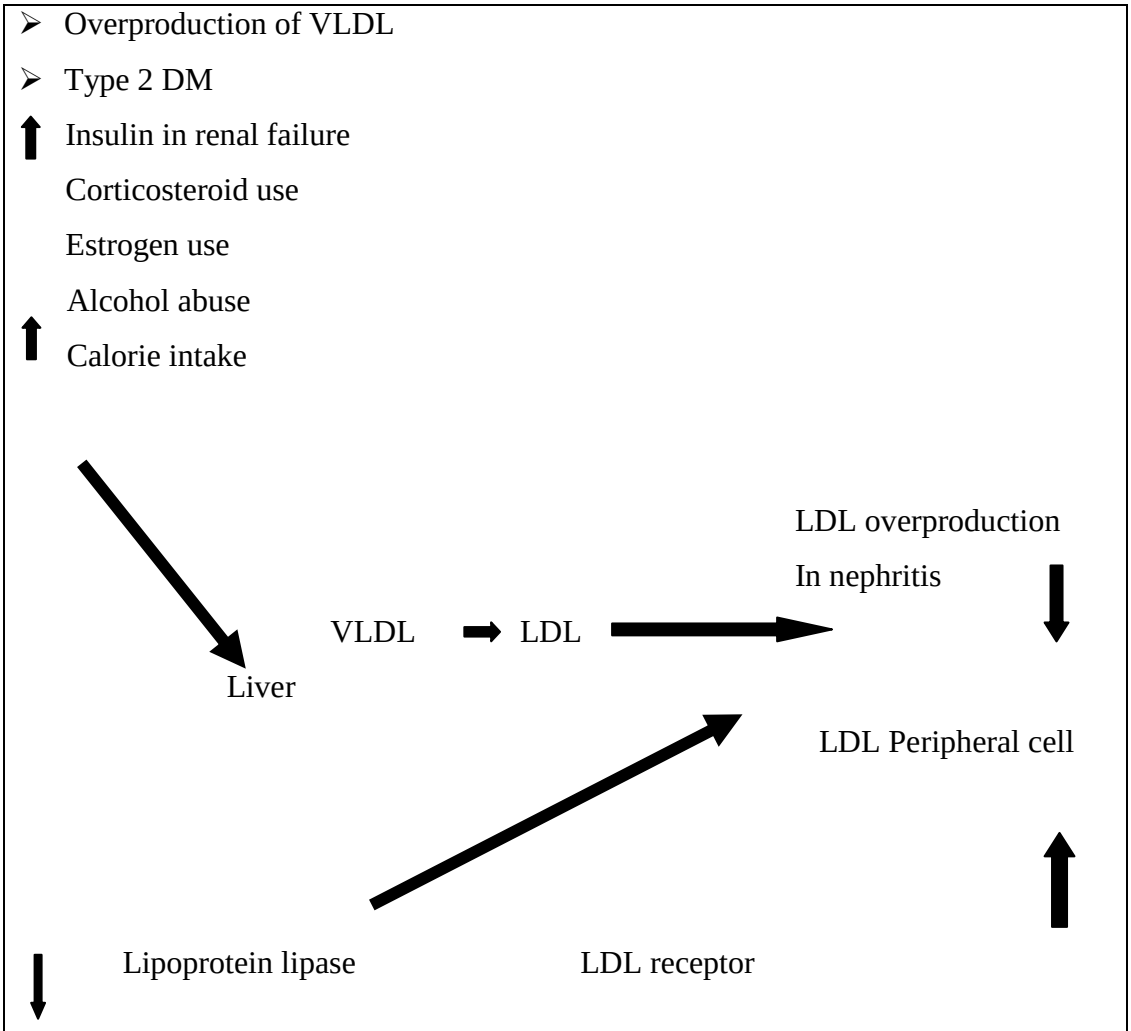
Disorder	**Principal plasma abnormality CorrespondingFredicks on Classification**	**Clinical Features**	**Estimated Frequency**
Heterozygous Familial Hypercholeste Rolemia	↑ LDL only (Inherited abnormality of the LDL receptor) (IIa)	Tendinousxathmous Corneal arcus, Premature CAD Hypercholesterolemi a	0.2 % of general population 5% ofMI survivors <60yr old. Autosomal codominant
Familial Defective Apolipoprotein B	↑ LDL (inherited abnormality of the LDL receptor) (IIa)	Some clinical features as heterozygous hypercholesterolemi a	Same frequency as heterozygous familial hypercholestremia
Familial combined hyperlipidemia	1/3: ↑ LDL only (IIa) 1/3: ↑ VLDL only(I) 1/3: ↑ LDL & VLDL(IIb) Apo-B overproduction is common.	Usually >30yr old often overweight. Usually no xanthomas, premature CAD different generations have different lipoprotein abnormalities	0.5% of general population 15% of MI survivors. <60yrs old.Autosomal dominent.
PolygenicHypercholes terolemia	↑ LDL(IIa)	Premature CAD, no xanthomas, no F/H/o hypercholesterolemi a	Unknown
Familial Hypertriglyceridemia	↑ *VLDL only (highVLDL production,*	Often overweight >30yr old. Often	01%of general population 05 % of

Disorder	Principal plasma abnormality CorrespondingFredicks on Classification	Clinical Features	Estimated Frequency
(200-1000 mg/dl)	*Decreased lipoprotein lipase activity) (IV)*	diabetic hyperuricemic.May or may have not premature CAD. Determined by family history & HDL –C	MI survivors.
Severe Hypertriglyceridemia (>1000 mg/ dl)	↑ *Chylomicrons & VLD* L(high VLDL production, decreased lipoprotein lipase activity (V)	Usually middle aged often obese. Often hyperuricemia. Usually diabetic. Risk of recurrent pancreatitis	Unknown
Familial hyper *α lipiproteinemia*	HDL (<30 mg/ dl) in males, <35 mg /dl in females)(decreased apo-A-I production)	Premature CAD.	01% of general population 25-305 of patients with premature CAD.Autosomal dominent
Dys β – lipoproteinemia (TG-250-600mg /dl, TC-250-500mg/dl)	↑ *LDL*, ↑ *chylomicron*	Yellow palmar crease, palmar xanthomus.Tuberoer uptive xanthomus, premature CAD	Uncommon 03% of MI survivors,Autosoma l recessive

Secondary Dyslipidaemia:

The most common cause of secondary dyslipidaemia in adults is the sedentary life style coupled with excessive intake of saturated fat, cholesterol & trans fats. Other causes of dyslipidaemia includes diabetes mellitus, hypothyroidism , Alcohol abuse & chronic renal disease.

Sycondary dyslipidaemia are more prevelent in the Asciatic population . Common secondary causes of dyslipidaemia & the mechanism by which these conditions or the therapies alter lipid levels are represented in fig. given below:



• Antibodies to lipoprotein - LDL	Gamma globulin binding to Glycosylation of LDL, leading to
• Lipase in dysgammaglobulinemia	decreased receptor binding

• Lipoprotein deficiency due to Hypothyroidism (number of receptors) Insulin deficiency in type 1 DM
• Decreased activity with Estrogen use, Thiazides, α blockers • Decreased T4

Fig.42 Mechanism Of Lipid Alteration In Various Disorders

Table No. 14 – Secondary Causes Of Dyslipidemia

Affected Lipids	**Conditions**
Total Cholesterol & LDL- C ↑	❖ Hypothyroidism ❖ Nephrosis ❖ Progestin or anabolic steroid treatment ❖ Obstructive liver disease due to abnormal lipoproteins,as in pri. Biliary cirrhosis ❖ Anorexia nervosa (hypercholesterolemia occurs as a result of mobilçation of cholesterol from tissue) ❖ Dys gammaglobulinemia (SLE, multiple myeloma)
Total triglycerides & VLDL ↑	❖ Chronic renal failure ❖ Diabetes mellitus –type 2 ❖ Obesity ❖ Excessive Alcohol intake ❖ Hypothyroidism ❖ Oral contraceptives ❖ Pregnancy ❖ Oral estrogens ❖ Corticosteroid therapy ❖ Severe stress (↑ endogenous

	corticosteroids) ❖ Antihypertensive medications(thiazides,α-adernergic blocking agents) ❖ Bile acid sequestrants
Low HDL –C	❖ Sedentary life style ❖ Anabolic steroids ❖ Cigarette smoking ❖ Verry low fat diet ❖ Secondary to hypertriglyceridemia regardless of cause (except of alcohol & estrogen induced hypertriglyceridemia) ❖ MI or major surgery temporarily lower HDL

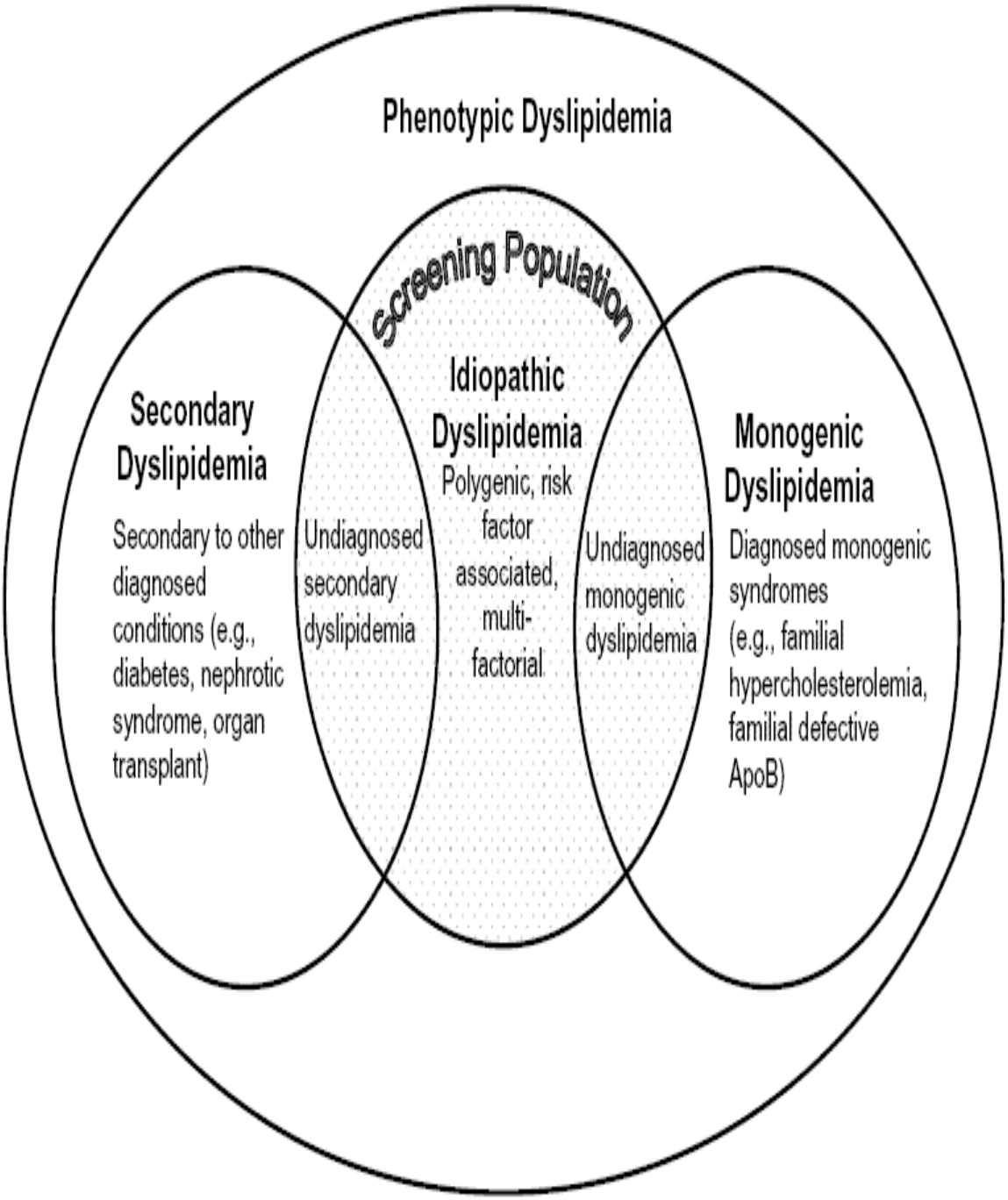


Fig. No. 43- Types of Dyslpidaemia

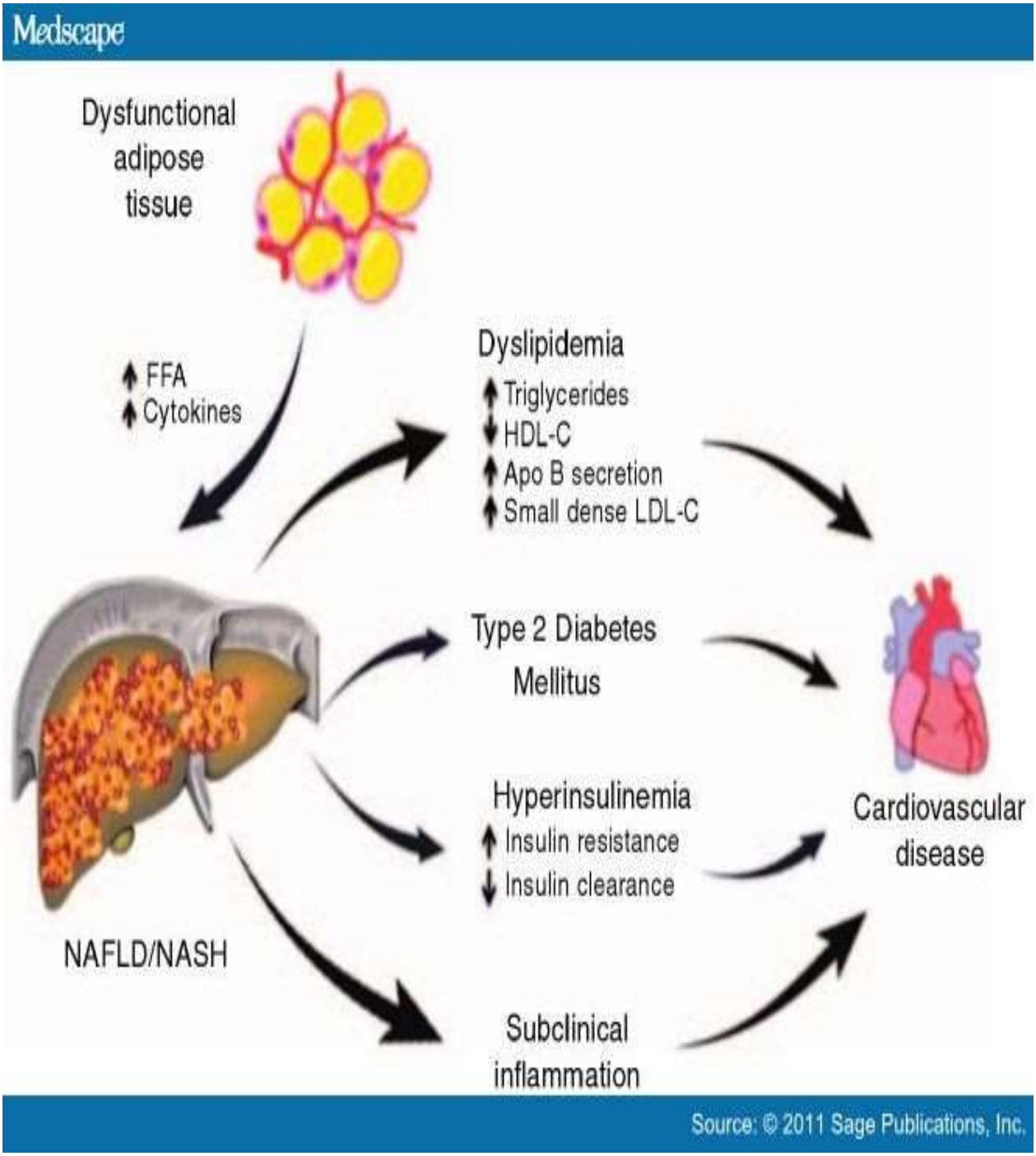


Fig. No. 44-Showing the role of Lipid & Liver in the development of Cardiovascular disorders

Atherosclerosis and Dyslipidaemia

Dyslipidaemia is a major risk factor for atherosclerosis & thus Coronary Artery Disease (CAD). WHO defines atherosclerosis as variable combination of focal accumulation of lipids, complex carbohydrates, blood & its constituents, fibrous

tissues & calcium deposites combined with its changes of media. (API text book of medicine, 2003)

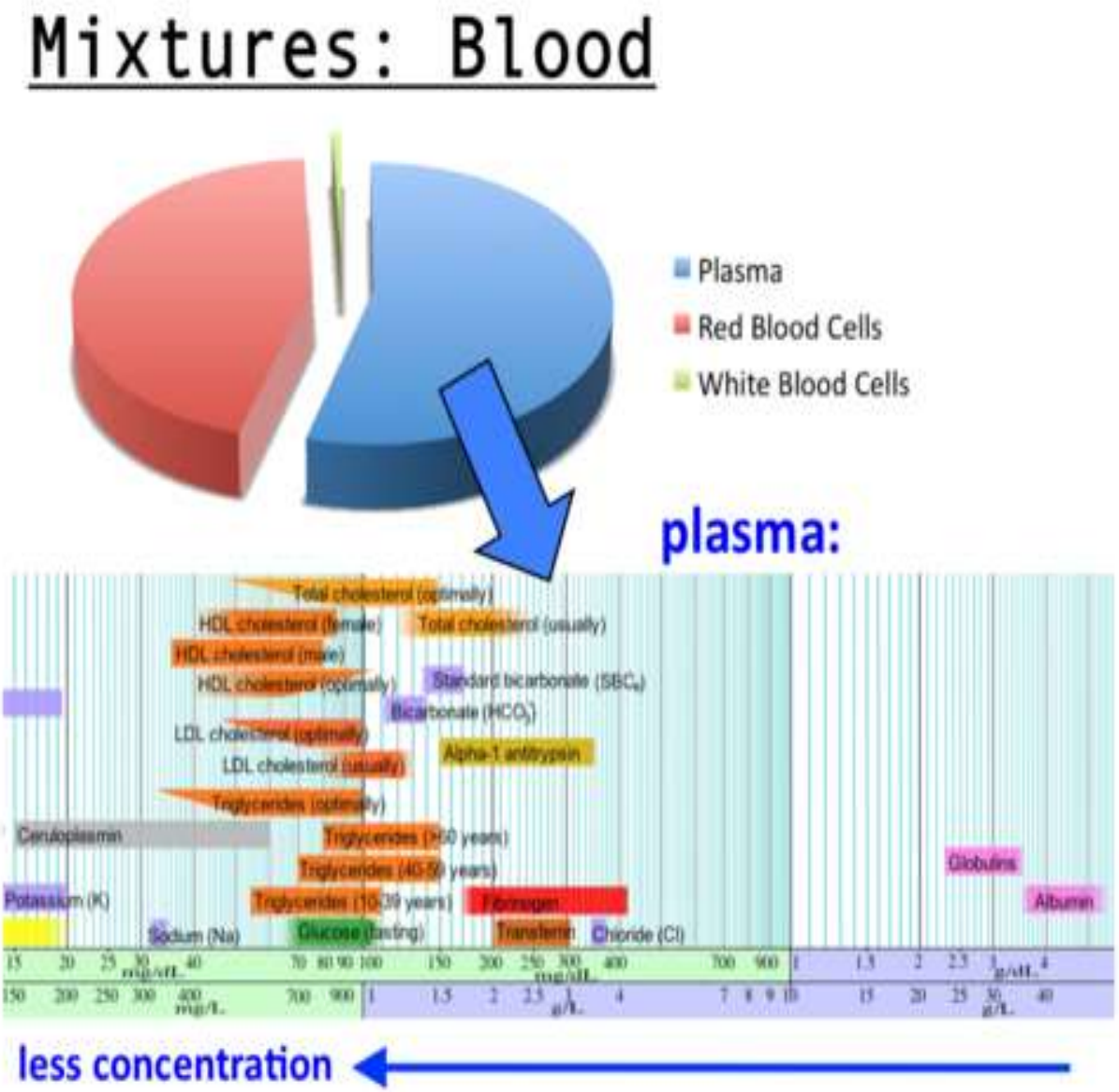


Fig . No. 45-Composition of Blood & Plasma

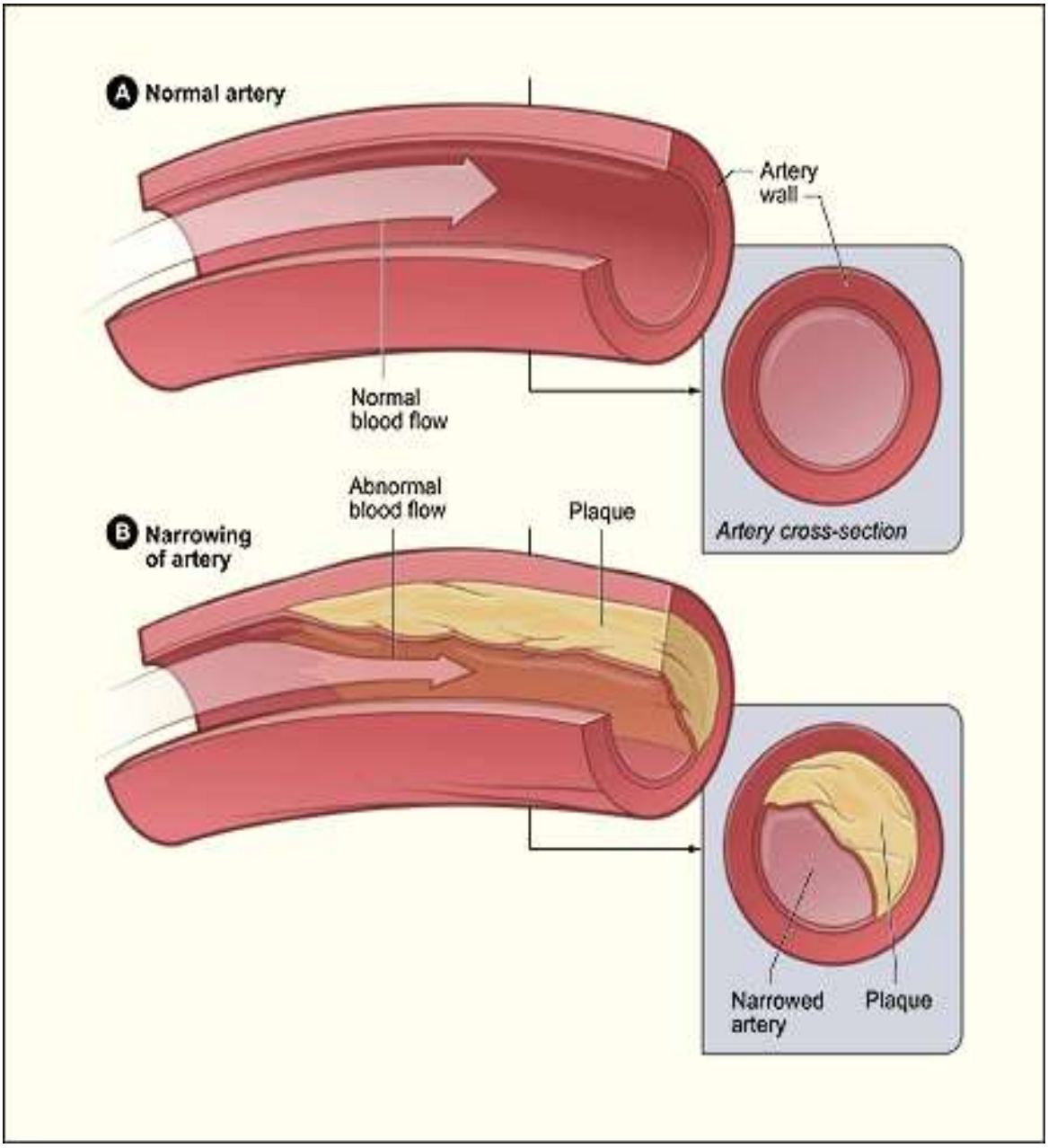


Fig. No. 46-Cross sections of a. normal artery & b. Obstructed artery due to Atherosclerosis

Risk Associated With Atherosclertosis

Atherosclerosis is a multifactorial disease with hypercholesterolemia being a major risk factor in the indian climates. Increased triglycerides with low HDL cholesterol has been found to be atherogenic with low density Lipoprotein (LDL) being the most potent factor.

Atherosclerosis due to dyslipidaemia is an important risk factor for Peripheral Vascular Disease (PVD) as it leads to insulin resistence which in turn results in increased level of serum triglycerides & low density lipoprotein & decreased HDL cholesterol. Patients with common lipipd triad i.e. increased triglycerides, high LDL-C & low LDL- C have a high risk for it. When lipid triad is accompanied by insulin resistence, a precoagulation state & hypertension a condition known as Cardiovascular dysmetabolic Syndrome.

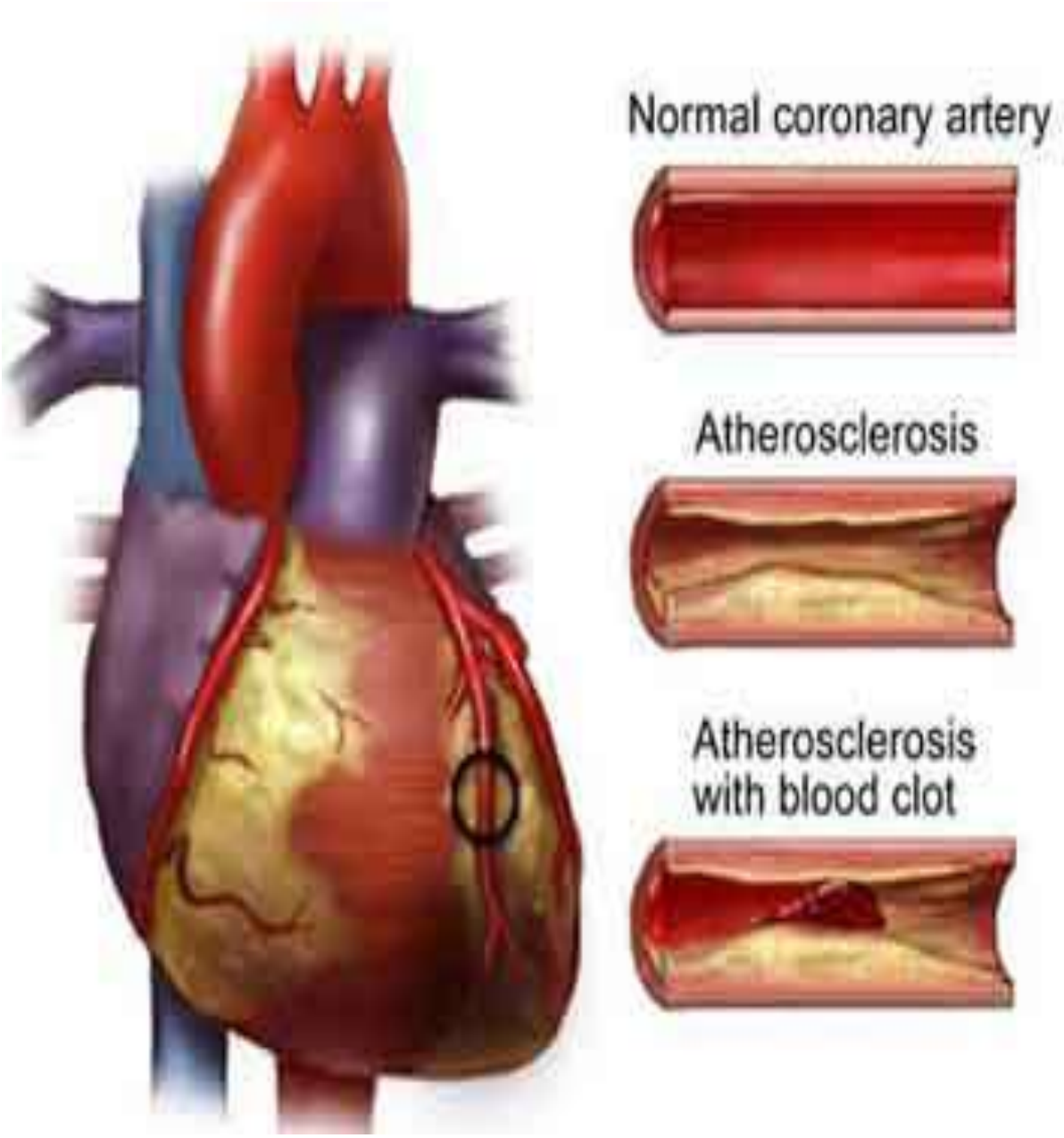


Fig. No. 47- Showing Normal & Constricted artery due to Atherosclerosis

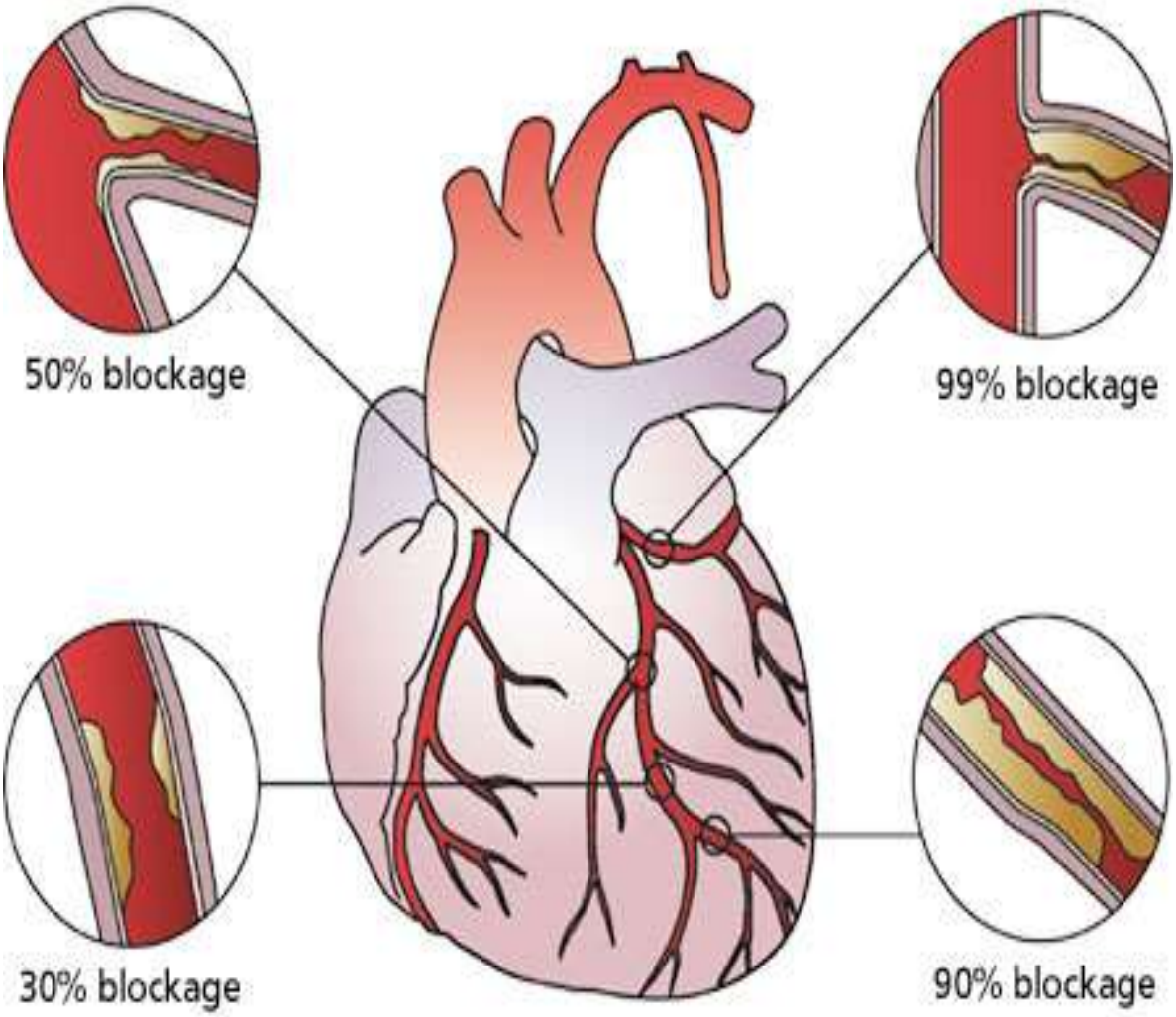


Fig. No. 48- Presentation of Different % Of Blockage in different coronary arteries

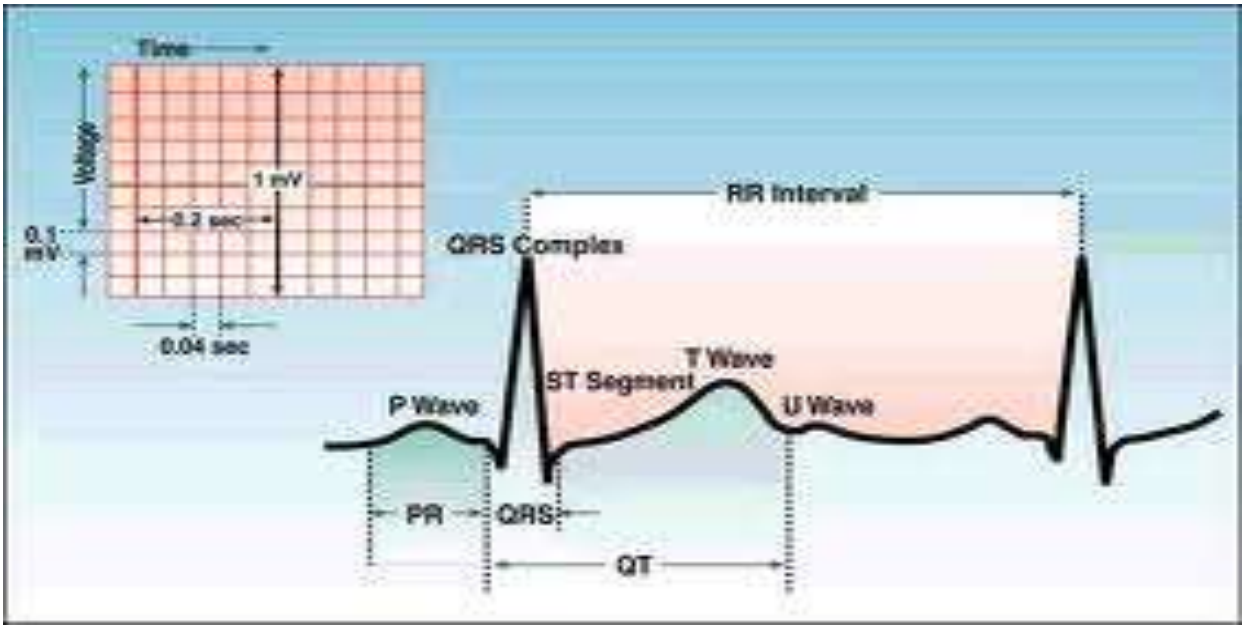


Fig. No. 49-Normal E.C.G. Pattern

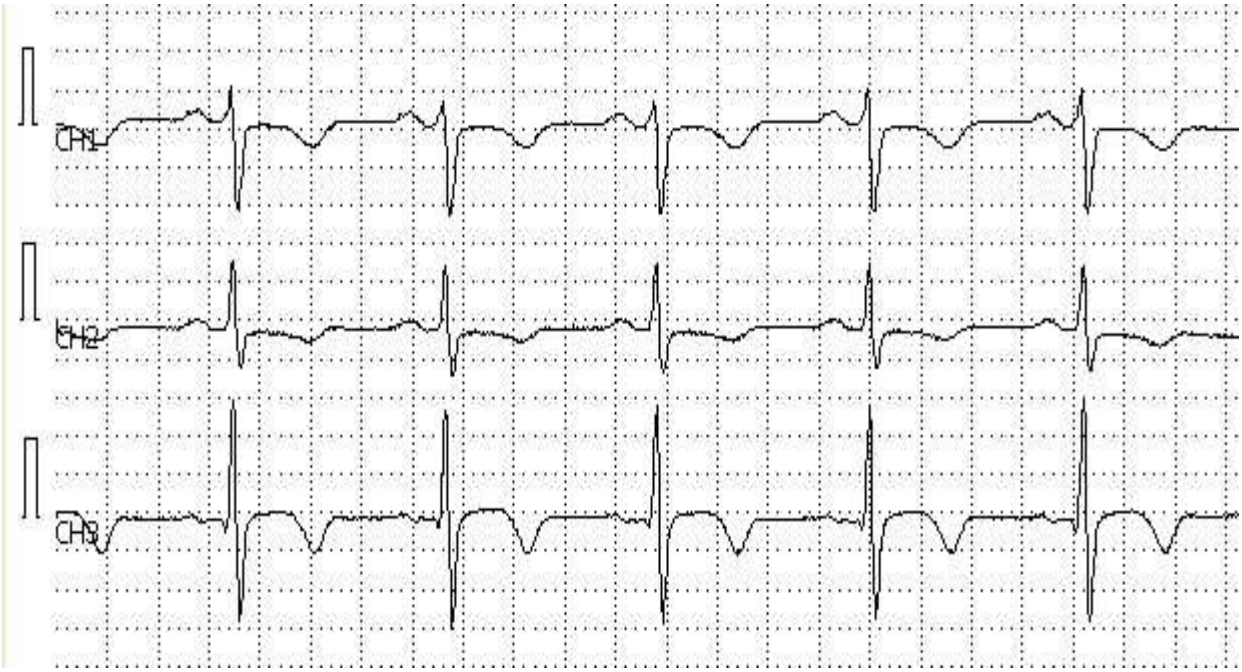


Fig. No. 50-E.C.G. Showing IHD, ST-segment depression & T wave Inversion, rate- Normal Heart

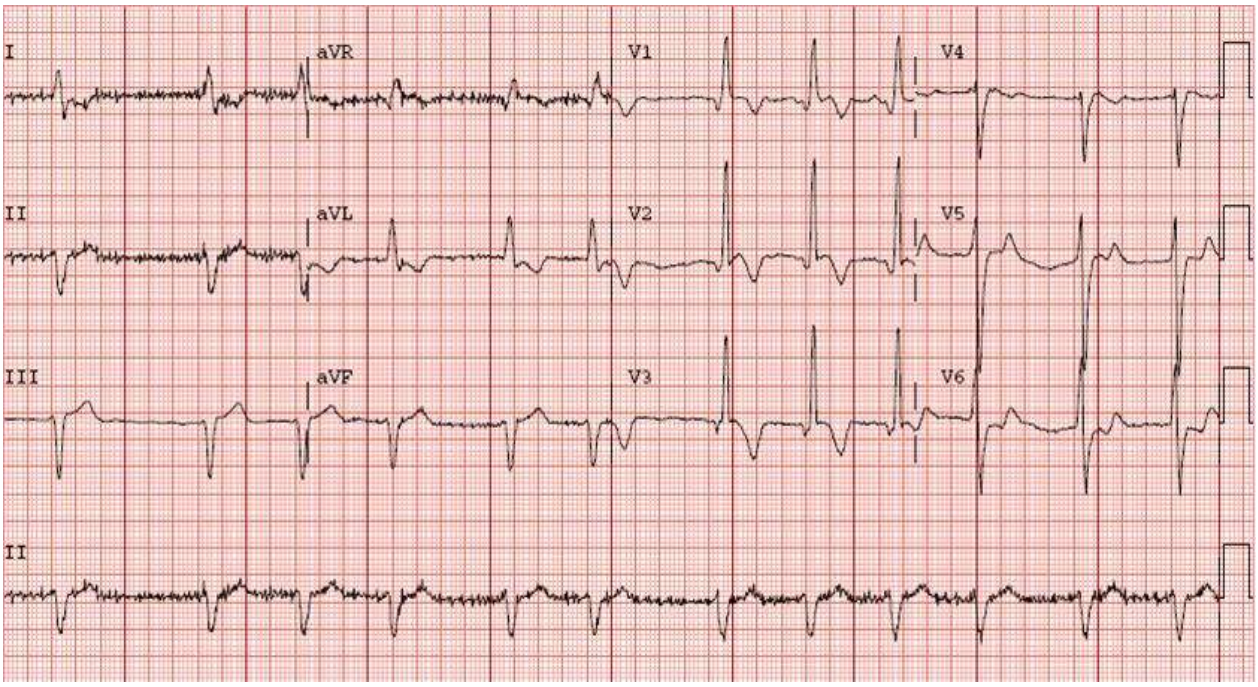


Fig. No. 51-E.C.G. Showing IHD (Ant.wall ischaemia) with RVH(Right Ventricular Hypertrophy) Upright R wave in lead V1& V2, T Wave inversion in leads V1—V4.

Diagnosis and Risk Assesment

Although dyslipidaemia usually doesnot cause symptoms, it can lead to symptomatic vascular disease, which includes coronary artery disease (CAD) & peripheral artery disease. Those with a high concentration of LDL in their blood can experience symptoms such as Xantholesmas on eyelid (yellow/ white growth) & Arcus corneae (an abnormal white/ grey opaque ring at the outer edge of the cornea).

Identification of risk factors enables the physician to start the appropriate therapy for dyslipidaemia to each patients risk level & the thereby maximçe treatment effectiveness.

Step-1 Screening tests:

Lipid Profile i.e.fasting total cholesterol, triglycerides & HDL- C profile is essential to determine the patient at risk. The Framingham study showed that 80 % of patient s with CAD had total cholesterol level s equivalent to those without CAD and high serum HDL – C level may oerestimate total cholesterol level. Hence 12-14 hours fast is essential to avoid the effect of food intake on chylomicron, VLDL &triglycerides. Non fasting profiles that reveal total cholesterol level 200 mg /dl or an HDL- C concentration <35mg /dl or both dictate need for a fasting lipid profile. LDL – C can then be calculated by using Friedewald equation:

LDL - C = Total cholesterol – HDL- C- TG / 5

The results may very ± 10 %. It is valid upto TG levels of 200 mg/ dl in fasting state only. Above this, values become inaccurate.

Fasting TG levels exceeding 250-300 mg /dl:

In such case, direct LDL – C assay may be useful. If it is not feasible, the non HDL – C (total serum cholesterol - HDL – C) can be useful to determine treatment goal.

Fasting TG levels marginally increased (150-200 mg / dl) :

Two additional lipid evaluations may be required, first direct measurement of the LDL & secondarily evaulation of post prandial TG level. A normal post prandial TG reference range has not yet been established. (Summary of NCEP, JAMA, 2002)

Step-2 : Assesment of Lipid Related Risk:

Secondary causes of dyslipidaemia must be ruled out with athrough medical & dietary history as well as laboratory testing for glucose, thyroid, liver & renal fuctions as treatment of underlying contributing disease may alleviate the lipid abnormality. A waist Circumfernce > $0 inches in males and > 36 inches in females (truncal obesity) also predisposes to dyslipidaemia. A 12 - 14 hours fasting TG study exceeding 150 mg / dl should also be considered for cardiovascular dysmetabolic Syndrome.

Non HDL –C Evaluation:

Many patients with cardio dysmetabolic syndrome have increased LDL & VLDL levels. A simple way to estimate risk from LDL & VLDL as well as IDL & Lp (a) in patients with moderate hypertriglycerideemia is to determined HDL – C content. An LDL- C concentration of 130 mg / dl or higher is considered above normal while Non HDL – C concentration of 160 mg / dl or more should raised clinical suspecion.

Patients with CAD & Relatively Normal lipid profile:

Measurement of total plasma apo-B can be useful in the assesment of patient with CAD having relatively normal level of lipids. A high apo-B level (130 mg / dl) & LDL – C < 160 mg / dl with or without increased Tg identify hyper apobetalipoproteinemia, which is the cause of premature CAD.

MANAGEMENT OF DYSLIPIDAEMIA:

The followings are the cornerstone in the dyslipidemia management:

1. Diet therapy
2. Regular aerobic Physical exercise
3. Weight reduction
4. Smoking Cessation
5. Control of Blood Pressure

6. Anticoagulants
7. Management of associated metabolic condition (diabetes mellitus, thyroid function & anaemia etc.)

Dietary & Life Style Modifications:

Modification of life Style including food habits, avoid smoking & alcohol beverages, weight control &regular aerobic exercise, is not only necessary to attain normal lipid level but is the first step in the management of hyperlipidemia.

Frequent snacks & commercially available precooked food (fast foods) & soft drinks (pepsi, coke etc.) should be abandoned as they are rich in fat & cream as well as refined carbohydrates.

Alcohol supplies empty calories, its use should be resticted for a patient of dyslipidemia & in others should not exceed 30 ml /day.

Nutrition therapy should be prescribed for at least 3 - 6 months before started drug therapy, unless the patient is at a high risk. The American Heart Association (AHA) & NCEP have reccomnded step 1 & step 2 diets . Step 1 is reccomnded for healthy population & step 2 for the patients of CAD.

Low fat diets high in fibre (oat, pectin, barely, moderate consumption of alcohol ic beverages & diet containing **2 - 4 gms of fish oils (Omega - 3 fatty acids) have been found to yield cholesterol reduction by 10-15 % & LDL – C levels by 15-20 %. Fish oils were found to decrease triglycerides by 20-25 % or more without sufficient effect on HDL – C.**

A standard step 1 cholesterol lowering diets recommends reducing total fat to 30 % and saturated fat to 10 % of calories. Dietary cholesterol should be limited to 300 mg / day. The step 2 diet further restricts saturated fat to 7 % of calories & dietary cholesterol to 200 mg / day. These diet replace fat, perticularly saturated fat with carbohydrate.

Exercise & weight loss may reduce the LDL cholesterol & increase the HDL cholesterol.

Pharmacological Therapy:

NCEP guidelines recommnded that consideration be given to the use of cholesterol lowering agents if lipid levels remin high after 6 monthes of continuous dietary therapy. The drug therapy may consist of one, two or in cases of very more dyslipidemia, three agents may be used. Currently used lipid lowering agents include Nicotinic acid (niacin), Bile acid sequestrants (resin), Hydroxyl methyl glutaryl co-enzyme A (HMG-Co A) reductase inhibitors (statins) & fibric acids derivatives.

In most patients with hyper cholesterolemia, HMG Co-A reductase inhibitors are the drug of choice because they reduce LDL cholesterol most effectively.

Gemifibrozil (lopid) or nicotinic acid may be latter choices in patients with significant hypertriglyceridemia.

Primary Lipid lowering drugs classes with their effects and main side effects are given in table

Table No. 15 –Primary Lipid Lowering Drugs classes

Drug Class	Metabolic Effect	Side effects
Niacin (Nicotinic Acid)	↓ LDL-C 10-20%, ↓ TG 20-30%, ↑ HDL 10-35% by hepatic synthesis of LDL-C& VLDL-C Lp-C to less atherogenic form.	Deleterious effect on serum glucose at higher doses.Hyperuricemia, Hepatotoxicity,Peptic ulcer, flushing,Ab.discomfort.Only 50-60% of Patients can tolerate in effective dose for Prolonged time.
Bile Acid Sequestrants. (Cholestramine,Colestipol)	Primarily ↓ LDL-C 10-30% by binding acids at intestinal level.	May ↑ TG, GIT troubles, which can reduce compliance. May absorption of folic acid& fat soluble vita. A,D,K
HMG-CoA reductase Inhibitors (Statins)	Primarily ↓ LDL-C 15-45% by competitively inhibiting rate limiting	Monitoring of Liver function required. Muscles Aches & Fatigue in small proportion of

Drug Class	Metabolic Effect	Side effects
	step of cholesterol syn. In liver. ↑ HDL-C 2-12%	proteins.
Fibric Acid derivatives (Gemfibrozil,Fenofibrate)	Primarily ↓ TG 30-55%, HDL-C 15-25% by stimulating lipase activity. Finofibrate may ↓ TG & LDL-C 20-25%. ↓ Both VLDL & LDL, causing reciprocal rise in LDL-C, transform LDL-C into less atherogenic form.	Gemifibrozil may ↑ LDL-C 10-15%. GIT symptoms, Cholelithiasis & myopathy when used with other agents. May potentiate effect of Anticoagulants. Gemfibrozil may fibrinogen level. Gemfibrozil & Fenofibrate can homocystine independent of vita.concentrations.Rhabdomyolosis when used with statin

Table No.16Guidelines For Selection Of Hyperlipidaemic Drugs

Hyperlipidaemia	Monotherapy Drugs	Drug Combination
Elevated LDL & Normal TG	a) Statins b) Bile acid Sequestrants. c) Nicotinic acid d) ERT in women	a) Statins+ Bile acid sequestrants. b) Bile acid sequestrants +niacin. c) Statins + Niacin
ElevatedLDL- C & TG	Statins Gemfibrozil Niacin	
Elevated TG	a) Gemfibrozil b) Niacin	

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