Introduction
Sleep apnoea is a sleep induced pulmonary disorder characterized by snoring and respiratory pauses, often accompanied with respiratory obstruction. Two third of patients are obese, and male population is 20 times more frequently affected as compared to females. Idiopathic sleep apnoea (SAS) affects 1% of adult population in USA, affecting more commonly in the age group between 40-60 years. SAS has become a major research field in pulmonary physiology in the recent years; nevertheless, the significant realization of SAS causing morbidity and mortality has not been spotted by physicians in this part of the country. SAS is defined as the presence of more than five apnoeas or hypoapnoeas per hour of in a young or middle aged patients sleep in conjunction with at least two major symptoms. There is cessation of airflow for at least 10 seconds and a hypoapnoea as a 50% reduction in thoracoabdominal movements for at least 10 seconds. Respiratory disturbance index (RDI) is the number of apnoea or hypoapnoea per hour of sleep. In other words, SAS is diagnosed if respiratory events occur during both NREM and RM sleep with a RDI of greater than five. Severity of SAS is denoted by RBI and degree of oxygen desaturation accompanying apnoeic episodes. Mild cases have RDI below 20/hour with oxygen saturation remaining above 85% while severe cases of SAS have RDI above 50/hour with oxygen saturation falling below 65% or cardiac arrhythmia accompanying apnoeic episode.

There are two types of sleep apnoeas; obstructive variety characterized by frustrated respiratory efforts to breathe against blocked upper airways; and central apnoea, where there is simple cessation of breathing without ventilatory efforts i.e. cessation of all air ow simultaneous with cessation of respiratory muscle activity. And those with obstructive apnoea having no initial respiratory efforts, when start having thoracic efforts as the apnoea progresses, is called mixed apnoea. All the apnoeas can occur in SAS, the obstructive variety is the commonest one. Familial tendency in SAS has also been documented. Obstructive variety has only been recognized in the past decade, although it is as common as asthma. Although exact prevalence of obstructive variety is not known, it is roughly estimated around 1-5%.

Pathophysiology
Almost all the apnoeas originate from airway occlusion around the level of soft palate. There is an
active contraction of muscles such as genioglossus, geniohyoid and omohyoid with each inspiration. During sleep, the muscle tone drops, creating a greater tendency of upper airway to narrow on inspiration. Turbulent flow occurs, resulting in the vibration sound of snoring. If the airway narrows subcritically, hypoapnea results if the airway occludes almost totally and apnoea results with total occlusion. Predisposing factors including obesity, enlarged tonsils, hypothyroidism, macro glossia, retrognathia etc. for pharyngeal encroachment, recurrent rhinitis and deviated nasal septum for nasal obstruction and alcohol, benzodiazepines etc. for decreased activity of upper airway dilator muscle. Possibly, upper airway behaving like a starling resistor may tend to collapse within a limited upper airway segment in sleep apnoea / hypoapnoea patients.\textsuperscript{4} Anatomical and functional mechanisms lead to development of critical collapsing pressure, the most important being a small lumen in the upper airways and the reduction and instability of drive to the dilator and abductors of upper airways. The latter is the characteristic of REM sleep and light NREM sleep. Excessive day time sleepiness (EDS) and snoring are the clinical hallmarks of SAS. Most of the patients find falling asleep while being idle or doing monotonous work such as driving a vehicle on lonely road. Vehicular accidents are likely to be more in such cases. Children with SAS are likely to lower their potential in studies. Often there is a history of snoring in these patients is quite old and may be intermittent; absence of snoring, however, does not exclude SAS. Often history of unsatisfying sleep, unrefreshed awakening, disorientation and generalized headache. Gradual onset of excessive day time sleepiness means it is underdiagnosed for years.\textsuperscript{5} Occasional episodes of nocturnal choking are quite disturbing. Even homicidal behavior has been described during sleep apnoea.\textsuperscript{6} Possibly because of atrial natriuretic peptide secretion being stimulated by apnoea, nocturia may appear due to excess of salt and water excretion.\textsuperscript{7} Enuresis has been documented in younger patients. Recently association of cherubism with OSA and mental retardation has been documented.\textsuperscript{8,9} Decrease in libido is yet another problem, impotence in males is often associated with long lasting and severe SAS. Majority of the patients with SAS are males, role of testosterone may be important both in hypogonadal patients of both sexes.\textsuperscript{1}

Although SAS is commonly seen in overweight patients yet obesity is not a prerequisite to establish the diagnosis. When sleep apnoea is associated with a significant fall of 74% in arterial oxygen saturation, this leads to repeated apnoea. Long standing severe cases, however, precipitate hypoxemic complications of cor pulmonale, secondary polycythemia and day time carbon dioxide retension, especially in the presence of coexisting pulmonary disease.\textsuperscript{10} Coexisting systemic as well as pulmonary hypertension and cardiac arrhythmias have been documented with SAS. Higher incidence of SAS in COPD as overlap syndrome has been recorded and these patients hence are at higher risk of developing respiratory insufficiency and cor pulmonale.\textsuperscript{11} Personality changes, impaired memory and motor performance are quite commonly encountered in SAS. Recently two separate studies have documented the evidence of a link between obstructive sleep apnoea and insulin resistance, independent of degree of obesity.\textsuperscript{12-14} Significant higher levels of fasting glucose and insulin levels have been demonstrated when compared with weight matched controls. Further studies have identified impaired metabolism as one of the consequence of sleep loss, suggesting that decrements in sleep duration and / or quality may increase the severity of metabolic disorders, particularly diabetes mellitus. Sleep disturbances are common in type 2 diabetes mellitus. Infact there has been a significant association of sleep disturbances with the presence of dyspnoea, nocturnal cramps, paresthesia and burning of sole.\textsuperscript{15} But findings of sleep disturbed breathing in these patients are mostly observed in obese patients with a mean BMI of 35.6 (SD: 9.9) kg /m\textsuperscript{2} in an Indian study. Therefore, a large community based epidemiological study is required to identify the true prevalence of sleep apnoea in Indian population. During clinical examination, obesity should be noticed, nose and orpharynx should be examined, especially by ENT surgeon, assessment for hypothyroidism and acromegaly and for complications such as cyanosis and congestive cardiac failure should be made. There will be sleep fragmentation also. Observations should be recorded, leaving the patient alone on the examination table, especially for induction of sleep while sitting idle (waiting for the doctor).
When air flow stops at the mouth and nose for a period of 10 seconds or more (apnoea) and is accompanied by continuing respiratory movements is called obstructive apnoea. And occurrence of 5 apnoeas per hour or more than 35 per night, during sleep, with frequent arousals, unrestful sleep, followed by daytime hypersonmolence constitutes obstructive sleep apnoea syndrome. The classic patients with OSAS is an overweight, middle aged person (although it can occur at any age and obesity is not necessarily present). A majority of these patients have some degree of upper airways obstruction. Alcohol is often a co-factor. Apnoea is associated with oxygen desaturation and a marked fall in tissue oxygen concentration during sleep. Typical snoring of OSAS is loud and spouse-disturbing. With the progress of disease, period of silence may alternate with vigorous breathing efforts of irregular intervals, with a terminal explosive snore, accompanied with restlessness and flailing of limbs. Recurrent arousals and insomnia cause unrefreshing effect of sleep, leading to mental fogginess, disorientation and tiredness during the day. Even headache is noted in 1/5 of these patients. Intermittent snoring and arousals occur due to combination of hypoxia and hypercapnia. Day time excessive sleepiness may result in social consequence due to deterioration in intelligence, impairment of memory and decreased sense of appropriate judgment. These patients may become instable, irritable and loose temper on little provocation. Systemic hypertension may be associated in 50% of these patients. Progressive asphyxia and pulmonary hypertension are also associated. Though pulmonary artery pressure returns to normal level during wakefulness in majority of cases, the nocturnal event may be accentuated in patients with obstructive airways disease, associated with sustained daytime hypoxaemia, hypercapnia, and polycythemia in 10-15% of patients. Such an event leads to right heart failure, cardiac arrhythmias such as ventricular premature beats and ventricular tachycardia may occur and may cause death during sleep (but further corroborative data is lacking). Nevertheless, daytime sleepiness has to be differentiated in case of narcolepsy, hypometabolic states such as hypothermia and hypothyroidism, uremia, cerebrovascular diseases, depression and use of sedatives.

Management
SAS cannot be diagnosed alone by clinical history or clinical observations, oximetry has been applied by man to diagnose SAS from the characteristic pattern of recurrent arterial oxyhaemoglobin desaturation. Again simple pulse oximetry is not disease-pictorial although it may be useful for population screening. At the same time, false negative results are obtained when oximetry employed even for screening. Ideal diagnostic choice for SAS includes over night polysomnography, recording oxygen saturation by an oximeter, thoracoabdominal movement by surface techniques, air ow by temperature or carbon dioxide sensors, electro encephalogram, electromyogram and electro-occulogram. This help in differentiating other causes of excessive daytime sleep i.e. narcolepsy. It is observed that severity of apnoea increases as the night progresses, possibly due to lengthening of mean apnoea duration. Forced oscillation technique has recently been used as a diagnostic tool to accurately & non-invasibly quantify respiratory obstruction during sleep. Elevated serum amyloid a has been suggested to be a risk factor for cardiovascular & neuronal dysfunction in patients with obstructive sleep apnoea. Treatment starts with a piece of advice to loose weight and avoid alcohol and sedative use. This sufficient for mild form of disorder. Appropriate treatment should be provided for myxodema, acromegaly, retrognathia and naso-pharyngeal problems. Drug therapy as such has found no place in the treatment of SAS. However, tricyclic antidepressant protriptyline has been used. It is thought to increase the tone of upper airways muscle and also help in reducing the REP sleep. Tracheostomy that used to be original 100% effective treatment, has now been obsolete. Increasing the pharynx size through surgical operation-Uvulopalatopharyngoplasty prevents snoring but it is not effective in treating SAS.

CPAP therapy has been found to be effective treatment in SAS using positive pressure between 7.5-20 cm H2O. It works by physically blowing the back of the throat open. It acts by splinting the
upper airways and preventing its collapse during inspiration. Results are equally good using subcritical pressure with lesser side effect.\textsuperscript{22} OSAS can be managed using combination of surgical technique and oxygen therapies.\textsuperscript{23,24} Continuous positive airway pressure therapy improves subjective & objective measures of sleepiness more than placebo in OSA.\textsuperscript{25} Short term beneficial effect on blood pressure & cardiac functions have been observed in these patients; long term vascular consequences of OSA are in unceded by CPAP is yet to be confirmed.\textsuperscript{26,27} Regression in left ventricular hypertrophy in patients with severe OSA has also been observed after six months of nasal CPAP therapy.\textsuperscript{28} However, CPAP therapy is ineffective in those who have not been benefited by Uvulopalatopharyngoplasty. Also, cost factor is another major hurdle in our country. Treatment of central sleep apnoea is more difficult. Often seen in the elderly, it is associated with a little of snoring. There is a lack of drive to upper airway muscles and diaphragm, which may be primary or due to reflex elicited by stimulation of mucosal sensory receptors by passive airway closure. Earlier treatment included diaphragm pacing with tracheostomy. Recently CPAP has been effectively used Acetazolamide in low dose may reduce the number of apnoeas.\textsuperscript{29} Suspecting and diagnosis SAS early and referring for sleep clinic, to start earlier benefiting from modern, effective and straight forward treatments to lead better social life\textsuperscript{16} is the need of the present time. President of ACCP Dr Udaya B S Prakash, while describing complications of sleep apnoea such as hypertension, heart disease & other cardiovascular complications, has remarked that the advancement in sleep medicine in diagnosing & treatment in the recent years should reach to primary care & speciality physicians so as to enable them to educate patients on the health effects of sleep disorders.

References
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