CHAPTER 1

INTRODUCTION AND REVIEW OF LITERATURE

Energy is an integral part of a human society. It plays an important role in socio-economic development. As the population increases, the energy demand also increases to fulfil the daily needs. Approximately 70% of the country’s whooping population lives in rural areas. There is a tremendous demand on resources such as fuel wood, agricultural residues, etc. to meet the daily fuel requirements. In many developing countries biomass is the major source of energy, nearly accounting for 30% of the country’s energy use (Ramachandra and Kamakshi, 2005). This component is much higher in rural areas. Biomass fuels serve 85-90% of the domestic energy demands and 75% of the rural energy demand (Natarajan, 1985).

1.1 State of India’s environments: The rapidly burgeoning population, with increased economic development, rapid industrialization and urbanization has burdened India’s infrastructure, and its environment. Deforestation, soil erosion, air & water pollution and land degradation continue to get worse (EIA, 2001). Apart from these, there are other environmental issues e.g. improper availability of potable water and energy related environmental problems etc. (Countrywatch, 2001).

1.2 Population and Poverty: The population of the world has grown from 6.134x10^9 in 2001 to approximately 7x10^9 in 2011. India has grown from approximately 1.027 x10^9 in 2001 to 1.21x10^9 in 2011. The population
growth can be caused by a decrease in the death rate, with no fall in birth rates (Fig. 1.1). This happens because many developing countries have adopted western medical technology, which reduces death from contagious and other diseases (Reidhead et al., 1998).

**Fig. 1.1-** Trends in birth and death rates (Registrar general 1991).

On the basis of URP-Consumption (URP-Consumption= Uniform recall period consumption in which the consumer expenditure data for all the items are collected from 30-day recall period) the Planning Commission of India has estimated that 27.5% (301.72 x10^6) of the population (all India) was living below the poverty line in 2004–2005. The source for this was the 61st round of the National Sample Survey and the criterion used was monthly per capita consumption expenditure (all India) below ₹356.30 for rural areas and ₹538.60 for urban areas. In the rural areas of India the monthly per capita consumption expenditure was ranged from below ₹292.95 to ₹478.02. In the same survey it was estimated that 33.4% of rural population and 30.6% urban populations were living below the poverty line. In Uttar
Pradesh the monthly per capita consumption expenditure ranged from below ₹ 365.84 for rural areas and below ₹ 483.26 for urban areas (Government of India, 2007).

Poverty and non affordability of clean fuel have forced the low socioeconomic people to fulfil the energy demands from low grade fuel i.e. biomass fuel.

1.3 Classification of fuels: The fuels used in rural areas for domestic cooking and heating are divided into two groups:

1. Solid fuels: Solid fuels contain coal, biomass fuels (also known as traditional biomass fuels [TBFs]). TBF is subdivided again into two classes-(i) agricultural waste-consists of wood, crop-residue (cotton stalks, oil seed stalks, rice and wheat straw, bagasse, sugarcane leaves, coir-pith, ground nut shell / coconut husk, coffee husk, bark), and (ii) animal waste includes animal dung, cattle manure, poultry litter etc.

2. Nonsolid fuels: It consists of kerosene, liquefied petroleum gas (LPG), electricity etc.

The sources of energy and fuels can also be divided into renewable and nonrenewable. Renewable sources of energy are derived from sources that are essentially inexhaustible, such as solar energy, wind, and biomass fuels. Nonrenewable source of energy includes fossil fuels, such as petroleum and nuclear energy. Biomass contains the stored energy from the sun through photosynthesis. This energy in plants gets passed on to animals who eat them. When biomass is burned, the chemical energy is released as heat. Biomass also contains minerals, which are incorporated into the plant during their growth. When they are burnt, some are lost as volatiles into the environment.

Biomass can be converted into methane gas (CH₄) also called "landfill gas" or "biogas" (main ingredient of natural gas). It is also converted into
transportation fuels like ethanol (by fermentation of corn and sugar cane) and biodiesel from food products (vegetable oils and animal fats).

World Energy Council (WEC), the Intergovernmental Panel on Climate Change (IPCC), Shell and the Stockholm Environmental Institute (SEI), indicate that the biomass has the potential to make a large contribution to the world's energy supply. Recently there has been a renewed interest in biomass as a commercial and sustainable source of energy (Faaïj, 1997). Currently, the commercial use of biomass to generate electricity is limited mainly to the utilization of low-cost biomass waste or residues. The biofuel is also tested in airlines. Recently Brazilian renewable jet fuel was tested in Azul flight (http://www.blackseagrains.net/data/news/brazilian-renewable-jet-fuel-tested-in-azul-flight dated 21.07.2012) down loaded on 27.07.2014. “Azul Brazilian Airlines (ABA) hosted a demonstration jet flight using fuel made from locally-sourced sugarcane recently”). Soon the biofuel will be used in other flights, too.

Approximately 80% of all national fuel combustion occurs under cooking pots in many poorest countries (Smith et al., 2000b). Approximately half of the world's population and mainly 90% of rural household in developing countries rely on traditional biomass fuels (WRI, 1998; Reddy and Williams, 1997; Bhattachayra and Salam, 2002; Mulaudzi, 2006; Rahman, 2010; World Energy Outlook, 2006). Nation-wide about 78% of the population relied upon the traditional biomass fuels (NFHS, 1995; Rahman, 2010). According to census, 75% of household energy consumption is accounted for by these TBFs (Census, 2001) because the majorities of population lives in rural areas and subsist on agriculture. In the energy ladder bio mass fuels like animal dung, crop residue etc. occupied lowest runk. Such types of
biomass use are greater among the class V people i.e. antyodaya group (poorer among the poor) of population.

In most developing countries, wood fuel and biomass are burnt in inefficient traditional mud stoves (efficiency ~20%) in poorly ventilated kitchens by women who also shoulder the responsibility for biomass collection and formation of dung cake. Most of the fuel wood used in rural areas is collected from common lands, panchayat land, privately land etc. Mostly women, adolescent girls and children transport wood and other biomass fuels on their head.

India is the second largest consumer of biomass fuels in Asia and uses the most animal waste (in absolute terms). According to 1996-97 information, total TBF consumption was 538 MT/yr. Out of this rural consumption was 54.46%. In a rough estimate the dung cake consumption was 121 MT/yr. Out of 121 MT, Uttar Pradesh alone contributing 40 MT/yr (Fig. 1.2) (Reddy and Venkataraman, 2002).

**Fig. 1.2-** Biomass burning estimates for India, for 1996–1997.
Reports from rural India indicate that biomass fuels are the major source of cooking-energy and fulfil about 85-90% energy demand (Kumar A, 2009) of which wood accounts for 56%, crop residues for 16%, and dung cakes 21% (TEDDY, 1998-1999). The rest of energy uses is divided among biogas (1.6%), kerosene (1.3%), LPG (1.3%) and coal (1.4%) (Fig. 1.3) (TERI, 2002).

**Fig. 1.3-** Energy use in the rural domestic sector in India.

The 13% (largest part) of bio-fuel consumption is from Uttar Pradesh. The contribution of dung cake and fuelwood is 7% and 4% respectively followed by Andhra Pradesh (11%, fuelwood-5%; crop waste-4%);, Bihar (10%, fuel wood- 7%; dung-cake- 1%) and M.P. (9%, fuel wood- 7%; dung-cake- 1%) (Reddy and Venkataraman, 2002).

In another estimate, in U.P. the consumption of fuel wood, crop residue and animal waste, were estimated to be 303.3 PJ, 82.7PJ and 475.4 PJ (petajoule-10¹⁵Joules) respectively aggregating 861.3 PJ consumption of biomass fuel (Streets and Waldhoff, 1999). The monthly per capita consumption of firewood increased from 16 to 17 kg over the period 1987/88 to 1993/94. The use of firewood in the form of logs has tripled in 15 years as shown in Fig. 1.4 (Natarajan, 1997).
1.3.1 Consumption of other fuels: Petroleum products like LPG consumption were around 8-9% in rural areas as compared to 62% in urban areas (NSSO, 2010). Hence the large imports of petroleum products only marginally benefit the rural population. Similarly is the case with the Kerosene. Nearly 62% of rural household uses kerosene for lighting purposes and only 1.3% use it for cooking (NSSO, 2010). About 65% of the households in electrified villages do not receive the benefits of electricity. The result is that about 70-80x10^6 households depend upon kerosene lamps for lighting (http://iis-db.stanford.edu/evnts/3920/REHMAN_data_ppr.pdf). The total kerosene consumption in India during 2000/01 was estimated to be at around 10.5x10^6 tonnes out of which about 60% was for the rural areas (TERI, 2002).

LPG is used mainly for cooking. Since 1985, the consumption of LPG has grown from over 100x10^6 tonnes in 1985 to over 6000x10^6 tonnes in 1999. The number of LPG customers up to 01.01,2000 was 43.6x10^6 while in rural areas it was 1% of the total households (MoPNG, 2000). Due to non-availability of traditional fuels, and LPG, being a convenient, the government of India has decided to supply the LPG in subsized rate on rural areas.
1.3.2 Chemistry: The TBFs are composed of complex organic matters, vegetable protein and carbohydrate incorporating carbon, nitrogen, hydrogen etc. (UNEP, 1980). All biomass has the same elemental composition. They contain carbon (C), Hydrogen (H) and Oxygen (O). Cellulose constitutes 40-45% of the mass of dry wood. Hemicelluloses are bound to the cellulose skeleton as a matrix substance and make up 20-35% of dry wood mass (Parham and Gray, 1984). The lignin is also present in wood. Cellulose, hemicelluloses and lignin are collectively called lignocellulose (Pettersen, 1984).

1.3.3 Constituents of wood smoke particles: The compounds emitted during wood combustion are aromatic hydrocarbons, furans, aromatic carbonyls, phenol and substituted phenols, n-Alkanes, branched alkanes, n-Alkenes, branched alkenes, cycloalkanes, cycloalkenes, aliphatic alcohols, aliphatic ketones, ester, amine etc (Wu et al., 2002). Almost a similar composition was found by Khalil et al. (1983) and McDonald et al. (2000). Apart from these, Schauer et al. (2001) also found guaiacol and substituted guaiacols, syringol and substituted syringols, alkanoic acids, resin acids, and levoglucosan. Plant sterols, n-alkanols, and some sugars are also found in wood smoke particles (Nolte et al., 2001). Percent wise, the particles generation by biomass burning were particulate organic material (POM) with associated organic matter (e.g. H, N, and O) 80%, black carbon 5 to 9% and trace inorganic species 12 to 15%.

Smith et al. (1983) and Aggarwal et al. (1982) also found the presence of BaP exposures during the cooking time in four villages in India. It was equivalent to smoking nearly 20 packs of cigarettes/day. This indicates that the burning of solid fuels in open stoves exposes the subjects to various harmful pollutants. Previous studies have shown that the level of carboxyhemoglobin (COHb) was more (2.5-13%) among people exposed to
biomass smoke (Rahman, 2010). COHb level should be less than 2.5% (WHO, 1999).

1.3.4 Fundamentals of particle formation: Smoke particles are composed of about 50–60% organic carbon and about 5–10% black carbon. Chemistry of biomass-burning combustion has been discussed by many authors (Tillman, 1981; Lobert and Warnatz, 1996; Simoneit, 2002). Approximately 90% of the carbon emitted during the burning of traditional biomass which is oxidized to carbon di oxide (CO$_2$) / (carbon mono oxide (CO) and carbon (<5%) being released as particulate matter (Radke et al., 1991; Ferek et al., 1998; Andreae et al., 1988). The particle formation process in flames begins with the creation of condensation nuclei such as polycyclic aromatic hydrocarbons (PAH) and a variety “soot-like” species (Glassman, 1977; Frenklach, 2002). As the PAH molecules grow, these particles become condensation nuclei for other pyrolyzed species, and may experience considerable growth. Subsequently, many of these particles may reduce in size through further oxidation in the interior of the flame zone (Glassman, 1977). Improper availability of oxygen into the flame leads to secondary condensation growth phase of many particles and is emitted in the form of smoke.

Smoldering combustion is a surface process which begins when most of the volatiles have been expelled from the cellulose fuel. Oxygen diffuses to the surface and reacts exothermally with carbon. If temperatures are increased, carbon mono oxide (CO) can convert into carbon di oxide (CO$_2$). The above processes show that it is the condensation process which forms particles during the biomass burning (Radke et al., 1991; Reid and Hobbs, 1998).
1.4 STUDIES ON HEALTH EFFECTS

According to World Bank report, the indoor air pollution is responsible for almost 50% of the burden of total diseases resulting from poor household environments in developing countries (World Bank, 1993).

Reliance on TBF for domestic cooking and heating houses expose many women, infants and young children including adolescent females in developing countries. Epidemiological studies revealed that TBF smoke exposure has a risk factor for the development of respiratory diseases, adverse pregnancy outcomes, ocular diseases, cardiac diseases etc. (Murry and Lopez, 1996; Smith, 1987).

The health effects on the exposed subjects depend upon the type of fuel used, ventilation, types of stove, time spend during cooking, area of cooking space etc. Climatic conditions also influence the exposure to emission of TBF. The major categories of health problems associated with exposure and biomass fuel smoke are divided in two major groups:

1.4.1 Respiratory illness:
   1.4.1.1 in children
   1.4.1.2 in adults

1.4.2 Non-respiratory illness:
   1.4.2.1 in children
   1.4.2.2 in adults

1.4.1.1 Respiratory illness in children:

1.4.1.1.1 Acute lower respiratory tract infection (ALRI): ALRI is a common acute infection in children. Young children, living in households exposed to smoke of TBF, have 2 to 3 times greater risk of developing ALRI
as compared to those living in households using cleaner fuel (Smith et al., 2000a). Acute respiratory infection (ARI) is one of the chief killers of children in the developing countries and a major cause of death in the world. Six per cent disease and mortality was accounted for worldwide due to ARI and mostly in the developing countries. ALRI are one of the most important causes of death globally in children under five years (Von Schirnding, 2000). In children under 5 years, the mortality attributable to ALRIs is approximately $2 \times 10^6$ per year (Black et al., 2003; Murray et al., 2001; Rudan et al., 2004). Globally approximately $1.5 \times 10^6$ deaths occur annually from respiratory infections and are attributable to the environment. It includes at least 42% of lower respiratory infections and 24% of upper respiratory infections in developing countries. A study of Europe revealed that 4.6% of all deaths due to ALRI and 3.1% of all DALYs (Disability adjusted life year) in children aged 0-4 were attributable to the indoor air pollution from solid fuel use alone (Valent et al., 2004). According to Smith (2000), ARI is the largest disease for India, accounting for 8.5% of the global burden.

Thomas et al. (1999) reported that, on average, 14% of households had children (<6 years) who were usually present when their mothers were cooking with polluting fuels. It was found that exposure to indoor air pollution more than doubles the risk of pneumonia (Muller et al., 2003). Similarly Burki (2011) found the link of indoor air pollution to pneumonia. Globally, pneumonia and other ALRI are responsible for about $\frac{1}{3}$ of all deaths among children under 5 years of age in developing countries (Bruce et al., 2000; WHO, 2002). The infants/ young children are at the greatest risk of serious respiratory illnesses because of incomplete development of lungs and immune system (Koenig, 2000; Braga et al., 2001). These children are also more susceptible to environmental toxicants because the dose of inhaled pollutant per unit body weight is likely to be greater (Cerna et al., 1998;
Gilliland et al., 1999). It is known that exposure to CO impairs the oxygen-carrying capacity of the blood while PM$_{10}$, PM$_{2.5}$, PAH, volatile organic compounds (VOC), nitrogen di oxide (NO$_2$) and sulphur di oxide (SO$_2$) cause the inflammation of the airways and lungs and impair the immune response (Patterson et al., 2002; Naheer et al., 2007; Fortoul et al. 2011).

According to Smith (2000a), many studies have been conducted including India that give quantitative estimates of the relative risk of severe ARI for children living in biomass-burning households. Studies conducted in Nepal (Pandey, 1989), Zimbabwe (Collings, 1990), Nigeria (Johnson, 1992), and Gambia (Armstrong and Campbell, 1991) document very interesting findings. Data from Guatemala have suggested that symptoms of wheeze are more frequent amongst households that use an open fire compared with a stove with a chimney (Schei et al., 2004).

Many studies in developing countries have reported on the association between exposure to indoor air pollution and ALRI (Robin, 1996; Armstrong and Campbell, 1991). A case control study denotes that solid fuel use for domestic cooking (OR 3.97) and poor economic status (OR 4.95) were associated with high-risk pneumonia (Mahalannabis et al., 2002). Indoor cooking smoke associated with childhood pneumonia and bronchiolitis was reported in Nigeria (Sofoluwe, 1968). Similar findings were reported by Ezzati and Kammen (2001) from Kenya. Chakravathy et al. (2002) reported 18% asthma among children below 12 years of age. Studies conducted in Kenya, Malaysia and China have shown the childhood asthma because of indoor pollution from solid fuel use (Mohamed et al., 1995; Azizi et al., 1995; Xu et al., 1996a).

1.4.1.1.2 Deterioration in lung function in children: Data from Ecuador reveal the deterioration in lung function among children exposed to BMF
A review from China has shown deterioration of FEV$_1$, FVC and peak flow among children associated with domestic coal use (Zhang and Smith, 2007).

1.4.1.2 Respiratory illness in adults:

1.4.1.2.1 Chronic obstructive pulmonary disease (COPD). The prevalence of COPD is very high in India (Vishwanathan and Singh, 1977). Studies from Nepal and Northern India have defined the problem of chronic bronchitis and COPD and the role of biomass fuel combustion (Master, 1974). In a meta-analysis, Hu et al. (2010) concluded that exposure to biomass fuel is a risk factor for COPD. Symptoms of chronic respiratory problems among Indian women cooking with open biomass stove have been reported by Malik (1985) and Gupta (1997). Smith (2000) reported 1.8% of the national burden of disease for women in India.

BMF smoke is also responsible for COPD in the non-smoking women of rural areas (Ezzati, 2005; Orozco-Levi et al., 2006; Smith et al., 2004). In an estimate among the rural women of Turkey the COPD attributed to exposure to BMF smoke was 23.1% (Ekici et al., 2005). Malik and Behera (1989) also reported that chronic bronchitis and respiratory symptoms were very common among the non-smoking women exposed to domestic fuels during cooking on 'Chullah'.

In a study in Nepal, Pandey (1984a) reported that the high level of domestic air pollution was responsible for chronic lung diseases. Similarly studies from other countries have reported on the association between exposure to biomass smoke and chronic bronchitis or COPD (Qureshi, 1994; Dutt, 1996; Ellegard, 1996; Norboo et al., 1991b; Perez-Padilla et al., 1996).
Based on NFHS-2 (1999) (after controlling all confounders) the effect of cooking smoke reveals that the risk of asthma in elderly of above 60 years was 1.59 times (women 1.83 and men 1.32 times) among rural household who use biomass fuel for cooking. Numbers of peoples of village of Chuchot Shamuna (Leh) were suffering from chronic bronchitis probably due to exposure to the burning of wood and dried yak dung (Misra, 2003).

1.4.1.2.2 Cor pulmonale: Cor pulmonale is the failure of the right side of the heart caused by prolonged high blood pressure in the pulmonary artery and right ventricle of the heart. It is a serious heart condition. There was a close relation between exposure to indoor air pollution and chronic obstructive lung disease causing corpulmonale (Padmavati and Pathak 1959; Padmavati and Joshi, 1964). Padmawati and Joshi (1964) attributed the prevalence of chronic bronchitis and chronic cor pulmonale among women in rural India exposed to BMF emission during domestic cooking. Similar observations were reported by Pandey (1984b) and Pandey et al. (1988) in Nepal. The chronic cor pulmonale was much more common in the northern states of India as compared to the southern states. This higher prevalence of chronic cor pulmonale in women was due to exposure to domestic air pollution. It is because the BMF is used by very poor people. In winter the rural women are forced to prepare the meals inside the house and also use BMF for room/space heating. Because of these two reasons the exposure period is increased leading to chronic bronchitis. If heavy exposure continued, the emphysema might develop and result into cor pulmonale. In south there is no winter and so women are not forced to prepare the meal inside the house and room/space heating is not required so exposure period is reduced. Because of this reason the prevalence of chronic cor pulmonale is lesser in southern
state. Malik (1985) also reported chronic bronchitis associated to exposure to domestic cooking fuels.

1.4.1.2.3 **Interstitial lung diseases:** Interstitial lung disease due to BMF smoke is also called ‘hut lung’ (Gold et al., 2000; Grobbelaar and Bateman, 1991). It is a form of pneumoconiosis. It is described as ‘Transkei silicosis’. It develops due to the presence of silica particles. Silicosis also has been reported and attributed to such exposures, usually in association with soil dust (Saiyed et al., 1991; Norboo et al., 1991a).

1.4.1.2.4 **Pulmonary tuberculosis in women:** There are many evidences which show that the incidence of TB is increased amongst the BMF exposed women. On the basis of an analysis of the data of Indian adults during the 1992-1993 NFHS, Mishra et al (1999a) reported the association between the use of biomass fuels and pulmonary tuberculosis. Persons exposed to biomass fuels smoke were reported to have higher prevalence of tuberculosis (odds ratio [OR] = 3.56; 95% confidence interval [CI] = 2.82-4.50) than persons living in households that use cleaner fuels, with an adjustment of confounding factors. The prevalence of tuberculosis attributed to cooking smoke was 51% among people of 20 years’ age and above (Mishra et al., 1999a). Gupta et al (1997) have reported a similar finding from the nearby area of Lucknow with an adjustment of confounding factor for age. Chaudhury and Thatte (2003) reported the prevalence of Indian tuberculosis 138x10^5 while the world average is 59.7x10^5.

In a rough estimate in 2005, more than 3.2x10^6 people were living with tuberculosis in India. In India one person dies every minute due to tuberculosis (Country Profile: India, 2008). It is known that BMF smoke impairs alveolar macrophage function (Aam and Fonnum, 2007; Arredouani
et al., 2006; Zhou and Kobzik, 2007). Alveolar macrophages are not only the target of *Mycobacterium tuberculosis* infection but also contribute an important early defense mechanism against bacteria. So it is thought that BMF smoke also leads to increased incidence of tuberculosis.

Experimental studies show that the exposure to wood smoke may lead to lung infection (Houtmeyers, 1999). Thomas and Zelikoff (1999) in animal studies showed that exposure to wood smoke altered local and systemic immune response associated with bacterial infection.

1.4.1.2.5 Lung Cancer: Lung cancer is well known among Chinese women cooking on open coal stove (Smith and Liu 1994; Mumford et al., 1995; Xu et al., 1995; Xu et al., 1996b; Luo et al., 1996; Shen et al., 1996; Dai et al., 1996; Wang et al., 1996). In studies from India and Mexico, non-smoking women exposed to BMF smoke for long time may develop adenocarcinoma of lung (Behera and Balamugesh, 2005; Hernandez-Garduño et al., 2004). Gupta (1998) reported about 67% of lung cancers among non-smoking women in India. The presence of pulmonary tuberculosis is a risk factor for development of lung cancer (Wu, 1995). PAH, formaldehyde etc. present in BMF smoke are well known for their mutagenic and carcinogenic activities. Smith et al. (1983) found average BaP exposures during the cooking period in four villages in western India was nearly 4000 ng/m³ which is equivalent to smoking about 20 packs of cigarettes per day. The exposure to suspended particulate matter reached over 55000 μg/ m³ and averaged about 7000 μg/ m³ while WHO recommended maximum 100-150 μg/ m³ in 24-hour. Sobue (1990) in Japan have established an association of biomass fuel use and cancer (OR=1.8. Aggarwal et.al. (1982) conducted a study at Ahmedabad (India) and found similar levels of BaP and SPM concentration near cooking stoves. He concluded that houses where solid fuels are burnt in open stove
contribute high concentration of harmful chemical/ pollutants in the environment.
Animal studies show that the chemical presents in BMF smoke are known for causing cancer (Cooper, 1980). In a study conducted in Kenya, it was thought that nasopharyngeal cancer might be due to exposure to biomass smoke (Clifford, 1972). In Kenya, carcinoma of nasopharynx occurs more among people in the highland where cooking is done indoor than people of the hotter areas where food is prepared out of door (Clifford, 1965). Analysis of indoor air samples from Kikuya village hut of Kenya had showed the elevated concentration of carcinogenic hydrocarbons i.e. BaP etc. (Hoffman and Wynder, 1971).
Hong Kong study showed that the relationship to inhaled emissions from cooking stove has higher relatively risk (2.21) of bronchial irritation among those with a past history of using wood burning stoves. Behera and Balamugesh (2005) in a study show that the risk of development of lung cancer was highest among non-smokers exposed to BMF [OR= 5.333 (95% CI:1.700-16.731)]. Similar studies conducted in Brazil have shown a strong relationship with upper aerodigestive tract cancers and use of wood stove [OR=2.68 (95% CI:2.2-3.3)] (Pintos et al.,1998).
The International Agency for Research on Cancer (IARC) recently termed the biomass smoke a ‘probable carcinogen’ (Group 2a) to humans (Straif et al., 2006).

1.4.1.2.6 Deterioration in lung function in adults: Malik and Behera, (1989) reported in their study that the ventilatory abnormalities were more marked in rural subjects exposed to BMF smoke during cooking on 'Chullah'. Davidson et al. (1981) reported impairment in ventilatory capacity among rural women using such fuel. Behera et al. (1994) also show the lung
function impairment due to indoor air pollution caused by domestic cooking, more so with biomass fuel.

1.4.1.2.7 Other respiratory problems: The study conducted by National Institute of Occupational Health (NIOH) Ahmedabad, India shows the increased prevalence of cough, cough with expectoration and dyspnoea among women exposed to BMF smoke and kerosene. Roentgenic studies have showed 18% abnormalities among the women exposed to BMF smoke while it was 5.8% among the kerosene users. Long-term exposure to biomass fuel smoke may damage the lung because of inflammation, possibly leading to hypoxia and elevated erythropoietic response (Samet and Tielsch, 2007).

1.4.2 Non respiratory illness:

1.4.2.1 Non respiratory illness in children: There are many studies which have linked the tobacco smoke and ambient air pollution to adverse pregnancy outcomes. BMF has nearly the same pollutants found in the tobacco smoke and ambient air. Evidence shows that the exposure to BMF smoke has adverse effects on different birth outcomes (Sram et al., 2005) e.g. low birth weight, premature birth, intrauterine mortality, congenital abnormalities/ birth defects, and infant mortality and neonatal deaths etc.

1.4.2.1.1 Low birth weight (LBW): LBW is defined as a birth weight of <2,500 g. LBW is an important public health problem in developing countries. It has strong relationship between birth weight and infant mortality and morbidity. Studies showed that baby less than 2,500 g at birth are at greater risk of neonatal mortality. Maternal risks e.g. demographic risks (poor socio economic status, low level of education/ illiteracy); medical
risks in pregnancy (anemia), behavioral and environmental risks (poor nutritional status) are also associated with delivering low birth weight baby (Institute of Medicine 1985; Brown, 1985). All the risks mentioned above are associated with BMF smoke exposure directly or indirectly. An association was made by Boy et al., (2002) between the birth weight and the type of fuel used in Guatemala. The open fire produced average levels of PM$_{10}$ of 1000 µg/m$^3$. In rural Guatemala, the babies of mothers using open wood fires were on average 63 g lighter (p=0.05; 95% CI, 0.4-126) as compared to the babies of mothers using cleaner fuels. Levels of CO in the houses using biomass fuels for cooking were high enough. Mean 24-hour values CO and mean of CO during the use of fire were in the range 5–10 ppm and 20–50 ppm or more respectively (Dary et al., 1981; Norboo et al., 1991b; Naheer et al., 1996). The COHb levels were between 1.5% and 2.5% (Dary et al., 1981) and rising up to 13% (Behera et al., 1988). Such high levels of COHb have detrimental effects upon the fetal growth. A correlation between the maternal exposure to CO and low birth weight has been reported by Lewtas (2007) and Townsend and Maynard (2002). Rinne et al. (2007) showed a significant trend for a higher infant mortality among the household women who cooked with biomass fuel (P =0.008). Pereira et al (1998) showed the association between the levels of COHb in cord blood and ambient CO levels in children delivered by the non-smoking women. Wang et al. (1997) reported an increasing exposure-response relationship between SO$_2$ and TSP levels and LBW. Bobak and Leon (1999) found an association between pollutants with LBW [OR 1.10 (1.01 to 1.20), p=0.033] in the Czech Republic. There are many other studies and reviews that have evaluated links between air pollution and birth weight/ various birth outcomes. (Ritz and Yu, 1999; Roger et al., 2000; Maisonet et al., 2001; Ha et al., 2001; Lee et al., 2003; Gouveia et al., 2004; Basu et al., 2004; Parker
et al., 2005; Wilhelm and Ritz, 2005; AAP Committee, 2004; Maisonet et al., 2004; Sram et al., 2005).

Impact of PAH and fine particles on pregnancy outcome were reported by Dejmek et al. (2000). Dejmek et al. (1999) also reported the effect of particulate matter during pregnancy on fetal growth. They established the relationship between intrauterine growth retardation (IUGR) and exposure to pm \( \leq 10 \mu m \) and pm \( \leq 2.5 \mu m \) in early pregnancy at Teplice. Exposure-response relationship was also established. For each 10 ng increase of c-PAHs in the first gestational months (GM), the AOR was 1.22 (CI, 1.07-1.39). The study reveals that exposure to c-PAHs in early gestation may impair fetal growth. Similarly Jedrychowski et al. (2004) in a study find that high personal exposure to fine particles is associated with adverse effects on the developing fetus.

1.4.2.1.2 Premature birth/preterm baby: Very limited number of studies were conducted to find out the effects of air pollution on preterm baby (birth before the 37th week of gestation). BMF have many pollutants which have detrimental affects upon fetus resulting in prematurity. Bobak M (2000) analysed singleton (premature) live births registered by the Czech national birth register in 1991. Prevalence (4.8%) of prematurity (<37 weeks of gestation) were more associated with SO\(_2\) than TSP. The effects on prematurity were stronger in the first trimester. The AORs of prematurity were 1.27 (CI, 1.16-1.39) and 1.18 (CI, 1.05-1.31) for a 50 \( \mu g/m^3 \) increase in SO\(_2\) and TSP respectively. The findings of Bobak (2000) support the hypothesis that air pollution may increase the risk of adverse birth outcomes. In a cohort of 97,518 neonates Ritz et al. (2000) evaluated the effect of air pollution during pregnancy. The study shows 20% increase in preterm birth per 50-\( \mu g \) increase in ambient PM\(_{10}\) levels and a 16% increase when
averaging over the first month of pregnancy. He concluded that exposure to increased levels of ambient PM$_{10}$ during pregnancy may result in preterm births. Sagiv et al. (2005) also observed the effects of air pollution and preterm birth.

1.4.2.1.3 **Intrauterine mortality:** Intrauterine mortality includes missed abortion/ miscarriage and stillbirth. The association between intrauterine mortality and air pollution was studied by Pereira et al (1998) in Brazil. The association between intrauterine mortality and air pollution was strong for NO$_2$ (p<0.01) as compared to SO$_2$ (p<0.10) and CO (p<0.10). Another study conducted in Ahmedabad (India) reported a risk of 50% stillbirth [OR 1.5 (1.0–2.1)] among women using biomass fuel during pregnancy (Mavalankar, 1991).

To examine the association between the household use of biomass fuel, tobacco smoke and risk of still birth, Mishra et al. (2005) analysed a study and found the effects of cooking smoke and tobacco smoke on the likelihood of having a stillbirth. The findings indicate that women who cook with BMF are more likely to have experienced a stillbirth and the number of still birth were 2 or more as compared to those who cook with clean fuel [(OR=1.44; 95%CI: 1.04, 1.97) and (OR=2.01; 95%CI:1.11,3.62) respectively]. Similarly Mattison (2010) summarized studies indicating the relationship between paternal and maternal environmental exposures to chemicals to adverse fetal developmental outcomes.

1.4.2.1.4 **Congenital abnormalities/ birth defects:** A few studies are available on birth defects. Ritz et al. (2002) evaluated the effect of air pollution on the occurrence of birth defects. Odds ratios for ventricular septal defects increased with increasing CO exposure in 2$^{nd}$ month of pregnancy.
In a study Marshall et al. (2010) compared exposure to ambient air pollutants (CO, NO₂, Ozone, SO₂, PM\(_{10}\) in aerodynamic diameter and PM\(_{2.5}\) in aerodynamic diameter) during early pregnancy among mothers of children having oral cleft defects (cases) to that among mothers of controls. He found that the cleft palate showed limited evidence of an association with increasing SO₂ exposure.

1.4.2.1.5 Neonatal and infant mortality: Death of new born between the first and the 28th days of life is called neonatal deaths. Death of baby between the 28th days and one year of life is called infant death. There are strong evidences that air pollution is a risk factor for increased mortality in infants and young children. Lin et al. (2004) show association between air pollutants and neonatal deaths. Chay and Greenstone (2003) found interesting findings during recession time. During recession there was reduction in air pollution (especially TSPs). Reduction in air pollution results in 0.35% reduction in the infant mortality. In a recent study Son et al. (2011) investigated the association between long-term exposure to different sizes of particles and infant mortality. Their study supported the hypothesis that long-term exposure to PM during pregnancy increases the risk of infant mortality. Lipfert et al. (2000) in an analysis mentioned that air pollution has been one of the suspected risk factors for sudden infant death syndrome (SIDS), and other causes of infant mortality. Woodruff et al (1997 and 2006) in their study show that infant mortality in the USA showed excess perinatal mortality associated with higher PM\(_{10}\) levels for the high pollution group (mean 44.5 µg/m\(^3\)) versus the low pollution group (mean 23.6 µg/m\(^3\)). In infants of normal birth weight, high exposure group was associated with SIDS [(OR=1.26 (1.14–1.39)]. They concluded that air pollution PM effect on post neonatal infant mortality due to respiratory problems (Woodruff et
al. 1997 and 2006). Similarly a study conducted in Mexico City shows the relationship between fine particles and the infant mortality The strongest effect was with PM$_{2.5}$ at 3–5 days before death, when an increase of 10 µg/m$^3$ was associated with a 6.9% (95% CI: 2.5–11.3) excess increase in infant mortality (Loomis et al. 1999). Many others studies and reviews concluded that PM exposures are associated with intrauterine developmental effects and post neonatal mortality (Ha et al., 2003; Glinianaia et al., 2004; Lacasana et al., 2005; AAP committee, 2004; Maisonet et al., 2004; Dales et al., 2004; Sram et al., 2005; Tong and Colditz, 2004).

1.4.2.1.6 **Nutritional deficiency:** A report of Mishra and Retherford (2007) suggested that exposure to BMF smoke in young children contributes to nutritional deficiencies including anaemia and stunted growth in India.

1.4.2.2 **Non-respiratory illnesses in adult:**

1.4.2.2.1 **Cataract in women:** India has a larger burden of blindness. About 33% cataracts occur in India. The cataracts are responsible for 80% blindness in country (Thylefors et al, 1995). In a study conducted by Mohan et al. (1989) in Delhi, they found an excess cataract risk (about 80%) among people using biomass fuel (OR-1.6). The 58th round data from the NSSO (2002) revealed that, of all the disabled individuals in India, 10.88% were blind and 4.39% had low vision. Animal experiments show that cataracts can be caused by wood smoke (Shalini, 1994; Rao, 1995). It is believed that pollutants present in biomass smoke absorbed systematically and accumulate in the lens. Absorption of toxins into lens lead to oxidative changes and damage the protein of lens. The damaged protein aggregates and precipitates resulting into opacity of lens i.e. cataract (Bruce et al., 2000; Mishra et al.,
Siwitabau (1978) showed the complaints of sore inflamed eyes in women who were exposed to wood smoke.

1.4.2.2.2 Effects on endocrine system: According to European Workshop on the Impact of Endocrine Disrupters on Human Health and Wildlife (1996), there are some chemicals and pollutants in the environments which affect human health. Exposure to such chemicals and pollutants with steroid-like activities can disrupt normal endocrine function leading to the various types of reproductive impairments. (McLachlan, 1993; Colborn, 1995; Jensen et al., 1995; Safe, 1995; Vom Saal, 1995; Wu et al., 2002).

1.4.2.3 Reproductive health impairments:

1.4.2.3.1 Impairments in menstrual cycle: There are various types of menstrual impairments e.g. delay in menarche, changes in length of cycle, changes in duration of cycle/bleeding duration (menses phase), variations in amount of flow etc. The causes of irregular menstrual cycle are mostly related to stress, underweight, poor nutrition, use of certain medication, chronic illness like tuberculosis, gynecological problems (polycystic ovarian syndrome, inflammation of the uterus, cervical tumors etc), miscarriage/still birth etc.

Cigarette smoking is also associated with irregular menstrual cycles. Smoking shortens the follicular phase. Heavy smoking also shortens luteal phase. An excess intake of alcohol is also responsible for impairment in menstrual cycle. Endocrine disrupting chemicals which are present in emissions of biomass fuel may alter the hormonal chemistry resulting in the impairment of the menstrual cycle.

There are two types of irregularity in menstrual cycle: long term and short term. In long-term irregularities changes occur in length from month to
month. It may manifest as heavy bleeding, no cycle for a few months, very painful periods etc. Short-term irregularity may be due to fatigue, stress, and over exercise etc. There are various terms which are used in menstrual impairment

<table>
<thead>
<tr>
<th>Terms</th>
<th>Interval</th>
<th>Duration</th>
<th>Amount flow</th>
</tr>
</thead>
<tbody>
<tr>
<td>Menorrhagia</td>
<td>Regular</td>
<td>Prolong</td>
<td>Excess</td>
</tr>
<tr>
<td>Metrorrhagia</td>
<td>Irregular</td>
<td>±Prolong</td>
<td>Normal</td>
</tr>
<tr>
<td>Menometrorrhagia</td>
<td>Irregular</td>
<td>Prolong</td>
<td>Excess</td>
</tr>
<tr>
<td>Hypermenorrhea</td>
<td>Regular</td>
<td>Normal</td>
<td>Excess</td>
</tr>
<tr>
<td>Hypomenorrhea</td>
<td>Regular</td>
<td>Normal or less</td>
<td>Less</td>
</tr>
<tr>
<td>Oligomenorrhea</td>
<td>Irregular or infrequent</td>
<td>Variable</td>
<td>Scanty</td>
</tr>
<tr>
<td>Amenorrhea</td>
<td>Absent</td>
<td>No menses for 90 days</td>
<td>Absent</td>
</tr>
</tbody>
</table>

1.4.2.2.3.2 Menopause: Menopause is a normal phenomenon. It is defined as the permanent cessation of menses due to loss of ovarian follicular activity. Menopause generally occurs during the age of late 40s or early 50s. Menopause can be declared in an adult woman when there has been absence of menstruation for one complete year provided there should be no pregnancy, no lactation and no surgical removal of uterus. Early menopause may be due to illnesses, chemotherapy, radiation and the surgical removal of the uterus and/or both ovaries. Exposure to chemicals and pollutants with steroid-like activities can disrupt normal endocrine function leading to various reproductive problems (Wu et al., 2002).

During climacteric (climacteric is the phase of warning ovarian activity, and may begin 2-3 years before menopause and continue for 2-5 years after it), ovarian follicle declines, ovulation does not occur and there is no formation of corpus luteum and progesterone is not secreted by the ovary. Development of graafian fallicles does not occur. There is reduction in
oestrogenic activity, and endomaterial atrophy. These changes lead to amenorrhoea. In the Western world the average age for the last period is 51 years (Kato et al., 1998). In India and the Philippines, the median age of natural menopause is about at 44 years (Ringa, 2000). Smoking women experience the menopause earlier than the non-smokers (Kaufman et al., 1980).

### 1.4.2.2.3.3 Premature menopause:
Menopause setting before the age of 40 is known as premature menopause. Some times ovaries stop working at early age. This is known as premature ovarian failure (POF). The POF may be due to autoimmune disorders, thyroid diseases, chemotherapy, indoor air pollution and radiotherapy. POF is confirmed by high blood levels of follicle stimulating hormone (FSH) and luteinizing hormone (LH) on at least 3 occasions at least 4 weeks apart (Kalantaridou et al., 1998).

### 1.4.2.2.3.4 The hormonal context of menopause:
The menopause transition is characterized by marked variations in FSH and estradiol levels. The stages of the menopause transition have been classified according to a woman’s reported bleeding pattern, supported by changes in the pituitary FSH levels.

During normal menstrual cycle the ovaries produce estradiol, testosterone and progesterone in a cyclical pattern. It is under the control of FSH and LH. Blood estradiol levels generally remain unchanged. Some time the blood estradiol levels may increase before the menopause. It may be due to in response to elevated FSH levels. Menopause depends upon the cessation of estradiol and progesterone production by the ovaries. After menopause, estrogen continues to be produced by bone, blood vessels and brain also. The sudden fall in estradiol during menopause, the levels of total and free
testosterone, androstenedione, dehydroepiandrosterone sulfate (DHEAS) decline more or less steadily with age (Davison et al., 2005).

1.4.2.2.3.5 Perimenopause: The time around menopause is called perimenopause. It is the transitional period from normal menstrual periods to no menstrual periods at all. Perimenopause is defined as the time from which menses start to become irregular and FSH levels have increased, through until 12 months after the last menstrual bleed. During perimenopause phase production of ovarian hormone becomes irregular and fluctuates causing a large number of symptoms and reduction in fertility level. Women realise physical changes due to hormonal fluctuations. The most common symptom is the "hot flash". In hot flash there is sudden and rapid rise in body temperature for a small period followed by weakness and heavy sweating. Some times women may suffer with depression, anxiety, irritability, disturbed sleep, lack of concentration etc. Some women also feel crawling, itching, or tingling skin sensations. Many of the symptoms disappear with the disappearance of perimenopause transition period.

Due to skipped ovulations, the woman has irregular menstrual cycle. The timing of the flow becomes uncertain, the duration of the flow may be shorter or longer, and the flow may be heavy, light or spotting. Some times periods disappear for 2-3 months even up 6 months followed by a heavier period. When a woman of menopausal age had no periods or even spotting for 12 months, she is supposed to be one year into post-menopause.

1.4.2.2.3.6 Miscarriage: Miscarriage means the loss of an embryo or fetus before the 20th week of pregnancy. It is also called intrauterine fetal death (IUFT)/fetal demise. It is the most common complication of early pregnancy (Petrozz and Inna, 2006; Kabyemela, 2007). Approximately 20% of
pregnancies end in miscarriage and mostly occur during the 14 weeks of pregnancy. It may be due to chromosomal abnormalities or birth defects which make impossible for the baby to survive. They probably occur during the development of the egg or sperm.

In about 17% of cases, miscarriage is caused by abnormal hormonal imbalance. This abnormal hormonal imbalance interferes with the uterus to support the growing embryo. This is known as luteal phase defect. In some cases, the problem may be with the structure of the uterus or cervix. Smoking, indoor air pollution, infection, exposure to toxins, multiple pregnancy, poorly-controlled diabetes etc. increase the risk of miscarriage.

The most common symptom of miscarriage is bleeding from the vagina in the first 3 months of pregnancy. During miscarriage the uterus attempts to push out the pregnancy tissue. A woman may feel cramps in the abdomen. If both the symptoms are present, the chance of miscarriage is more likely. Some time fever and chills may occur.

1.4.2.2.3.7 Stillbirth (also called sudden antenatal death syndrome or SADS): Loss of pregnancy after the 20th week in which the baby passes away before birth. Many stillbirths occur at full term. The exact causes of large percentage of stillbirths are not known. There are many causes which can precipitate the problem of stillbirth e.g. bacterial infection, birth defects (pulmonary hypoplasia), chromosomal aberrations, growth retardation, maternal diabetes, hypertension, consumption of alcohol/nicotine, use of contraindicated drugs in pregnancy, preeclampsia, postdate pregnancy, physical trauma, Rh disease, indoor air pollution, umbilical cord accidents (Collins et al. 2009; 2010) etc.
1.5 ANATOMY OF THE RESPIRATORY SYSTEM

The respiratory system has two parts:

1. Upper respiratory tract, and
2. Lower respiratory tract

The upper respiratory tract extends from the external nares to the junction of the larynx and trachea while the lower respiratory tract extends from the laryngotracheal junction to the distal alveoli (Parkes, 1994).

1.5.1 Nose and the nasal cavity: This is an organ for smelling. It consists of two parts: an outer, the external nose, which projects from the center of the face; and an internal, the nasal cavity, which is divided by a septum into right and left nasal chambers. The external nose is pyramidal in form, and its root is connected directly with the forehead. Its free angle is termed the apex. Its base is perforated by two elliptical orifices called nares. The nares are separated from each other by an antero-posterior septum, known as columna. The margins of the nares are provided with a number of hairs called vibrissae. Vibrissae arrests the entry of foreign substances carried with the current of air. The upper part of the dorsum is supported by the nasal bones. The lateral surface ends below in the ala nasi. The frame of the external nose is composed of bones and cartilages. It is covered by the integument, and lined with mucous membrane. The nasolacrimal duct opens into inferior meatus.

1.5.2 Pharynx: The pharynx is the part of the digestive tube. It is placed behind the nasal cavities, mouth, and larynx. It is 12.5 cm long fibro muscular chamber. It is attached above to the base of skull and continuously below with oesophagus. In front it communicates nasal, oral and laryngeal cavities. It is sub divisible into three parts upper (nasal), middle (oral), and lower (laryngeal) parts.
1.5.3 **Larynx:** It is an organ of phonation, an air passage, extends from the root of the tongue to the trachea. It open above into laryngeal part of pharynx and below with the trachea. It is lined with mucous membrane. The skeletal framework of larynx is formed by cartilages, connected by ligaments and membranes. The chief cartridges of the larynx are:

1. The thyroid cartilage
2. The cricoid cartilages
3. The arytenoid cartilages
4. The corniculate cartilages
5. The cuneiform cartilage
6. The cartilage of epiglottis

1.5.4 **Trachea:** The trachea is a cartilaginous, membranous tube, about 11 cm long and diameter of the index finger (2 to 2.5 cm). It extends from the level of the 6th cervical vertebra to the upper border of the 5th thoracic vertebra. At this level it bifurcates into two bronchus, right bronchus (for right lung) and left bronchus (for left lung). The trachea is divided into cervical part and thoracic parts. The cervical part of trachea is covered with the skin, superficial and deep fasciae anteriorly while the thoracic part descends through the superior mediastinum up to its bifercation (Fig. 1.5 and 1.6).

1.5.4.1 **Structure of trachea:** The trachea consists of 16-20 incomplete rings of hyaline cartilages, fibrous tissue, muscular fibers, mucous membrane, and glands. Hyaline cartilages are placed horizontally one above another, and are separated by small spaces. They are highly elastic. In extra-pulmonary bronchi the cartilages are shorter and narrower than those of the trachea. The first and the last tracheal cartilages differ from each other. The
first cartilage is broader and divided at one end while the last cartilage is thick and broad in the middle (Fig. 1.5 and 1.6).

**Fig. 1.5**- Diagram of the respiratory system (Grant’s method of Anatomy 9th ed)

![Diagram of the respiratory system](image)

**Fig. 1.6**- Cartilage and ligaments of the larynx, side view (Grant’s method of Anatomy 9th ed).

![Cartilage and ligaments of the larynx](image)
**Fibrous membrane:** The fibrous membrane is double-layered, and covers the cartilages. At the end the membrane forms a single layer.

**Muscular tissue:** It consists of two layers of non-striated muscle; external longitudinal fibers consist of a few scattered bundles and single thin-layered internal transverse fibers which extend transversely between the ends of the cartilages.

**Mucous Membrane:** It is continuous with the larynx above, and extends below with that of the bronchi. It is made of areolar, lymphoid tissue, and basement membrane. A layer of longitudinal elastic fibers and small amount of areolar tissue are present below the basement membrane. The submucous layer of connective tissue contains large blood vessels, nerves, and mucous glands.

**1.5.5 The lungs** (Fig. 1.5, 1.7, 1.8, 1.9, 1.10): The lungs are organs of respiration. They are dark and slaty-gray, 2 in number and conical in shape. Each lung is situated on either side within the thorax and is separated from each other by the heart and the contents of mediastinum. The surface of lungs is smooth, shining and marked out into numerous polyhedral areas, indicating the lobules of the lungs. The weight of the right lung and the left lung are about 625gm and 565gm respectively. The lungs are heavier in male than in the female. The apex of lung extends into the root of the neck. The base is broad and concave. It rests upon the convex surface of diaphragm.

**1.5.5.1 Surfaces:** 1. Costal surface is smooth, convex and corresponds to the form of the cavity of the chest. It is in contact with the costal pleura. Mild grooves are present because of the overlying ribs. 2. Mediastinal surface is inner surface of the lung and is in contact with the mediastinal pleura. It has a deep concavity called cardiac impression, which
accommodates the pericardium. It is larger and deeper on the left lung. Above and behind this concavity there is a triangular depression called hilum, where the structures forming root of the lung enter and leave the viscus. On the right lung above the hilus is an arched furrow. This furrow is produced by the azygos vein. Behind the hilus and the attachment of the pulmonary ligament there is a vertical groove (for esophagus). On the left lung above the hilus, there is a curved furrow of the aortic arch, and toward the apex is a groove of left subclavian artery. Behind the hilus and pulmonary ligament there is a vertical groove produced by the descending aorta (Fig. 1.8, 1.9).

1.5.5.2 Borders: The inferior border is sharp and thin. It separates the base from the costal surface. It also divides the base from the mediastinal surface. The posterior border is broad and rounded, and is received at the deep concavity of the vertebral column on either side. It is much longer than the anterior border. The anterior border is thin and sharp, and overlaps the front of the pericardium. The anterior border of the right lung is almost vertical.

1.5.5.3 Fissures and lobes of the lungs: The left lung is divided into two lobes by an inter-lobular fissure, an upper one and a lower one. The superior lobe lies above and in front of this fissure. It includes apex, the anterior border, a part of the costal surface and mediastinal surface of the lung. The inferior lobe is situated below and behind the fissure, and comprises base, costal surface, and posterior border.

The right lung is divided into three lobes by two interlobular fissures. These are superior, middle, and inferior lobes. One of these 2 fissures separate the inferior lobe from the middle and superior lobes. Another fissure separates the superior lobe from the middle lobe.
1.5.5.4 **The Root of the lung:** The root connects lungs to the heart and the trachea. The chief structures composing the root of each lung are arranged from before backward on both sides, viz., the upper of the two pulmonary veins (front); the pulmonary artery (middle); and the bronchus with the bronchial vessels (behind).

1.5.5.6 **Divisions of the bronchi**

1.5.5.6.1 **Right bronchus:** Right bronchus is wider, smaller and more vertical than the left, and enters the right lung at the level of 5\(^{th}\) thoracic vertebra. After commencing about 2 cm it gives off a branch to the upper lobe of the right lung. It arises above the right pulmonary artery (eparterial branch) then passes below the artery (hyparterial branch) and is divided into two branches. One for middle lobe and another for lower lobe and descends giving of bronchus which in turn branch and rebranch like a tree.

1.5.5.6.2 **Left bronchus:** Left bronchus has smaller caliber, more longer (2 inches) than the right and enters the root of left lung at the level of 6\(^{th}\) thoracic vertebra. It has no eparterial branch. It gives branch to superior lobe. The 1\(^{st}\) branch arises about 5 cm from the bifurcation of trachea. The main stem enters in the inferior lobe. Here it divides itself into ventral and dorsal branches. All of its branches are hyparterial.

1.5.5.6.3 **Structure:** The lungs are composed of serous coat, subserous areolar tissue and the pulmonary substance or parenchyma. The serous coat is the pulmonary pleura. It is external, thin, transparent, and covers the entire organ including the root. The subserous areolar tissue contains a large number of elastic fibers. It covers the entire surface of the lung. It also
extends inward between the lobules. The parenchyma is composed of secondary lobules of different sizes. These secondary lobules are closely connected together by an interlobular areolar tissue. Each secondary lobule is composed of several primary lobules. The primary lobule is composed of an alveolar duct with air spaces, blood vessels, lymphatic and nerves. Secondary lobules on the surface are large and pyramidal form. Secondary lobules of interior site are smaller and of various forms. The intrapulmonary bronchi divide and subdivide throughout the entire organ, the smallest subdivisions constituting the lobular bronchioles of diameter approximately 0.2 mm. Each bronchiole divides into two or more than two respiratory bronchioles. The respiratory bronchioles are again divide into many alveolar ducts. A greater numbers of alveoli are connected to each alveolar duct. Each alveolar duct is connected with a number of spherical spaces, the atria. Two to five alveolar sacs are connected with each atrium, which bears on all parts of their circumference alveoli or air sacs. The alveoli are lined by a simple squamous epithelium layer. The cells of alveoli are united by a cement substance. Outside the epithelial lining there is a little connective tissue with many elastic fibers and a close net-work of blood capillaries (Fig. 1.10).
Fig. 1.7 – Front view of heart and lungs.
(http://education.yahoo.com/reference/gray/subjects/subject/240)

Fig. 1.8 – Mediastinal surface of right lung.
(http://education.yahoo.com/reference/gray/subjects/subject/240)
Fig 1.9- Mediastinal surface of left lung. 
(http://education.yahoo.com/reference/gray/subjects/subject/240)

Fig. 1.10- Schematic longitudinal section of a primary lobule of the lung; 
r. b., respiratory bronchiole; al. d., alveolar duct; at., atria; 
a. s., alveolar sac; a, alveolus or air cell; p.a.: pulmonary artery: 
p. v., pulmonary vein; l., lymphatic; l. n., lymph node. (Miller) 
(http://education.yahoo.com/reference/gray/subjects/subject/240)
1.6 ANATOMY OF FEMALE REPRODUCTIVE SYSTEM

In the reproductive process, two kinds of gametes (sperm and ovum) meet in the reproductive system to create a new individual. The female reproductive system is composed of the vagina, cervix, uterus, fallopian tubes or uterine tubes and ovaries (fig. 1.11, 1.12).

1.6.1 Vagina: It is the most inferior region of the female reproductive tract. It is a thick walled, fibromuscular tube lined by stratified epithelium. It extends from the vestibule to the cervix of the uterus. It is situated between the urinary bladder and the rectum. In a mature female its length is 7.5 cm at its anterior wall and 9 cm at its posterior wall. It is the exit canal for blood discharge during menstruation and the baby during a vaginal childbirth. It connects the uterus and other internal reproductive organs to the external genitalia via cervix. (fig. 1.11, 1.12).

The structure of vagina consists of single internal mucous lining and single muscular coat. The mucous membrane is also called tunica mucosa. Tunica mucosa is continuous above with lining of the uterus. The inner surface of tunica mucosa contains two longitudinal ridges called columns of vagina. From these ridges many transverse ridges arise which extend outward on either side. The mucous membrane is covered by a type of stratified squamous epithelium. The submucous tissue contains a large number of veins. These veins form a plexus with smooth muscular fibers of muscular coat. The muscular coat also called tunica muscularis. It is made of two layers: (1) external layer is longitudinal. The fibers of this layer are in continuous with the superficial muscular fibers of the uterus. (2) Internal layer is circular. Both the layers are connected by oblique placed decussating
fasciculi. The lowest part of vagina is surrounded by muscular fibers of bulbocavernosus muscle.

1.6.2 Cervix: The cervix is the narrow, cylindrical and inferior part of the uterus and situated between the vagina and the uterus. The portion projecting into the vagina is exocervix which has a convex surface. The opening of exocervix is called the external os. The passageway between the external os and the uterine cavity is called endocervical canal. The endocervical canal ends at the internal os. Internal os is the opening of the cervix inside the uterine cavity (fig. 1.11, 1.12). There are 3 main functions of cervix. 1. It provides passage for semen; 2. It provide passage during the vaginal delivery for newcomers; 3. It provides passage for the shedding of endometrium during menstruation.

1.6.3 Uterus: The uterus is a hollow pear-shaped structure with an upside-down measuring 3 inches x 2 inches with a thick lining and muscular walls. It is located near the floor of the pelvic cavity. It allows the fertilized egg (blastocyte) to implant and grow. It also allows for the inner lining of the uterus to build up until a fertilized egg is implanted. It is sloughed off during menses (Fig. 1.11). The uterus has 3 coats: (1) The serous coat (external) is derived from the peritoneum. It mainly covers the fundus and the whole of the intestinal surface of the uterus. (2) The muscular coat (middle) is the chief bulk of the uterus. It is made of bundles of unstriped muscular fibers with areolar tissue, blood vessels, lymphatic vessels, and nerves. The layers of muscular coat are three in number: external layer, middle layer, and internal layer. The external and middle layers constitute the muscular coat proper, while the inner layer is greatly hypertrophied muscularis mucosæ. (3) The mucous membrane
The mucous membrane of the body of the uterus is soft, smooth, pale red in color, lined by columnar ciliated epithelium. There are many orifices of tubular follicles on the surface. The tubular uterine glands are small in the unimpregnated uterus and large and elongated in impregnation uterus. In the upper two-thirds of the canal, the mucous membrane has a many deep glandular follicles, which secrete viscid alkaline mucus. There are a number of occluded and distended (with retained secretion) little cysts in the canal. They are the ovula Nabothi. The epithelium of the upper 2/3rd is cylindrical and ciliated, but below it is unciliated and changes to stratified squamous epithelium. A fertilized egg implants itself into the wall of the endometrium where it develops throughout the pregnancy. The muscles of the uterus expand and contract to accommodate a growing fetus and then help push the baby out during labor.

**1.6.4 Fallopian (uterine) tubes:** The fallopian tubes are paired and extend superiolaterally off the uterus and are connected with the respective ovaries. They transport ova from the ovaries to the cavity of the uterus and fertilized ovum to uterus for implant. Each uterine tube is about 10 cm long and about as wide as a piece of spaghetti. Each tube has three parts: (1) Isthmus (2) dilated ampulla and (3) Infundibulum surrounded by finger like projections called fimbriae near the ovary. These fimbriae help to collect mature eggs released by the ovaries. The released egg sweeps into the lumen of the uterine tube by the frimbriae. The oocyte takes 4 to 5 days to travel down the length of the uterine tube. The fertilization takes place in the uterine tube. The fertilized ovum is then passed on into the uterus (fig. 1.11, 1.12).
1.6.5 Ovaries: The ovaries are paired, greyish-pink in color, oval organs located within the pelvic cavity and are situated either side of the uterus. The mature ovary is a roughly bean-shaped weighing about 14 grams having dimensions of about 3 cm long, 1.5 cm wide and about 1 cm thick (fig 1.11,1.12). Apart from this the ovary is an endocrine structure also. It secretes hormones.

1.6.5.1 The structure of ovary: Each ovary is invested with a layer of columnar cells, which constitutes the germinal epithelium of Waldeyer. The ovary consists of numerous vesicular ovarian follicles imbedded in the meshes of a stroma (frame-work). The stroma consists of spindle-shaped cells and small amount of ordinary connective tissue which resembles non-striated muscle cells. On the surface the tissue is condensed and forms a layer called tunica albuginea. The ovary, after puberty, has a thick cortex which contains the ovarian follicles and corpora lutea and surrounds richly vascular medulla (except at the hilus).

The cells of the ovarian follicle are the oocyte, granulosa cells and the cells of the internal and external theca layers. Every month ovary releases a mature egg (oocyte). Granulosa cells surround the oocyte. The granulosa cells are enclosed in a thin layer of extracellular matrix (follicular basement membrane or basal lamina) (fibro-vascular coat in figure). Outside the basal lamina, the layers of theca interna and theca externa are found (fig.1.13).
Fig.1.11 - Female reproductive system
(Source: http://www.patient.co.uk/diagram/Uterus-and-Ovaries.htm)
Fig. 1.12- A median sagittal section through a human female pelvic (Gray’s Anatomy 35 th ed, edited by R Warwick and PM Williams, Longman 1973, reprint 1975, Great Britain)
1.6.5.2 **Primordial follicle:** At the time of birth the surviving primary oocytes are surrounded by thin but single layers of follicular epithelial cells. These are delimited from the rest of the ovarian stroma by a thin basal lamina. The primordial follicles always form the follicles in the ovary (Fig. 1.14 A).

1.6.5.3 **Primary follicle:** In the transition of the primordial follicles into primary follicles the follicular epithelium that surrounds the oocyte becomes iso- to highly prismatic (Fig. 1.14 B)

1.6.5.4 **Secondary follicle:** When the primary follicles survive, the secondary follicle with follicular epithelium surrounds, multiplies and produces many rows. This is called stratum granulosum. In the secondary follicles a glycoprotein layer (the pellucid zone), is visible between the oocyte and follicular epithelium. Cytoplasmic processes of the granulosa cells reach the oocyte through the pellucid zone and maintain its function. Outside the basal lamina the stroma ovarii organizes itself to become theca folliculi cells (Fig. 1.15).
**Fig. 1.14-** Development from primordial follicle to primary follicle

A. Primordial follicle;  
B. Primary follicle  
1. Oocyte; 2. Follicular epithelium

**Fig. 1.15-** Secondary follicle: in the transition from primary to secondary follicle the stratum granulosum is engendered from the cells of the follicular epithelium. The stroma ovarii organizes itself around the secondary follicle to become the theca folliculi (interna and externa)  

1.6.5.5 **Tertiary follicle:** On the successful survival of the secondary follicles, tertiary follicles are generated. It has a fluid-filled cavity called the antral follicle. The oocyte lies at the edge of granulosa epithelial cells, the cumulus oophorus. During this time it has grown and its cellular nucleus has achieved the size of a primordial follicle. The connective tissue around the follicle differentiated itself into theca interna and theca externa. The theca interna is supplied with capillaries, lipid-rich cells and theca externa contains larger vessels. Decisive for a successful follicle growth is a well-developed net of capillaries in the theca interna. Before ovulation the growth of the tertiary follicles takes place. Graafian follicle corresponds to a large tertiary follicle that can be expected to be enough for ovulation (Fig. 1.16).
Fig. 1.16- In a tertiary follicle the theca can be subdivided into an interna (hormone production) and an externa (transition to the ovarian stroma).

1.7 PHYSIOLOGY OF PUBERTY: Puberty is the process of physical maturation by which an individual becomes physiologically mature into an adult and is capable of sexual reproduction. It is supposed that the biological changes that occur during puberty are initiated by hormonal signals from the brain to the ovaries. The ovaries produce hormones that stimulate libido and the growth and transformation of the various body organs e.g. brain, bones, muscle, skin, hair, breasts, and sexual organs and their endocrine as well as exocrine secretions.

1.7.1 Activation of the hypothalamic-pituitary-gonadal axis (hpg axis): Activation of the hpg axis induces and enhances the progressive ovarian sex hormone secretions that are responsible for the biological, morphological, and psychological changes. After birth the gonads respond to stimuli such as follicle-stimulating hormone (FSH) in girls up to the age of 12-24 months (Fig. 1.17).

Fig 1.17- Activation of the hypothalamo-pituitary-gonadal axis (Forest MG et al., 1973)
1.7.2 **Adrenarche:** Adrenarche is the onset of androgen dependent signs of puberty, including the growth of pubic and axillary hair, acne, and the emerging of adult body odor. Adrenal androgens dehydroepiandrosterone (DHEA) and its sulfate (DHEA-S) production from the adrenal zona reticularis lead to the adrenarche. In girls, DHEA and DHEA-S, begin as early as 2 years of age, accelerated at 7 to 8 years of age (initiation of adrenarche) and continue up to 15 years of age (Korth-Schutz et al 1976; Ducharme et al 1976; Lee et al, 1976). The accelerated increases in adrenal androgens begin about 2 years before the increase in gonadotropin and gonadal sex steroid secretion when the hypothalamic-pituitary-gonadal till then is still functioning at low prepubertal level (fig 1.17, 1.18). Premature adrenarche reduces the adult height.

**Fig 1.18-** Circulating DHEA and DHEAS (Source: Auchus J et al. Clinical Endocrinolgy 2004)
1.7.3 Hormonal Changes of Puberty:

1.7.3.1 Gonadotropins (Gn): Gonadotropins are hormones secreted by gonadotrope cells of the pituitary gland. Gonadotropins are released under the control of gonadotropin-releasing hormone (GnRH) from hypothalamus. The first demonstrable biological change of puberty is the appearance of pulsatile luteinizing hormone (LH) release during sleep. As puberty progresses, the frequency and amplitude of LH secretory peaks increase, although peaks are also found during the wake period. At the end of puberty, the difference between sleep and wake LH secretory patterns disappear.

1.7.3.2 Gonadotropin-releasing hormone (GnRH): GnRH is a unique hormone and is also known as Luteinizing-hormone-releasing hormone (LHRH). By 10 weeks of gestation, Gonadotropin-releasing hormone (GnRH) is present in the hypothalamus, and LH and FSH are present in the pituitary gland. Gonadotropins levels are elevated before birth. The levels of FSH are also higher. Several of the hormonal changes associated with pubertal development begin before any of the physical changes are obvious. Sleep-entrained increases in LH and FSH early in puberty. LHRH is the controlling factor for gonadotropins secretion. GnRH is synthesized and released from neurons within the hypothalamus. These GnRH-secreting neurons project axons that terminate on the portal vessels at the median eminence where GnRH is secreted for delivery to the anterior pituitary. The nocturnal increase in gonadotropin levels are followed by an increased secretion of the estradiol next day. Basal levels of both FSH and LH increase through puberty. The identity of GnRH was clarified by the Nobel Laureates Roger Charles Louis Guillemin (physiologist), Andrew Victor Schally (endocrinologist) and Rosalyn Sussman Yalow (medical physicist) in 1977:
pyroGlu-His-Trp-Ser-Tyr-Gly-Leu-Arg-Pro-Gly-NH₂.

The gene that encodes GnRH produces 92 amino acid precursor proteins, which contain GnRH decapeptide and 56 amino acid peptide known as GnRH-associated peptide (GAP). GAP is a potent inhibitor of prolactin secretion as well as stimulator of gonadotropin release.

1.7.3.3 Pulsatile secretion: GnRH controls the secretion of FSH and LH from the anterior pituitary. It is secreted in a pulsatile fashion. GnRH influences the release of two gonadotropins. The continual pulsatile secretion of GnRH is essential because the half life of GnRH is about 2-4 minutes. The pulsatile secretion of GnRH varies in frequency as well as amplitude throughout the menstrual cycle and it is strictly regulated (fig 1.19). Frequent and small-amplitude pulse of GnRH secretion is found in the follicular phase. There is an increase in frequency and amplitude of pulses in the late follicular stage. In the luteal phase, the interval between pulses is increased. Similarly the amplitude is also higher in the luteal phase. Pulsatile activity is disrupted by hypothalamic-pituitary diseases i.e. hypothalamic suppression, trauma, tumor etc.
Fig. 1.19- The pulsatile secretion of GnRH in the follicular and luteal phase of the cycle (Soules MR et al., 1985 & Filicori M et al., 1986).

1.7.4 Physical changes of Puberty: After birth the gonads respond to LH and FSH. Puberty proceeds through 5 stages from childhood to maturity as described by Marshall and Tanner (1969). These stages reflect the pubertal changes. The secondary sex characteristics appear at a mean age of 10.5 years in girls.

1.7.4.1 Secondary sex characteristics: The secondary sexual development involves the enlargement of the ovaries, uterus, vagina, labia, breasts and growth of pubic hair (Marshall and Tanner, 1969). Increase in breast size, pubic hair, and genital development with the vaginal mucosa becomes more humid, of a darker pink colour, and takes on a secretory appearance which will follow. The uterus increases in size. Between 11 and 14.5 years of age, the adolescent growth takes place.
Stages of development

<table>
<thead>
<tr>
<th>Stages</th>
<th>Breast development</th>
<th>Pubic hair</th>
<th>growth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1 (P1)</td>
<td>Prepubertal</td>
<td>Prepubertal (can see velus hair similar to abdominal wall)</td>
<td>5-6cm/ year</td>
</tr>
<tr>
<td>Stage 2 (P2)</td>
<td>Breast bud stage with elevation of breast and papilla; enlargement of areola</td>
<td>Sparse growth of long, slightly pigmented hair, straight or curled along labia</td>
<td>7-8cm/ year</td>
</tr>
<tr>
<td>Stage 3 (P3)</td>
<td>Further enlargement of breast and areola; no separation of their contour</td>
<td>Darker, coarser and more curled hair, spreading sparsely over junction of pubes</td>
<td>8cm/ year</td>
</tr>
<tr>
<td>Stage 4 (P4)</td>
<td>Areola and papilla form a secondary mound above the level of breast</td>
<td>Hair adult in type, but covering smaller area than in adult; no spread to medial surface of thighs</td>
<td>7cm/ year</td>
</tr>
<tr>
<td>Stage 5 (P5)</td>
<td>Mature stage: projection of papilla only, related to recession of areola</td>
<td>Adult in type and quantity, with horizontal distribution (&quot;feminine&quot;)</td>
<td>No further height after 16 years</td>
</tr>
</tbody>
</table>

1.7.4.1 Uterine and ovarian development: At prepubertal stage, the uterus is tubular, 2-3 cm in length, having volume 0.4 ml -1.6 ml and single layered endometrium. At pubertal stage it becomes pear-shaped and attains more length (5-8cm), more volume (3-5ml), and thickness of endometrium increases. These changes occur due to production of estrogens. The gradual rising level of plasma gonadotropins induces ovary to produce excess amount of estradiol. The estradiol is responsible for the development of secondary sexual characteristics. The ovarian volume in prepubertal and
during puberty ranges from 0.2 ml to 1.6 ml and 2.8 ml to 15 ml respectively and becomes multicystic at puberty.

1.7.4.2 Initiation of menarche: Increased level of plasma Gonadotropins stimulate ovary. The ovary produces excess amount of estradiol which reflects successive waves of follicular development that fails to reach the ovulatory stage. The proliferation of uterine endometrium is affected by these changes, until a point is reached when substantial growth occurs so that the withdrawal of estrogen results in the menarche. Menarche is the first menstrual cycle in the female human beings.

1.7.5 Menstrual cycle: The menstrual cycle is the stage in the fertile female human beings in which various physiological changes occur. It is controlled by the endocrine system and is necessary for reproduction.

1.7.5.1 Age of menarche: It is the age of the beginning of first menstruation. Generally menarche occurs around 13 years of age in our country. Variations in the age of menarche may depend upon nutritional status, geographical location, environmental conditions and socio economic status etc. In most of the girls 80% cycles are anovulatory in the first year after menarche followed by 50% and 10% in the 3rd and 6th year respectively. Severe uterine pain occurs during menstruation (dysmenorrhea). It is common among adolescents and younger women.

1.7.5.2 Length of cycle: It is the length of the starting of continuous two menses. It is measured from the start of one menses to the start of the next one. The average menstrual cycle takes 28 days.
1.7.5.3 **Amount of flow:** The average blood loss during one menstruation is 35 milliliters (Healy 2010). Roughly the volume of blood loss can be estimated by asking the number of pads soaked per cycle.

1.7.5.4 **Phases of cycle** (Fig. 1.20): The menstrual cycle is divided into many phases. The length of each phase is shown below. The first two phases are related to changes in the lining of the uterus and the last two are related to the process occurring in ovary. Between 2\textsuperscript{nd} and 4\textsuperscript{th} phase there is a dividing phase known as ovulation.

<table>
<thead>
<tr>
<th>Name of phase</th>
<th>Days</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Changes in the lining of uterus</strong></td>
<td></td>
</tr>
<tr>
<td>Menstrual phase</td>
<td>1–4</td>
</tr>
<tr>
<td>Follicular phase (or proliferative phase)</td>
<td>1–13</td>
</tr>
<tr>
<td>Ovulation (an event dividing phases)</td>
<td>14</td>
</tr>
<tr>
<td><strong>Process occurring in ovary</strong></td>
<td></td>
</tr>
<tr>
<td>Luteal phase (or secretory phase)</td>
<td>15–26</td>
</tr>
<tr>
<td>Ischemic phase</td>
<td>27–28</td>
</tr>
</tbody>
</table>

1.7.5.4.1 **Menstrual phase or menses phase:** It starts from 1\textsuperscript{st} day of bleeding to the last day of bleeding (Fehring et al., 2006). Eumenorrhea denotes normal, regular menstruation that lasts for usually 3 to 5 days. Decreasing levels of progesterone trigger menstruation and the beginning of the cycle.

1.7.5.4.2 **Follicular phase or proliferative phase:** The follicular phase starts from 1\textsuperscript{st} day of bleeding to the estimated day of ovulation (Fehring et al., 2006). In this phase lining of the uterus proliferates (Losos et al., 2002). Increase FSH during the 1\textsuperscript{st} days of the cycle stimulates ovarian follicles.
These follicles begin folliculogenesis (primordial follicles into large preovulatory follicles). Under the influence of hormones, one dominant follicle matures (Graafian follicle- which forms the ovum) (Losos et al., 2002). This follicle secretes excess amounts of estrogen. The estrogens start the formation of a new layer of endometrium.

1.7.5.4.3 Ovulation: For ovulation LH surge is required, which starts around the cycle day 12 and lasts 48 hours. After maturation of egg, the required levels of estradiol stimulate production of LH. Under the influence of LH the egg matures and weakens the wall of the follicle and secondary oocyte is released. The secondary oocyte matures and finally becomes a mature ovum (Losos et al., 2002). After being released from the ovary and into the peritoneal space, the egg is picked up by respective fallopian tube by the fimbria. After fertilization the embryogenesis starts. In about 6 days the developing embryo becomes blastocyst. At this stage it is implanted into the endometrium (Losos et al., 2002). The unfertilized egg disintegrates itself in the fallopian tube (Losos et al., 2002).

1.7.5.4.4 Luteal phase (or secretory phase): The luteal phase starts from the 1st day after estimated day of ovulation to the day before the next expected menstrual cycle (Fehring et al., 2006). In this phase the corpus luteum continues to grow for some time and produces significant amount of progesterone (steroid hormone) (Losos et al., 2002). The progesterone makes the lining of the uterus thick for implantation of the blastocyst. After ovulation, FSH and LH transform the remaining parts of the follicle into the corpus luteum. The higher level of progesterone induces the formation of estrogen. The corpus luteum continues to produce progesterone until the placenta begins to take over progesterone production. This period is about 10
weeks of gestation. The hormones produced by the corpus luteum also suppress production of the FSH and LH resulting into quickly fall of FSH and LH leading to atrophies of corpus luteum. (Losos et al., 2002).

1.7.5.4.5 Ischemic phase: Ischemic phase lasts for 1 to 2 days. Ischemic phase begins if fertilization does not take place. Regression of the corpus luteum results in to sudden fall of estrogens and progesterone levels. The endometrium undergoes involution. The constriction of the arterioles results ischemia of the basal layer. The ischemia leads to the diminishing of the supply of oxygen and nutrients. After vascular constriction the endometrial arterioles dilate resulting in hemorrhage, and menstrual flow begins
Fig 1.20 - Phases of menstrual cycle (Menstrual cycle.mht)