REVIEW OF LITERATURE

Obstructive sleep apnoea is the most common breathing disorder during sleep. It is characterised by repetitive episodes of upper airway obstruction and apnoea, which results in disruption of sleep and hypoxaemia.

In 1837, Charles Dickens, a novelist, well recognized in his own time as an astute observer of medical conditions captured the essentials of the condition in 'Joe the fat boy' in Pickwick papers. It was published in a series of papers entitled the 'Posthumous papers of the Pickwick club' (20). Pulmonologist notified the importance of this observation by stating that hypercarbia could be the reason though hypercarbia per say does not cause somnolence; it causes altered cerebral functions at higher levels of carbon dioxide tension.

In 1918, Sir William Oslar described a syndrome of obesity, hypersomnolence, and cyanosis and coined the term Pickwickian syndrome (21). In 1956, Burwell described a patient so somnolent that having been dealt a pocker hand of three aces and two kings; he dropped off to sleep and failed to take advantage of his opportunity. He included obesity, hyper-somnolence, periodic breathing with hypoventilation and corpulmonale in the syndrome (22). In 1956, Gastaut first described multiple respiratory pauses occurring during sleep in a pickwickian syndrome (23). This was the beginning towards research work on sleep apnoea syndrome. Gastut, Tassinari and Duron in France (24) and Jung and Kuhlo in Germany independently discovered sleep apnoea (25). In 1967, Henri Gasteau and Elio Lugaresi organised a symposium, published as *The abnormality of sleep in Man* which included wide range of topics covering pathological sleep in humans (26).

In 1970, C Alberio Tassinari a neurologist from France joined the Italian neurologist Elio Lugaresi and performed a crucial series of clinical sleep investigations and provided a complete description of the sleep apnoea syndrome including the first observations of the occurrences of sleep apnoea in non-obese patients (27).

In spite of all the clinical research, the clear concept of all night sleep recording as a clinical diagnostic test did not emerge unambiguously. The important reasons were that disease was not considered that serious enough requiring investigation and also it was unprecedented nature of an all night diagnostic test, particularly if it was conducted on an outpatient. Moreover, it was time and labour

intensive, and also not cost effective in terms of its basic expenses. There was also reluctance of non-hospital clinical professionals to work at night.

In 1970, the development of the sleep disorder clinic at Stanford University was a microsome of how sleep medicine evolved throughout the world. In 1972, Christian Guilleminault a French neurologist and Psychiatrist joined the Stanford group. He introduced respiratory and cardiac sensors in their all night sleep study. Starting in 1972, these measurements became a routine part of the all night diagnostic test, which was named polysomnography in 1974 by Dr Jerome Holland, a member of the group. The disability and cardiovascular complications of sleep apnoea were alarming. The treatment options were limited to weight loss and chronic tracheotomy. The results of tracheotomy were dramatic in ameliorating the symptoms and complications of obstructive sleep apnoea.

As the decade of the 1970 drew to a close, the consolidation and formalization of the practice sleep disorders medicine was largely completed. American sleep disorders Association was formed and provided home for professionals interested in sleep and particularly, the diagnosis and treatment of sleep disorders. This organization began as the association of sleep disorders centres with five members in 1975. The organization fostered the settings of standards through centre accreditation and examination for practitioners by which they were designated, accredited clinical polysomnographers. The diagnostic classification of sleep and arousal disorder was published in the end of 1979 after 03 years of extraordinary effort by a small group of dedicated individuals who composed the nosology committee chaired by Howard Roffwarg (28). In the beginning of 1981, the strategy for treatment of obstructive sleep apnoea was changed to surgical and mechanical from conventional chronic tracheostomy. The surgical approach was uvulopalatopharyngoplasty (UPPP) (29). The second and mechanical approach to treatment of obstructive sleep apnoea was continuous positive pressure airway technique, which was introduced by the Australian pulmonologist Colin Sullivan (30). The combination and high prevalence of obstructive sleep apnoea and effective treatment fuelled a strong expansion of centres offering the diagnosis and treatment of sleep disorder to patients. The decade was capped by the publication of sleep medicines first textbook, 'Principles and practice of sleep medicine' (31).

The importance of obstructive sleep apnoea in anaesthetic practice has been realized very late. Even today it is not a standard protocol in pre anaesthetic evaluation to enquire about history of sleep apnoea as we ask about many medical diseases. It is no wonder that many post operative cardio respiratory events causing morbidity and even mortality may be due to complications occurring due to obstructive sleep apnoea, not recognized preoperatively and due care is not taken during immediate post operative care. Several animal studies have indicated that anaesthetic agents both intravenous (32) and inhalational (33) and benzodiazepines (32) cause greater depression of the activity of upper airway muscles than diaphragm. In normal human subjects, Diazepam causes a selective decrease in genioglossus activity relative to that of the diaphragm favouring inspiratory upper airway obstruction (34). Another study in healthy subjects demonstrated that Midazolam in sedative doses markedly increased supraglottic upper airway resistance and induced central as well as obstructive sleep apnoea events (35). This increase in upper airway resistance could be due to diminished upper air way muscle tone. Some of the studies have also shown that central and obstructive sleep apnoea can occur in normal subjects breathing, although the effects of this agent on the upper airway muscles have not been explored.

The site of upper airway obstruction in anaesthetized normal humans has been investigated using a flexible bronchoscope (36). The subjects were spontaneously breathing under deep halothane anaesthesia. They demonstrated that upper airway obstruction was associated most consistently with contact between the epiglottis and the posterior pharyngeal wall. The motion of epiglottis and hyoid are closely associated and controlled by neck strap muscles. Drummond made the similar observation using surface EMG of tongue and neck muscle (37). Upper airway obstruction during Thiopentone induction was associated with loss of tonic activity in the neck strap muscles with consequent dorsal displacement of the hyoid. A more recent radiographic study has implicated the soft palate as the major site of upper airway obstruction in anaesthetized patient (38). Secondary collapse of the pharynx with multiple sites of obstruction occurred when the patients attempted inspiration similar to what has been observed in obstructive sleep apnoea syndrome.

There had been no direct studies of the effects of opioids analgesics on upper airway muscles. The study by Catley demonstrated that intravenous infusions in healthy patients can results in episodic obstructive sleep apnoeas with pronounced

oxygen de-saturations (6). They suggested that opioids preferentially depress upper airway muscle activity in way similar to the effect of sleep. It is speculated that both dose and route of administration of opioids are important for alteration of upper airway muscle function Robinson demonstrated a lack of selective depression of upper airway muscle function by small doses of oral opioids administered to healthy adults (8). Although there have been no systematic studies of the effects of intravenous narcotics on breathing during sleep. There have been anecdotal reports of clinically significant number of episodes of upper airway obstruction developing following administration of narcotic drugs in premedication (7, 8, 39, 40, 41, 42, 43, 44, 45, 46). Despite these concerns, one study of 12 normal adult subjects failed to demonstrate a change in breathing during sleep after administration of oral hydro morphine hydrochloride in 2 and 4 mg doses (8). It may not be valid to extrapolate these data to larger doses of narcotics or to their administration to patients with pre-existing upper airway dysfunction during sleep.

It is generally believed that epidural opioids are therapy of choice for patients with sleep apnoea for pain relief (47). The reports of three sudden post operative respiratory arrests associated with epidural opioids in patients with sleep apnoea, fatality ii all cases have opened up new thinking about safety of epidural opioids (48). On review of literature, out of 15 patients with sleep apnoea syndrome, 10 had severe respiratory problems due to postoperative analgesia; only one report was associated with epidural analgesia to which now another three cases can be added (48). It appears that sleep apnoea patients are particularly at high risk of postoperative respiratory depression from any mode of analgesic therapy. There are case reports of 16 patients with documented obstructive sleep apnoea who were listed for various type of surgery and given usual premedication and analgesia but with continuous nasal continuous positive airway pressure preoperatively and 24 – 48 hours postoperatively in all patients except two, did well and had uneventful recovery without any major complications due to opioids (49).

With these conflicting reports and no direct systematic study on parenteral use of opioids as premedicants, this area remains open for further exploration and research.