

## OBESITY AND RESPIRATORY COMPLICATIONS

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**Abstract:** Obesity has detrimental effect on almost all organ systems of the body. It involves respiratory system by altering respiratory mechanics; airflow resistance, breathing pattern, respiratory drive and causing impaired gas exchange. The obesity hyperventilation syndrome (OHS) was originally described in 1955 in subjects with obesity, chronic daytime hypercapnia, hypoxemia, polycythemia, hyper somnolence and right ventricular failure. Obesity causes various changes in respiratory mechanics. Reductions in lung chest wall and total respiratory system compliance is seen in especially OHS group. Respiratory system resistance is elevated in obese subjects i.e. around 30% increase in Simple Obesity and 100% increase in OHS. Respiratory resistance increases further in supine position as compared to upright body position. In OHS there is a decrease in respiratory drive even though there is extreme increase in respiratory work. The impairment in gas exchange depends on severity of the obesity. Impaired respiratory drive and increased work of breathing leading to hypoventilation contributes to hypercapnia and hypoxemia in OHS. Sleep related respiratory complications have been categorized under reading of obstructive sleep disorder breathing syndrome, which includes entire spectrum ranging from primary snoring to obstructive sleep apnea syndrome (OSA). In OSA syndrome, there is a complete cessation of airflow for at least 10 seconds and is associated with 4% fall in oxyhemoglobin saturation while in primary snoring there is no apnea, no sleep arousal and no reduction in oxygen saturation. Sleeplessness, fatigue, irritability and personality change has been observed in all forms. The diagnosis is made by focused history taking and physical examination of obese patients' who report of sleep difficulties and increased daytime sleepiness. The diagnosis can be confirmed by polysomnography. The therapeutic strategies for patients with sleep apnea involve conservative management, which includes avoidance of factors that increase severity of upper airway obstruction. Of the medical intervention that is done mainly includes the role of positive pressure ventilation in the form of CPAP, drugs like Protriptylene, Fluoxetine, Medroxyprogesterone, Acetazolomide have been tried with varying benefits. In resistant cases, tracheostomy and airway reconstruction in form of uvulopalatopharyngoplasty have been advised. Obese patients are also prone to other respiratory ailments such as pulmonary embolism, increased risk of gastric aspiration and difficulties during surgical and anesthetic procedures.

### INTRODUCTION

Obesity has detrimental effect on all most all the organ systems of the body<sup>1,2</sup>. It involves respiratory system by altering respiratory mechanics, airflow resistance, breathing pattern, respiratory drive and causing impaired gas exchange. Obesity can be divided into simple obesity (SO) & obesity hypoventilation syndrome (OHS) depending on the absence or presence of day time hypercapnoea. **The obesity-hypoventilation syndrome (OHS)** was originally described in 1955 in subjects with obesity, chronic daytime hypercapnia and hypoxemia, polycythemia, hypersomnolence, and right ventricular failure. In 1956, Burwell et al coined the term Pickwickian syndrome for these patients.<sup>3</sup> The majority of people with OHS have OSA.

The criteria of OHS include<sup>4</sup> : 1) BMI >30 kg/ m<sup>2</sup> ; 2) Daytime PaCO<sub>2</sub> > 45 mm Hg ; 3) Excessive daytime sleepiness; 4) Severe OSA (AHI >30) or sleep hypoventilation or both; 5) Absence of other known causes of hypoventilation

Various respiratory alterations and complications in an obese person can be broadly classified in to three main headings.

- 1) Alteration in respiratory system mechanics
- 2) Sleep related respiratory complications in obese
- 3) Respiratory complication in obese during critical illness
- 4) Other respiratory problems

Other diversified effects in each of the broad categories are shown in table 1.

**Table 1 :** Effects of obesity on respiratory system

A	Alteration in respiratory system mechanics
	1) Decrease in respiratory system compliance 2) Increase respiratory system resistance 3) Alteration in respiratory drive 4) Impaired gas exchange 5) Altered respiratory muscle strength and endurance 6) Increase work of breathing 7) Abnormality in pulmonary function tests.
B	Sleep related respiratory complications
	1) Obstructive sleep disordered breathing syndrome (OSDB) - Primary snoring - Upper airway resistance syndrome - Sleep hypopnea syndrome - Obstructive sleep apnea syndrome (OSA)
C	Respiratory complication in obese during critical illness
	1) Risk of deep venous and pulmonary thrombosis 2) Risk of aspiration 3) Difficult endotracheal intubation and mechanical ventilation
D	Other respiratory problems
	1) Exacerbation of bronchial asthma 2) Anesthetic and perioperative difficulties.

### CHANGES IN RESPIRATORY MECHANICS IN OBESITY

Obesity causes various changes in the respiratory mechanics of a person leading to impaired respiratory compliance and increased respiratory resistance. Reductions in lung, chest wall and total respiratory system compliance is seen in especially in OHS.<sup>5-7</sup> group. Lung compliance is reduced due to increased pulmonary blood volume and closure of dependent airways<sup>8</sup>.

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Chest wall compliance is reduced because of excess weight on the thorax and abdomen. Thus a more negative pleural pressure must be generated by the respiratory muscles to initiate airflow.<sup>5-8</sup>

Respiratory system resistance is elevated in obese subjects i.e. around 30% increase in SO and 100% increase in OHS. This increase is mainly due to increase in the chest wall resistance.<sup>6,7</sup> Respiratory resistance increases further in the supine position as compared to upright body position possibly due to compression of the supralaryngeal airway by fat and increased intrapulmonary blood flow leading to further airway narrowing.<sup>7,8</sup>

In OHS there is a decrease in respiratory drive even though there is extreme increase in respiratory work. Hypoventilation and tolerance of a higher  $P_{aCO_2}$  thus occurs & the set point of the CNS chemoreceptors is adjusted to a higher  $P_{aCO_2}$  with further decrease in respiratory drive. There is 25% greater respiratory rate and 25% lower  $V_T$ .<sup>9</sup> The impairment in the ventilatory responses to hypoxia<sup>10</sup> &  $CO_2$  is more as compare to those with SO.

The impairments in gas exchange depends on the severity of the obesity. Mildly reduced  $P_{aO_2}$  and widened alveolar-arterial oxygen difference ( $P_{aO_2}-P_{aO_2}$ )<sup>8,11</sup> is much more severe in people with OHS than with SO. Impaired respiratory drive and increased work of breathing leading to hypoventilation contributes to the hypercapnia & hypoxemia in OHS.

The abnormality in pulmonary function test depend on both the magnitude of the obesity, as well as the distribution of body fat (central/truncal vs peripheral predominance). People with central fat distribution have greater reductions in FVC, FEV<sub>1</sub>, and TLC<sup>13</sup>. It is because of the mechanical effect of the additional fat in the chest, abdominal wall and within the abdomen which compresses the thoracic cage and the diaphragm thus decreasing the lung volumes. In OHS FEV<sub>1</sub> / FVC ratio remains normal while in SO TLC remains normal.<sup>12</sup> Diffusing capacity also decreases slightly in proportion to the degree of obesity.<sup>13</sup> Reduction in the expiratory reserve volume (ERV) is the most common abnormality in PFT. ERV diminishes in proportion to the severity of obesity and is particularly abnormal in supine position.<sup>13</sup> with reductions found more in OHS patients.

## SLEEP RELATED RESPIRATORY COMPLICATIONS

The term **obstructive sleep disordered breathing syndrome (OSDB)** better describes this entire spectrum. With increasing order of severity they are:

- 1) Primary Snoring
- 2) Upper Airway Resistance Syndrome (UARS)
- 3) Sleep Hypopnea Syndrome
- 4) Obstructive Sleep Apnea Syndrome (OSA)

The basic pathogenic mechanism of OSDB is the upper airway narrowing. In obese patients, increased adipose tissue in the neck predispose the airway to narrowing which is confirmed

by MRI scans in patients with sleep apnea.<sup>14</sup> Also there is increased collapsibility of the pharynx due to impaired function of upper-airway dilator muscles. These events are generally most prominent during rapid-eye-movement (REM) sleep because of the hypotonia of the upper-airway muscles characteristic of this stage of sleep.

### **Obstructive sleep apnea**

- 1) Complete Cessation of airflow for atleast 10 seconds despite continuing ventilatory effort (apnea).
- 2) 5 or more episodes per hour of sleep.
- 3) Usually associated with a decrease of 4% in oxyhemoglobin saturation.

### **Obstructive sleep hypopnea**

- 1) Decrease of 30–50% in airflow for 10 seconds (hypopnea).
- 2) 15 or more episodes per hour of sleep.
- 3) May be associated with a decrease of 4% in oxyhemoglobin saturation.

### **Upper-airway resistance syndrome**

- 1) No significant decrease in airflow- i.e. no apnea / hypopnea.
- 2) 15 or more episodes of arousal per hour of sleep.
- 3) No significant decrease in oxyhemoglobin saturation.

### **Primary snoring**

- 1) No apnea / hypopnea.
- 2) No arousal during sleep.
- 3) No significant decrease in oxyhemoglobin saturation.

**Clinical features of OSDB** are attributed to arousal from sleep and/ or oxyhemoglobin desaturation and hypercarbia. Most commonly reported symptoms are loud habitual snoring, nocturnal choking episodes, morning headaches, and excessive daytime sleepiness (EDS). Sleepiness, fatigue, irritability, and personality change have been attributed to both nocturnal desaturation and the chronic sleep deprivation caused by sleep fragmentation

**Diagnosis** is made by a focused history taking and physical examination of every obese patients who report of symptoms of sleep disorders. This further helps in identifying the people at risk. The presence of certain physical characteristics like retrognathia and discrete upper-airway abnormalities, such as an enlarged soft palate or tonsillar hypertrophy, are clinical clues. An increased body-mass index, hypertension, and increased upper-body obesity, which is reflected by the neck circumference are good predictor of sleep apnea. The characteristics which are strongly associated with OSDB include, male sex, age > 40 years, BMI >25 kg/m<sup>2</sup>; or neck circumference >= 17 inches in men and >=16 inches in women, habitual snoring, nocturnal gasping, choking, or resuscitative snorting, observed apnea, history of systemic hypertension. Sleep disordered breathing can be diagnosed in-laboratory by polysomnography (PSG). This involves recording of multiple variables during sleep, including the neurological variables like electroencephalogram (EEG), electro-oculogram (EOG) and electromyogram (EMG) and cardiorespiratory variables

like airflow, respiratory effort, oxygen saturation, snoring and ECG.

The **differential diagnosis** of increased day time sleepiness that need to be considered are insufficient sleep, circadian rhythm disorder (eg jet lag, shift work) insomnia, drugs (eg sedatives), depression, CNS abnormalities, post traumatic hypersomnia, sleep fragmentation, periodic limb movement disorder & narcolepsy.

**Therapeutic strategies** for patients with sleep apnea may be grouped into three general categories: conservative, medical, and surgical. The goals of treatment are to establish normal nocturnal oxygenation and ventilation, abolish snoring, and eliminate disruption of sleep due to upper-airway closure. Conservative treatment includes avoidance of factors that increase the severity of upper-airway obstruction — such as sleep deprivation<sup>15</sup> the use of alcohol, sedatives, and hypnotic agents; and increased weight. Positive airway pressure delivered through a mask is the initial medical treatment of choice in patients moderate to severe sleep apnea. *Continuous positive airway pressure* (CPAP) is applied to the upper airway with a nasal mask, nasal prongs, or a mask that covers both the nose and mouth. The level of positive pressure required to sustain patency of the upper airway during sleep is determined in a sleep laboratory. Patients treated with nasal CPAP have repeatedly demonstrated improvement in neuropsychiatric function and a lessening of daytime sleepiness. Nocturnal desaturation, ventilatory-related arousals, nocturnal dysrhythmias, pulmonary hypertension, and right-sided heart failure have also been effectively treated.

*Drugs* like protriptyline and fluoxetine decrease the amount of REM sleep and increase tone of upper airway muscles, and may be useful in mild OSA intolerant to CPAP.<sup>16</sup> Medroxyprogesterone in a dose of 60 mg /day may be useful for treatment of hypoventilation in patients with OHS by increasing the ventilatory response to hypercapnea. Acetazolamide in a dose of 250 mg/ day may be used to reduce the serum bicarbonate level and the resulting metabolic acidosis increases the minute ventilation and reduces the PaCO<sub>2</sub>.

*Tracheostomy* and *upper airway reconstruction* are also required in some patients. The most commonly performed procedure, uvulopalatopharyngoplasty, is curative in less than 50 percent of patients.<sup>17</sup>

## OTHER RESPIRATORY PROBLEMS

Obese adults are at increased risk for many chronic medical conditions, and this increases the likelihood of admission to an ICU & presents with many problems. They are:

- 1) Obesity is a major independent risk factor for pulmonary embolism and venous thromboembolism (VTE).<sup>18</sup> The risk of pulmonary embolism rises as BMI increases.
- 2) Risk of aspiration is higher among obese patients, as compared with non-obese patients.<sup>18</sup> Higher volumes, lower pH of gastric fluid, delayed gastric emptying, and increased intraabdominal pressure leading to a high incidence of gastroesophageal reflux account for the increased risk of aspiration.
- 3) Obesity poses an increased risk of complications and difficulties during anesthesia and perioperative period.

First, gas exchange disturbances such as hypoxia and hypercarbia, may be exaggerated during anesthesia<sup>18</sup> Second, the incidence of postoperative pulmonary complications such as VTE, aspiration pneumonia, atelectasis, worsened gas exchange and respiratory failure is greater among obese patients.

- 4) Endotracheal intubation and mechanical ventilation is also difficult in obese patient. Factors associated with difficult intubations include large neck circumference, limited neck mobility, small oropharyngeal opening and difficulty in mouth opening.

## CONCLUSION

Disturbances of respiratory function, including reduced respiratory system compliance, increased small airways resistance, impaired respiratory muscle function, increased work of breathing, impaired gas exchange, exercise intolerance, sleep-disordered breathing, and increased risks of venous thromboembolism and aspiration are common, particularly among severely obese patients. These changes are independent of any underlying parenchymal lung disease. Weight loss can significantly decrease the risk and severity of obesity-related respiratory disturbances & should be considered for inclusion in a structured rehabilitation program with dietary, behavioral, and exercise components in an effort to improve functional capacity and quality of life.

## REFERENCES

1. **Lean MEJ, Seidell JC.** Impairment of health and quality of life in people with large waist circumference. *Lancet* 1998; 351: 853-856.
2. **Mokdad AH, Ford ES, Bowman BA, et al.** Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. *JAMA* 2003; 289:76-79
3. **Kessler R, Chaouat A, Schinkewitch P, et al.** The obesity-hypoventilation syndrome revisited. *Chest* 2001; 120:369-376
4. **Martin R. TJ, Sanders MH.** Chronic alveolar hypoventilation: a review for the clinician. *Sleep* 1995; 18:617-634
5. **Fontaine KR, Redden DT, Wang C, et al.** Years of life lost due to obesity. *JAMA* 2003; 289:187-193
6. **McCool FD, Rochester DF.** Nonmuscular diseases of the chest wall. In: Fishman AP, Elias JA, Fishman JA, et al, eds. Vol 2. 3ed ed. *Pulmonary diseases and disorders*. New York, NY: McGraw-Hill; 1998:1541-1560
7. **Rochester DF.** Obesity and pulmonary function. In: Alpert MA, Alexander JK, eds. *The heart and lung in obesity*. New York, NY: Futura Publishing Co; 1998:109-131
8. **Sharp JT, Henry JP, Sweany SK, et al.** The total work of breathing in normal and obese men. *J Clin Invest* 1964; 43:728-739
9. **Burki NK, Baker RW.** Ventilatory regulation in eucapnic morbid obesity. *Am Rev Respir Dis* 1984; 129:538-543
10. **Sampson MG, Grassino A.** Neuromechanical properties in obese patients during carbon dioxide rebreathing. *Am J Med* 1983; 75:81-90
11. **Zwillich CW, Sutton FD, Pierson DJ, et al.** Decreased hypoxic ventilatory drive in the obesity-hypoventilation syndrome. *Am J Med* 1975; 59:343-348
12. **Jenkins SC, Moxham J.** The effects of mild obesity on lung function. *Respir Med* 1991; 85:309-311
13. **Kress JP, Pohlman AS, Alverdy J, et al.** The impact of morbid obesity on oxygen cost of breathing (V o 2 resp) at rest. *Am J Respir Crit Care Med* 1999; 160:883-886
14. **Davies RJO, Stradling JR.** The relationship between neck circumference, radiographic pharyngeal anatomy, and the obstructive sleep apnoea syndrome. *Eur Respir J* 1990; 3:509-14.
15. **Neilly JB, Kribbs NB, Maislin G, Pack AI.** Effects of selective sleep deprivation on ventilation during recovery sleep in normal humans. *J Appl Physiol* 1992; 72:100-109.
16. **Hanzel DA, Proia NG, Hudgel DW.** Response of obstructive sleep apnea to fluoxetine and protriptyline. *Chest* 1991; 100:416-421
17. **Shepard JW Jr, Olsen KD.** Uvulopalatopharyngoplasty for treatment of obstructive sleep apnea. *Mayo Clin Proc* 1990; 65:1260-1267.
18. **Pietrantonio C, El Solh AA.** Weighty issues. *Advance for Managers of Respiratory Care* 2003; 12:33-36.