INTRODUCTION

‘Asthma’ is a Greek word which means ‘breathlessness’ or ‘to breathe with open mouth’. Asthma is a chronic inflammatory illness of the bronchi. It usually manifests itself through attacks of breathlessness accompanied by wheezing, chest tightness and coughing particularly at night or early morning. The frequency of asthma attacks varies according to the severity of the disease. Many asthma sufferers have attacks that awaken them during deep sleep. Sitting up in bed allows them to breathe more freely. Asthma attacks are usually harmless, but certain severe attacks put life of asthma sufferers at risk. Airway inflammation produces air flow limitation through acute bronchoconstriction, chronic mucus formation and air wall swelling or remodeling. These symptoms may be relieved either spontaneously or after treatment.

Asthma is a chronic condition in which airways undergo changes when stimulated by allergens or other environmental triggers. These changes in the airways cause patients to cough, wheeze, and experience shortness of breath (dyspnea), the classic symptoms of asthma. Such changes are characterized by two specific responses:

- The hyper reactive response (also called hyper responsiveness)
- The inflammatory response
Hyper reactive Response

In the hyper reactive response, smooth muscles in the airways of the lungs constrict and narrow excessively in response to inhaled allergens or other irritants. Everyone's airways respond by constricting when exposed to allergens or irritants, but a special hyper reactive response occurs in people with asthma.

Inflammatory Response

The hyper reactive stage is followed by the inflammatory response, which generally contributes to asthma in the following way:

- In response to allergens or other environmental triggers, the immune system delivers white blood cells and other immune factors to the airways.
- These so-called inflammatory factors cause the airways to swell, to fill with fluid, and to produce thick sticky mucus.
- This combination of events results in wheezing, breathlessness, inability to exhale properly, and a phlegm-producing cough.

Inflammation appears to be present in the lungs of all patients with asthma, even those with mild cases, and plays a key role in all forms of the disease. Airways inflammation, airflow obstruction, and bronchial hyper responsiveness are characteristic phenotypic features of asthma. Clinically, airflow obstruction in asthma often is not fully reversible, and many asthmatic subjects experience an accelerated and progressive loss of
lung function over time. Histopathologic studies of the asthmatic airways have demonstrated stereotypic changes that might explain the loss of lung function that many patients with asthma experience. The notion of airway remodeling in asthma postulates that the alteration of the structure and function of key airway constituents, including airway smooth muscle, epithelium, blood vessels, and mucus glands, might explain, at least in part, the progressive loss of lung function that is observed clinically (Pauscal & Peters, 2005). Inflammation and remodeling with increase in wall thickness; changes in myofibroblasts and airway smooth muscle have been reported in young as well as older asthmatics (Jeffery, 1992).

Onset of asthma can occur at any age but children and young adults are the commonly affected age groups and 5-10 percent of children with mild asthma may develop severe asthma later in life. Asthma cannot be cured, but it can be controlled by proper management. Allergic rhinitis and skin allergy may coexist with or precede the onset of asthma. Environmental exposures during the early years and airway obstruction that develop during this time, in conjunction with genetic susceptibility, are important factors in the development of persistent asthma in childhood (Panettieri et al., 2008).

**Etiology and risk factors of asthma**

Asthma comprises a range of heterogeneous phenotypes that differ in presentation, etiology and pathophysiology. The risk factors for each recognized phenotype of asthma include genetic, environmental and host factors. Although a family history of asthma is
common, it is neither sufficient nor necessary for the development of asthma (Burke et al., 2003).

There are two general categories for classifying asthma: extrinsic and intrinsic depending upon the types of stimuli that trigger attacks. Extrinsic asthma is caused by a type of immune system response to inhaled allergens such as pollen, animal dander or dust mite particles. Intrinsic asthma is caused by inhalation of chemicals such as cigarette smoke or cleaning agents, taking aspirin, a chest infection, stress, laughter, exercise, cold air, food preservatives or a myriad of other factors.

The substantial increases in the incidence of asthma over the past few decades and the geographic variation in both prevalence rates and the magnitude of the increases clearly reveal that environmental changes play a larger role in the current asthma epidemic. Furthermore, environmental triggers may affect asthma differently at different times of a person’s life, and the relevant risk factors may change over time.

There are surely reasons or risk factors that predispose one to asthma and respiratory problems. Asthma does not just happen randomly to anyone without asthma risk factors. The common risk factors are:

**Family History** It is thought that three-fifths of all asthma cases are hereditary. According to Centre for Disease Control report (2008), if a person has a parent with
asthma, one is three to six times more likely to develop asthma than someone who does not have a parent with asthma.

**Gender**  Childhood asthma occurs more frequently in boys, followed by greater incidence in girls during adolescent period. Around age 20, the ratio of asthma between men and women is the same. At age 40, more females than males have adult asthma.

**Airway Hyper-reactivity**  Airway hyper-reactivity is another risk factor for asthma. In asthma, the airways are hyper-reactive and become inflamed when they meet such asthma triggers as allergens or cold air. Not all people with airway hyper-reactivity develop asthma, but in those who do have it, the airway hyper-reactivity appears to increase the risk of asthma.

**Environmental Factors**  Indoor air pollution such as cigarette smoke, mold, and noxious fumes from the kitchen and from household cleaners and paints can cause allergic reactions and asthma. Environmental factors such as pollution caused by burning of fodder after harvesting of wheat and paddy in the fields, sulfur dioxide, nitrogen oxide, ozone, low temperature, and high humidity are all known to trigger asthma in susceptible individuals. In fact, asthma symptoms and hospital admissions are greatly increased during periods of harvesting, heavy air pollution and winters. Ozone is the major destructive ingredient in smog. It causes coughing, shortness of breath, and even chest pain and can boost the susceptibility to infection. Sulfur dioxide, another component of smog, also irritates the airways and constricts the air passages, resulting in asthma
attacks. Weather changes can also result in asthma attacks in some people. For instance, cold air causes airway congestion, bronchoconstriction, secretions, and decreased mucociliary clearance. Increases in humidity may also cause breathing difficulty in some individuals.

**Smoking** Several studies confirm that smoking is linked with an increased risk for developing asthma. There is also evidence that cigarette smoking among adolescents increases the risk of asthma. Even more findings link secondhand smoke exposure with the development of asthma in early life. Smoking has been associated with airway hyper-responsiveness in a number of surveys as well as in clinical studies (Morrison, 1976). The association is stronger in the elderly or in those with a greater lifetime exposure to cigarettes, which is strongly correlated with age in smokers. Children with asthma who are exposed to tobacco smoke at home are reported to require an increased use of emergency rooms for the management of asthma (Evans et al., 1987). Maternal smoking during pregnancy has also resulted in lower pulmonary functions in infants.

**Obesity** In both adults and children, the incidence of obesity and asthma has been increasing in parallel over recent years. Studies report a strong association between the two conditions. Some experts suggest that excess weight pressing on the lungs may trigger the hyper reactive response in the airways typical of asthma. Others believe that asthma leads to obesity by inhibiting physical activity, although several studies have found no difference in activity levels between people with or without asthma. Some
studies suggest that many obese people may be misdiagnosed as having asthma when in fact they are simply short of breath, possibly because of the increased effort required for breathing.

A number of studies have associated obesity and asthma in adults (Seidell et al., 1986; Camargo et al., 1999; Gibson., 2000; Harik-Khan et al., 2001; Litonjua et al., 2002; Jarvis et al., 2002; Sin et al., 2002; and Huovinen et al., 2003). Obesity has also been linked with impaired pulmonary function and airway hyper responsiveness, (Santana et al., 2001; Weiner et al., 1998; Amara et al., 2001 and Mohamed et al., 2002) but not in all studies (Jarvis et al., 2002 and Chlif et al., 2005). The parallel increase in the prevalence of obesity and asthma in the past 2-3 decades has led some researchers to postulate a causal relationship between the two conditions.

Trigger factors

Asthmatic people have airways that are highly sensitive to certain things which do not bother people without asthma. These things are called “triggers”. When an asthmatic comes in contact with them, an asthma episode starts. The airways become swollen, produce too much mucus, and are tightened up. Common triggers of asthma can be classified as follows:

a. Allergens (Individual specific)

i. Outdoor allergens

- Pollens - from grass, flowering plants and trees
- Molds - of some fungi
ii. Indoor Allergens

- House dust mites
- Dander (or flakes) - from the skin, hair or feathers of warm-blooded pets (dogs, cats, birds, rodents, etc.)
- Molds - harboured in vacuum cleaners, air-conditioners, humidifiers.
- Insects – Cockroaches

iii. Food Allergens rarely cause an asthma attack. Though some of the foodstuffs may cause allergic manifestations in some people. It is not wise to ban allergy producing foods in general for an asthmatic but those specific foods which evoke an asthma/allergy attack within few minutes or hours after intake must be avoided

b. Irritants (More generalized)

i. Tobacco smoke

ii. Wood smoke

iii. Strong odours, perfumes and sprays, cosmetics, paints, cooking (especially with oils and spices)

iv. Air-pollutants - Smoke and toxic gases from automobiles and factories

c. Upper respiratory tract infection - viral infections, common cold

d. Exercise - strenuous physical activities

e. Certain Drugs - e.g. beta-blockers (even some eye drops), aspirin, non steroidal anti inflammatory drugs etc.

f. Seasonal/Time Triggers. Some people with asthma experience more problems with symptoms at certain times of the day (during sleeping time when airways tend to close),
certain times of the month (hormonal changes in women during pregnancy or menstruation), or certain times of the year (when pollen levels are high such as in the fall and the spring or the harvest season). Hot and humid conditions are known to aggravate asthma and similarly cold, foggy, dusty and rainy conditions may also trigger it.

g. Stress due to

i. Emotion - e.g. Laughing, Sobbing, Mental depression
ii. Surgery
iii. Pregnancy

h. Gastro Esophageal Reflux Gastro Esophageal Reflux Disease (GERD) is a common exacerbating factor in some patients with asthma, possibly via esophageal acid-induced reflex bronchoconstriction or by microaspiration of acid.

Pulmonary Functions in Asthma

Asthma isn't a single disease. Instead, it's a set of related symptoms that spring from a variety of underlying processes, both environmental and genetic. These different processes influence the rate of progression, lung function decline and response to therapy.

The main function of the lungs is to provide continuous gas exchange between inspired air and the blood in the pulmonary circulation, supplying oxygen and removing carbon dioxide, which is then cleared from the lungs by subsequent expiration. Survival is dependent upon this process being reliable, sustained and efficient, even when challenged by disease or an unfavourable environment (Roberts, 2000).
Asthma is characterized by the presence of reversible airflow obstruction; however, irreversible airflow obstruction develops in some patients. Moreover, accelerated loss of lung function over time has been reported in groups of patients with asthma in longitudinal prospective and retrospective studies (Peat et al., 1987; Lange et al., 1998; Sears et al., 2003 and Covar et al., 2004). Sears et al. (2003) reported that in view of pathogenic changes of airway remodeling lung function in asthmatics is expected to decline gradually over a period of time in spite of treatment.

Lange et al. (1998) have reported in their study on ventilatory function in adults with asthma that people who identified themselves as having asthma had substantially greater decline in forced expiratory volume in one second (FEV1) over time than those who did not.

Accelerated decline in lung function does not occur in all patients. The risk factors identified for accelerated decline in lung function include young age (Cover et al., 2004), male gender (Covar et al., 2004 and Lee et al., 2007), longer duration of disease (Lee et al., 2007) more prominent eosinophilic airway inflammation (Covar et al., 2004), asthma exacerbations (Bai et al., 2007), and smoking (Lee et al., 2007).

Many asthmatics retain normal or close to normal lung function throughout life, showing reversibility from acute worsenings and return to previous function. Conversely, some patients develop “irreversible” asthma, as seen in population-based studies (Peat et al., 1987) and in specialist-treated patients whose obstruction persisted despite bronchodilators and oral corticosteroids (Brown et al., 1984). In the latter patients, lung
function decreased with age, and with duration and severity of asthma. Progressive loss of function can be inexorable despite aggressive therapy, resulting in end-stage respiratory failure that occasionally justifies lung transplantation (Corris & Dark, 1993).

The function of the lung reflects the health status of the whole organism. The most convenient indices of body size are weight and stature. Stature is positively correlated with lung size usually to a greater extent than any other anthropometric index and is the reference variable of choice for most purposes. In addition, the sitting height is also informative (American Thoracic Society, 1991).

Harik-Khan et al. (2004) have studied the effect of anthropometric factors on the racial differences in lung function. African-Americans have lower lung function than Whites. In their study it was found that sitting height accounts for the largest portion of the racial differences in lung function in both sexes.

Chen et al. (1993) have reported that the body weight provides an alternative index of lung size; however the component of the weight which is due to the bone may be correlated positively with lung size. The component which is due to the adipose tissue is correlated negatively with this variable. For most indices of lung function the best reference variables are the age, sex, stature and ethnic group.

Higher BMI is associated with both increased fat mass (FM) and muscle mass or fat free mass (FFM), which have been shown to have opposite effects on lung functions (Lazarus et al., 1998 and Santana et al., 2001). Most population studies that examined the relations between obesity and lung function using body mass Index (BMI) as a
measure of overall adiposity, and non significant or weak associations have been reported, with diminished lung function at both extremes of the BMI distribution (i.e. thin or obese) (Lazarus et al., 1998 and Maiolo et al., 2003). However BMI does not take into account the pattern of fat distribution or body composition and cannot adequately distinguish between fat mass (FM) and fat free mass (FFM). In the past few years, it was suggested that FFM is positively associated with pulmonary function (Lazarus et al., 1998; Santana et al., 2001; Amara et al., 2001; Mohamed et al., 2002; Maiolo et al., 2003 and Wannamethee et al., 2005).

In Asian populations, abdominal or central obesity is more common than obesity defined by BMI (McKeigue et al., 1991) and health risks associated with overweight and obesity occur at lower levels of BMI than in North America or Europe (Ko et al., 1999 and Deurenberg-Yap et al., 1999).

There is a clear association between dyspnea and obesity (Sin et al., 2002). Obesity increases the work of breathing because of the reduction in both chest wall compliance (Sharp et al., 1964 and Chlif et al., 2005) and respiratory muscle strength (Weiner et al., 1998 and Chlif et al., 2005). This creates an imbalance between the demand on the respiratory muscles and their capacity to generate tension, which leads to the perception of increased breathing efforts (Le Blanc et al., 1986). Patients with obesity frequently report dyspnea and wheezing and are therefore often given therapy for asthma without objective diagnostic confirmation by pulmonary testing.
Much of the research linking obesity and asthma to date have been carried out in developed countries. In developing countries, with continuing high levels of under-nutrition and high prevalence of communicable diseases, very little attention has been paid to the rapidly growing problems of obesity and asthma. Previous research in India has related obesity with diabetes, hypertension and heart disease (Venkataramana and Reddy, 2002; Misra et al., 2001 and Gopinath et al., 1994) but there are few studies linking obesity and asthma.

Little is known about pulmonary function and anthropometric profile of bronchial asthma patients in this part of the region as patients are often treated on the basis of clinical history and sign and symptoms of the disease and their lung function is rarely assessed. It is important to study anthropometric profile and pulmonary functions of bronchial asthma patients as these have profound effect on the disease progression and management. Therefore, the present research work has been undertaken with the following aims and objectives:

1) To assess the anthropometric profile – body mass index, waist hip ratio, body fat and lean body mass of bronchial asthma patients.

2) To assess the pulmonary functions of bronchial asthma patients.

3) To study the relationship of selected anthropometric indices and pulmonary functions of bronchial asthma patients

4) To study the relationship of pulmonary functions of bronchial asthma patients with duration and severity of disease.