Leak Monitoring in Noninvasive Ventilation

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Nasal mask ventilation has been shown to be effective, but outcomes do not always match expectations because of mouth leaks, patient-ventilator asynchrony, or decreased upper airway patency. These developments are detected when they lead ultimately to circuit leaks that lower the effectiveness of ventilation through pressure loss, poor inspiratory triggering, and prolonged inspiratory time. The quality of sleep is affected, and adverse effects and treatment intolerance may arise.

A number of ways to detect leaks and their practical consequences are proposed in this article.

We applied 310 leak-detection procedures to 177 patients who had disappointing clinical, gasometric, or polysomnographic outcomes of ventilation. The leak-detection procedures varied according to the type of ventilation and the supposed underlying pathophysiological mechanism. Significant leaks were detected in 132 patients (76%); therapeutic changes were then prescribed to optimize outcomes.

We present a practical method to apply in patients with suboptimal ventilation outcomes. If leaks can be detected during treatment, the probable cause of treatment failure can sometimes be established and possible pathophysiological mechanisms better understood. With this knowledge, it may be possible to improve ventilation.

Key words: *Noninvasive ventilation. Monitoring. Leaks. Respiratory insufficiency. Sleep apnea-hypopnea syndrome.*

Monitorización de las fugas en ventilación no invasiva

La ventilación por mascarilla nasal ha dado sobradas pruebas de su eficacia. Sin embargo, en ciertos casos los resultados no son los esperados. Tres mecanismos pueden explicar estos fallos: apertura bucal, desincronización paciente-respirador y disminución de la permeabilidad de la vía respiratoria superior. Éstos pueden detectarse por su manifestación última: las fugas en el circuito, que reducen la eficacia de la ventilación (fallo de presurización, disfunción del *trigger* inspiratorio y prolongación del tiempo inspiratorio), alteran la calidad del sueño y producen efectos adversos e intolerancia al tratamiento.

Proponemos aquí varias técnicas de detección de fugas y sus consecuencias prácticas.

Se sometió a 177 pacientes, con resultados de la ventilación inferiores a los esperados (clínicos, gasométricos o poligráficos), a 310 procedimientos de detección de fugas, con montajes que variaron según la modalidad ventilatoria y el mecanismo fisiopatológico juzgado como responsable. Se detectaron fugas significativas en 132 pacientes (76%), lo cual impuso modificaciones terapéuticas para optimizar los resultados.

Presentamos un método de aplicación práctica en casos en que se asista a resultados insuficientes de la ventilación. La detección de fugas bajo tratamiento ofrece la posibilidad de establecer la causa probable del fracaso, comprender el mecanismo fisiopatológico potencialmente responsable e intervenir en consecuencia.

Palabras clave: *Ventilación no invasiva. Monitorización. Fugas. Insuficiencia respiratoria. Síndrome de apnea del sueño.*

Introduction

The extensive development of nasal mask ventilation (NMV) has led to a considerable increase in intermittent positive pressure ventilation treatment for patients with chronic hypercapnic respiratory insufficiency and in continuous positive airway pressure (CPAP) therapy for patients with obstructive sleep apnea/hypopnea syndrome (OSAHS). The effectiveness of these treatments is no longer questioned. However, their widespread use has brought to light that a certain number of patients have unsatisfactory or unsuccessful outcomes with these treatments despite correct application. The failure rate varies according to the type of NMV and is estimated to be between 5% and 40%.[1] NMV differs from ventilation by tracheal intubation or cannulation in two respects which might explain the lack of success. First, the system is not airtight and, second, the ventilator-lung system cannot be considered a single compartment because of the variable resistance of the upper airway that is also present.[2] In effect, NMV differs

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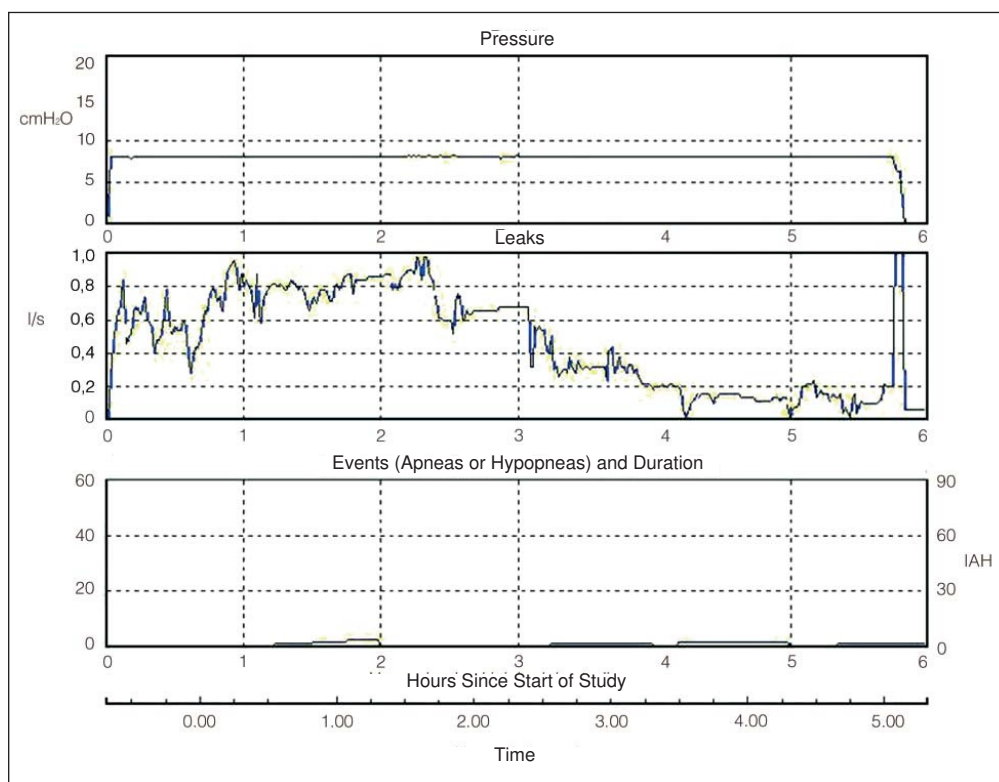
Figure 1. Leak detection in a patient on continuous positive airway pressure therapy —nighttime polygraph trace of a patient with sleep apnea-hypopnea syndrome. Note the presence of leaks greater than 0.4 L/s for most of the night at the preset optimal pressure (7 cm H_2O). AHI indicates apnea-hypopnea index.

essentially from invasive ventilation in that there is a discontinuity between the ventilator and the airway. This peculiarity—and its potential consequence, that is, leaks in the system—may explain failures with NMV to a large extent. In the case of OSAHS, leaks may render treatment ineffective if CPAP therapy (which effectively involves continuous pressure generation) does not manage to sufficiently pressurize the circuit and stabilize the upper airway. Adequate pressurization is essential to neutralize the obstructive episodes. In the case of intermittent positive pressure therapy, the fact that the system is not airtight will lead to different consequences depending on the type of ventilator used. With a volume ventilator, the entire volume generated may not reach the patient, whereas with bilevel pressure nasal ventilation (BPNV) (BiPAP® or similar systems), the ventilator may not be able to offer the desired pressure support.

For NMV to be effective, the upper airway must be patent, the interface must be correctly applied, the mouth must be kept closed, and the patient and ventilator must be synchronized. If one or more of these conditions are not met, ventilation is imperfect, whether clinically (no improvement), paraclinically (no correction of blood gases), or instrumentally (unsatisfactory nighttime polygraph or oximetric traces during ventilation).

If the technique is working correctly with no leaks at the interface between the mask and the patient, 3 mechanisms may reduce the effectiveness of NMV, namely, reduced upper airway patency, mouth opening, and patient-ventilator asynchrony. These 3 mechanisms

are detected when they lead ultimately to circuit leaks. When the circuit is sufficiently sealed and the mask is adapted to the patient, these leaks can be assumed to be secondary to mouth leaks.

Thus, it would be instructive to develop noninvasive techniques to detect and quantify these potential leaks and to establish the underlying mechanism. This article draws on our own experience to propose several techniques for leak detection and to highlight the practical consequences of such detection.

Material and Methods

The methods used for leak detection depended on the type of ventilation used by the patients.

For patients treated with CPAP for OSAHS, we initially used the hospital version of the Autoset Resmed system (Resmed Ltd., North Ryde, Australia) before changing to the Autoset T system (Resmed Ltd., North Ryde, Australia). These systems can record various parameters during ventilator use, and can also estimate leaks from measurement of the "intentional leaks" through expiratory ports in the nasal masks which the system recognizes by settings for given pressures. The leaks detected are recorded and a plot can be made of the whole ventilation period.

When a mouth leak was suspected, we used these systems at the preset pressure (optimal pressure) to record the parameters mentioned above. With these data, we could then quantify the circuit leaks. Leaks larger than 0.4 L/s are defined by the manufacturer as excessive leaks, that is, leaks able to compromise the effectiveness of treatment (Figure 1).
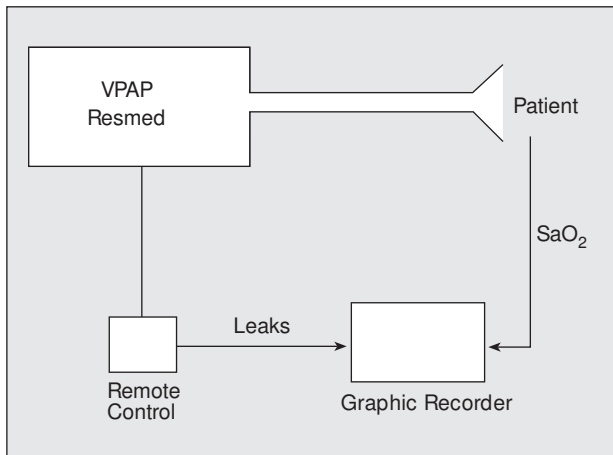
Figure 2. Set-up for leak detection in patients with bilevel pressure nasal ventilation. SaO$_2$ indicates arterial oxygen saturation.

Ltd., North Ryde, Australia) with a liquid crystal display can be added to the system for remote titration and monitoring of several parameters, including circuit leaks in excess of the intentional leak. This remote control has several analogue interfaces for real-time data acquisition. A serial connection to a multichannel graphic recorder (Linseis Gmbh, Selb, Germany) can plot leaks in real-time on graph paper (Figure 2). We used a two-channel system coupled to an oximeter (Datex-Ohmeda Corp., Helsinki, Finland) with an analogue interface that allowed simultaneous display with the arterial oxygen saturation curve. Figure 3 shows 2 examples of traces obtained with this set-up. The criteria for defining leaks as significant were the same as those used for patients treated with CPAP for OSAHS.

In patients treated with a volume ventilator and in some ventilated with BPNV, we used a graphic recorder of respiratory variables with a computerized polysomnograph (Respisomnograph, Mallinckrodt Nellcor Puritan Bennet, St. Louis, MO, USA) which simultaneously recorded the
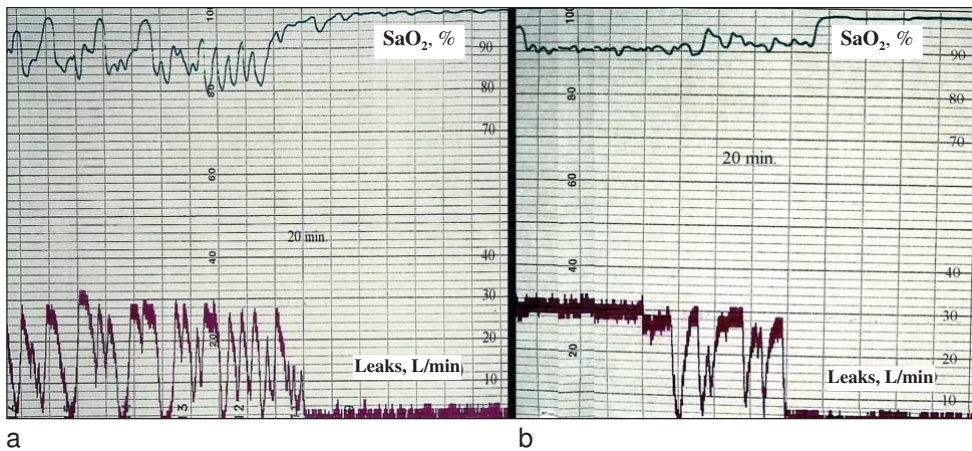

Figure 3. Simultaneous recording of leaks and arterial oxygen saturation (SaO$_2$) trace during sleep in patients on bilevel pressure nasal ventilation with a VPAP II ST device.
a) **Desaturation peaks "mirroring" intermittent leaks; and** *b)* **regular desaturation resulting from a permanent leak. (The traces read from right to left.)**
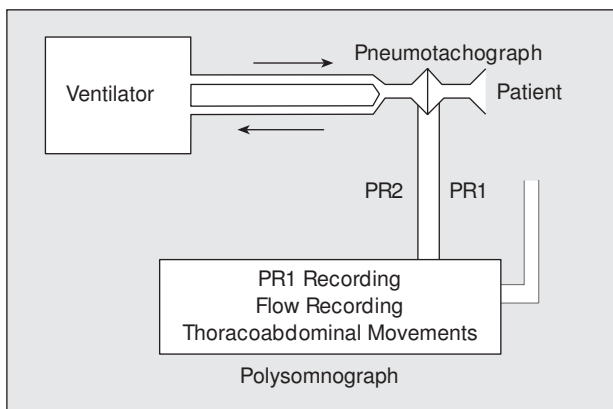

Figure 4. Respiratory polygraph set-up for leak detection in patients with volume ventilation or bilevel pressure nasal ventilation. PR1 and PR2 indicate circuit pressures.

following parameters: *a)* airflow through a Fleisch pneumotachograph connected between the mask and the expiratory valve; *b)* thoracoabdominal movements measured by elastic belts coupled to piezoelectric sensors; and *c)* pulse oximetry. This system—already described by other groups[3,4]—allows simultaneous recording of the flow insufflated by the ventilator (constant in the case of volume ventilation and decreasing with pressure-limited ventilation), the flow expired by the patient, the pressure in the mask, and thoracoabdominal movements. The system is shown schematically in Figure 4.

Leaks are revealed through a decrease in expiratory flow with respect to flow insufflated by the ventilator. This is usually accompanied by a decrease in the amplitude of thoracoabdominal movements. If passive mouth opening were the primary leak mechanism, the leak would cause the pressure in the mask to decrease with respect to normal cycles from the start of insufflation, and depressurization would occur (Figure 5a). If, on the other hand, the mouth leaks were related to an increase in pressure in the circuit, due to a decrease in the upper airway patency for example, the mask pressure would be higher than normal from the start of insufflation and would drop quickly when leaks occurred (Figure 5b). In such instances, we assumed that the probable mechanism for pressure increase was reflex glottic closure

We therefore considered leaks greater than 0.4 L/s for at least 10% of the trace to be significant.

We used the VPAP II ST system (Resmed Ltd., North Ryde, Australia) in patients treated with BPNV (BiPAP® or similar systems). A remote control module (UCU 1, Resmed

during ventilation, as described by Jounieaux et al[3,5-7] (see Discussion, below), but here we use the term "inspiratory block" given that our method cannot determine the underlying pathophysiological mechanism.

Results

From 1998 onwards, our group started to use this method systematically for detecting leaks whenever partial or total ventilatory failure occurred. Between June 1998 and December 2001, 177 patients—119 men and 58 women with a mean (SD) age of 67(10.9) years—underwent 331 leak detection procedures (1.87[1.2] procedures/patient) that varied according to the type of ventilation and probable pathophysiological mechanism.

Detection of Leaks in Patients With Nasal Continuous Positive Airway Pressure

The method described earlier was used 153 times in 100 patients (1.64[0.9] traces/patient). All patients had OSAHS, diagnosed by polysomnography, and were candidates for CPAP treatment according to consensus criteria.[8] Nine patients (9%) also had chronic obstructive pulmonary disease, constituting an overlap syndrome according to the definition of Flenley.[9] A further 2 patients had restrictive syndrome related to

thoracoplasty sequelae and one had kyphoscoliosis. No patients had daytime hypercapnia. In 5 patients, the sleep apnea syndrome had a significant central component. CPAP titration was performed during a night of therapy in the hospital using an automatic titration system (Autoset T) and the recorded optimal pressure was subsequently verified by complete nighttime polysomnography in the hospital. Our experience and that of others is that this titration method is more reliable for calculating the optimal CPAP than is manual titration under polysomnographic control.[10-13] The interface used in all cases was a factory-made nasal mask adapted for each patient. The initial preset pressure used by the patient population was 11.4(1.9) cm H_2O. Table 1 shows the reasons that led to investigation of leaks in these patients.

Significant leaks, according to the definition given above, were detected in 69 patients (69%). The results of leak detection in these patients and subsequent therapeutic interventions are presented in Figure 6. It is of note that 4 of the patients treated initially with CPAP changed to BPNV because of hypercapnia. Three of these were also retrospectively classed as suffers of obesity-related hypoventilation, which presented in one of them as overlap syndrome. In all 4 cases, leak detection was subsequently performed during BPNV, and the patients were included in the following section.
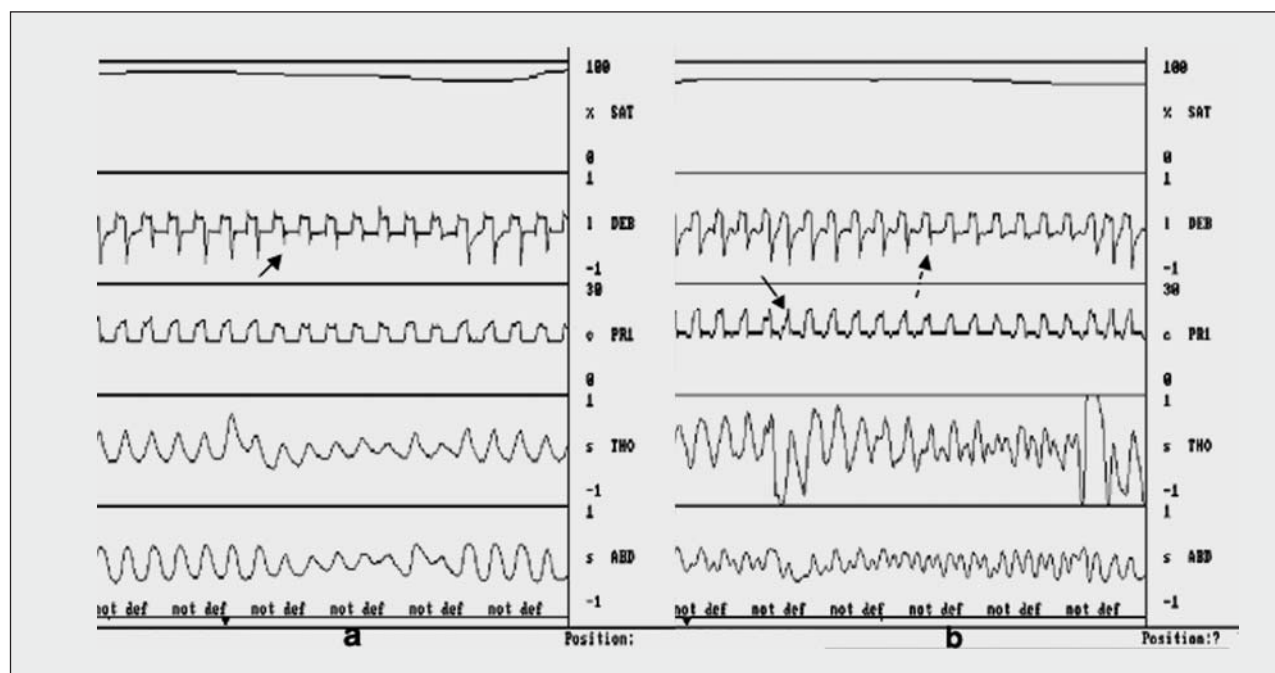

Figure 5. Respiratory polygraph traces under volume ventilation in assist-control mode (1-minute pages). *a)* "Primary" leaks caused by mouth opening—the circuit leak is evidenced by the decrease in expiratory flow (DEB) (rising part of the curve), whereas the inspiratory flow (falling part of the curve) is maintained (arrow). The circuit pressure (PR1) drops at the same time, proving that the circuit is no longer airtight because of "primary" leaks; and *b)* secondary leaks due to "inspiratory block"—in this case the pressure rises sharply (solid arrow) from the start of the cycle (normally it rises steadily during insufflation) and the expiratory flow is accelerated, showing that insufflation is obstructed. The expiratory flow (DEB) and pressure decrease when leaks occur as a result of overpressure in the circuit (dashed arrow). In both cases, thoracoabdominal expansion decreases with subsequent oxygen arterial saturation (SAT). THO indicates thoracic movements; ABD, abdominal movements.
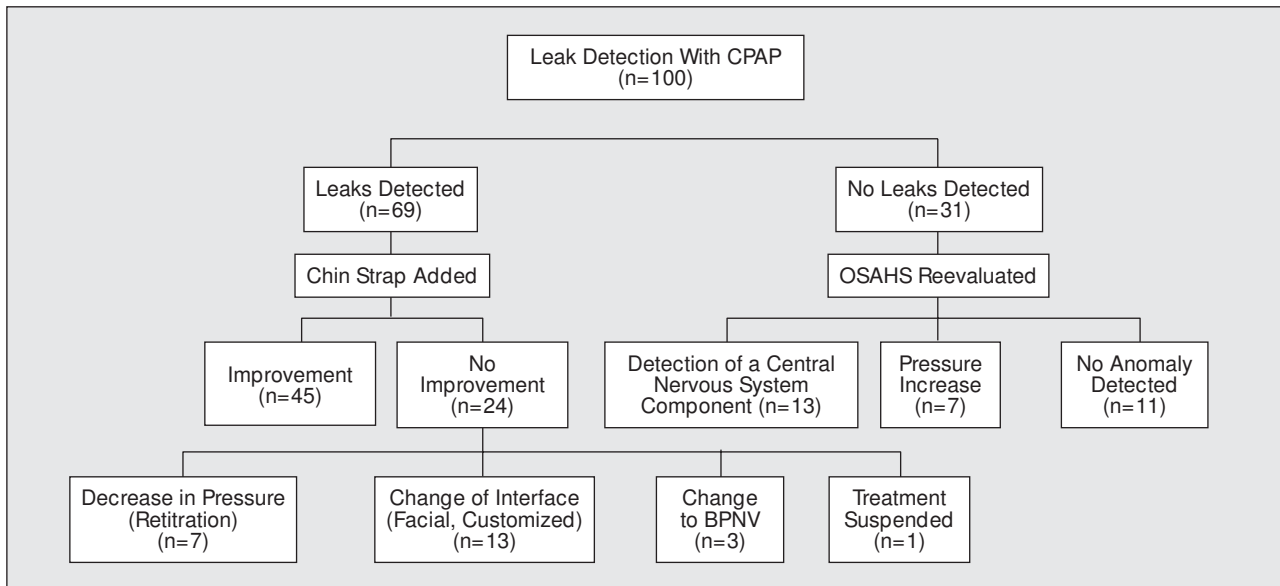
Figure 6. Results of the leak investigation and subsequent therapeutic modifications in patients on nasal continuous positive airway pressure (CPAP) therapy. OSAHS indicates obstructive sleep apnea-hypopnea syndrome; BPNV, bilevel pressure nasal ventilation.

Leak Detection in Patients Treated With Bilevel Pressure Nasal Ventilation

The leak detection protocol was followed 141 times in 75 patients (1.9[1.3] traces/patient). Most patients were being ventilated in spontaneous-time, assist-control mode. The mean titration values used were inspiratory positive airway pressure, 17.3(2.4) cm H_2O; expiratory positive airway pressure, 5.16(2.8) cm H_2O; and respiratory rate at rest, 12.2(2.33) cycles/min. In 25 patients (34%), at least one of the procedures was performed in our respiratory intensive care unit during acute noninvasive ventilation because we suspected that leaks might be reducing the effectiveness of ventilation. The interface used in all cases was a factory-made nasal mask chosen personally for each patient. The various diseases causing the respiratory insufficiency that led to prescription of BPNV in patients who underwent a leak detection procedure are shown in Table 2. The reasons that led to a search for leaks in these patients can be found in Table 1.

Leaks considered to be significant in at least one of the traces were found in 57 patients (76%). In 7 of these, the presence of leaks and the shape of the persistent desaturation peaks during ventilation, with no documented obstructive apneas during spontaneous ventilation, led us to suspect reflex glottic closure as the cause (see Discussion, below).

Changes in therapy as a result of leak detection findings are shown in Table 3.

The measures adopted in response to leaks depended on the suspected mechanism responsible for insufficient ventilation. A chin strap was used when "primary" leaks were suspected, the difference between inspiratory and expiratory pressures (Δ pressure) was decreased when reflex glottic closure during ventilation was suspected

TABLE 1
Reasons for Leak Detection, Classed According to Methods Used*

Reason	No. of Traces	Percentage of All Traces
Patients with CPAP†	153 (100)	
Repeated desaturation peaks or uncompensated SaO_2 at the original pressure	81 (51)	52.9
Mouth dryness or leaks indicated by the patient or those responsible for him or her	34 (25)	22.2
Persistence of excessive daytime somnolence	22 (16)	14.3
Intolerance of CPAP	19 (12)	12.4
Regular periods of desaturation with CPAP	5 (4)	3.2
Patients with BPNV‡	141 (75)	
Repeated desaturation peaks or uncompensated SaO_2 during NMV	71 (39)	50.3
Periods of regular desaturation during NMV	33 (14)	23.4
Persistence of alveolar hypoventilation ($PaCO_2$>50 mm Hg) during NMV	31 (19)	21.9
Intolerance of ventilation	10 (6)	6.0
Patients with BPNV or volume ventilation (respiratory polygraph set-up)	37 (13)	
Repeated desaturation peaks or uncompensated SaO_2 during NMV	6 (4)	16.2
Persistence of alveolar hypoventilation ($PaCO_2$>50 mm Hg) and/or regular desaturation during NMV	11 (4)	29.7
Both findings in the same patient	20 (5)	54.1

*Data expressed in percentages (number of patients). CPAP indicates continuous positive airway pressure; SaO_2, oxygen arterial saturation; BPNV, bilevel pressure nasal ventilation; NMV, nasal mask ventilation.
†Multiple reasons in 8 of the patients.
‡Multiple reasons in 3 of the patients (4 traces).

TABLE 2
Classification by Underlying Disease of Patients With Bilevel Pressure Nasal Ventilation Who Underwent a Leak Monitoring Procedure*

Diagnosis	No. of Patients, %
COPD[†]	19 (25.6)
Overlap syndrome	19 (25.6)
OSAHS combined with obesity–related hypoventilation[‡]	13 (17.5)
Tuberculosis sequelae (parietal)	8 (10.8)
Kyphoscoliosis[§]	5 (6.7)
OSAHS complicated by alveolar hypoventilation[‖]	4 (5.4)
Myopathies	3 (4)
Obesity-related hypoventilation[¶]	2 (2.7)
Lateral amyotrophic sclerosis	1 (1.3)

*COPD indicates chronic obstructive pulmonary disease; OSAHS, obstructive sleep apnea-hypopnea syndrome.
[†]Defined according to criteria in the Global Initiative for Chronic Obstructive Lung Disease (GOLD).[14]
[‡]Patients with OSAHS who remained hypercapnic when receiving continuous positive airway pressure, indicating that hypercapnia is due, at least partly, to another underlying mechanism, that is, obesity.
[§]Three patients also met criteria of COPD and one had OSAHS.
[‖]OSAHS complicated with alveolar hypoventilation—patients with OSAHS whose hypoventilation depends only on this entity (normalization of arterial carbon dioxide pressure with continuous positive airway pressure).
[¶]Patients with hypoventilation due exclusively to obesity. Other groups denominate this "obesity-hypoventilation syndrome" but, given that the scope of this term is not clear in the literature, we prefer the term "obesity-related hypoventilation."[15-17]

and increased when there was evidence of residual hypoventilation, the expiratory pressure level was increased when uncompensated OSAHS was suspected, or the type of ventilation was changed if this improved outcome.

TABLE 3
Therapeutic Modifications According to the Findings From Leak Monitoring in Patients on Bilevel Pressure Nasal Ventilation*

Therapeutic Modification	No. of Patients, %
Presence of leaks[†]	57
Use of a chin strap	36 (63)
Modification of ventilatory parameters	
Reduction in Δ pressure	6 (10.5)
Increase in Δ pressure	3 (5.2)
Increase in PEP	3 (5.2)
Successive modifications of different ventilatory parameters	5 (8.8)
Change of interface	
For a face mask	9 (15.8)
For a customized nasal mask	1 (1.7)
Replacement by CPAP	2 (3.5)
Suspension of treatment	3 (5.2)
Absence of leaks	15
Replacement by a volume ventilator	2 (13.3)
Modification of ventilatory parameters	
Increase in PEP	2 (13.3)
Increase in pressure support	3 (20.0)
Change of interface	
For a customized mask	2 (13.3)
No modification	6 (40.0)

*The trace could not be interpreted in 3 patients. Δ pressure indicates pressure difference between positive inspiratory pressure and positive expiratory pressure; PEP, positive expiratory pressure; CPAP, continuous positive airway pressure.
[†]Multiple modifications in 11 patients.

TABLE 4
Classification by Disease of Patients With Nasal Mask Ventilation Who Underwent Leak Monitoring With a Respiratory Polygraph Set-up*

Diagnosis	No. of Patients
Tuberculosis sequelae (parietal)	5
Kyphoscoliosis	3
COPD	1
Overlap syndrome	1
Myopathies	1
Cerebral palsy	1
Amyotrophic lateral sclerosis	1

*See Table 2 for definitions. COPD indicates chronic obstructive pulmonary disease.

Leak Detection in Patients With Volume Ventilation or Bilevel Pressure Nasal Ventilation Using Respiratory Polygraphy

The device described earlier was used 37 times in 13 patients (2.84[1.3] traces/patient). The polygraph traces were recorded during volume ventilation in 4 patients and during BPNV in 5 patients. The 4 remaining patients were evaluated on volume ventilation and BPNV successively. The mean titration values used for patients on BPNV were 15.6(3.7) cm H_2O for inspiratory positive airway pressure, 5(2) cm H_2O for expiratory positive airway pressure, and 13.8(2.1) cycles/min for basal respiratory rate. The mean titration values for patients on volume ventilators were 636(80) mL for tidal volume and 14.3(1.7) cycles/min for respiratory rate. Seven patients on BPNV had already undergone evaluation with the system described in the previous section. The diagnoses that led to the monitored patients receiving NMV are given in Table 4. The reasons for searching for leaks can be found in Table 1. Significant leaks were detected in at least one of the traces in 12 patients. In all cases, a detailed analysis of the respiratory polygraph traces according to the method described above allowed us to determine which leaks were caused by an increase in pressure in the airway. Secondary leaks indicated that the probable mechanism was reflex glottic closure. Therapeutic changes were made according to the same criteria as those named in the previous section and are given in Table 5.

Discussion

Leaks are common in patients with NMV[18-20] and comprise one of the main problems with this treatment because they can reduce the effectiveness of ventilation and treatment compliance. Generally, a leak of less than 0.4 L/s can be tolerated.[21,22] Leaks can arise due to passive mouth opening (known as "primary leaks" or "passive leaks") or as a result of an increase in airway resistance (known as "secondary leaks" or "reflex obstruction during ventilation").[21] Oropharyngeal closure mechanisms regulated by downward movement of the soft palate and mouth closure can prevent the first

TABLE 5
Therapeutic Changes According to Findings in Patients Who Underwent a Respiratory Polygraph Procedure*

Therapeutic Change	No. of Patients
Use of chin strap	2
Decrease in Δ pressure[†]	3
Increase in PEP[†]	1
Decrease in tidal volume[‡]	3
Change in type of ventilation	2
Replacement by oxygen therapy	1
Change in interface for face mask	1

*Δ pressure indicates difference between positive inspiratory pressure and positive expiratory pressure; PEP, positive expiratory pressure.
[†]Patients on bilevel pressure nasal ventilation.
[‡]Patients on volume ventilation.

type of leak and so ensure effective NMV in almost all patients. It has been suggested that these mechanisms may be mediated by central nervous system triggering of muscle activity to prevent leaks.[18] Thus, mouth leaks may arise because of masseteric hypotonia or because the soft palate and mouth musculature are unable to counteract the overpressure caused by insufflation. With regard to "secondary leaks," several years ago, Delguste et al[23] reported episodes of upper airway obstruction in patients with NMV. Obstruction occurs in the glottic region, as subsequently demonstrated by the same group in healthy volunteers through measurement of glottic opening during ventilation.[5] Such episodes, reported for both volume and pressure-limited ventilation, appear as hypopneas or central apneas in the polygraph traces (characterized by a reduction in the effective volume with a decreased or nonexistent thoracoabdominal signal despite the obstructive cause of the leak). Alternatively, a pattern similar to periodic breathing may be seen ("checkerboard" appearance or waxing-waning pattern of the thoracoabdominal signal) corresponding to cyclic closure and opening of the glottis.[3,5,7] It has been proposed that these represent a reflex response to hyperventilation. Many pathophysiological mechanisms have been proposed, including a central reflex inhibition triggered by relative hypocapnia, stimulation of baroreceptors in the airway as a result of positive pressure, or a reflex arc mediated by thermoreceptors that respond to insufflated air at a lower temperature as a result of a decrease in the effectiveness of humidity and temperature regulation by the nostrils at high air flows.[5,21] In central inhibition, therefore, efferent signals are thought to be sent to the glottis as well as the respiratory muscles, thus producing a reflex decrease in diameter. Such a system would comprise a truly complex reflex arc. In this situation, an increase in the volume delivered by the ventilator would, paradoxically, lead to a decrease in the effectiveness of ventilation, with the subsequent increase in leaks in the circuit because the effective tidal volume is the result of a complex interaction between the applied pressure, airway resistance, elasticity of the system, and inspiratory time.[3] The glottis, which influences airway resistance, would play a critical role in the effectiveness

of the NMV and, given that one of the characteristics of this technique is that the system is not airtight, an increase in resistance would lead to leaks. The resulting apneas and hypopneas can be detected by the presence of desaturation peaks during NMV.[7,18] Oximetry, which we use systematically to monitor patients with NMV, forms a fundamental part of screening for leak detection. In our experience, these desaturation peaks during ventilation, probably resulting from "inspiratory block," appear frequently in the first days of noninvasive ventilation or in patients on long-term ventilation with acute decompensation. Desaturation peaks, however, are much less common in ventilated patients who are stable. These episodes end spontaneously with hyperventilation, which may arise from 2 different mechanisms. There may be no inspiratory activity of the patient (in which case variation corresponds exclusively to changes in glottic opening—probably a reflex response to the change in $PaCO_2$). Alternatively, there may be recovery of the electromyographic inspiratory activity (in this case, disruption of central inhibition would affect inspiratory muscles and glottic muscles at the same time), possibly accompanied by an arousal.[3,18,21] Thus, episodes of glottic closure could be at least partly ruled out as the cause of poor quality of sleep suffered by patients with leaks.

These observations confirm that the upper airway muscles and the respiratory muscles have the same function in this situation and that they have an important efferent function in ventilatory control.[5]

Our findings have led us on numerous occasions to reduce the tidal volume or insufflation pressure. An increase in positive expiratory airway pressure could theoretically be useful by stabilizing the upper airway (according to the pathophysiological model of OSAHS) and thus obstructive phenomena would be prevented. In practice however, the measure has not been effective, as the pressure levels have been insufficient to keep a cartilaginous structure such as the glottis open. In contrast, in cases of a "true" additional OSAHS that was not diagnosed when NMV started, the increase in positive expiratory pressure was useful in our experience for neutralizing obstructive episodes.

Leaks influence the effectiveness of NMV in a number of ways, and their negative effect on the effectiveness of ventilation varies according to the type of ventilator. Volume ventilators are unable to compensate for leaks, so the volume received by the patient is affected. This shows up in practice as a pattern of regular desaturation in the oximetric trace during NMV[18] (Figure 3). In the case of ventilators that provide pressure support, leaks affect ventilation less because the ventilators are able to compensate by increasing the turbine speed.[24,25] Most ventilators offering pressure support are able to compensate a leak of less than 0.4 L/s above the intentional leak,[22] but larger leaks may affect the ability of the system to pressurize the circuit. Finally, in the case of CPAP, a large leak can reduce the effectiveness of treatment if

the system does not manage to pressurize the circuit properly and it may also lead to treatment intolerance.

With a large leak, the system may not trigger correctly in response to the inspiratory effort of the patient during the inspiratory phase (trigger failure).[22] Most bilevel pressure ventilators respond appropriately even when leaks are present, but response varies according to ventilator.[24] Trigger failure substantially reduces the effectiveness of ventilatory support, in turn increasing respiratory effort, asynchrony, and the number of arousals.

Barometric ventilators with pressure support cease inspiratory assistance when there is a preset drop in peak percentage flow. With small leaks, the system does not detect the decrease in flow and continues support, leading to a prolongation of inspiratory time and subsequent patient-ventilator asynchrony, and an increase in respiratory effort.[1,26-28] This may have important consequences, above all in patients with chronic airway obstruction, because hyperinflation may result from the reduced expiratory time, and the inspiratory trigger failure caused by the leak may be aggravated.[24,29] However, it should be noted that most new portable ventilators with pressure support limit this problem by allowing a maximum inspiratory time to be established or by using a set inspiratory time.[26] Excessive leaks can also generate autocycling in some ventilators because the system interprets the leak as the start of inspiration by the patient.[30]

Mouth leaks essentially alter the 3 main elements of good patient-ventilator synchrony, namely, the inspiratory trigger, the pressurization capacity of the system, and the end of support.

Episodes of mouth leaks often follow arousals. Arousals are more frequent during light sleep (stages I/II) and reduce the quality of sleep by causing fragmentation.[1,18-20] On the other hand, it has been clearly shown that sleep disruption has, in turn, negative effects on ventilatory control because the response to hypoxia and hypercapnia is reduced, and thus a vicious circle develops.[31]

Leaks cause discomfort and treatment intolerance in patients with NMV. Odynophagia, dry mouth, eye irritation, nasal symptoms, and noise may result, all of which reduce therapeutic compliance.[1,20,22] Furthermore, it has been shown that, in the presence of leaks, there is an increase in nasal resistance, which reduces the effectiveness of ventilation.[22,32,33] When NMV fails, the causes often implicated are lack of cooperation from the patient, mask or pressure intolerance, or an inappropriate patient screening for ventilation. Poor interaction between patient demand and ventilator response is rarely mentioned, though a leak in such a situation poses a greater problem.[26,30]

When pressure-support ventilation is being used, the greater flows needed to pressurize the circuit in the presence of leaks can make it difficult to obtain an inspiratory oxygen fraction suitable for the needs of the patient who needs supplemental oxygen. The

TABLE 6
Consequences of Leaks in Nasal Mask Ventilation*

Inconsequential leaks (of around 0.4 L/s)
   Discomfort and treatment intolerance
   Adverse effects (eye irritation, mouth dryness, nasal symptoms)
   Failure in the reliability of calculation of optimal pressure of automatic CPAP titration systems
   Poor functioning of automatic CPAP titration systems
   Decrease in ventilation effectiveness (volume ventilation)
Significant leaks
   Impossible to maintain optimal pressure in treatment with CPAP
   Sleep fragmentation
   Decrease in ventilation effectiveness (pressure-support ventilation)
   Inspiratory trigger failure
   Prolongation of inspiratory time (end of inspiration of the patient not detected)
   Greater requirement of oxygen in the circuit

*CPAP indicates continuous positive airway pressure.

inspiratory oxygen fraction obtained in these cases depends on factors such as the mixing of air supplied by the system and the oxygen in the circuit. If greater flow is needed to pressurize the circuit, high oxygen concentrations are harder to reach even with high flow supplements.[22]

Finally, in the specific case of CPAP titration in sleep apnea, a large leak can cause poor functioning of automatic titration systems. Generally, titration is considered acceptable for the calculation of optimal pressure in this type of device if the leak is less than 0.4 L/s.[11] If these devices are used for calculation of the optimal pressure of CPAP, the reliability of the titration may be affected. Poor functioning takes on even greater importance in patients treated at home with automatic CPAP systems, given that leaks can greatly reduce the effectiveness of treatment.

Several options are available for reducing or neutralizing leaks. These include chin straps, devices to support the chin, or adhesive tapes to ensure mouth closure, face masks, and ventilation through mouth interfaces.[20] These measures are only effective, however, if the leaks are due mainly to passive mouth opening. The use of chin straps have been reported to reduce the size of the leak by less than 50% on most occasions,[22] but in our experience, their use was effective at significantly reducing leaks in a large number of patients. At times, a thermal humidifier added to the circuit proved useful because the tendency to open the mouth decreases as nasal resistance decreases. Another alternative we have resorted to in patients on BPNV is to reduce inspiratory pressure in order to reduce circuit pressure and thus lower the chance of leaks. It might seem paradoxical, but a reduction in positive inspiratory pressure may even improve the effectiveness of ventilation. More recently, now that face masks with a safety valve have become available, we have resorted to the use of face masks
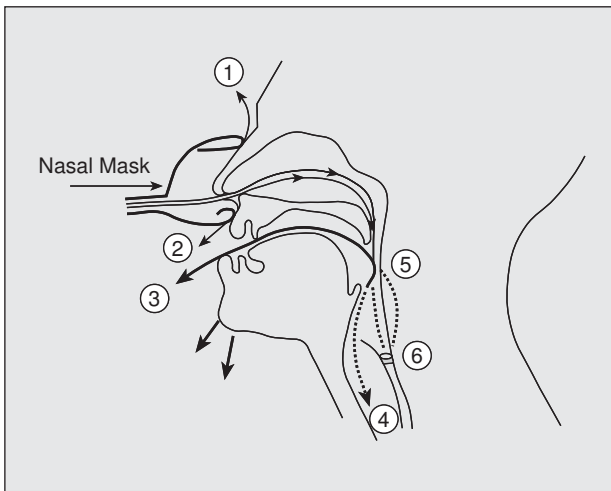
Figure 7. Potential sites for leaks in a patient on nasal mask ventilation. External leaks: 1 and 2 are the most common sites for leaks at the mask interface with the patient, and 3 indicates mouth leak. "Internal" leaks: 5 indicates the formation of an oropharyngeal reservoir and 6 shows the passage of air to the digestive tract. Pathway 4 corresponds to effective ventilation (Thanks to D. Rodenstein, modified by D. Robert.)

after other methods have failed. However, tolerance of this interface for long-term ventilation is slightly lower than tolerance of a nasal mask.

We have proposed a simple, noninvasive, diagnostic method for detection of a frequent problem that can lower the therapeutic effectiveness of NMV. Although reports of other methods for measuring leaks in NMV can be found in the literature, they require complex assemblies or mathematical equations that hinder daily use.[19,20] Our techniques can be easily applied in day-to-day practice and do not require material that is complex or that will significantly affect quality of sleep. They may also provide a rough indication of the pathophysiological mechanisms underlying partial or total failure of NMV. Nevertheless, the limitations of our techniques are evident. Among the techniques that might complement ours, we might particularly mention such noninvasive techniques as continuous monitoring of expired tidal volume with the signal from a Fleisch pneumotachograph coupled to the expiratory valve,[4] or by combining chest and abdominal signals with an inductance plethysmograph,[2] or by monitoring expired carbon dioxide pressure or transcutaneous carbon dioxide pressure.[34] Complementary invasive techniques include measuring esophageal pressure or hypopharyngeal pressure,[20] possibly in combination with polysomnographic assessment of quality of sleep. Although these methods can be very useful in complicated cases or within the framework of clinical research, they are hard to apply in day-to-day clinical practice.

One limitation of our method is that it cannot detect so-called "internal leaks." In this case, the upper airway or the digestive tract—compliant structures reached by the insufflated air from the ventilator when the system is not leak-proof—act as a kind of "air reservoir" and so no external leak is detected. Part of the insufflated tidal volume is diverted to these "reservoirs" where it is unable to participate effectively in alveolar gas exchange[21,31] (Figure 7). Distension in the region of the neck is often observed in such patients during inspiration, suggesting that a large part of the insufflated air reaches compliant tissues of the oropharynx causing distension. Studies have shown that, for an insufflation pressure of 20 cm H_2O, up to 130 mL of air can be stored in such reservoirs.[6] Insufflated air may also reach the digestive tract. The esophageal sphincter resists pressures that range from 15 cm H_2O to 20 cm H_2O, thus at higher pressures some of the tidal volume may be diverted to the digestive tract. This not only reduces the effectiveness of ventilation, but also produces aerophagia and increases the elastic work of the ventilation.[22]

In conclusion, this paper presents an original and practical method that can be applied to patients suspected of inadequate ventilation evidenced by clinical symptoms (memory loss, dry mouth, treatment intolerance) or by uncompensated arterial gas levels and/or nighttime oxygen saturation (desaturation peaks during NMV, persistent hypoventilation). If leaks are suspected, detection during treatment with our method may determine the probable cause of failure and help establish the possible pathophysiological mechanism. With this knowledge, it may be possible to improve ventilation.

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