

Monitoring of Non-Invasive Ventilatory Support in Overlap Syndrome

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Doi: 10.5958/j.0973-340X.7.1.002

Indian J Sleep Med 2012; 7.1, 6-10

Abstract

Sleep is associated with important changes in upper airway, lung mechanics as well as respiratory control. The significance of monitoring of various physiological parameters during sleep are discussed.

Sleep is associated with deep changes in upper airway resistance, respiratory control and lung mechanics. Sleep-related hypoventilation may also occur. In patients with sleep-disordered breathing (as in the case of overlap syndrome), abnormal respiratory events occur during sleep. Sleep-disordered breathing (mainly obstructive sleep apnoea [OSA]) and chronic obstructive pulmonary disease (COPD) are among the most common pulmonary diseases¹⁷. Overlap syndrome patients, who have a lethal combination of both these disorders, would seem to be the ideal candidates for non-invasive ventilation (NIV), since their standard treatment already involves the discomfort of positive airway pressure and they are chronically hypercapnic¹⁷. NIV settings, which are appropriate for ventilating patients who are awake, may not work appropriately during the night. Sleep itself increases non-intentional leaks, patient-ventilator asynchrony and periodic breathing or glottic closures. Hence, it becomes extremely important to monitor various physiological parameters during sleep. Although several guidelines from various professional societies are

in place, implementation of these guidelines may require clinician education, additional health care personnel, organizational change or additional resources (equipment or beds with cardiopulmonary monitoring) to ensure safe and appropriate application of non-invasive positive-pressure ventilation (NPPV) and continuous positive airway pressure¹.

Which Parameters Should Be Monitored?

Monitoring should include a blend of physiological and clinical parameters.¹⁶ These should be used to formulate a management plan and, within the first 4 h of NIV, to assist in the decision to the need to proceed to intubation.^{2,14,15} Staff involved in the care and monitoring of NIV patients should be appropriately trained and experienced. A host of parameters need to be recorded and be used to formulate a logical management plan. Baseline observations should include arterial blood gas (ABG), respiratory rate and heart rate. Continuous pulse oximetry and electrocardiogram should be recorded during the first 12 hours. Repeat ABG estimations may be carried out after 1 h of NIV therapy and 1 h after every subsequent change in settings. Further ABG estimations are performed at 4 hours or earlier in patients who are not improving clinically. Frequent clinical monitoring of acutely ill patients needs to be conducted every 15 min in the first hour, every 30 min in the 1- to

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4-hour period and hourly in the 4- to 12-hour period. Observations should include respiratory rate, heart rate, and level of consciousness, patient comfort, chest wall movement, ventilator synchrony and accessory muscle use. Patient comfort and enhanced compliance are key factors in determining the outcome of this therapeutic modality.¹² Synchrony of ventilation should be checked frequently. Clinical assessment of mask fit to include skin condition and degree of leak (particularly onto the corneas) should be performed at the same time. Table 1 summarizes the criteria included in monitoring patients undergoing NIV in acute and chronic care settings.

Table 1: Criteria Included in Monitoring of Patients Undergoing NIV in Acute and Chronic Care Settings

Acute Setting	Chronic Setting
<ul style="list-style-type: none"> • Patient comfort • Mask fit and leak • Patient–ventilator synchrony • Sternocleidomastoid muscle activity • Vital signs: heart and respiratory rate, systemic blood pressure • Continuous oximetry until stabilized • Occasional blood gas measurements: initial and after 30–120 min, then as clinically indicated 	<ul style="list-style-type: none"> • Patient comfort • Mask fit and leak • Hours of use • Problems with adaptation (e.g., nasal congestion, dryness, gastric insufflation, conjunctival irritation, inability to sleep) • Symptoms (e.g., dyspnoea, fatigue, morning headache, hypersomnolence) • Gas exchange: daytime, nocturnal oximetry, blood gases measured periodically to assess PaCO₂ • Polysomnography, if symptoms of sleep disturbance persist or nocturnal desaturation persists without clear explanation

Adaptation and Monitoring

In a COPD patient, a decrease in respiratory rate and reduction in sternocleidomastoid muscle activity are important physical signs that should be apparent early on and are commonly accompanied by good patient–ventilator synchrony. With long-term NPPV, the patient may be instructed to initiate NPPV at home for 1- or 2-h trial periods during the daytime and then to try to fall asleep

with the device at bedtime. Several methods have been described to monitor patients during sleep. They include pulse oximetry, capnography — the built-in monitoring software in the ventilator — and autonomic markers of sleep fragmentation. Various respiratory events take place during sleep in a patient undergoing NIV. Prominent among these are repetitive leaks, upper airway instability and residual obstructive events, recurrent decreases in ventilatory command (with/without closure of the glottis) and patient–ventilator asynchrony.^{3,19}

Physiological Basis of Upper Airway Patency and Ventilatory Drive During NIV

The following factors may form the physiological basis of upper airway patency and ventilatory drive during NIV:^{13,18,20}

- a) **Nasal obstruction** is an important factor.
- b) **Pharyngeal collapse:** Passive collapse of the upper airway results from the inability of positive pressure ventilation to stabilize the upper airway throughout the respiratory cycle. Out-of-phase or paradoxical thoracoabdominal movements are characteristic of these persistent efforts.
- c) **Glottic closure:** Adduction of the vocal cords resulting in progressive closure of the glottis has been described in response to ventilator-induced hyperventilation.
- d) **Ventilatory drive:** Ventilatory control is physiologically altered during sleep. There is usually a decreased responsiveness to chemical, mechanical and cortical inputs. If the NIV settings lead to hyperventilation, bursts of central apnoeas or hypopnoeas can occur, particularly during transitions between sleep onset and wakefulness.

Pulse Oximetry

The various advantages and disadvantages of pulse oximetry as a monitoring parameter are outlined in Table 2.³

The morphological pattern of SpO₂ desaturations cannot accurately separate central from obstructive events. Recurrent desaturations may reflect upper airway instability and residual obstructive events, decreases in ventilatory command (with or without glottic closure) as well as repetitive leaks interrupted by microarousals.³

Furthermore, in subjects with oxygen supplementation, pulse oximetry is unreliable for the detection of hypoventilation. Despite limitations, pulse oximetry is a valuable screening tool for patients established on home ventilation who do not appear to have any problems related to NIV and who are not receiving supplemental oxygen.³

Table 2: Advantages and Disadvantages of Pulse Oximetry in Monitoring Patients Undergoing NIV

Advantages	Disadvantages
<ul style="list-style-type: none"> • Simplicity of application • Short set-up time • Short time response (seconds) • Low cost • Ease of basic interpretation • Possible use in telemedicine programmes and transmission of devices with recordings by mail 	<ul style="list-style-type: none"> • Motion artefacts • Sensitivity to perfusion • Dependence on signal averaging time and sampling frequency (high-speed signal averaging also improves detection of motion artefacts) • Accuracy of SpO₂ measurements reported in sleep studies is 2–6% compared with 75–100% in case of SaO₂

Maintaining an adequate nocturnal SpO₂ most probably decreases secondary pulmonary hypertension, improves respiratory muscle function and, in patients with diurnal hypoxaemia, enhances survival. A reasonable goal would be to adjust ventilator settings to obtain a mean nocturnal SpO₂ ≥90%, with only <10% of the total recording time displaying SpO₂ <90% after correction of leaks.⁴

Oxygen supplementation should be provided only in case of ventilation/perfusion (V/Q) mismatch incompletely treated by NIV. Daytime arterial blood gases (ABGs) may reflect nocturnal values of arterial carbon dioxide tension (PaCO₂) poorly. An acceptable average SpO₂ may be associated with important nocturnal respiratory events or poor control of nocturnal hypoventilation. Thus, visual inspection of oximetry traces is important for the detection of nocturnal respiratory events.

Pulse oximetry (including visual inspection of traces) should be used as a screening tool in stable home-ventilated patients without supplemental oxygen to identify and exclude such patients who do not require more detailed, expensive and time-consuming investigations.³

Microarousals and Sleep Fragmentation

Microarousals are associated with momentary increases in sympathetic activity (heart rate, blood pressure, pulse

transit time and pulse wave amplitude [PWA]). PWA, measured by photoplethysmography, can be easily recorded by using conventional pulse oximeters. Peripheral vasoconstrictor responses associated with microarousals are visually identifiable by marked reductions in PWA.³

Non-Invasive assessment of nocturnal arterial CO₂ Tension

Repeated sampling of arterial blood does not reliably assess the control of nocturnal hypoventilation because the patient will usually wake up; in addition, there is a need for costly equipment, specially trained personnel, and an intensive care unit.^{3,14} A simple approach would be to measure PaCO₂ by arterial puncture at the end of the night, which would document night-to-morning increases in PaCO₂. However, blood is most often sampled after arousal; a normal morning PaCO₂ would not actually reflect the abnormal time course of PaCO₂ during the night.^{5,6} The relationship between PaCO₂ and PETCO₂ depends on the physiological dead space (V_D) and the patients' ventilatory mode (tidal volume or V_T): End-tidal PaCO₂ (PETCO₂) is estimated by the following equation: PETCO₂ = PaCO₂ (1 - V_D/V_T). During NIV, ventilation is not homogeneous, and the expired CO₂ does not reach an adequate plateau. It is technically difficult to measure these values with the continuous flow through the mask related to bi-level pressure support. Recent devices, which combine transcutaneous CO₂ (PtcCO₂) and SpO₂ in earlobe sensors, have been validated in acute care and chronic clinical settings.⁷⁻⁹ They are designed for continuous recording over 8-h periods without requiring recalibration and are feasible for routine use. However, all PtcCO₂ sensors have a lag time (approximately 2 min), which precludes the monitoring of rapid changes in PaCO₂ such as those that could be associated with recurrent apnoeas, hypopnoeas or brief leaks.¹⁰ It provides no information pertaining to the cause of hypoventilation (e.g., inappropriate settings and leaks).

Data Available from NIV Machines

Currently used NIV equipment includes data-recording devices inbuilt into the software.³ Such downloaded data can be differentiated into three different categories. The first is a synthesis report that displays the trends of each parameter recorded during a given period. This includes

compliance, ventilator settings, estimations (not absolute values) of leaks, tidal volume, respiratory frequency, minute ventilation and respiratory cycles triggered by the patient. The second category is a detailed data analysis in which raw data of a given parameter can be analyzed cycle by cycle. The third category is a polygraphic data analysis. In addition to the signals already stored by the device, physiological parameters such as oxygen saturation, heart rate and respiratory effort can be recorded and displayed by adding an external module connected to the machine.

Compliance and pattern of daily use of ventilators can thus be determined. These include average daily use of the ventilator, special patterns of ventilator use, suggesting inappropriate settings and patient discomfort (e.g., fragmented nocturnal use, multiple short periods of ventilator use).

Tidal volume (V_T), as reported by ventilator software, is an indicator of effective ventilation. However, it is subject to variability in accuracy depending on leaks. A high variability of V_T may suggest leaks or periodic breathing, either spontaneous or ventilator induced.

Respiratory rate (RR) analysis allows an estimation of the difference between spontaneous RR and the preset back-up RR. Back-up RR is usually set at approximately two breaths below spontaneous RR; this can be easily adjusted using RR monitoring by a ventilator.³

Leaks

The average value of leaks as well as its 95% percentile can be estimated from the data available from the NIV machine. Adjustment of interface and ventilator settings can be aimed at maintaining the 95th percentile of leak values under a predetermined threshold value.^{3,11}

Integrated Multichannel Modules

Certain bi-level ventilators can be coupled to multichannel modules, providing trend data on minute ventilation, respiratory rate, air leaks, SpO₂ and heart rate. Data can be stored on a memory card and transferred to a computer for simultaneous onscreen viewing.^{3,11}

Several criteria have been used to define ineffective ventilation. They are outlined in Table 3.

Table 3: Criteria to Define Ineffective Ventilation

- Leakage >24 L/min for >20% of the trace duration
- Prolonged desaturations (>30% of the tracing with an SpO₂ <90%), whether or not accompanied by a simultaneous reduction of minute ventilation (>10% reduction compared with baseline) in the absence of significant leaks
- Cumulative desaturation dips (>3%) for >10% of the trace duration

Polygraphy and Polysomnography Tracings of Patients During NIV

This modality is at times extremely useful in detecting leaks, changes occurring in ventilatory drive, upper airway patency and several other events occurring during sleep in patients undergoing NIV. The minimal prerequisites that must be met for tracing analysis include mask pressure, flow rate in the circuit measured close to the mask, thoracic and abdominal movements (piezoelectric strain gauges, respiratory inductive plethysmograph effort belts) and pulse oximetry (SpO₂). The optional parameters for monitoring of respiratory efforts are diaphragmatic electromyogram (EMG), cervical muscle EMG, oesophageal pressure, pulse transit time and suprasternal notch pressure.¹³

Conclusions

Physiological and pathological changes that are brought about by sleep on upper airway necessitate close monitoring of several parameters highly significant.

References

1. **Keenan SP**, Sinuff T, Burns KEA, Muscedere J, Kustogiannis J, Mehta S. et al. *CMAJ* 2011; 183(3):E195–214
2. **Elliot MW**. Non-invasive ventilation during sleep: time to define new tools in the systematic evaluation of the technique. *Thorax* 2011; 66:82–84.
3. **Janssens J-P**, Borel J-C, Pepin J-L. Nocturnal monitoring of home non-invasive ventilation: the contribution of simple tools such as pulse oximetry, capnography, built-in ventilator software and autonomic markers of sleep fragmentation. *Thorax* 2011; 66:438–445.
4. **Gonzalez C**, Ferris G, Diaz J, et al. Kyphoscoliotic ventilatory insufficiency: effects of long-term intermittent positive-pressure ventilation. *Chest* 2003; 124:857e62.
5. **Janssens JP**, Heritier-Praz A, Staneczak O, et al. Agreement between daytime measurement of arterial blood gases (ABG), nocturnal pulse-oximetry (NPO) and transcutaneous capnography in home mechanical

- ventilation. *Eur Respir J* 2002;20:155S.
6. **Paiva R**, Krivec U, Aubertin G, et al. Carbon dioxide monitoring during long-term noninvasive respiratory support in children. *Intensive Care Med* 2009; 35:1068e74.
 7. **Senn O**, Clarenbach CF, Kaplan V, et al. Monitoring carbon dioxide tension and arterial oxygen saturation by a single earlobe sensor in patients with critical illness or sleep apnea. *Chest* 2005; 128:1291e6.
 8. **Bendjelid K**, Schutz N, Stotz M, et al. Transcutaneous PCO₂ monitoring in critically ill adults: clinical evaluation of a new sensor. *Crit Care Med* 2005; 33:2203e6.
 9. **Parker SM**, Gibson GJ. Evaluation of a transcutaneous carbon dioxide monitor ("TOSCA") in adult patients in routine respiratory practice. *Respir Med* 2007; 101:261e4.
 10. **Janssens JP**, Howarth-Frey C, Chevrolet JC, et al. Transcutaneous PCO₂ to monitor noninvasive mechanical ventilation in adults: assessment of a new transcutaneous PCO₂ device. *Chest* 1998; 113:768e73.
 11. **Rabec C**, Georges M, Kabeya NK, et al. Evaluating noninvasive ventilation using a monitoring system coupled to a ventilator: a bench-to-bedside study. *Eur Respir J* 2009; 34:902e13.
 12. **Rabec C**, Rodenstein D, Leger P, Rouault S, Perrin C, Gonzalez-Bermejo J. Ventilator modes and settings during non-invasive ventilation: effects on respiratory events and implications for their identification. *Thorax* 2011; 66:170e178.
 13. **Gonzalez-Bermejo J**, Perrin C, Janssens JP, Pepin JL et al. Proposal for a systematic analysis of polygraphy or polysomnography for identifying and scoring abnormal events occurring during non-invasive ventilation. *Thorax* 2012; 67(6):546–552.
 14. **Pepin JF** et al. Sleep and NIV: monitoring of the patient under home ventilation. In: *Noninvasive ventilation*, 2nd ed, Muir JF, Ambrosino N, Simonds AK (Eds), *Eur Respir Monograph*, 2008, 41, 350–366.
 15. **Goldstein RS**, Davis L. The importance of overnight monitoring in the management of chronic respiratory failure. In: *Ventilatory Support for Chronic Respiratory Failure, Lung Biology in Health and Disease*, 2008, 225, N Ambrosino and RS Goldstein Ed; Informa Healthcare, 371–388.
 16. **Chawla R**, Khilnani GC, Suri JC, Ramakrishnan N, Mani RK, Prayag S, Nagarkar S, Kansal S, Sidhu US, Kumar V. Guidelines for non-invasive ventilation in acute respiratory failure. *Indian J Crit Care Med* 2006; 10(2):117–147.
 17. **Owens RL**, Malhotra A. Sleep-disordered breathing and COPD: the overlap syndrome. *Respir Care* 2010; 55(10):1333–1344.
 18. **Ahmed MM**, Schwab RJ. Chronic non-invasive positive pressure ventilation: considerations during sleep. *Sleep Med Clin* 2008; 3:557–568
 19. **Berry RB**. Non-invasive titration and treatment initiation in patients with chronic hypoventilation syndromes. *Sleep Med Clin* 2010; 5(3):485–505
 20. **Dempsey JA**, Veasey SC, Morgan BJ, et al. Pathophysiology of sleep apnea. *Physiol Rev* 2010; 90:47e112.