Noninvasive positive pressure therapy of the obesity hypoventilation syndrome OHS

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INTRODUCTION

Obesity Hypoventilation Syndrome (OHS) **occurs** in:

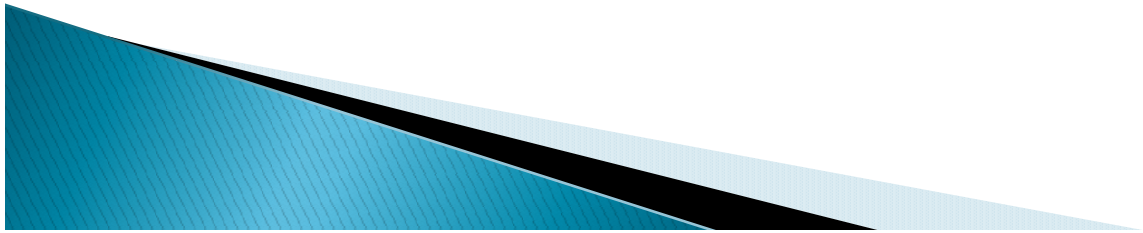
1-obese body mass index (BMI) $>30 kg/m^2$ patients

2-when awake alveolar hypoventilation ($PaCO_2 >45$ mmHg)

3- **cannot be attributed** to pulmonary parenchymal disease, skeletal restriction, neuromuscular weakness, or pleural pathology .

- Application **of positive airway pressure** is the mainstay of therapy although the effect on survival is unclear , together with

- **weight loss**
  are the initial first line therapies for patients with OHS, regardless of the presentation.



Weight loss and lifestyle modifications :

All patients with OHS should begin a weight loss program.

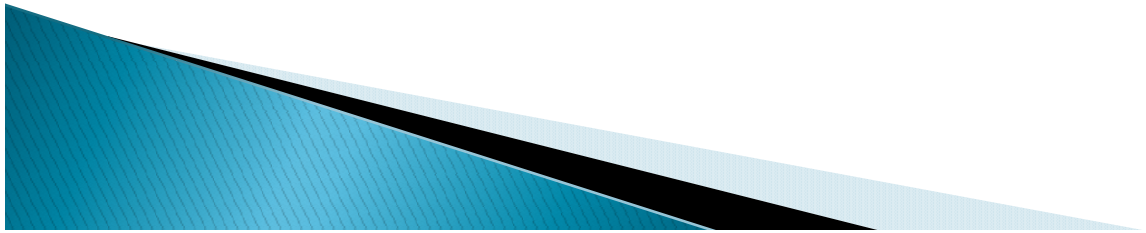
•Weight loss **improves alveolar ventilation** (sometimes normalizing the awake arterial carbon dioxide tension [$PaCO_2$] and arterial oxygen tension [PaO_2]),

•reduces the **risk of cardiorespiratory complications** (eg, pulmonary arterial hypertension and left ventricular function),

- improves nocturnal **oxyhemoglobin saturation,**

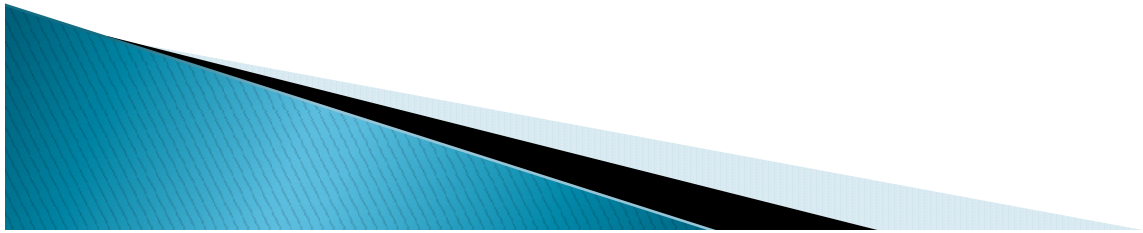
- decreases the frequency of respiratory events **AHI** during sleep if the patient has coexisting OSA,

- and improves **pulmonary function.**



- These benefits appear to occur **regardless** of whether the weight loss was due to lifestyle modification **diet** , **exercise** or **surgery**.

- Lumachi F, Marzano B, Fanti G, et al. Hypoxemia and hypoventilation syndrome improvement after laparoscopic bariatric surgery in patients with morbid obesity. In Vivo 2010; 24:329.



A comprehensive and multidisciplinary approach utilizing experts in

obesity,

sleep, and

pulmonary medicine is recommended.

For patients with OHS, we recommend **noninvasive positive airway pressure (PAP) therapy during sleep rather than lifestyle modifications alone** in order to improve symptoms and parameters of awake ventilation (ie, arterial partial pressure of carbon dioxide [$PaCO_2$]).

This recommendation is based upon the rationale that OHS will progress if not treated with PAP and improvement is dependent upon optimal compliance with therapy.

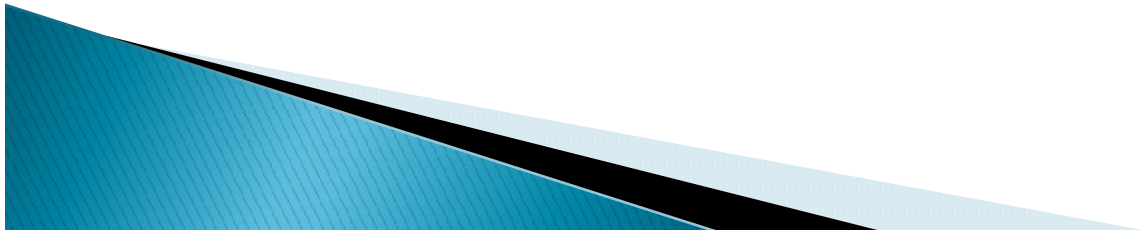
CHOOSING A MODE OF NONINVASIVE POSITIVE PRESSURE THERAPY

Mode selection for initial PAP therapy is **determined by the presence or absence of comorbid obstructive sleep apnea (OSA)** based on the results of in-laboratory **polysomnography** (PSG).

- All patients with **OHS** have some form of sleep disordered breathing, typically **obstructive sleep apnea %90** or

- sleep-related hypoventilation **10 %** ,

PAP therapy should **not** be delayed while the patient tries to **lose weight**.

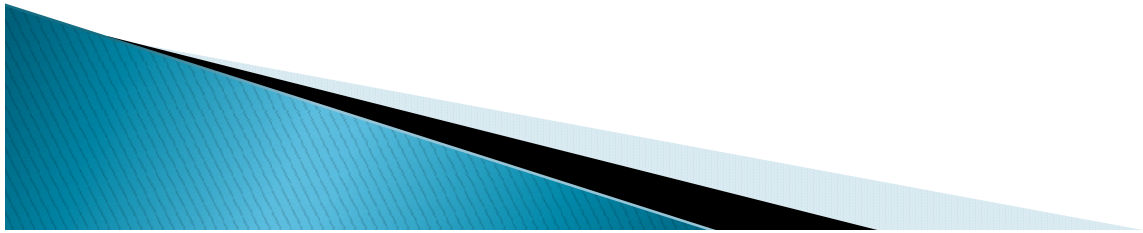


Approximately 90 % of patients with **OHS** have coexisting obstructive sleep apnea (**OSA**),
in which case continuous positive airway pressure (**CPAP**) is the initial mode of choice.

- For patients with **OHS and sleep-related hypoventilation**

- and patients with **acutely decompensated** OHS,

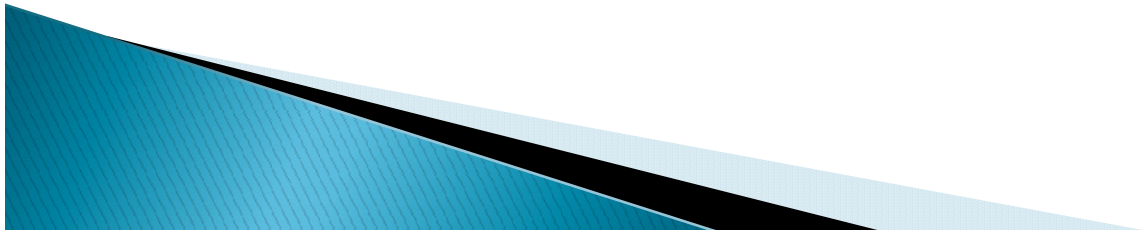
- Patients with OHS and OSA who **fail or do not tolerate** CPAP are **treated with BPAP.**



MODES OF NONINVASIVE POSITIVE PRESSURE THERAPY :

There are **two major categories** of noninvasive positive pressure therapy that are used to treat patients with OHS:

1-**Continuous positive airway pressure** (CPAP)



2-**Noninvasive positive pressure ventilatin (NPPV)**

Among the available modes of NPPV, there is :

- bilevel positive airway pressure **(BPAP)** and
- volume cycled positive pressure ventilation **(VCPPV)**.

Noninvasive positive pressure therapy is typically administered during sleep via

- nasal mask,
- full face mask (covering the nose and mouth),
- nasal pillows,
- or hybrid mask (oral mask with nasal pillows).
- NPPV can also be administered via mouthpiece.

But during an episode of acute decompensation, it may be necessary to apply NPPV during wakefulness.

OBESITY HYPOVENTILATION AND OBSTRUCTIVE SLEEP APNEA

Continuous positive airway pressure
CPAP

Continuous positive airway pressure

In patients with **coexisting OHS and OSA,**

CPAP is the typical mode chosen for treatment,

while BPAP, usually indicated in those who fail CPAP.

nocturnal CPAP may improve

- alveolar ventilation during sleep while also
- treating the OSA.

Several **case reports** and prospective series initially reported reduction of **awake** arterial carbon dioxide tension ($PaCO_2$) after the initiation of **nocturnal** CPAP

nocturnal CPAP does not directly augment ventilation other than by maintaining upper airway patency.

the CPAP-related improvement of **hypercapnia** during both wakefulness and sleep may be due to

-relief of ventilatory muscle fatigue

and/or

-augmentation of central ventilatory drive

The **ventilatory drive** may be facilitated by relief of nocturnal

-asphyxia اختناق or

-sleep fragmentation

Nocturnal CPAP therapy does not benefit all patients with OHS .

The failure of CPAP to eliminate nocturnal oxyhemoglobin desaturation remains a frequent indication for NPPV.

This was illustrated by a **study** that matched

-23 patients with OSA alone to
-23 patients with OHS plus OSA
ALL WERE PUT ON CPAP
according to their BMI, AHI, and spirometry .
43% of the patients with OHS plus OSA continued to spend more than 20 % of their total sleep time with an SpO2 <90 % .
even after their CPAP had been titrated to a level that significantly improved the
- AHI,
-rapid eye movement (REM) sleep duration,
-and sleep fragmentation.

- Banerjee D, Yee BJ, Piper AJ, et al. Obesity hypoventilation syndrome: hypoxemia during continuous positive airway pressure. Chest 2007; 131:1678.

9 patients with OHS plus OSA had to be placed directly on BPAP because initial CPAP failed

-(significant oxyhemoglobin desaturation and/or

-carbon dioxide retention),

emphasizing يؤكد the importance of close observation during the initiation of CPAP in patients with OHS.

Patients who benefit from nocturnal CPAP therapy tend to have a

1- higher baseline apnea hypopnea index (AHI),
2- less restrictive physiology on spirometry,
3- and less severe oxyhemoglobin desaturation during baseline polysomnography .

There are limited clinical data comparing CPAP to NPPV in patients with OHS.

One **trial** randomly assigned **36 patients** with :

-**coexisting** OHS and OSA,

as well as

-mild residual hypoventilation on CPAP,

they were put on either continue CPAP or BPAP

The trial defined

- **mild residual hypoventilation** as
  - nocturnal oxyhemoglobin desaturation to < 80 % for fewer than 10 minutes
  - and nocturnal CO_2 retention of less than 10 mmHg

After **three months**,

the patients had similar improvement in their **awake $PaCO_2$**, regardless of whether they received **CPAP or BPAP**

although subjective **sleep quality** and psychomotor **(vigilance**اليقظة)testing improved in the **BPAP group.**

The observations that nocturnal **CPAP** therapy **does not benefit all patients** with OHS suggest that **some patients with coexisting OHS and OSA** **probably have**

-abnormal control of breathing and
-persistent hypoventilation even when the upper airway is patent

Moreover,
the relative contributions of **the underlying** pathophysiologic **mechanisms** that lead to chronic hypoventilation probably differ among patients who benefit from CPAP compared to those who do not respond to CPAP .

Thus, patients who use nocturnal CPAP **should be monitored for**

-evidence that nocturnal **hypoventilation** still exists despite compliance with therapy.

Clinical symptoms and signs suggestive of persistent sleep-related **hypercapnia** include nocturnal dyspnea, a sensation of smotheringاختناق at night, chronic morning headaches, or failure of awake arterial blood gases to improve.

patients who **do not** improve with CPAP **will require BPAP**

Second line therapies

Noninvasive positive pressure ventilation
NPPV

Noninvasive positive pressure therapy (NPPV) is indicated for patients with OHS because untreated disease may result in

- –progressive hypercapnia,
- –hypoxemia, and numerous
- adverse effects, including poor neurocognitive function,
- –pulmonary hypertension,
- and cor pulmonale.

NPPV improves

- nocturnal alveolar ventilation and may also
- stabilize or improve daytime alveolar ventilation in patients with OHS .

available modes of NPPV

- BPAP and
- VCPPV
- Hybrid modes also exist.

Administering positive pressure

Advantages and disadvantages of the different methods for administering positive airway pressure

Mode of positive pressure ventilation	Advantages	Disadvantages
CPAP	Inexpensive	Lack of inspiratory pressure support
	Widely available	
Bi-level	Widely available	Tidal volume may may be limited by patient-related factor
	Can provide inspiratory pressure support to augment tidal volume	
	Leak tolerant	
Volume-cycled	Can set specific respiratory parameters	More expensive
		Less widely available
		Less well-tolerated than pressure support devices
		Leaks lead to loss of tidal volume

Bilevel positive airway pressure

During BPAP therapy, an inspiratory positive airway pressure (IPAP) and an expiratory positive airway pressure (EPAP) are independently titrated and set.

Tidal volume correlates with the **difference between the IPAP and the EPAP.**

As an example, tidal volume is greater using an IPAP of 15 cm H_2O and an EPAP of 5 cm H_2O (difference of 10 cm H_2O), than an IPAP of 10 cm H_2O and an EPAP of 5 cm H_2O (difference of 5 cm H_2O).

Alveolar ventilation is enhanced by a larger tidal volume, assuming that the respiratory rate is constantثابت.

Nocturnal BPAP therapy usually:

- decreases nocturnal $PaCO_2$,
- daytime $PaCO_2$, and
- daytime sleepiness in patients with OHS
- restored **sleep architecture** .
- It may also improve long-term survival .

Randerath W, Verbraecken J, Andreas S, et al. Definition, discrimination, diagnosis and treatment of central breathing disturbances during sleep. Eur Respir J 2017; 49.

BPAP probably offers several advantages compared to **CPAP**, These include :

1-active ventilation rather than pneumatic splinting,

2- a lower mean airway pressure (which may lead to **better tolerance** of the therapy),

3-better rest of the ventilatory muscles,

4-more rapid improvement of respiratory acidosis, and

5-a rapid return of normal **ventilatory control** or chemoreceptor function.

BPAP appears to be effective, even in the presence of a small leak at the patient interface, because the inspiratory airflow will continue until the target pressure is reached.

In the case of severe upper airway obstruction, the EPAP and IPAP can be simultaneously مباشرةincreased.

- Volume cycled Positive Pressure ventilation VCPPV

In some patients with OHS, upper airway obstruction or decreased respiratory system , **compliance** may be so severe that

sufficient alveolar ventilation cannot be achieved with CPAP or BPAP

- volume cycle positive pressure ventilation **(VCPPV) reserved** for when

sufficient alveolar ventilation cannot be achieved with BPAP.

VCPPV can be considered in this situation.

Grade 2C

VCPPV insures adequate ventilation by **generating pressures high enough** to overcome the physiologic limitations presented by the patient.

Although this modality may be useful during an episode of acute **decompensation**, the high interface pressures required limit acceptance of this mode as chronic therapy.

Short-term nocturnal VCPPV has been reported to improve

daytime respiratory failure, with most patients eventually "لاحقا"able to return to long-term CPAP or BPAP therapy.

Acute decompensated عدم المعاوضة OHS :

Patients who present with an acute decompensation of OHS should have noninvasive positive pressure ventilation (NPPV) initiated expeditiously بسرعة in a monitored inpatient setting,

assuming that they are acceptable candidates for this therapy

-able to cooperate,
-protect their airway,
-hemodynamically stable,
-arterial pH greater than 7.20

- **Bilevel** positive airway pressure (BPAP) is the mode of NPPV that is generally tried **first,**
- with volume cycle positive pressure ventilation **(VCPPV) reserved** for when sufficient alveolar ventilation cannot be achieved with BPAP.
- CPAP **should not** be used in this setting.

Grade 2C

There is no universal strategy for determining the initial settings

For BPAP,

-initial IPAP and EPAP are usually started at 8 and 4 cm H_2O, and then

- increase the IPAP every FIVE minutes in increments of 2 cm H_2O until the patient appears:

-more **comfortable** and there is an

- acceptable **respiratory rate** <30 breaths per minute,
- oxyhemoglobin **saturation** ≥ 90 % ,
- **heart rate** ≤ 100 beats per minute,
- and degree of ventilation **pH** >7.30 on serial arterial blood gases.

- maximum IPAP is typically 20 to 30 cm H_2O for adults.

For VCPPV,

We should select :

-the ventilator mode,

-respiratory rate,

- tidal volume,

- inspired oxygen concentration, and

-end-expiratory pressure.

An assist-control mode is typically used to fully augment spontaneous respiratory efforts.

-The **largest tidal volume** that consistently maintains an airway pressure **less than 30 cm** H_2O is generally chosen,

-and the respiratory rate is then set to achieve a **minute ventilation of 6 to 10 L/min.**

-The **respiratory rate** can be adjusted as needed to achieve the ventilatory goals **pH >7.30** on serial arterial blood gases.

-The inspired **oxygen concentration** should be titrated to maintain adequate oxyhemoglobin saturation (eg, ≥90 percent) and

-the end-expiratory pressure should be equivalent to the prior CPAP or EPAP settings.

-High interface pressures may cause sleep fragmentation, discomfort, intolerance, or an interface leak when VCPPV begins

In this situation, reducing the tidal volume and raising the respiratory rate will decrease the interface pressure while maintaining the desired minute ventilation.

In a retrospective series of patients having an **acute ventilatory decompensation** of OHS, use of NPPV successfully

-**averted** يجنب**endotracheal intubation** in all patients and was associated with

- **enhanced survival**

Chronic compensated OHS :

Patients who present with chronic compensated OHS or who have been stabilized after an acute decompensation should undergo

nocturnal polysomnography.

Polysomnography detects coexisting (OSA), which guides the initial **mode of therapy**.

It also allows the **titration** of positive airway pressure device settings.

Strategy for Patients with both OHS and OSA

- Patients with OHS and OSA can be initially treated with nocturnal **CPAP** .

- Beginning at a level of 4 cm H_2O, the CPAP is increased in small increments زيادات(eg, 2 cm H_2O) until obstructive events are eliminated

- Patients who have **persistent alveolar hypoventilation** despite the elimination of obstructive events (AHI)should be **converted from CPAP to NPPV.**

- This conversion is generally done using BPAP because the CPAP settings can be used as the starting point for the titration of BPAP.

- Beginning with IPAP and EPAP settings identical to the CPAP level at which obstructive events were eliminated,

Patients with OHS only

Patients with OHS alone should be treated with **nocturnal NPPV** because **CPAP is unlikely** to be effective in the absence of OSA.

- **Blood gas** analysis is the **gold standard** method of assessing alveolar ventilation.

- Frequent arterial blood gases may be required to verify that improvement in arterial gas exchange is occurring.

- However, **overnight blood gas analysis requires placement of an indwelling arterial catheter** or multiple arterial blood draws, both of which are impractical in most sleep laboratories.

- The **indicator of hypoventilation** that is most commonly used is **oxyhemoglobin desaturation** unrelated to obstructive events.

- Transcutaneous measurement of arterial carbon dioxide ($PaCO_2$) , calibrated respiratory inductance plethysmography, or end tidal CO_2 are alternative techniques, but they are controversial because their accuracy is uncertain.

Follow-up

- Once nocturnal positive pressure therapy has been initiated,
  periodic awake arterial blood gases are useful to verify that alveolar hypoventilation has improved.

- Repeat polysomnography should be considered in the following situations:

- There are symptoms or signs suggestive of **persistent alveolar hypoventilation** (eg, nocturnal dyspnea, a sensation of smothering at night, chronic morning headaches, failure of awake blood gases to improve) **despite** noninvasive positive pressure therapy.

- Persistent alveolar hypoventilation suggests that the **type or level of positive pressure therapy may need to be changed**

Supplemental oxygen

- Hypoxemia is common in patients with OHS, especially those with coexisting OSA

- Supplemental oxygen should be administered whenever positive pressure therapy alone is insufficient to eliminate hypoxia.

- Supplemental oxygen during sleep is titrated during polysomnography to eliminate hypoxemia or severe oxyhemoglobin desaturation after the optimal settings of positive pressure therapy

- Daytime supplemental oxygen can be titrated using oximetry at rest and with exertion.

Supplemental oxygen alone (without positive pressure therapy)
is inadequate therapy for OHS.
Although it may:

- improve nocturnal oxyhemoglobin desaturation,
- it does not relieve upper airway obstruction
- Doesn't augment ventilation,
- and it may acutely worsen carbon dioxide retention in patients with OHS

conclusion

Obesity Hypoventilation Syndrome (OHS) is a **serious underestimated disease** we should think of it when meeting **obese patient** with unexplained

hypoxia or

carbon dioxide retention , to diagnose and treat for better lives.

Thank you