	LÖWENSTEIN medical
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# **Ventilation Strategies**

A Monograph with Practical Tips.







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## 1 Ventilation, where are you going?

Mechanical ventilation is becoming increasingly important in the treatment of respiratory and ventilatory disorders.

Ventilation therapy is now focused primarily on ventilatory failure (hypercapnic insufficiency, Type II) and secondly on respiratory failure (hypoxemic insufficiency, Type I).

Today most chronically ill patients are effectively treated with non-invasive ventilation based on state-of-the-art ventilation technology and delivered via high-quality masks.

In addition to treating well-known indications such as neuromuscular and thoracic wall disorders, non-invasive ventilation is being administered to patients with Chronic Obstructive Pulmonary Disease (COPD) and Obesity-Hypoventilation Syndrome (OHS). In sleep medicine, a borderline case is the ventilation of patients with the type of periodic breathing known as Cheyne-Stokes Respiration (CSR).

Ventilation specialists receive increased support from new technologies which, based on adaptive algorithms and modern biofeedback systems, continuously adjust ventilation parameters to the needs of the patient.

Furthermore, medical professionals can optimize their work processes in ventilation units with assitance from electronic data processing. Another trend involves telemedical connections for ventilators. With cloud-based solutions, medical experts have completely automatic access to daily therapy information from anywhere – all for the good of the patient.

# 2 Ventilation – State of the Art

In addition to blood circulation and consciousness, respiration is a vital function that sustains human life. A disorder or breakdown of any vital function is lifethreatening.

It is therefore no surprise that medical practitioners have long attempted to devise a therapeutic means of providing artificial respiration. Hippocrates (460 - 377 B.C.) and Paracelsus (ca. 1493 - 1541 A.D.) left accounts of their experiments in the field. In 1876 the Frenchman Eugene Joseph Woillez presented his prototype of an iron lung, which he called a "spirophore"1 to the French Academy.

The polio epidemic in the early 1950s gave rise to significant developments in artificial respiration. Among other things, hospitals set up Intensive Care Units (ICUs) in response to the newly emerging medical requirements. At that time anesthesiology contributed greatly to the further development of mechanical ventilation.

Ventilation supports inadequate respiration or fills in when spontaneous breathing ceases. The treatment of chronic respiratory failure or ventilatory failure by means of ventilation technology is considered an important therapeutic measure in reducing the morbidity and mortality of the patients affected.<sup>2, 3, 4</sup> Furthermore, ventilation has a positive effect on the patients' quality of life.<sup>5</sup>

The increasing use of mechanical ventilation is developing in step with options for the use of Non-Invasive Ventilation (NIV). The quality of the patient interface in the form of varying mask systems plays a critical role.

These days many patients with chronic respiratory failure or ventilatory failure are treated with NIV. That makes it easier to treat patients who require mechanical ventilation outside the hospital in their own homes. Because many devices are small and manageable, patients gain a certain degree of mobility.

Table 1 provides an overview of the attributes of non-invasive and Invasive Ventilation (IV) in acute situations. The contraindications of NIV are shown in Table 2.

## **Decision Support in Acute Situation**

Characteristics of non-invasive and invasive ventilation based on S3 guideline "Non-Invasive Mechanical Ventilation in Acute Respiratory Failure"

Negative attribute

🗗 Positive attribute



Table 1<sup>6</sup>

The attributes of IV and NIV show that in most cases non-invasive ventilation is the better alternative.

## **Contraindications for Non-Invasive Ventilation (NIV)**

Although current developments in medical technology have made non-invasive ventilation the treatment of choice in most cases, consideration should be given to certain absolute and relative contraindications, which are listed below:



Table 2<sup>6</sup>

Given the contraindications, it can be concluded that invasive ventilation will retain some importance. The absolute and relative contraindications are to be used as the basis for treatment decisions.

In a study that analyzed the use of home mechanical ventilation in several European countries, <sup>7,8</sup> the prevalence of mechanical ventilation was estimated at 6.6 per 100,000 residents. Some medical experts maintain that the prevalence in some of those countries is much higher.

Furthermore, according to the study,<sup>7</sup> some countries report significant variances in the distribution percentages of some of the indications. The diseases in the standard categories are subdivided as follows:

- Lungs/airways (COPD)
- Thoracic wall disorders (kyphoscoliosis of thoracic spine)
- Neuromuscular diseases

These categories can be further expanded to include Obesity-Hypoventilation Syndrome<sup>9</sup> and Cheyne-Stokes Respiration, although the latter is generally treated within the scope of sleep medicine. Some overlap exists here (e.g., OHS). Quite often sleep medicine diagnostics are used to assess nighttime ventilation quality.

Since the end of the 1990s the absolute number of mechanically ventilated patients with neuromuscular disease and thoracic wall deformities has been relatively stable, but the number of COPD patients has risen considerably. A 2014 study showed a significant effect of NIV on life expectancy of hypercapnic, stable COPD patients.<sup>10</sup> The dramatic increase in obesity among the general population translates into rapid growth in the number of ventilated patients, many of whom have OHS.<sup>11</sup>

Parallel to the development of sleep medicine, effective ventilation concepts have been introduced for treatment of patients with central respiratory disorders like Cheyne-Stokes Respiration. <sup>12, 13</sup>

These days experts are looking critically at the PaCO<sub>2</sub> levels in COPD patients whose hypercapnia is not effectively resolved by ventilation.

Evidence is mounting that shows the reduction in PaCO<sub>2</sub> through the application of higher inspiratory pressures can have a positive effect on life expectancy.<sup>14</sup> One predictive indicator for life expectancy of COPD patients appears to be the six-minute walk test.<sup>15</sup> Furthermore, stable hypercapnic COPD patients show better tolerance of high ventilation pressures accompanied by a more effective reduction in nocturnal PaCO<sub>2</sub>.<sup>16</sup>



The number of patients requiring mechanical ventilation is increasing considerably along with the indications of Obesity-Hypoventilation Syndrome and COPD.

Device technology alone does not determine the quality of ventilation. Expert care of the patient at home with the support of family members is just as important<sup>8</sup>.

A key to gaining patient acceptance of therapy is proper patient briefing. Medical personnel have to act with sensitivity and understand that patients requiring ventilation often suffer from shortness of breath and feelings of claustrophobia when a mask is placed over the nose or nose and mouth. It is important that the patient quickly develop trust and confidence in the therapy and see it as a source of relief and protection.

Modern ventilation technology based on intelligent ventilation solutions can contribute greatly to achieving this goal.

# **3 Indications for Ventilation**

The indication ranking for ventilation is based on the following parameters:

- underlying disease
- clinical picture
- blood gas levels

Disease	Indication for non-invasive ventilation	Indication for invasive ventilation*
Underlying neuromuscular disease	<ul> <li>Alveola hypoventilation in connection with at least one of the following parameters:</li> <li>chronic hypercapnia (PaCO<sub>2</sub> ≥ 45 mmHg) during the day and/or</li> <li>at night (≥ 50 mmHg) and/or</li> <li>normocapnia during the day with increase in PTc CO<sub>2</sub> ≥ 10 mmHg at night</li> <li>or rapid decrease in vital capacity</li> <li>at a Peak Cough Flow (PCF) ≤ 270 l/min for mechanical secretion management is required.</li> </ul>	<ul> <li>Requires ventilation and despite use of device, no sufficient benefit from NIV</li> <li>Dysphagy with recurring pneumonia</li> <li>NIV required &gt; 16 hours/day</li> </ul>
Thoracic restriction	<ul> <li>Hypoventilation symptoms</li> <li>Chronic daytime hypercapnia with PaCO<sub>2</sub> ≥ 45 mmHg</li> <li>Nocturnal hypercapnia with PaCO<sub>2</sub> ≥ 50 mmHg</li> <li>Daytime normocapnia with increase in PTc CO<sub>2</sub> ≥ 10 mmHg</li> </ul>	<ul> <li>Requires ventiation, but during treatment significant worsening of blood gas levels, severe acidosis (pH &lt; 7.35)</li> </ul>

Disease	Indication for non-invasive ventilation	Indication for invasive ventilation
Obesity Hypoventilations- Syndrome	<ul> <li>Obesity and hypercapnia despite adequate CPAP therapy</li> <li>≥ 5-minute increase of PTc CO2 ≥ 55 mmHg or PaCO2 ≥ 10 mmHg compared to waking state or Desaturation &lt; 80% SaO<sub>2</sub> over ≥ 10 minutes</li> <li>Desaturations &lt; 80% SaO<sub>2</sub> for ≥ 10 minutes</li> <li>If reevaluation after three months of CPAP therapy shows no clinical improvement and daytime normocapnia</li> </ul>	- Contraindications for NIV, e.g., dysphagia
COPD	<ul> <li>Symptoms of ventilatory failure and chronic hypercapnia and reduced quality of life</li> <li>Indication criteria (at least one additional criterion must be fulfilled)</li> <li>chronic daytime hypercapnia with PaCO<sub>2</sub> &gt; 50 mmHg</li> <li>nocturnal hypercapnia with PaCO<sub>2</sub> &gt; 55 mmHg</li> <li>stable daytime hypercapnia with PaCO<sub>2</sub> 46-50 mmHg and increase in PTc CO<sub>2</sub> &gt; 10 mmHg during sleep</li> <li>stable daytime hypercapnia with PaCO<sub>2</sub> 46-50 mmHg and at least two acute exacerbations with respiratory acidosis requiring hospitalization within the previous 12 months.</li> <li>directly subsequent to an acute exacerbation requiring ventilation, according to clinical evaluation</li> </ul>	<ul> <li>Requires ventilation, but in course of treat- ment, blood gas levels worsen considerably, severe acidosis (pH &lt; 7.35)</li> </ul>

Disease	Medical benefits from ventilation
Neuromuscular illness	<ul> <li>Unloading of respiratory muscles<sup>56</sup></li> <li>Reduction in respiratory complications<sup>49</sup></li> <li>Improvement in sleep quality<sup>74</sup></li> <li>Improvement in sleep-disordered breathing<sup>74</sup></li> <li>Improvement in quality of life<sup>5, 56, 74</sup></li> <li>Increased life expectancy<sup>56, 7</sup></li> <li>Reduction in daytime sleepiness<sup>56, 74</sup></li> </ul>
Thoracic- restrictive disorder	<ul> <li>Improvement in blood gases<sup>72,73</sup></li> <li>Improvement in lung volume<sup>72</sup></li> <li>Unloading of respiratory muscles<sup>2</sup></li> <li>Reduction in hypercapnia<sup>76</sup></li> <li>Regression of pulmonary arterial hypertension<sup>18,79</sup></li> <li>Improvement in strength of respiratory muscles<sup>73,78</sup></li> <li>Improvement in inspiratory muscle function<sup>72,78</sup></li> <li>Improvement in sleep quality<sup>18,78</sup></li> <li>Improvement in quality of life<sup>5</sup></li> <li>Increased life expectancy<sup>77,78</sup></li> </ul>
Obesity Hypoventilation Syndrome	<ul> <li>Normalization of ventilation during the day and night<sup>56</sup></li> <li>Improvement in blood gases<sup>56</sup></li> <li>Increased life expectancy<sup>56</sup></li> <li>Reduction in daytime sleepiness<sup>56</sup></li> <li>Improvement in lung function<sup>75</sup></li> <li>Improvement in sleep quality<sup>77</sup></li> </ul>
COPD	<ul> <li>Reduction in hypercapnia <sup>10</sup></li> <li>Improvement in quality of life <sup>2</sup></li> <li>Improvement in blood gases <sup>2</sup></li> <li>Improvement in sleep quality <sup>56</sup></li> <li>Increased life expectancy <sup>10</sup></li> </ul>
Cheyne-Stokes Respiration	<ul> <li>Improvement in sleep-disordered breathing<sup>66</sup></li> <li>Normalization of nighttime breathing<sup>66</sup></li> <li>Improvement in sleep quality<sup>66</sup></li> <li>Increase in ejections fraction <sup>12, 19</sup></li> <li>Improvement in heart failure <sup>12</sup></li> <li>Improvement in physical activity<sup>80</sup></li> </ul>

Table 4 Effects of mechanical ventilation

Patients with neuromuscular diseases or thoracic restriction can live for many years with the help of mechanical ventilation. According to a European study, those patients generally use a ventilator for more than six years.<sup>7</sup> Patients with OHS also benefit from non-invasive ventilation as far as pulmonary function and gas exchange are concerned. A decision in favor of non-invasive ventilation is based on the existence of hypercapnia despite CPAP therapy.<sup>9</sup> Negative prognostic factors for OHS are hypoxemia and elevated inflammation markers.<sup>20</sup>

# **4** Ventilation Technologies

Before a patient can be mechanically ventilated, the treating physician has to find the correct ventilation pattern. That is, the timing of the respiratory cycle with regard to pressure, flow and volume has to be determined. A basic distinction is made between pressurecontrolled and volume-controlled modes.

**Parameters to be set** on a pressurecontrolled device include:

- ventilation mode
- levels of inspiratory and expiratory pressure
- ventilation frequency
- respiratory time ratio (inspiration to expiration, I:E) or inspiratory time
- backup frequency (in S and ST modes and in PSV and aPCV modes)
- trigger sensitivity
- pressure rise and pressure decline speed
- target volume

In addition, pressure and/or volume alarms should be set.

**The required ventilation** pressures depend in part on the following:

- the mechanical characteristics of the pulmonary-thorax system (resistance and compliance)
- the pathophysiological factors.

In the pressure curve, **IPAP** (Inspiratory Positive Airway Pressure) is differentiated from **EPAP / PEEP** (Expiratory Positive Airway Pressure / Positive End-Expiratory Pressure). The pressure difference between IPAP and EPAP/ PEEP is described as the effective ventilatory pressure.

An external PEEP can be used to counteract the patient's intrinsic PEEP (e.g., in the case of COPD). It has the following effects:<sup>57</sup>

- holds open collapse-prone alveoli
- reduces the extent of intrapulmonary shunt
- in COPD patients: Reduction of intrinsic PEEP and thus reduced Work of Breathing
- Effects on hemodynamics: Reduction in the filling volume of the left ventricle, which can be disadvantageous, particularly in the absence of atrial contraction as a result of absolute arrhythmia and left heart failure.

An external PEEP also effects a splint of the upper airways during sleep. In autoST mode the EPAP is automatically adjusted to the lowest pressure volume required to prevent obstructive apnea and/or hypopnea.

#### **Inspiratory time**

Treatment success depends greatly on the setting for inspiratory time as it decisively influences the applied ventilation volume. If no firm setting is made for inspiratory time (as in aPCV mode, for example), the time is dependent on the resistance and compliance of the patient's respiratory system. The reason for that is found in the expiratory trigger, which is always a percentage of the inspiratory maximum flow (peak flow). When the setting for the expiratory trigger is reached, the ventilator switches to expiration, a process referred to as "cycling".



#### Figure 1

Inspiratory time (Ti) depends on resistance and compliance.

If resistance is high and compliance low, the maximum inspiratory flow—and with it, the expiratory trigger—is quickly reached, causing the device to cycle too early. The inspiratory time is shortened and, as a result, the ventilation volume is low.



#### Figure 2

High resistance and/or low compliance lead to a short inspiratory time and a low ventilation volume in COPD.

If, however, resistance is low and compliance high, cycling takes place later and the volume is correspondingly high.



#### Figure 3

At low resistance and/or high compliance, a longer inspiratory time is achieved; ventilation volume is high.

High leakage is a potential problem. In the presence of high leakage, the ventilator cannot recognize the expiratory trigger threshold. Without a pre-set maximum inspiratory time, the device is not in a position to cycle. For the good of the patient, this dependency can be ended

by setting a minimum inspiratory time (Ti min) and a maximum inspiratory time (Ti max). The therapeutic solution ensures a minimum ventilation volume with Ti min and proper cycling with Ti max:



#### Figure 4

Setting for Ti min: Ensure a minimum volume, prevent hypoventilation. Setting for Ti max: Prevent hyperventilation.

Assisted ventilation modes, like ST mode, are designed so that when the patient fails to breathe spontaneously, he/she is supported by controlled ventilation. For that purpose F and Ti timed settings are made for a backup frequency. Ti timed regulates the I:E ratio during controlled ventilation. Ti timed can be set either to a fixed time at which inspiration is ended or to auto. In auto mode, Ti timed lies between Ti min and Ti max, depending on the selected expiration trigger.



Figure 5 prisma VENT50

### **Ventilation frequency**

Respiratory rate or frequency depends on the patient's age and disease. The rates should be adjusted accordingly to these conditions.

Pathologically high respiratory frequency combined with low tidal volume (rapid shallow breathing) is the leading symptom of impending respiratory failure.<sup>58</sup>

The **respiratory time** is the relationship between the lengths of time for inspiration and expiration. The I:E ratio is set on the ventilator or is given by the combination of the parameters tidal volume, ventilation frequency and inspiratory time. The I:E ratio usually chosen for healthy lungs is 1:2 (corresponds to Ti / T of 33%) to 1:1. In the case of obstructive pulmonary diseases that can lead to hyperinflation, a prolonged expiratory time should be selected.<sup>57</sup>



Figure 6 Operating panel on ventilators prisma VENT30/40/50/50-C: I:E setting (Ti/T)

## I:E setting (Ti/T)

# Target volume in pressure-controlled ventilation

Many studies have shown that volumeand pressure-controlled ventilation technologies achieve comparable therapeutic effectiveness with regard to blood gases, breathing pattern and nocturnal oxygen saturation.<sup>21, 22, 23</sup>

Ventilators based on a pressure-controlled blower also offer the beneficial feature of leakage compensation. Furthermore, there are indications of better patient tolerance due to fewer gastrointestinal side effects. Pressure-controlled ventilators therefore are widely used. The devices should be capable of regulating to a target volume.

The function called **volume compensation" (target volume)**<sup>24</sup> can be used in pressure-controlled ventilation. It should ensure that the patient is adequately ventilated at all times, even when compliance in the lungs and chest varies

that the target volume has been reached and

changes the pressure by  $1.0 \text{ cmH}_2\text{O}$ .

as the result of mechanical influences on the chest (e.g., changes of position during sleep). Compensation can be made for longer term changes in the mechanics caused by exacerbation or a disease's progress.

Caution is advised in treating patients with different degrees of leakage (at night, for example). The ventilator could incorrectly interpret the signals and reduce the pressure in such cases. Measuring the PaCO<sub>2</sub> curve and the level of bicarbonate can be used to assess ventilation's sufficiency under volume compensation. The PaCO<sub>2</sub> curve should decline under ventilation. An increase, on the other hand, indicates underventilation.

A quickly changing breathing pattern (e.g., Cheyne-Stokes Respiration) could challenge the target volume algorithm.



If, at the respective speed, the pressure reaches a corridor around the target volume, the device switches to a precise regulation at (± 0,1cmH<sub>2</sub>O/100ml).

# Trigger/Managing the respiratory cycle

Ventilators control the patient's breathing when previously set thresholds (triggers) are exceeded. The mechanisms are referred to as pressure-, flow-, volume- and time-controlled.<sup>68</sup>

Both the acceptance and effectiveness of ventilation depend greatly on good synchrony between patient and device<sup>67,68</sup>, which is determined by the following: <sup>41</sup>

- 1. triggerung of the ventilator
- 2. phase of inspiration after triggering
- 3. transition from inspiration to expiration
- 4. end of expiration

**Trigger sensitivity** 

The patient triggers the ventilator during assisted ventilation. The advantage of triggering is that the patient can initiate the ventilator-delivered breath on his own. A disadvantage for patients whose respiratory muscles are exhausted is the amount of energy required to trigger the inspiration phase. Trigger settings that are insufficiently sensitive unnecessarily increase the patient's Work of Breathing and potentially lead to respiratory muscle fatigue. In worst case, patient triggering may fail completely and pressure support would fail too.<sup>57</sup>

It is therefore important to be able to adjust the trigger sensitivity to the needs of the patient. Well-chosen sensitivity unloads the respiratory muscles and gives the patient more personal freedom.<sup>67,68</sup>

Assisted ventilation is not recommended for advanced stages of neuromuscular diseases. For modes in which the ventilator controls the ventilation completely, it is important to suppress the dreaded patient "fighting" with the device and thereby avoid ansynchrony<sup>66,67</sup> of patient and device. In this situation, the patient "fights" the ventilator's rhythm. The original medical intention of fully unloading the patient's respiratory pump then comes to nothing.



#### Figure 8

Example: LUISA: Trigger setting in Manual and Auto. Ventilator efficiency is positively affected by a trigger sensitivity set specifically for a patient. The manual inspiratory setting is made in eight (8) levels.



Figure 9 Example: LUISA: Trigger setting: The expiratory setting from 5% to 95% is made in increments of 5%. Trigger Lockout can be set in order to prevent potential false triggering in the expiratory phase.

## 4.1 Ventilation Modes and Current Technological Solutions

The three basic ventilation modes differ in the extent to which the ventilator takes over the Work Of Breathing.

- Controlled ventilation in which the ventilator takes over all the work:
   T, PCV, VCV mode
- Controlled-assisted ventilation in which the ventilator takes over 50 to 100% of the work, depending on the setting selected: ST and PSV mode with backup frequency, aPCV, aVCV
- Spontaneous breathing in which the patient receives pressure or volume support: CPAP, S and PSV mode with-out backup frequency, MPV

In addition to these three basic forms of ventilation, High-Flow Therapy supports patients with respiratory failure by washing out the CO<sub>2</sub> from the upper airways.

These ventilation modes are explained in the following paragraphs:

#### 4.1.1 CPAP

Continuous Positive Airway Pressure (CPAP). A quality indicator is the pressure constancy that is maintained during spontaneous breathing. CPAP is primarily used to treat obstructive sleep apnea, mild forms of OHS, pulmonary edema and, to an extent, Cheyne-Stokes Respiration (CSR).

### **Use of CPAP mode**

CPAP applies a pneumatic splint to the airways and helps to improve oxygenation.

#### 4.1.2 BiLevel

Continuous positive airway pressure at two pressure levels. BiLevel is the basis for several different modes in which settings can be made for a higher inspiratory (IPAP) pressure level and a lower expiratory pressure (EPAP/PEEP) level.

#### 4.1.2.1 S Mode

The basic mode of bilevel ventilation is the **S mode** (S = spontaneous), which involves inspiratory (IPAP) and expiratory (EPAP) pressure support.

### 4.1.2.2 ST Mode

BiLevel ventilation also can be provided in **ST mode** (Spontaneous Timed). It combines assisted and when necessary, controlled ventilation. For the patient's safety, a backup frequency with a fixed I:E ratio is set in addition to the therapeutically required pressure level (IPAP/EPAP). The I:E ratio (Ti / T) normally lies below the patient's spontaneous breathing rate.

Spontaneous breathing is permitted in S and in ST modes. A trigger adapted to a specific patient's needs can be configured to provide optimum support of the patient's spontaneous breathing efforts.

	Ventilation	Prog 12
Program		1
Mode	S	т
IPAP	10	.0 cmH2O
EPAP	4	.0 cmH2O
E.	10	.0 /min
Ti min	0	.5 s
Ti max	1	.7 s
System	Ventilation	Report

ST mode. The example shows an IPAP of 10.0 cmH<sub>2</sub>O, an EPAP of 4.0 cmH<sub>2</sub>O and a backup frequency of 10.0/min.

At low effective ventilation pressures (i.e., with low PDiff = IPAP – EPAP), there is a risk that the patient could still suffer from dyspnea.

## 4.1.2.3 T Mode

T mode corresponds to controlled ventilation. The patient has no influence on the ventilation. Settings include IPAP and EPAP, respiratory frequency, inspiratory time and inspiratory pressure rise. Maximum unloading of the respiratory pump is achieved as long as the patient does not expend any effort. A "quasi" T mode exists when the selected frequency in ST mode lies slightly above the spontaneous frequency. This setting reduces the patient's WOB, with maximum freedom permitted above the backup frequency.



Figure 11 T mode (controlled ventilation). The example shows an IPAP of 22.0 cmH<sub>2</sub>O and an EPAP of 4.0 cmH<sub>2</sub>O. The patient is ventilated with a respiratory rate of 18/min. and an I:E ratio of 1:2.

## Use of T mode

T mode or controlled ventilation provides maximum unloading of patient's exhausted respiratory pump as long as the patient does not experience asynchrony with the ventilator. Under this mode, fatigued respiratory muscles should recover more quickly than they would under assisted ventilation.<sup>57</sup>

#### 4.1.2.4 autoST Mode

With **autoST** (autoST = autoEPAP + autoF) the patient is given an intelligent backup which combines pressure adjustment (auto-EPAP) with a continuously regulated backup frequency (autoF). If an inadequate flow is detected in this mode, obstruction recognition takes effect and adjusts the EPAP level to the patient's current needs. The EPAP adjustment takes place between EPAP min and EPAP max.

On the basis of the autoF setting, the ventilator prevents central apnea phases and desaturation by delivering mandatory breaths in the absence of spontaneous breaths. The volume supplied is monitored and the frequency is adjusted within a defined range (10 to 20 breaths per minute). The patient can breathe spontaneously at any time and thereby suppress the mandatory ventilation.

#### autoEPAP and autoF



#### Figure 12

A patient-specific adjustment between EPAP min-max and a set pressure delta (Pinsp.) permits an automatic pressure adjustment for the patient and helps to eliminate obstructions.



#### Figure 13

EPAP increases automatically to eliminate obstructions and adjusts continuously when upper airways are clear. Pressure support remains constant.

#### 4.1.3 PSV

In Pressure Support Ventilation (PSV) spontaneous breathing is coupled with mechanical ventilation. The patient triggers the device by means of his inspiratory effort. As soon as the trigger threshold is exceeded, the ventilator responds by increasing the inspiratory pressure to a pre-set level. If the flow decreases to a defined percentage of inspiratory peak flow during inspiration, expiration is triggered. The resulting tidal volume is dependent on

- the level of the set differential pressure,
- the intensity and duration of inspiratory effort,
- the compliance and resistance of the lungs.



Figure 14

The patient's inspiratory effort initiates an increase in ventilation pressure to a defined level. The patient triggers mechanical ventilation, resulting in a reduction in the Work Of Breathing.

### Use of PSV

PSV is used on patients with intact respiratory drive and sufficient respiratory muscle strength to trigger the device.

### 4.1.4 PCV

PCV stands for Pressure-Controlled Ventilation. In this controlled mode, inspiration is regulated at a pre-set pressure level (IPAP), which is maintained until the end of inspiration. At the end of inspiration time, the device automatically switches to expiration. In contrast to PSV, spontaneous breathing is not allowed. Changes to lung compliance and resistance affect tidal volumes.



Figure 15 PCV – a ventilation mode often used in home mechanical ventilation

### 4.1.5 VCV

Under Volume-Controlled Ventilation (VCV) the patient receives a specified tidal volume within a defined time. The applied ventilation pressure varies, dependent on the factors of lung compliance and resistance. It is therefore necessary to set alarms for ventilation pressure. Any spontaneous breathing by the patient is not supported in this mode.



Figure 16

VCV – at high inspiratory flow the volume to be applied is administered before the inspiratory time has elapsed. This causes a pause known as the "plateau phase" to occur. The flow should be set to make the end-inspiratory phase very brief.

#### 4.1.6 aPCV/aVCV

The modes aPCV and aVCV (assisted PCV and assisted VCV) are types of pressure- controlled or volume-controlled ventilation. The patient triggers a ventilator-delivered breath.

The modes aPCV and aVCV allow the patient to inhale during a specified time window.

The inspiratory time is set on the ventilator.

This is actually controlled ventilation with the option of inspiratory triggering.



#### Figure 17

Shown on the ventilator LUISA. In both modes aPCV/aVCV, the inspiratory time is firmly set.

#### Figure 18

aPCV/ aVCV, the invariable frequency setting is moderated by assisted inspiration. The critical factor is the setting of the trigger threshold. If it is set too high, the patient cannot trigger inspiration. Then controlled ventilation is administered to the patient.

### 4.1.7 SIMV

The ventilation mode Synchronized Intermittent Mandatory Ventilation (SIMV) combines spontaneous breathing with volume- (V-SIMV) or pressure-controlled (P-SIMV) ventilation. The patient may take spontaneous breaths between the mandatory ventilator-delivered breaths. In SIMV, breaths are triggered by the patient except when the patient is apneic. Breaths can be triggered, however, only within an expected time window.

<i>l</i> lodes for leakage and/or valve systems		
Modes for leakage system		
Acronym	Meaning	
CPAP	Continuous Positive Airway Pressure	
S	Spontaneous	
ST	Spontaneous Timed	
autoST	Automatic Spontaneous Timed	
т	Timed controlled	
	Modes for leakage and valve system	
PSV	Pressure Support Ventilation	
aPCV	assisted Pressure Controlled Ventilation	
PCV	Pressure Controlled Ventilation	
aVCV	assisted Volume Controlled Ventilation	
VCV	Volume Controlled Ventilation	
MPVp	Mouthpiece Ventilation pressure controlled	
MPVv	Mouthpiece Ventilation volume controlled	
HFT	High-Flow Therapy	
	Mode for valve system	
SIMV	Synchronized Intermittent Mandatory Ventilation	

Table 5

Overview of Modes, depending on patient circuit system in use

If the device does not detect spontaneous breathing activity during this window, an unsynchronized ventilator breath is delivered. In worst case, an incorrect device setting can hinder spontaneous breathing. If severe respiratory failure is present, the spontaneously breathed tidal volume might be low enough to cause alveolar hypoventilation.<sup>25</sup>

SIMV is used only for invasive ventilation and nowadays very infrequently.

### 4.1.8 MPVp/MPVv

Mouthpiece ventilation can be administered in two different modes, pressurecontrolled (MPVp) and volume-controlled (MPVv) ventilation. Very often large volumes (800 to 1,500 ml) are delivered to make it easier for the patient to speak, cough and use airor breath-stacking techniques.

The mouthpiece affixed to the wheelchair or bed is within patient's reach. Unlike NIV delivered via a mask or IV via a tracheal cannula, mouthpiece ventilation has no direct connection between device and patient.

Consequently, the patient has maximum freedom of movement and the option of using the mouthpiece to obtain a ventilator-delivered breath. Mouthpiece ventilation:

- simplifies speaking, eating and drinking
- improves quality of life by giving the patient more freedom and comfort during treatment.

Mouthpiece ventilation is particularly suited for treatment of patients with neuromuscular diseases and thoracic restriction such as:

- Muscular dystrophy (e.g., Duchenne)
- Amyotrophic Lateral Sclerosis (ALS)
- Spinal muscular atrophy I, II, III
- Musculoskeletal disorders (e.g., Kyphoscoliosis)



Figure 19 shows a flexible arm affixed to a wheelchair and holding a patient circuit and mouthpiece.

## 4.1.9 High-Flow Therapy (HFT)

In **High-Flow Therapy** (HFT) heated and humidified respiratory gas is applied in a high continuous flow. As needed, the gas can be enriched with oxygen. This type of therapy is suitable only for patients with their own respiratory drive.

In prisma VENT50-C, for example, a flow of 5 to 60 liters/minute can be set and supplemented with oxygen.

In contrast to non-invasive ventilation, HFT does not use a mask, but rather nasal cannula (infrequently a tracheostomy interface may be used). The warm and humid air is delivered to the patient via nasal prongs. For adults, the size of the prongs should cover about twothirds of the nasal opening; for children, only one-half of the area should be covered.

One of the most important mechanisms of HFT—the  $CO_2$  washout of the upper dead space—directs the expired gas alongside the prongs and through the mouth.

The effectiveness of HFT is flow and leakage dependent.<sup>26</sup> The higher the chosen flow, the better the CO<sub>2</sub> elimination.<sup>29</sup>

The major advantages of High-Flow Therapy are:

- washout of nasopharyngeal dead space and thus CO<sub>2</sub> elimination with consecutive decreases in tidal volume and the lowering of spontaneous breathing rate,<sup>29, 30</sup>
- increase of oxygen supplied to alveoli,<sup>28</sup>
- reduction in the Work of Breathing (WOB),<sup>31</sup>

- improvement in mucocillary clearance through humidifying and warming of upper airways,<sup>32</sup>
- good acceptance.

Although the system is open, PEEP can still develop under High-Flow Therapy. This PEEP is flow-dependent and generally low. At a flow of 40 liters/minute, PEEP is about 2 cmH<sub>2</sub>O; at a flow of 50 liters/minute, about 3 cmH<sub>2</sub>O.<sup>27</sup>

Because the patient can forgo the conventional ventilation mask, he/she can eat and speak during therapy. Many patients say the nasal cannula is more comfortable and speak positively about improvements such as less dyspnea and mouth dryness.<sup>28</sup>

High-Flow Therapy should always be conducted with an **active humidifier**, such as prisma VENT AQUA, which is used for non-invasive ventilation. When the humidifier is switched on, it automatically recognizes whether a heated tube system and a temperature sensor are connected and then starts up in the corresponding mode.

The easy-to-use prisma VENT AQUA is equipped with an extensive alarm management system.

A large selection of accessories, including tubes and nasal cannulas, is available for ventilation and for High-Flow Therapy.



Figure 20 The humidifier prisma VENT AQUA combined with the prisma VENT50-C ventilator. Three operating modes offer the user and patient a broad usage spectrum for conditioning respiratory gas during non-invasive ventilation.

#### 4.1.10 COPD Treatment options

# 4.1.10.1 AirTrap Control in Cases of Dynamic Hyperinflation

In mechanical ventilation, Positive End-Expiratory Pressure (PEEP) is used to hold open the alveoli and to prevent the airways from collapsing. An undesirable development, however, is intrinisic PEEP (also known as autoPEEP). It can occur when the respiratory rate or frequency is set too high or the set expiration time is too brief to allow complete expiration.

It can be seen in the flow curve when the flow does not fall back to "zero". COPD patients in particular tend to develop auto or intrinsic PEEP, which, in turn, can lead to dynamic hyperinflation.<sup>33</sup> The airways of COPD patients exhibit the following pathophysiological characteristics:

- bronchial obstruction
- instability in the small airways (as a result of changes caused by inflammation, for example)
- hypersecretion with cough and inflammation.

As a consequence, the airways collapse during forced expiration, trapping residual air in the alveoli.







Figure 22 The airways of COPD patients tend to collapse during inspiration.

## COPD patients are prone to dynamic hyperinflation

The Functional Residual Capacity (FRC) increases at the expense of vital capacity; the resting expiratory level shifts and intrinsic PEEP develops. <sup>34,35</sup>

Consequence: dynamic hyperinflation occurs.<sup>36</sup> The end-expiratory lung volume increases over the resting volume, which is connected to intrinsic PEEP (PEEPi) and lower compliance. The respiratory muscles fall into an unfavorable range of action in which the length of the diaphragm is shortened.

Sufficient ventilation under these conditions can be achieved only with increased respiratory effort.<sup>30</sup> A typical response of affected patients is thoracic breathing with help from auxiliary respiratory muscles. If the respiratory pump muscles become exhausted over the course of disease, the patient will suffer respiratory arrest, indicated by elevated PaCO<sub>2</sub> levels. The risk of dynamic hyperinflation is high in COPD patients. It should be prevented because

- 1. the efficiency of respiratory muscles is limited and
- 2. the Work Of Breathing (WOB) increases significantly.

Clinical signs of dynamic hyperinflation include dyspnea and limited physical capacity.<sup>38</sup> Gas exchange deteriorates.

Intrinsic PEEP is an undesirable condition during mechanical ventilation. If the patient wants to trigger the device, he/she has to generate a positive intrathoracic presure before he/she can generate negative intrathoracic pressure, which will then send a trigger signal to the ventilator. Very often the patient is unable to expend the respiratory effort required to trigger the device.<sup>39</sup>



Figure 23

In COPD patients the functional residual volume increases as a result of intrinsic PEEP.

Many different approaches are used to reduce intrinsic PEEP. Among them are medication with bronchodilators, a reduction of respiratory minute volume and a decrease in inspiratory time in relation to expiratory time<sup>40</sup> plus high inspiratory pressures, administration of external PEEP or a reduction in Respiratory Rate (RR) or frequency.

AirTrap Control is an approach intended to counteract dynamic hyperinflation. The principle of AirTrap Control involves the continuous measurement of flow rate during expiration. This measurement yields information about the patient's ideal respiratory rate. With an unchanging inspiratory time, the patient's expiratory time will be adapted to his needs (by reducing RR). That should cause intrinsic PEEP to decrease and shift the relaxed respiratory position toward a normal range. The result is efficient ventilation and a possible reduction in the effective ventilation pressure.



Figure 24 Use AirTrap Control when dynamic hyperinflation occurs

### How AirTrap Control works

AirTrap Control monitors ventilation for signs of air trapping and reacts to prevent overinflation of the lungs or dynamic hyperinflation. This function is particularly suitable in the treatment of COPD patients.

As soon as the volume and compliance curves indicate air trapping and an increase in intrinsic PEEP, the backup frequency is reduced. Inspiratory time is held constant.

To ensure that the patient is always adequately ventilated, AirTrap Control is equipped with a minimum safety level which must be met. The limit corresponds to a maximum prolongation of expiratory time measuring 50% or 0.8 seconds. When AirTrap Control is activated, the device responds to the patient's respiratory efforts by switching to inspiration in order to prevent dyspnea or asynchrony between patient and ventilator.



Figure 25

The effects of dynamic hyperinflation: Air trapping causes a shift in the resting expiratory level; intrinsic PEEP develops; tidal volume is reduced. Despite elevated pressure, it is not possible to transport significantly more volume into the lungs.



Figure 26 AirTrap Control causes intrinsic PEEP to decrease slowly.

#### 4.1.10.2 Trigger Lockout

The interaction of man and machine is just one aspect that greatly affects the quality of mechanical ventilation. Many different situations also influence quality and effectiveness.<sup>41</sup>

- Does the patient allow the machine to provide ventilation or does the patient attempt to fight the rhythm dictated by the device?
- Does the ventilator always register the triggering signal from the patient or does the patient have to expend extra effort to trigger a mechanical breath?
- Does faulty triggering of the device occur?

Faulty triggering is a ventilator malfunction. A fauty trigger arises through oscillation in the air column caused by instability of the small airways during expiration (in COPD patients) or by secretions (in neuromuscular patients). Another, premature triggering by the ventilator (referred to as "double triggering") can indicate an inspiratory time that is too short or an overly sensitive cycling. In these cases, the patient's inspiratory effort is not complete at the time of the cycling; the patient triggers the device anew.

Asynchrony between patient and machine is more than an a bothersome occurrence. It can have negative consequences for patient compliance and therapeutic effectiveness.

Synchrony is influenced by:

- selection of mode
- leakage
- patient interface
- patient's underlying disease



Figure 27 Faulty triggerung – Because a COPD patient has an exhausted respiratory pump, a sensitive trigger is required.



Figure 28

Assisted ventilation with false triggering caused by fluctuations in the flow curve with a sensitively set trigger without Trigger Lockout.



Figure 29

The setting of a suitable Trigger Lockout period prevents the ventilator from making a premature switch to inspiratory phase (see Figure 28).
The phenomenon of faulty triggering is often observed during ventilation of COPD patients. It is suspected that during expiration the instability of the airways gives rise to oscillations in the column of air. When the trigger has a very sensitive setting, the oscillations can cause a false positive signal for the start of patient's inspiration.

Oscillations that occur close to ventilation equipment (e.g., rubber lip on mask) are relevant in clinical routine.

When Trigger Lockout is used with a uniformally high trigger sensitivity, the device blocks the inspiration trigger for a defined period at the beginning of expiration. That stabilizes the patient's spontaneous breathing pattern. First, ventilation settings are made, including frequency and inspiratory time (Ti min and Ti max) and then a lockout time for inspiration within a physiological time frame is selected. Recommendation: start the Trigger Lockout at 50% of the expiratory time.

1

NOTE: The Trigger Lockout could influence the patient's respiratory rate. As Trigger Lockout time increases, the maximum possible respiratory rate decreases.



#### 4.1.11 Inspiratory Pressure Ramp

The pressure increase can be adjusted to the patient being treated in order to reach the selected pressure level in a reasonable amount of time during inspiration.

The speed of the pressure rise should be as high as possible as the increasing speed reduces the Work of Breathing. On the other hand, in assisted mode with flexible inspiratory time, an overly fast pressure increase can lead to a shortened inspiration and an elevated respiratory rate.<sup>57</sup> Consequently, it is important to tailor the pressure ramp to each patient.



#### Figure 31

The pressure rise can be personalized for the patient in order to reach the selected pressure level in a reasonable amount of time. A ramp can be set from 1 (steep) to 4 (flat) for the inspiration and the expiration phase\*.

\* with leakage system only

#### 4.1.12 Expiratory Pressure Ramp

An unrestrained expiration and a quick transition from high inspiratory ventilation pressure to expiratory pressure (EPAP) ina case of pulmonary emphysema can promote or cause local collapse of airways and flow limitation. The airway altered by disease is left to its own devices and subject to adverse mechanical conditions.

Figure 32 shows a corresponding flow curve in the presence of pulmonary emphysema, together with a ventilation pressure curve with a steep transition from inspiratory pressure to expiratory pressure. It is possible to protect the small airways from collapse with use of a quickly acting pneumatic splint at the start of expiration. For spontaneous breathing, for example, the German society of respiratory/pneumology experts (*Deutsche Atemwegsliga*) recommends the application of expiratory stenosis to bring about an increase in intrabronchial pressure.<sup>4</sup>

This pressure increase shifts the balance of forces onto the bronchial wall in favor of increased airway width and can keep the airways open longer or, in best case, constantly. A comparable effect can be achieved through a prolongation of the expiratory pressure ramp. The effect of a prolonged, flat ramp on the flow curve can be seen in Figure 33.

A slowly decreasing expiratory pressure ramp can be applied without the ventilator's use of an increased extrinsic EPAP.





A pressure ramp is particularly effective as the counter pressure helps during the phase in which the flow is large and the local thoracic pressure is high due to alveolar hyperinflation.

The risk of collapse in this early expiratory phase is very high. An expiratory ramp – similar to the effect of pursed lips breathing – is an effective countermeasure.

The alternative raising of end-expiratory pressure, on the other hand, can be disadvantageous because either the effective ventilation pressure (pressure difference between IPAP and PEEP) will be reduced or the inspiratory pressure will have to be increased further. The expiratory collapse can be counteracted by the intrabronchial pressure increase at the start of expiration and a carefully monitored reduction of the expiratory peak flow. The expiratory flow remains larger on average, the volume can be exhaled more easily and, as a result, the respiratory position can be lowered.





Effect of a flat transition between inspiratory pressure and expiratory pressure in the flow curve during expiration. The flow remains larger on average; the expiration volume can be increased by the temporary splint.



#### 4.1.13 softSTART and softSTOP

#### softSTART

The softSTART function is intended for patients who cannot tolerate high pressures when ventilation begins or who simply would like the start to be "softer". With this function the actual ventilation pressures are first reached after a pre-set time of T max (from five to 45 minutes in five-minute increments).

A start EPAP, also known as "EPAP min", also is set.

It defines at which pressure softSTART begins. A decision also can be made as to whether the effective ventilation pressure (Pdiff) should be applied (Pinsp soft is switched off) right at the beginning or both pressures (EPAP and IPAP) should be increased over the time. In that case, Pinsp soft is switched on and therapy begins with a pressure difference of 2 cmH<sub>2</sub>O from IPAP to EPAP (see Figure 35).



Figure 35 The example shows the following values:

T max: 30 min EPAP min: 8 cmH<sub>2</sub>O Therapy IPAP: 16 cmH<sub>2</sub>O Therapy EPAP: 10 cmH<sub>2</sub>O

#### softSTOP

Many patients are not ventilated around the clock, but only during the night. The moment the patients take off the ventilation mask and the ventilator thus ceases to provide support, they experience dyspnea or deventilation syndrome.<sup>59</sup>

Causes<sup>70, 71</sup> appear to be the following:

- acclimation of respiratory muscles to the support or to the phases of complete unloading provided by nighttime non-invasive ventilation
- asynchrony between patient's breathing and the device's respiratory support
- not exhaled, trapped air in the distal airways

Morning dyspnea can have a negative effect on therapy acceptance and adherence and thus on the treatment's effectiveness. Dyspnea can be prevented by activating a gentle end to therapy in the form of an inverse ramp (softSTOP) in ventilators equipped with that option. The therapeutic pressure level is not decreased suddenly. It is instead lowered to a minimum level of pressure support of IPAP =  $6 \text{ cmH}_2\text{O}$  and EPAP =  $4 \text{ cmH}_2\text{O}$ within a pre-set timeframe from five to 45 minutes in five-minute increments. When the end of the softSTOP ramp is reached, the ventilator operates at minimum pressure to ensure continuous CO<sub>2</sub> washout until the patient switches off the device.



Figure 36 The example shows the following values:

T max: 45 min

Target IPAP: 6 cmH<sub>2</sub>O Target EPAP: 4 cmH<sub>2</sub>O

## 4.2 Supplemental Oxygen

Patients with chronic respiratory failure and gas exchange disorders may require supplemental oxygen mixed with respiratory air in order to reach a sufficient level of arterial oxygenation.

It should be noted that a prolonged oxygen concentration of > 60% is toxic to an adult patient's lungs.<sup>61</sup> Oxygen toxicity results from the production of hyperoxide anions which become hydrogen peroxide due to hyperoxia.

Such high oxygen concentrations are not necessary for the ventilation of chronic cases in everyday hospital work. A toxic effect from long-term application of highly concentrated oxygen can can be seen at > 40% in premature babies and infants. Damage may occur in vascular endothelium and alveolar cells in the lungs.<sup>43,44</sup> Therefore, the maximum allowable oxygen concentration should be very carefully observed.

When oxygen is fed into the oxygen port at the rear of the ventilator, it is captured in the flow measurement path. When oxygen is introduced only at the end of the inspiratory phase or directly to the mask, the flow is not recorded, giving rise to the risk of a faulty flow trigger and incorrect volume measurement.



#### Figure 37

Rear view of prisma VENT30/40/50/50-C with oxygen port. The point at which oxygen is fed into the system is selected so that the oxygen will be captured in the flow measurement path.

## 4.3 Humidification

With relative humidity of 100% at 37 °C, the air in the alveoli is saturated with water vapor. This corresponds to a water content of 44 mg of water per one liter of air.

This area is referred to as the Isothermic Saturation Boundary (ISB). Approximately three-quarters of the heat and moisture comes from the mucous membranes in the nasopharynx with the remaining one-quarter generated in the trachea.

Ideal mucocilliary clearance occurs at a temperature of 37 °C and relative humidity of 100% (= absolute humidity of 44 mg / l). $^{62}$ 

Patients who are invasively ventilated —and whose nasopharynx is thus bridged—normally receive humidification of 100%. When a tracheal cannula or an endotracheal tube is used in mechanical ventilation, the ISB shifts down the respiratory tract; the physiological heat and moisture exchange in the nose is bypassed. As a result, about three times as much water and heat is drawn from the mucous membranes in the lower respiratory tract.<sup>45</sup> If water vapor saturation falls below 70%, mucocilliary clearance is severely restricted. Below 50% relative humidity, ciliary activity ceases.<sup>59</sup> If cold, dry air is introduced into the respiratory tract over a long period of time, the following complications may arise:

- Dehydration of mucosa
- Loss of ciliary activity
- Reduction of mucokinesis
- Secretion retention and secretion thickening (dyscrinism)
- Impairment of surfactant activity
- Development of obturation atelectasis with deterioration of gas exchange (Oczenski)
- Ulcers on mucous membranes
- Bronchospasm
- Hypothermia
- Infection

Caution also is advised when introducing heated air. At temperatures above 40 °C, there is a risk of damage to the ciliary epithelium, increased secretion production and deterioration in gas exchange.

On average an adult inhaling ambient air via the nose loses 250-300 ml of water daily through evaporation from the airways.

#### Active humidification systems

An active humidification system is based on vaporization that results when a heating element heats fresh tap water. For therapy at home, the use of sterile or boiled water is required only as an exception in some medical cases.<sup>46</sup> The vaporization of the water yields an atmosphere of saturated water vapor.

The inspiratory gas is directed over the surface of the heated water where it is warmed and enriched with water vapor. The electronically regulated water temperature can be set at different levels.

It is important to follow prescribed cleaning procedures in order to rule out the risk of bacterial contamination. Which patients benefit from the use of a humidifier?<sup>40</sup> As mentioned above, all invasively ventilated patients are treated with humidified air. The procedures for patients who receive non-invasive ventilation are not as clear-cut. Most of longterm non-invasively ventilated patients, however, benefit from a humidifier.

The decision should be based on the possible side effects of mechanical ventilation, such as mouth dryness.





prisma VENT40 with prismaAQUA – the prismaAQUA humidifier is attached to the ventilator with a click and used for non-invasive ventilation.

## 4.4 Secretion Management and Cough Support with LIAM

#### Background

#### **Physiology of cough**

The cough is the body's natural protective reflex for removing foreign material from the airways. In its most extreme form, the cough can be seen as forced expiration.

A cough has three distinct phases:

- 1. Deep inspiration comes first (up to 80% of vital capacity).
- 2. Thoracic pressure is generated by applying expiratory force against a closed glottis and contracting the expiratory muscles.
- 3. The glottis opens abruptly, air flows out at a high speed and the secretion is coughed up. The peak speeds in the large bronchii can reach more than 200 km/hr. This process requires the help of the expiratory muscles, which can be best employed when the subject is in a seated or semi-reclining position.

Sufficient strength in the inspiratory and expiratory muscles is required for the cough function.

A cough causes changes in the width of the large cartilage-encased bronchi. According to the Venturi effect, during forced expiration or cough, secretions are transported by means of an orally directed impulse. A cough is reflexively triggered by mechanical and inflammatory irritation of the pharyngeal area, trachea and carina of trachea to the 5th and 6th generations.

### Pathophysiology of cough

Varied pathophysiological processes<sup>63</sup> lead to a restriction of and change in the cough fuction.

They include:

Narrowing of airway lumen

Swelling of the mucosa and bronchospasms, which typically occur in asthma patients, cause the airway lumen to narrow. As a result, patients must expend more energy to generate an effective cough.

 Paralysis and deterioration of ciliary epithelium

Over the course of a viral or bacterial infection in the respiratory tract, mucocilliary clearance ceases to function. The cough function helps out as a replacement mechanism for bronchial cleaning.

### - Changes in secretions

In neuromuscular patients the aspiration of saliva leads to chronic bacterial colonization. COPD patients suffer from recurrent infections and increases in the quantity and viscosity of secretions.

#### - Muscle weakness

As a result of their underlying illness, neuromuscular patients have trouble generating the required Peak Cough Flow (PCF) for a normal cough function. In place of the abdominal muscles, they attempt to use the pectoralis and shoulder girdle muscles. The deglutition disorder which often affects these patients leads to chronic aspiration.

## Neuromuscular disease and limited cough function

Patients with underlying neuromuscular disease (e.g., muscular dystrophy Duchenne or Amyothrophic Lateral Sclerosis or ALS) suffer from weakness in the inspiratory and expiratory muscles. Consequently, they simply do not have the inspiratory and expiratory strength to inhale deeply and to generate the necessary peak flow of 160 to 270 liters/minute required for the normal cough function.<sup>64</sup>

A disorder of the cough function leads to a variety of pathophysiological changes. The increased retention of secretion causes a narrowing of the airway lumen, makes ventilation difficult in the presence of strong flow resistance and impairs gas exchange in the alveoli. This last issue may be made visible by rapid declines in the pulsoxymetrically measured oxygen saturation. Atelectasis occurs with greater frequency. The collapse gives rise to pathological germ cells which reduce the area available for gas exchange.

The increased bacterial colonization leads to frequent viral and bacterial infection in the airways, making the patient more susceptible to developing pneumonia.

Viral or bacterial infections can impair the functioning of the ciliated epithelium, so that mucociliary clearance is no longer guaranteed. The patient's susceptibility to infection increases again and the vicious circle repeats itself.

Severe secretion retention can result in ventilatory insufficiency in this patient group.

Under ventilation conditions too, secretion retention in neuromuscular patients should be prevented. Secretions can block the airways and jeopardize the success of mechanical ventilation.<sup>48</sup> Inadequate secretion mobilization is considered the most frequent cause of failure in ventilation treatment. Effective secretion management, on the other hand, reduces the hospitalization rate<sup>49</sup> and prolongs survival.<sup>50</sup>

#### **Therapy options**

Secretion mobilization is a therapeutic approach used for patients with neuromuscular disease whose cough function is diminished. A distinction is made between secretion-dissolving and secretion-transporting techniques. In the former, measures include percussion, vibration and oscillation. In percussion, a cupped hand held over the thorax is tapped, causing vibrations in the air column. Secretions are mobilized with this type of manual tapping or by means of IPV (Intrapulmonary Percussive Ventilation). Both exploit the physical principle that mechanical action makes the secretions less viscous and thus easier to remove.

As a neuromuscular disease progresses, manual techniques no longer suffice for effective removal of accumulated secretions.

Bronchoscopy is one secretion-transporting measure. This procedure, which has proven to be very helpful in acute situations, can be conducted during mask ventilation.

Secretion transport takes place through forced expiration. Among the oldest secretion mobilization techniques is postural drainage. Regularly changing the patient's position favors a homogenous ventilation/perfusion ratio. It is often used as a supplement to other techniques. These varied measures can be combined. For example, percussion used with postural draining moves secretions into the large airways. Gravity causes the secretions to flow out of the affected sections of the lungs. This method is not efficient in cases of pre-existing respiratory failure.

Therapy is indicated from a PCF of < 270 I / min!<sup>2</sup>

Cough may be considered the most extreme form of forced expiration. Cough support is an essential part of treatment management for patients with chronic respiratory failure.

In cases of neuromuscular disease, an efficient cough can avoid or significantly delay the need for ventilation or a tracheotomy!

## Therapeutic effects of secretion management

Secretion management often is required in treatment of patients with neuromuscular disease even before ventilation is indicated.

Secretion management accomplishes the following:

- delays the initiation of ventilation<sup>51</sup>
- ensures alveolar gas exchange and thus the ventilation's effectiveness<sup>48</sup>
- eliminates the need for hospital stays  $^{\scriptscriptstyle 48,\,49}$  and
- increases patients' life expectancy.<sup>50</sup>

#### LIAM (Lung Insufflation Assist Maneuver) – cough support

Ventilation therapy should be coupled with efficient secretion management for neuromuscular patients with severe respiratory failure.

In response to this therapeutic requirement, an innovative treatment concept was developed that integrates the secretion mobilization function in the ventilator.

Specifically, the unique cough support process LIAM (Lung Insufflation Assist Maneuver<sup>52</sup>) was integrated in the ventilators VENT50/50-C.

The process is based on an inspiratory maneuver in which the tidal volume (insufflation volume) is increased through insufflation and the lungs and thorax are "pre-loaded" with a deep inhalation. Thus, the subsequent cough flow can be significantly increased.<sup>53</sup>

#### How LIAM works

An inspiratory manuever is used during ventilation to overlay an additional defined mechanical breath ( $\Delta$  P) on IPAP. The increase in lung volume and the thoracic expansion thus generated cause an increase in Peak Cough Flow, or the maximum cough, which makes secretion elimination possible. The patient's cough is then more productive.

### **Scientific evidence**

The effectiveness of LIAM as cough support for patients with a variety of neuromuscular disorders was proven in a study <sup>53</sup> published in 2014. The objective of the study was to determine the individual optimum insufflation capacity for the subsequent cough.

Results showed that an insufflation capacity for the cough was about 90% of the individual achievable maximum capacity. The pressures generally required lay between 30 and 40 cmH<sub>2</sub>O, provided that an adequate insufflation time is set.

The study also documented that insufflation with LIAM resulted in a maximum insufflation capacity 150% (mean value) above baseline vital capacity. With an optimum insufflation volume, Peak Cough Flow of 110 l/min increased to 205 l/min (mean values). These results show that—even with severely limited muscle strength—the cough flow after deep insufflation was increased beyond the critical threshold of 160 l/min and that effective secretion management is possible with LIAM.

#### Settings are made on the device in the following steps:

1. Switch on LIAM (insufflation) in the Ventilation menu

- 2. Select deltaP LIAM → IPAPmax
- 3. Select Ti LIAM and Te LIAM
- 4. Set length of time for LIAM maneuver
- 5. Set intervals at which LIAM is repeated (optional)
- 6. Select the number of LIAM-supported breaths (insufflation)

LIAM key to activate/deactivate the LIAM function



Figure 39 prisma VENT50/50-C with LIAM key to activate/deactivate the LIAM function



#### Figure 40

How LIAM works: The IPAP pressure generated by the device is overlaid with deltaP LIAM, resulting in an IPAPmax. LIAM increases Ti LIAM to the pre-set pressure level and changes Te LIAM back to EPAP at the start of the expiratory phase.



Figure 41 Pressure curve with use of LIAM

If the LIAM function achieves the desired effect before all LIAM breaths have been delivered, the user can prematurely terminate the function by pressing the LIAM key again. If LIAM should be used for a longer period, it can be set to run from a minimum of one minute to continuous ∞ application.

A setting can be made for how often (from 15 seconds to 24 hours) a cycle of LIAM breaths should be repeated. If the patient needs several consecutive LIAM breaths to complete the maneuver successfully, up to 10 breaths can be administered.



Figure 42

If LIAM is to be applied over a prolonged period, settings can be made to meet specific patient needs for the number of consecutive breaths, the intervals at which they should be repeated and the duration of the application. This diagram shows two series of five consecutive LIAM breaths.

The process was developed in such a way to ensure that the set deltaP and deltaP LIAM have no additive effect when the target volume function is being used. This setting rules out the risk of undesired over-pressure. The LIAM function, in comparison to air- or breath-stacking, offers varied advantages.

LIAM can be used in all ventilation modes.

- The patient does not have to switch off the device for secretion management, unlike with other stand-alone devices.
- Because stacking is not required, the maneuver is significantly shorter than by air-stacking and therefore more comfortable for the patient.
- Especially important for ALS patients is the fact that insufflation does not require that the glottis be closed. However, glottis control remains an important prerequisite for an adequate cough with a purely positive pressure maneuver like LIAM.

- The patient retains a certain degree of independence; LIAM also can be triggered by the caregiver.
- An acoustic signal can be given to the patient that indicates when the LIAM plateau has been reached. At that point, forced expiration supported by LIAM is possible.
- An adjustable expiratory time under LIAM allows the maneuver to be further optimized with regard to cough or forced expiration.

The integration of the LIAM function is part of a holistic approach to ensure comfortable ventilation for patients with secretion retention issues.

If LIAM alone does not achieve an effective increase in cough flow—because the patient has no glottis control, for example—an alternative would be the use of an In-Exsufflator.

LIAM is an important therapeutic expansion to ventilation functions.

## **4.5 Monitor Connection**

Data transmission, which has been standard in intensive care ventilation for years, is becoming more important for home ventilation devices. Documentation of ventilation treatment time in particular is of special interest. The ventilators LUISA and prisma VENT

transmit settings, therapy data, including ventilation time and alarms, to the Philips IntelliVue monitors. From these monitors the data can be forwarded to Patient Data Management Systems (PDMS).





Figure 44 Single-patient use mask JOYCEclinic Full Face NV



Figure 45 Reusable full face mask LENA

> Figure 46 Reusable nasal mask CARA

### 4.6 Patient Interface

The quality of the patient interface in non-invasive ventilation plays a very important role in patient compliance with treatment. What matters most is a good fit, especially when high pressures are applied or when the pressure difference between inspiration and expiration is very large.

One critical element is the mask cushion. The anatomical shape should create a good seal and leave no pressure marks on the face as the mask has to be worn for up to 24 hours. When the right size is chosen, the mask will fit the patient comfortably.

Also important is the interplay between mask cushion and the headgear. The headgear material should be slightly inflexible so that it can hold the cushion on the face when it is subjected to high pressures or large pressure differences. The right setup allows no leakage and prevents any pumping of the mask. Soft materials and edges prevent scratching or pressure marks on the patient's cheeks.

Masks with an adjustable forehead support relieve pressure, especially on the bridge of the nose. The ideal distance between the bridge of the nose and the forehead contribute to a long-lasting comfortable mask fit.

#### Mask types – full face or nasal

Full face masks are used mostly in acute care situations. The advantage is that access to both airways is covered, making it easier for the patient to let go and "fall into" ventilation. For chronic stable patients, the nasal mask has the advantage of being lightweight. Furthermore, a nasal mask exerts less pressure on the face.

Other types of mask that are used include nasal pillows, mouthpieces, CPAP helmets and masks that cover the entire face.

#### Vented vs non-vented

Full face masks are offered as vented or non-vented variants. The choice depends on the ventilator and patient circuit in use. The leakage circuit system is always used with the vented mask and the valve circuit system with the non-vented variant. The diameter of the tubes and the mask connections are standardized, so that an incorrect connection cannot be made.

#### Hygienic reprocessing

In the hospital, the choice of mask for standard usage depends mostly on whether the mask can be hygienically reprocessed.

Reusable masks offer a good fit and a high degree of wearing comfort. If a patient requires long-term ventilation at home, the patient continues using the reusable mask previously adjusted specifically for him or her. At a change of patient in the hospital, the mask is hygienically reprocessed by means of disinfection or sterilization. If hygienic reprocessing of the masks cannot be guaranteed, single-patient use masks are used. Their price is generally lower than that of reusable masks, but their wearing comfort may be lower too.

Disposable masks are not used in the hospital.



Figure 47 Elbow variants: NV, NV + AAV, vented



Figure 48 Endoscopy adapter NV

#### **Endoscopy during ventilation**

An endoscopic examination of a patient can be conducted without interrupting the ventilation. For this purpose, a mask-specific elbow replaces the conventional elbow right on the mask. A silicone cap at the front of the endoscopy adapter is opened to allow the endoscope to be inserted. Upon completion of the examination, the cap is closed. Advantage: The patient suffers no dyspnea or shortness of breath while the doctor conducts the examination.

#### **Quick-release cord**

Both airways are covered by the full face mask. With nose and mouth covered, a disoriented may panic when he/she wakes at night. Or for diseaserelated reasons, a patient may be unable to handle the clips on the headgear without help. An optionally available quick-release cord can help in such situations. It is used in place of a clip. The patient need only pull the quick-release cord downwards to loosen the mask from the face.

# 5 Practical Usage Tips

## 5.1 Cases of Chronic Ventilatory Failure

## Acclimating chronic stable patients to ventilation

Preliminary patient settings are made on the ventilator when the patient is awake. The choice of the initial pressure depends on the patient's underlving disease and physical condition. As a rule, a low pressure is chosen for the beginning and is increased within minutes. In treatment of patients with Obesity-Hypoventilation Syndrome, a PEEP is always used. When determining the I:E ratio, the doctor usually starts with a relation of 1:2. For patients with an obstructive disorder, the expiratory phase is often prolonged (e.g., to 1:2.5). An automatic adjustment made by AirTrap Control can be helpful in such cases.

Some ventilation specialists have reported good experience with nighttime pressure regulation for a sleeping patient. If the patient cannot tolerate the high pressure regulation, he/she is sent home for four weeks to use a device set to his/her initial adaptive pressure with the objective of optimizing the pressure setting later. In cases of pre-existing hypercapnia, the goal is to achieve normocapnia.

As a rule, **ST or T mode** is selected as the first setting. The mode aPCV is also cosidered a standard ventilation mode. It should be noted that if the patient is allowed to trigger the device at the start of ventilation, there is a risk that the patient could be hyperventilated.

The decision about using minimum volume or **target volume** is often based on the results of a blood gas analysis. Close attention should be paid to substantial fluctuations in leakage, which can trick the ventilator into thinking that sufficient ventilation is being delivered even though the patient is underventilated. Because intelligent algorithms in modern ventilators like prisma VENT50 can differentiate leakage from volume compensation, faulty interpretations are prevented. Nevertheless, the fit of the mask should be checked in order to rule out substantial leakage.

During the setting phase, the **respiratory rate** is often set two breaths above the patient's own rate. Patients with an underlying neuromuscular disease acclimate themselves relatively quickly to ventilation (two to three days); thoracicrestrictive patients generally need five to seven days, while COPD patients may require up to 14 days.

The acclimation phase, i.e., the time it takes for a patient to become accustomed to ventilation, lasts two to 14 days, depending on the nature of the underlying disease.

### Mask fit and choice

Mask selection and acclimation to ventilation are important issues patients in a chronic stable state or with acute respiratory failure.

Chronic stable patients often do well with a nasal mask. Patients with acute respiratory failure are almost always fitted with full face masks.

The coverage of all upper airways makes it easier for the patient to relax, to "let go". Therapy pressures >  $20 \text{ cmH}_2\text{O}$ produce the same effect as the higher flow is distributed better over the entire surface of the upper airways. For fitting purposes, it should be noted that the larger contact surface can cause more pressure marks on the patient's face.

Other points to consider when fitting a patient with a mask:

- select the right mask size (for example, a suitable full face mask for patient who always sleeps with open mouth)
- make sure there is no leakage or unequal pressure distribution (If a patient with dentures uses a full face mask, he/she should wear a full set in order to prevent leakage from the sides of the mask.)
- brief the patient or caregiver in the use of the mask.

### Tip:

In some cases it may be helpful to acclimate the patient to the mask and ventilation step by step. First, place the mask without headgear on the patient's face and adjust the forehead support. Attach the tube so that the patient with shortness of breath can be readily ventilated. Then pull the headgear over the back of the head and fasten the clips while the patient holds the mask in place. This process allows the patient to get a feeling for the mask without causing any panic.

Make sure to adjust the headgear so that it is as loose as possible.

If possible, start with a low pressure and then increase it.

The mask symbol in the prisma VENT display indicates how well the mask fits (and seals):

Green solid:	No/minor leakage
Orange solid:	Up to 25 l/min leakage
Orange flashing:	More than 25 l/min leakage



Figure 49 Green mask symbol indicates a good mask fit.

### Nighttime Monitoring

For purposes of making ventilation settings, patients with chronic respiratory failure are diagnosed with the standard pulmonary function test and blood gas analysis and sometimes with polygraphy. As far as hypercapnic insufficiency is concerned, there seems to be little difference in the effect of daytime vs. nighttime ventilation<sup>54</sup>. However, it has been suggested<sup>55</sup> that nighttime monitoring could contribute to the prevention of hypoventilation phases and to an improvement in sleep quality. Some ventilation centers rely on long-term capnometry as a control parameter. The validity of this measurement, however, should be examined.

Why is it important to check ventilation quality during sleep?

First, most patients with chronic respiratory failure receive ventilation at night. It is therefore reasonable to check the conditions under which ventilation is administered to the patient.

Moreover, sleep is a physiological state to which respiratory regulation, muscle tone and consciousness all adapt.

Pathophysiological changes in respiration can often be detected early during sleep. One of the first signs of a chronic hypercaphic disorder is sleep-related hypoventilation during REM (Rapid Eye Movement) sleep. Furthermore, abnormal respiratory events frequently occur during sleep, so that ventilation parameters set during the day are often therapeutically inadequate at night. Asynchrony between patient and device tends to develop while the patient sleeps, leading to phases of periodic breathing, glottis closure and, because of the patient's changes in position, significantly more leakage. Nocturnal respiratory events give rise to sleep fragmentation and place an additional burden on the cardiovascular system<sup>55</sup>.

Nighttime monitoring serves to determine the patient's respiratory rate. As a rule, the frequency for the device is set one to two breaths above the patient's respiratory rate. This setting ensures that the patient is given controlled ventilation in ST mode during the night and his/her respiratory pump is unloaded to the greatest extent possible.

If the patient is awakened by a high mechanical respiratory rate and thinks it is too fast, the dotor can simply lower the frequency by one or two breaths per minute!

It is particularly difficult to find the proper settings for COPD patients. At high respiratory rates they are at risk of developing dynamic hyperinflation. A solution to this problem is controlled ventilation or use of the ventilation function AirTrap Control.

Continuous monitoring of oxygen saturation takes place during the setting night. Blood gases are normally checked at night and in the morning after the patient wakes. Device software is used to check whether phases of asynchrony and faulty triggering occur during the night. If that is the case, the use of Trigger Lockout is recommended, especially for COPD patients.

A further recommendation is to check whether patients with chronic stable respiratory failure have indications of an obstruction in the upper airways (i.e., obstructive sleep apnea). In this case EPAP or PEEP should be set at the level of the therapeutically required CPAP.

Polygraphy/polysomnography have proven effective as a means of monitoring ventilation patients at night. Supplemental video recordings also are recommended as they provide valuable information about nighttime events.

During the course of treatment, the parameters can be monitored offline via the device software or remotely via a cloud connection.

## Settings for ventilation in hospital – an example for trial intervals:

- first day: 15 20 minutes under doctor's supervision
- next several days: 4 to 5 hours of acclimation
- thereafter, nighttime ventilation.

During the setting phase the effectiveness of ventilation is judged on the basis of symptoms and blood gas analysis. Ideally, pulse and blood pressure are checked along with oximetry and/or PTc  $CO_2$  levels and measurement of tidal volume<sup>2</sup>.

The delta IPAP-EPAP increase should be made slowly until normocapnia is achieved.

As a rule, patients may be released after five to 14 days. The prerequisite for release is that the patient can tolerate non-invasive ventilation for several hours per day, depending on the underlying disease.

After six weeks of ventilation at home, the patient is again examined. At that point it is often necessary to adjust the ventilation parameters. Depending on the indication, it is recommended that parameters be checked subsequently once or twice per year.

## 5.2 Pediatric Aspects in Ventilation

A number of genetic or acquired diseases can lead to chronic respiratory failure in children. In the past, the tracheotomy was considered the only treatment solution, but today noninvasive ventilation is being used with increasing frequency and success.

Respiratory failure in children is generally just one part of a complex clinical picture. These children are often treated with mechanical ventilation at specialized hospitals.

The diseases which lead to chronic respiratory failure and long-term outpatient ventilation differ from those that affect adults.

The most frequent indications for outpatient ventilation of children and juveniles are:

- genetic diseases of nerve and muscle system, e.g., Duchenne, spinal muscular atrophy
- chronic pulmonary disease, e.g., cystic fibrosis, bronchopulmonary dysplasy
- thoracic deformities, e.g., thoracic scoliosis
- central respiratory regulation disorders, e.g., Undine Syndrome.

About three children/juveniles per 1000 residents require long-term ventilation. Approximately two-thirds of that number can be treated at home. Note: The same contraindications for non-invasive ventilation that apply to adults also apply to children (see Table 2, Page 7).

In light of a child's growth and the disease's progress, regular checks should be made of the ventilation's effectiveness and of the ventilation settings, which are then adjusted as needed <sup>60</sup>.

The treating physician is advised to consider the psychological aspects of treatment and involve the child in all decisions related to therapy. Given a child's limited capacities for understanding and cooperation, it is critical to the acceptance and successful ventilation that the child does not feel restricted by the technology and sees the benefits from treatment. A great deal of experience is demanded for ventilation of seriously ill children, infants and toddlers. Only a very few specialized centers offer this service.

When given proper briefing and care, children requiring ventilation have good therapy compliance – with help from family members – and a good quality of life. Mobility is very important for children.

Another treatment option besides ventilation is High-Flow Therapy. It can be applied with a nasal cannula, which lies more lightly and loosely on the face than a mask (more information about High-Flow Therapy is in 4.1.9).

Special attention must be paid to the following in the treatment of children:

### **Patient Interface**

- The child may not be able to put on and take off the mask without help.
- To prevent midface hypoplasia, use maks with very low contact pressure on infants and small children.
- Because of the danger of CO<sub>2</sub> rebreathing, the smallest possible amount of dead space should be selected.
- Special tubes have to be used for small volumes.

### **Technical features**

- Pressure-controlled assisted ventilation, possibly with volume safeguard
- Sensitive inspiration trigger
- Small tidal volumes (50 ml)
- Flow and time-controlled inspiratory time with backup frequency

# Children's acceptance of ventilation

Ventilation of a child works well when the treatment is comfortable for the patient, the respiratory disorder can be normalized, the patient acknowledges the benefit of treatment and the family accepts the treatment.

# 6 Product Solutions for Ventilation

## 6.1 Product Concept for Hardware shown with LUISA



Figure 50 LUISA, life-support ventilation

### 6.2 prisma VENT30 prisma VENT40

## Mobility and convenience at all levels

- Pressure range up to 30 cmH<sub>2</sub>O (prisma VENT30)
- Pressure range up to 40 cmH<sub>2</sub>O (prisma VENT40)
- Use with leakage circuit systems (15 mm / 22 mm)
- With autoST mode (autoEPAP, autoF)
- Helpful functions for COPD treatment: AirTrap Control, Expiratory Ramp and Trigger Lockout
- Integrated oxygen feed port
- Three stored ventilation programs
- Complete alarm management
- Battery capacity for up to 12 hours of operation



prisma VENT30 and prisma VENT40

# 6.3 prisma VENT50 and prisma VENT50-C\*

#### **Top-level ventilation therapy**

All the features in prisma VENT30/40 are also in prisma VENT50 and prisma VENT50-C\*, along with the following:

- Leakage and single patient circuit system with patient valve, which allows treatment of a broad spectrum of diseases
- Integrated secretion management and cough support LIAM
- Mouthpiece ventilation



Figure 52 prisma VENT50/50-C

\* with High-Flow mode

### 6.4 LUISA

#### For life-support ventilation

- Simplest operation
- Two operating positions: horizontal or vertical
- No adapter
- Brilliant touch screen
- Functionality: Up to 30 ml tidal volume, High-Flow mode (optional)
- Can be used with all circuit systems: leakage system, single patient circuit with valve, double patient circuit
- Pressure-controlled and volume-controlled ventilation
- Mobility for use at home and in hospital



### 6.5 LUISA App

The LUISA App lets the user keep an eye on ventilation treatment at all times. If a seond ventilator is used, it too can be connected to the app. The following information is available in the LUISA App:

- current device status
- display of battery capacity
- current measurements from ongoing treatment
- display of ventilation program
- device statistics





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Figure 55 prisma CLOUD

### 6.6 prisma CLOUD

# Telemedical connection for experts and patients

The prismaCLOUD permits simple, flexible and secure monitoring of treatment. Automatically provided information about the current day's treatment can be retrieved from anywhere. If necessary, treatment settings for a prisma VENT device can be adjusted immediately via the CLOUD. For LUISA, safety is the number one issue. Read only, no settings.

### 6.7 prismaTS Fast and flexible patient management

Quick overview or detailed analysis of treatment

The flexible, intutitively used software offers many options, including patient management, dynamic report generation and device management with remote control.



Figure 56 prismaTS

### 6.8 Ventilation Settings with Sonata/Scala (PSG/PG)

Coupling sleep therapy devices with home ventilators has become standard operating procedure in polysomnography (PSG) and polygraphy (PG) in the past several years.

In such a setup, measurements (e.g., flow, pressure, leakage, volume) from the therapy device are fed as real-time signals into the PG/PSG and displayed synchronically with their signals in graphics. The CO<sub>2</sub> parameter, which is so important for ventilator settings, is fed as an external analog signal from a transcutaneous measuring device, for example, in synchronous form as well.

Furthermore, it is possible to join the measurements to a synchronized video and audio recording. That paves the way for meaningful therapy settings or monitoring with regard to all relevant parameters such as sleep, oxygen saturation, work of breathing and flow, position, movements, ECG, pulse, pressure support, mask leakage, CO<sub>2</sub>.

The devices Sonata (PSG) and Scala (PG), equipped with the latest and most innovative technology from the diagnostics field, are available for this purpose.

Different solutions for implementation can be selected to suit the user's routine work and spatial infrastructure.

One option is to install all components on a rolling stand, which can be used in different rooms or stations as needed. The other is a permanent installation of the camera module, PG/PSG and therapy device technology in a designated room.



Figure 57 Sonata (PSG) and Scala (PG)

## 7 Outlook

Non-invasive ventilation has become firmly established in the treatment of chronic respiratory failure and ventilatory failure.

The prevalence of typical indications for NIV such as neuromuscular diseases and thoracic wall disorders appears to be relatively stable. Significant increases can be observed, however, in the number of COPD patients and patients with Obesity-Hypoventilation Syndrome. No reversal of this trend is currently expected.

Ventilators also are used to treat patients exhibiting periodic breathing (Cheyne-Stokes Respiration) and clinical symptoms once all drug therapies and alternative ventilation treatment methods (e.g., anti-cyclical servoventilation) have been exhausted.

The varied respiratory and ventilatory disorders differ in their pathophysiological characteristics. It seems reasonable to respond in each case with targeted therapeutic means. The functions AirTrap Control, Trigger Lockout and LIAM – to name just a few – help to improve treatment and outcomes. Many of these innovative technologies employ biofeedback systems that continuously adapt to the needs of patients with respiratory or ventilatory failure. The overall goal is optimizing medical care. One way to encourage greater patient compliance is through the patient interface. In recent years the quality of standard masks has clearly improved to the point that they can replace more expensive specially produced masks.

Most patients with chronic respiratory failure are ventilated overnight. It is therefore no surprise that polygraphy or polysomnography is becoming more important during the ventilation settings phase.

Everyone involved in ventilation treatment can now be networked by means of a cloud connection. In the future, all parties are expected to get involved in the ongoing design and increased use of cloud-based computing.

## 8 Glossary

ARF	Acute Respiratory Failure	
aPCV/aVCV	assisted PCV/VCV	
COPD	Chronic Obstructive Pulmonary Disease	
СРАР	Continuous Positive Airway Pressure	
CRF	Chronic Respiratory Failure	
CSR	Cheyne-Stokes Respiration	
EOM	Equation Of Movement	
LIAM	Lung Insufflation Assist Maneuver	
NIV	Non-Invasive Ventilation	

PCV	Pressure-Controlled Ventilation
PEEP	Positive End-Expiratory Pressure
PSV	Pressure Support Ventilation
S	Spontaneous
SIMV	Synchronized Intermittent Mandatory Ventilation
ST	Spontaneous Timed
т	Timed
VCV	Volume-Controlled Ventilation
WOB	Work Of Breathing

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