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.

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

In addition to treating classic indications such as neuromuscular and thoracic wall disorders, non-invasive ventilation also 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 therapy is now focused primarily on ventilatory failure (hypercapnic insufficiency, Type II) and secondly on respiratory failure (hypoxemic insufficiency, Type I).

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, with the use of ventilation technology and electronic data processing, medical professionals can optimize their work processes in ventilation units – all for the good of the patient.

2 Ventilation – State of the Art

Along with blood circulation and consciousness, respiration is a vital function which sustains human life. A disorder or breakdown of any vital function is life-threatening.

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. Patients suffering from hypoxemic respiratory failure, e.g., in cases of pulmonary edema, depend on the help of a ventilator.

Patients who require mechanical ventilation can now be adequately ventilated outside the hospital in their own homes. Furthermore, as many devices are small and manageable, patients gain a certain degree of mobility.

Use of mechanical ventilation is increasing along with the potential represented by 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 insufficiency are treated with NIV.

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.

The treatment of chronic respiratory failure or ventilatory insufficiency by means of ventilation technology is considered an important therapeutic measure to reduce the morbidity and mortality of the patients affected. [2, 3, 4]. Moreover, ventilation has a positive effect on the patient's quality of life[5].

Decision Support in Acute Situation

Attributes of Non-Invasive and Invasive Ventilation

⊖ Negative attribute ⊕ positive attribute

Complications and clinical aspects	Invasive Ventilation (IV)	Non-Invasive Ventilation (NIV)
Ventilator-associated or tube-associated pneumonia	⊖ Risk increase from third or fourth day of ventilation	⊕ Rare
Tube-associated increase in Work of Breathing	⊖ Yes (during spontaneous breathing and with insufficient tube compensation)	⊕ No
Early and late tracheal damage	⊖ Yes	⊕ No
Sedation	⊖ Often necessary	⊕ Rarely required
Intermittent application	⊖ Possible	⊕ Often possible
Patient can cough effectively	⊖ No	⊕ Yes
Patient can eat and drink	⊖ Difficult with tracheo stoma, with intubation: no	⊕ Yes
Patient can speak	⊖ Difficult	⊕ Yes
Patient can sit upright	⊖ To a limited extent	⊕ Often possible
Weaning from ventilator is difficult	⊖ In 10 - 20% of all cases	⊕ Rare
Access to airways	⊕ Direct	⊖ Difficult
Pressure points on face	⊕ Not with intubation, but may appear in corners of mouth	⊖ Often
CO_2 rebreathing	⊕ No	⊖ Rare
Leaks	⊕ Hardly	⊖ More or less, depending on mask fit
Aerophagy	⊕ Hardly	⊖ Often

Table 1[6]
The attributes of IV und 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:

Absolute Contraindications

- No spontaneous breathing, gasping
- Fixed or functional blockage of the airways
- Gastrointestinal bleeding or Ileus
- Coma

Relative Contraindications

- Hypercapnic coma
- Severe Hypoxemia or acidosis (ph < 7,1)
- Massive secretion retention despite bronchoscopy
- Hemodynamic instability (cardiogenic shock, myocardial infarction)
- Severe agitation
- Anatomical and/or subjective interface-incompatibility
- Directly after upper gastrointestinal OP

Table 2 [6] (= see Westhoff M. et al., 2015)
Given the contraindications, it can be concluded that invasive ventilation will retain some significance. 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 [7, 8] 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 [7], some countries report significant variances in the distribution percentages of some of the indications. The diseases in the "classic" 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 [9] 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. The dramatic increase in obesity translates into rapid growth in the population of ventilated patients, many of whom have Obesity-Hypoventilation Syndrome[10].

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 [11, 12].

These days experts are looking critically at the $PaCO_2$ levels in COPD patients whose hypercapnia is not effectively resolved by ventilation. A study made in 2009[13] shows that non-invasive ventilation has a life-prolonging effect in COPD patients with chronic ventilatory insufficiency. However, the patients under NIV had poorer quality of life. It should be noted that ventilation pressures were used that were too low.

Evidence is accumulating that the reduction in $PaCO_2$ through the application of higher inspiratory pressures can have a positive effect on life expectancy[14]. One predictive indicator for life expectancy of COPD patients appears to be the six-minute walk test [15]. Furthermore, stable hypercapnic COPD patients show better tolerance of high ventilation pressures accompanied by a more effective reduction in nocturnal $PaCO_2$ [16].

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 [8].

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**

Indications for Non-Invasive and Invasive Ventilation

as treatment of chronic respiratory failure / ventilatory insufficiency (taking into account therapy decision in cases of acute exacerbation).

Disease	Indication for Non-Invasive Ventilation	Indication for Invasive Ventilation*
Underlying neuromuscular disease	Alveola hypoventilation in connection with at least one of the following parameters: - chronic hypercapnia ($PaCO_2$ ≥ 45 mmHg) during the day and/or - at night (≥ 50 mmHg) and/or - normocapnia during the day with increase in PTc CO_2 by ≥ 10 mmHg at night - or rapid decrease in vital capacity - at a Peak Cough Flow (PCF) ≤ 270 l/min for mechanical secretion management is required.	- Requires ventilation and despite use of device, no sufficient benefit from NIV - Dysphagy with recurring pneumonia - NIV required > 16 hours/day
Thoracic restriction	- Hypoventilation symptoms - Chronic daytime hypercapnia with $PaCO_2$ ≥ 45 mmHg - Nocturnal hypercapnia with $PaCO_2$ ≥ 50 mmHg - Daytime normocapnia with increase in PTc CO_2 of ≥ 10 mmHg	- Requires ventiation, but during treatment significant worsening of blood gas levels, severe acidosis (pH < 7.35)

* when wanted by patient

Disease	Indication for Non-Invasive Ventilation	Indication for Invasive Ventilation
Obesity Hypoventilation Syndrome	Obesity and hypercapnia despite adequate CPAP therapy - ≥ 5-minute increase of PTc CO_2 ≥ 55 mmHg or $PaCO_2$ ≥ 10 mmHg compared to waking state or Desaturation < 80% SaO_2 over ≥ 10 minutes - If re-evaluation after three months of CPAP therapy shows no clinical improvement and daytime normocapnia	- Contraindications for NIV, e.g., dysphagia
COPD	Symptoms of ventilatory failure and chronic hypercapnia and reduced quality of life Indication criteria (at least one additional criterion must be fulfilled) - chronic daytime hypercapnia with $PaCO_2$ > 50 mmHg - nocturnal hypercapnia with $PaCO_2$ > 55 mmHg - stable daytime hypercapnia with $PaCO_2$ 46-50 mmHg and increase in PTc CO_2 by > 10 mmHg during sleep - stable daytime hypercapnia with $PaCO_2$ 46-50 mmHg and at least two acute exacerbations with respiratory acidosis requiring hospitalization within the previous 12 months. - directly subsequent to an acute exacerbation requiring ventilation, according to clinical evaluation	- Requires ventilation, but in course of treatment, blood gas levels worsen considerably, severe acidosis (pH < 7.35)

Table 3 [17, 2]
Indications NIV and IV

Therapeutic Effects of Mechanical Ventilation

Disease	Medical Benefits from Ventilation
Neuromuscular illness	- Improvement in blood gases - Reduction of respiratory complications - Improvement in sleep qualityt - Better quality of life - Increased life expectancy
Thoracic restriction	- Unloading of respiratory musclesr - Reduction of respiratory stimuli - Improvement in sleep quality [18] - Reduction of atelectases - Decrease of pulmonary arterial hypertension [18] - Better quality of life [5] - Increased life expectancy
Obesity Hypoventilation Syndrome	- Normalization of ventilation day and night - Improvement in blood gases - Improvement in sleep quality
COPD	- Reduction of hypercapnia - Improvement in sleep quality - Increased life expectancy
Cheyne-Strokes Respiration	- Normalization of nighttime breathing - Improvement in sleep quality - Improved physical capability - Preliminary indications of increased life expectancy[19]

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, the length of ventilator use by these patients is more than six years[7]. Patients with Obesity-Hypoventilation Syndrome (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 presence of hypercapnia despite CPAP therapy[9]. Negative prognostic factors for OHS are hypoxemia and elevated inflammation markers[20].

4 Ventilation Technologies

Before a patient can be mechanically ventilated, the correct breathing pattern has to be found. That is, the timing of the respiratory cycle with respect to pressure, flow and volume has to be determined. A basic distinction is made between pressure-controlled and volume-controlled modes.

Parameters to be set on a pressure-controlled device include:

- ventilation mode
- levels of inspiratory and expiratory pressure
- tidal volume
- respiratory rate (frequency)
- respiratory ratio (inspiration to exspiration, I:E) or inspiratory time
- backup frequency (in S and ST mode)
- trigger sensitivity
- pressure rise time

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

Ventilation pressures depend on the following:

a. the mechanical characteristics of the pulmonary-thorax system

b. tidal volume

c. inspiratory flow.

IPAP (Inspiratory Positive Airway Pressure) is differentiated from **EPAP / PEEP** (Expiratory Positive Airway Pressure / Positive End-Expiratory Pressure) in the pressure curve. 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:

- holds collapse-prone alveoli open and thereby increases the area available for gas exchange
- and reduces the extent of intrapulmonary shunt
- in COPD patients: Reduction of intrinsic PEEP and thus reduced triggering effort
- Effects on hemodynamics: Reduction of 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.

Ventilation Frequency

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

> *Pathologically high respiratory frequency combined with low tidal volume (rapid shallow breathing) is the leading syndrome of impending respiratory failure.*

The **respiratory ratio** is the relationship between the time for inspiration and for 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 flow. For healthly lungs the I:E ratio usually chosen is 1:2 (corresponds to Ti/T of 33 %) to 1:1. In the case of obstructive pulmonary diseases, which can lead to hyperinflation, a prolonged expiratory time should be selected.

Figure. 1
prsima VENT50

Setting I:E (Ti/T)

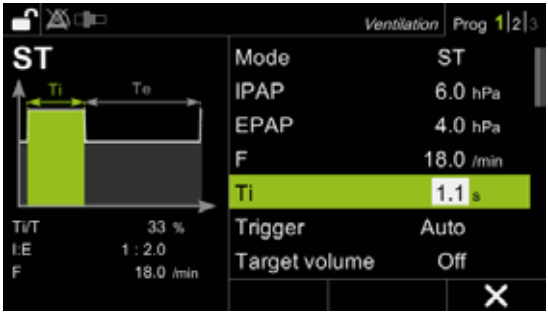


Figure 2
Operating panel on ventilator prisma VENT 30/40/50: Setting I:E (Ti/T)

Pressure or volume-controlled?

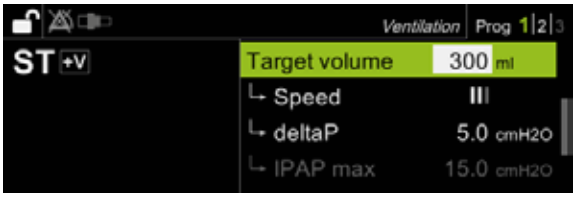
Many studies have shown that volume and pressure controlled ventilation technologies (bellows or blower) achieve comparable therapeutic effectiveness with regard to blood gases, breathing pattern and nocturnal oxygen saturation.[21, 22, 23]. Ventilators based on a pressure-controlled blower, however, offer the beneficial feature of leakage compensation. They also should be capable of regulating to a target volume.
A function called **"volume compensation"** [24] can be set for this purpose. It ensures that the patient is always sufficiently ventilated.

Caution is advised when non-constant leakage is observed in sleeping patients with activated volume compensation. In this case it could look as though the target volume had been reached and the pressure might then be incorrectly decreased. The patient is then under-ventilated. A solution is offered by the dual-pressure variation (IPAP - EPAP), which guarantees basic ventilation despite a non-constant leakage.

Modern ventilators like prismaVENT 30, 40 or 50, which is equipped with intelligent automatically regulating algorithms, can differentiate between leakage and volume compensation. Therefore, the false interpretation described above is successfully prevented and patient ventilation appropriate to the situation is guaranteed.

A critical factor in assessing the suficiency of mechanical ventilation under volume compensation is the $PaCO_2$ curve or, better yet, the level of bicarbonate. These values should sink after use of volume compensation. A rise, on the other hand, indicates under-ventilation of the patient.

Volume compensation – Three different speeds can be set.



Volume compensation:

off
off

slow

medium

fast

Figure 3
Volume compensation in prisma VENT 30/40/50. The speed can be adjusted at three different speeds to satisfy the patient's needs.

Managing the Respiratory Cycle

The change-over from the inspiration phase to the expiration phase contributes decisively to the quality of ventilation treatment. Ventilation technology uses pressure, flow, volume and time-controlled mechanisms.

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. It is therefore important to be able to adjust the trigger sensitivity to the needs of the patient. That achieves a high degree of unloading the respiratory muscles and gives the patient plenty of personal freedom.

If trigger sensitivity is set too low, the patient is forced to expend too much respiratory effort and may then suffer from exhaustion.

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. 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.

Trigger Sensitivity

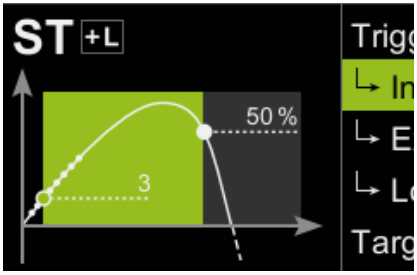


Figure 4
Trigger settings in Manual and Auto,
Trigger sensitivity adjusted for the patient improves ventilation efficiency.
Inspiratory settings are made in eight levels and expiratory settings from 95% to 5% (in 5% increments).
To prevent potentially false triggering in the expiration phase, Trigger lockout also can be set.

4.1 Ventilation Modes and Technical Solutions

The difference in the three basic modes of ventilation is the extent to which each ventilator takes over the Work Of Breathing.

- Controlled ventilation: the ventilator takes over all the work:
  T-, PCV-, VCV mode
- Controlled-assisted ventilation: the ventilator takes over 50 to 100% of the work, depending on the setting selected:
  ST mode, PSV incl. back-up frequency, aPCV, aVCV
- Spontaneous breathing: The patient receives pressure or volume support:
  CPAP, S and PSV mode without back-up frequency

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, which should be 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 split 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 a higher inspiratory (IPAP) pressure and a lower expiratory pressure (EPAP/PEEP) can be set.

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 can also be provided in **ST mode** (Spontaneous Timed). It combines inspiratory pressure support and 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 for a specific patient's needs can be configured to provide optimum support of the patient's spontaneous breathing efforts.

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, I:E ratio 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 back-up frequency.

ST Mode Setting

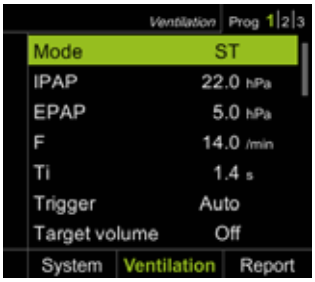


Figure 5
ST mode. Selected from the menu: IPAP of 22.0 hPa and EPAP of 5.0 hPa.

If ventilation pressures are too low to be effective,the patient may be at risk of suffering from dyspnea.

T Mode Setting

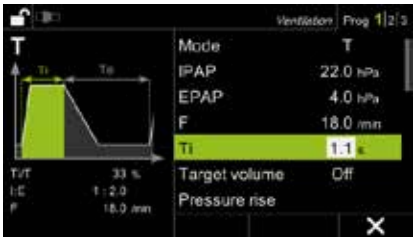


Figure 6
T mode (controlled ventilation) is set with inspiratory pressure of 22.0 hPa and EPAP of 4.0 hPa. The patient is ventilated with a respiratory frequency of 12 / min and Ti/T ratio of 33%.

Use of T Mode

T Mode or controlled ventilation provides maximum unloading of patient's exhausted respiratory pump.

4.1.2.4 autoST-Mode

With **autoST** (autoST=autoEPAP + autoF) the patient is given an intelligent backup which combines pressure adjustment (autoEPAP) 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 thus takes place between EPAP min and EPAP max with clear upper airways.

With the autoF setting as the basis, the ventilator prevents central apnea phases and desaturation by delivering mandatory breaths in the absence of spontaneous breaths. The volume provided 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 7
A patient-specific adjustment between EPAP min and max and a set pressure delta (ΔPinsp.) make possible an automatic pressure adjustment to eliminate obstructions.

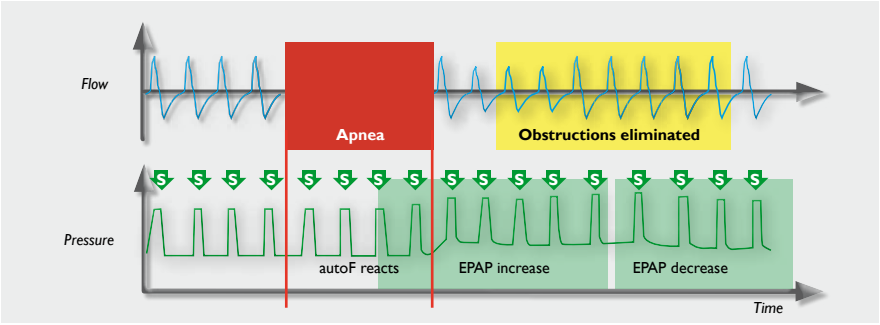


Figure 8
EPAP increases automatically to eliminate obstructions and adjusts continuously when upper airways are clear. Pressure support remains constant.

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 preset 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.

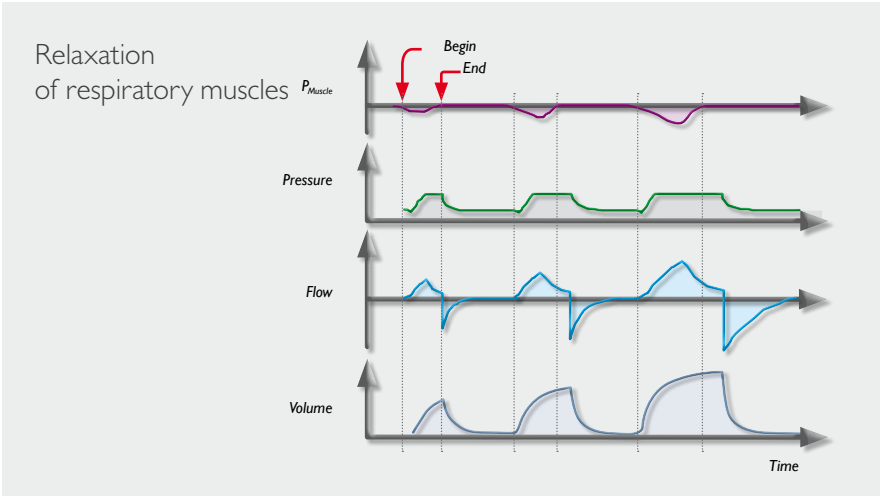


Figure 9
The patient's inspiratory effort initiates an increase of ventilation pressure to a defined level. The patient triggers mechanical ventilation, which reduces the Work Of Breathing.

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 that point, the device automatically switches to expiration. In contrast to BiLevel, spontaneous breathing is not allowed. Changes to lung compiance and resistance affect tidal volumes.

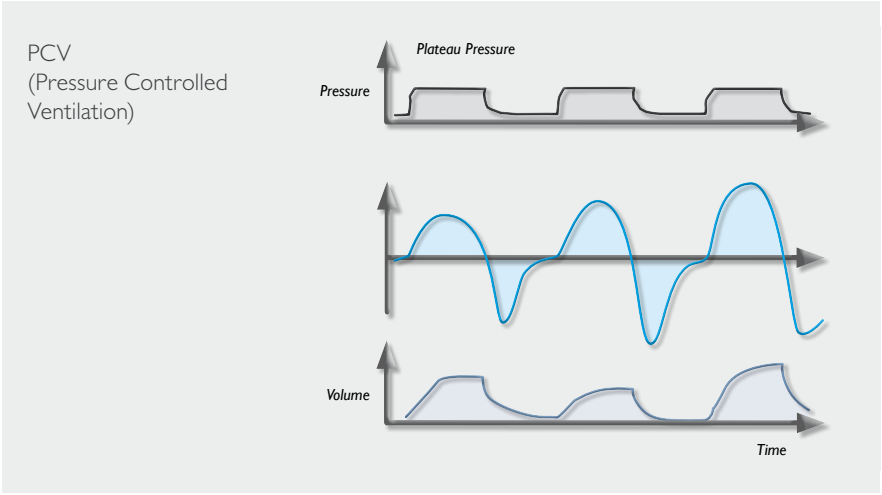


Figure 10
PCV – a ventilation mode often used in home mechanical ventilation

Pressure Increase

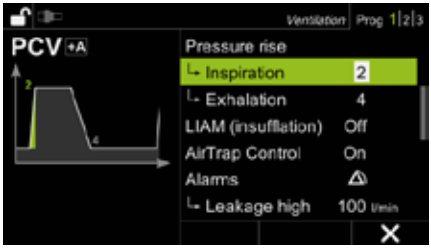


Figure 11
The pressure increase can be set for each patient in order to reach the selected pressure level in a reasonable amount of time. The settings for inspiration and expiration phases can be made from 1 (steep) to 4 (flat).

4.1.5 VCV

Under Volume-Controlled Ventilation (VCV) the patient receives a specified tital 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.

VCV
(Volume Controlled Ventilation)

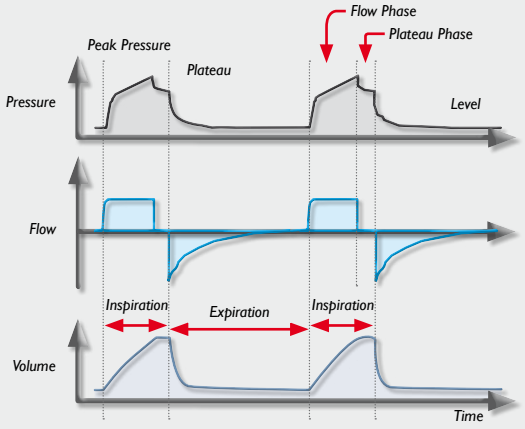


Figure 12
VCV – at high inspiratory flow the volume to be applied is administered before the inspiratory time has elapsed. This causes a pause known as the "plateau phase" to occur. The flow should be set to make the end-inspiratory phase very brief.

Use of VCV

Patients with neuromuscular disease are sometimes ventilated with VCV because the application of specified volume permits air stacking for secretion mobilization.

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.

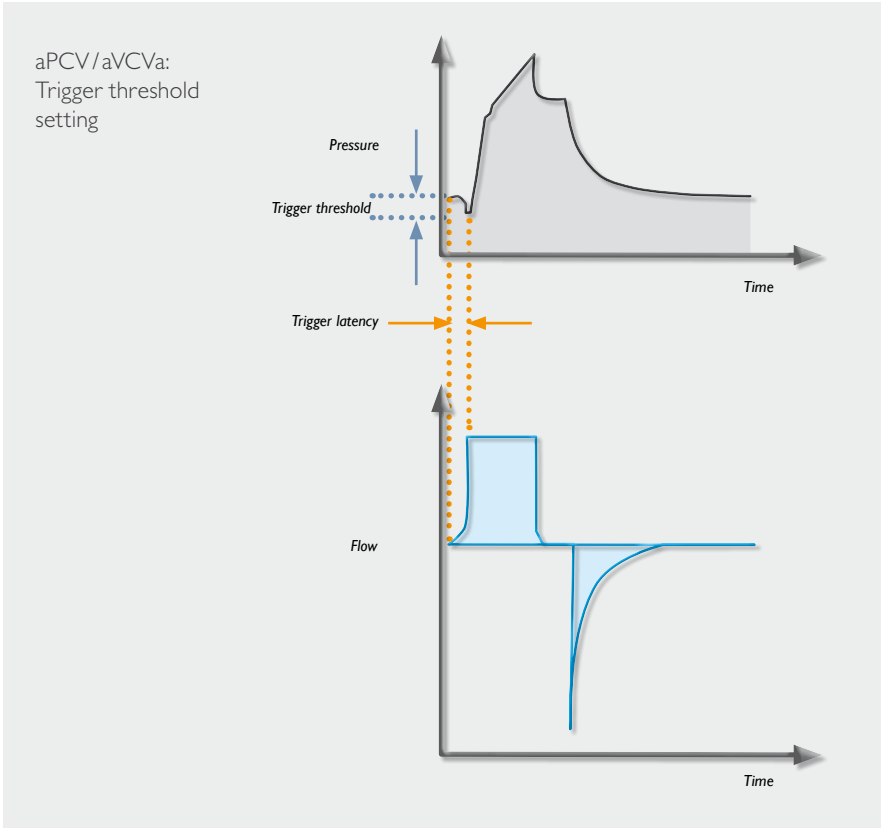


Figure 13
aPCV / aVCV The rigid frequency setting is moderated by assisted inspiration. The critical factor is the setting of the trigger threshold. If it is set too high, the patient cannot trigger inspiration. Then controlled ventilation is administered to the patient.

4.1.7 SIMV

The ventilation mode Synchronized Intermittent Mandatory Ventilation (SIMV) combines spontaneous breathing with volume or pressure controlled 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. If

Modes for Leakage and/or Valve Systems

Modes for Leakage System		
Acronym	**Name**	**Use**
CPAP	Continuous Positive Airway Pressure	OSA or well compensated OHS
S	Spontaneous	No CPAP compliance, high CPAP pressures
ST	Spontaneous Timed	ARI and CVI with lack of acceptance for T mode
autoST	automatic Spontaneous timed	during central apnea phases and/or danger of obstruction
T	Timed controlled	CVI
Modes for Leakage and Valve System		
PSV	Pressure Support Ventilation	Hypercapnic ARI, weaning
aPCV	assisted Pressure Controlled Ventilation	With low acceptance of PCV, weaning
PCV	Pressure Controlled Ventilation	Weaning, course of acute treatment, when patient condition has improved
aVCV	assisted Volume Controlled Ventilation	With low acceptance of VCV
VCV	Volume Controlled Ventilation	More likely for patients with neuromuscular disorders and thoracic restriction; no leakage compensation
MPVp	Mouthpiece Ventilation pressure controlled	For patients with neuromuscular disorders and thoracic restriction
MPVv	Mouthpiece Ventilation volume controlled	For patients with neuromuscular disorders and thoracic restriction
Mode for Valve System		
SIMV	Synchronized Intermittent Mandatory Ventilation	for invasive hospital ventilation

Table 5
Potential uses of mode combinations

the device does not detect spontaneous breathing activity during this window, an unsynchronized ventilator breath is delivered. A false device setting can interfere with spontaneous breathing. If severe respiratory failure is present, the spontaneously breathed tidal volume could be so low that alveolar hypoventilation can occur[25].

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, pressure-controlled (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 air or 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 14 shows a flexible arm affixed to a wheelchair which is holding a patient circuit and mouthpiece.

4.1.9 The TriLevel Principle

TriLevel ventilation is an optional feature which is used to treat patients who have high expiratory pressure needs or complex Sleep-Disordered Breathing. Conditions treated include Cheyne-Stokes Respiration and other central respiratory disorders, possibly combined with obstructions in the upper airways.

The name "TriLevel" reflects the use of three pressure levels, IPAP, EPAP and EEPAP. In BiLevel mode, the device provides IPAP during inspiration and EPAP during early phases of expiration. In TriLevel mode, EEPAP (End-Expiratory Positive Airway Pressure) is applied at the end of expiration when the upper airways are prone to collapse. The difference between IPAP and EEPAP represents the adaptive mechanical breath (PDIFF). The EEPAP functions like CPAP and prevents obstructions in the upper airways while providing additional support that makes exhaling more comfortable for the patient.

This therapeutic approach has proven effective for patients who require respiratory support and a pneumatic splint to keep their airways open and prevent obstructions. TriLevel is available as an option in prismaLINE devices. An anti-cyclical algorithm is used to treat Cheyne-Stokes Respiration, a type of periodic breathing accompanied by hyperventilation. That means that in phases of hyperventilation, the mandatory respiratory support is decreased to PDIFF =0 and is automatically increased in phases of hypoventilation.

How TriLevel Mode Works

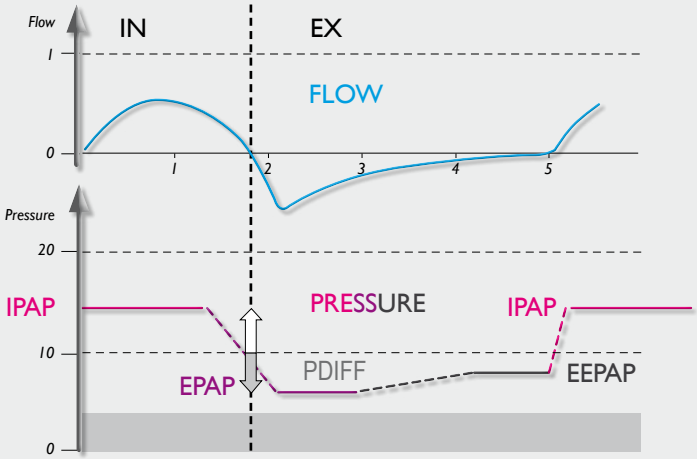


Figure 15
TriLevel-Modus in prismaCR – over the course of a breathing cycle, the three pressure levels are automatically adjusted to the current needs of the patient. The Anticyclical Modulated Ventilation (ACMV or ASV) stabilizes the patient's breathing. At the end of expiration – the phase in which airwas tend to collapse – the EEPAP (=autoCPAP function) detects impending events in time to prevent them.

Figure 16
prismaCR

Use of TriLevel

The optional TriLevel combines adaptive ventilator breaths (PDIFF) with the autoCPAP function and offers comfortable respiratory support to patients who need both support and a splint to keep open the upper airways.

4.1.10 AirTrap Control

In mechanical ventilation a Positive End-Expiratory Pressure (PEEP) is generated 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[26].

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.

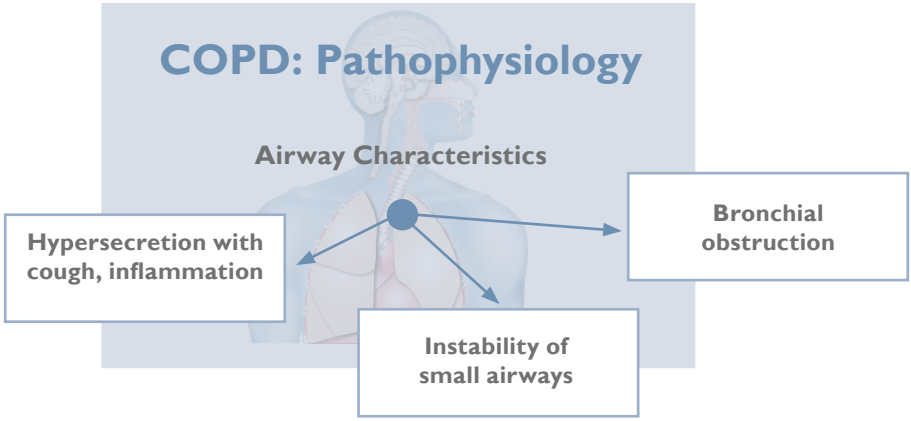


Figure 17
COPD pathophysiology

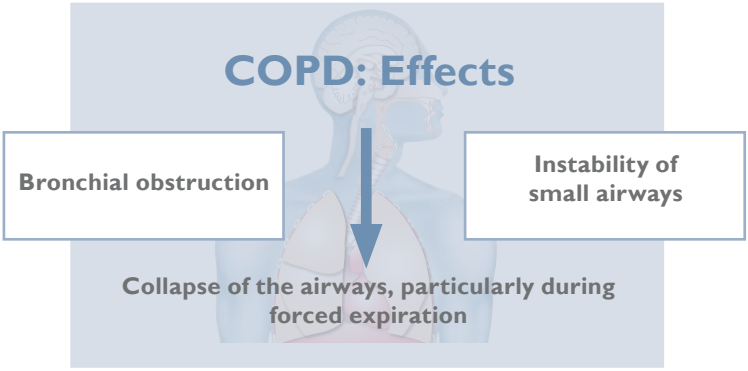


Figure 18
The airways of COPD patients tend to collapse during inspiration.

COPD patients are prone to dynamic hyperinflation

The Functional Residual Capacity (FRC) increases at the expense of vital capacity, the resting expiratory position shifts and intrinsic PEEP develops [27] [28]. Consequence: dynamic hyperinflation occurs [29]. Respiration shifts into higher regions of the airways which are less compliant. The respiratory muscles then operate in an unfavorable range of the length-tension curve.

Sufficient ventilation under these conditions can be achieved only with increased respiratory effort[30]. 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 $PaCO_2$ levels in the blood.

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[31]. Gas exchange is also restricted.

Intrinsic PEEP is an undesirable condition during mechanical ventilation. If the patient wants to trigger the device, he has to generate a positive intrathoracic presure before he 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 [32].

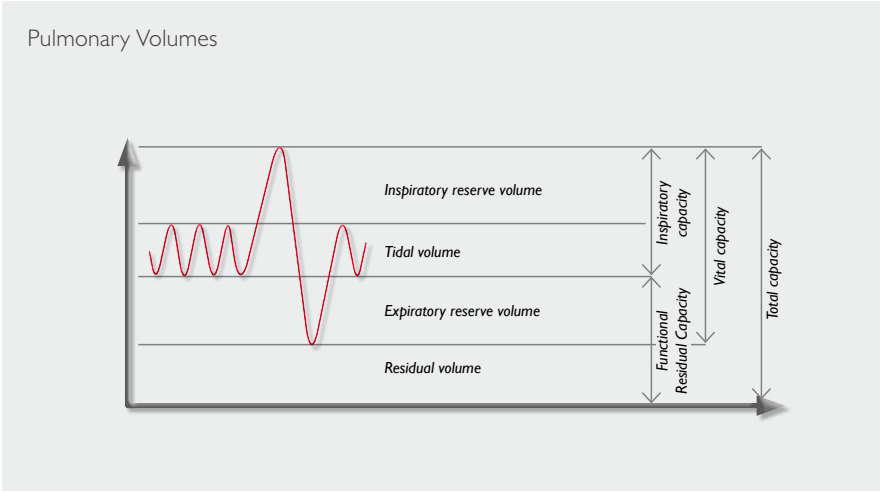


Figure 19
In COPD patients the functional residual volume increases as a result of intrinsic PEEP.

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 [33] plus high inspiratory pressures, administration of external PEEP or a reduction in Respiratory Rate (RR) or frequency.

AirTrap Control is a new approach which can effectively 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 (through the reduction of RR). This causes a decrease in intrinsic PEEP and shifts the resting respiratory position toward a normal range. The result is efficient ventilation and a possible reduction in the effective ventilation pressure.

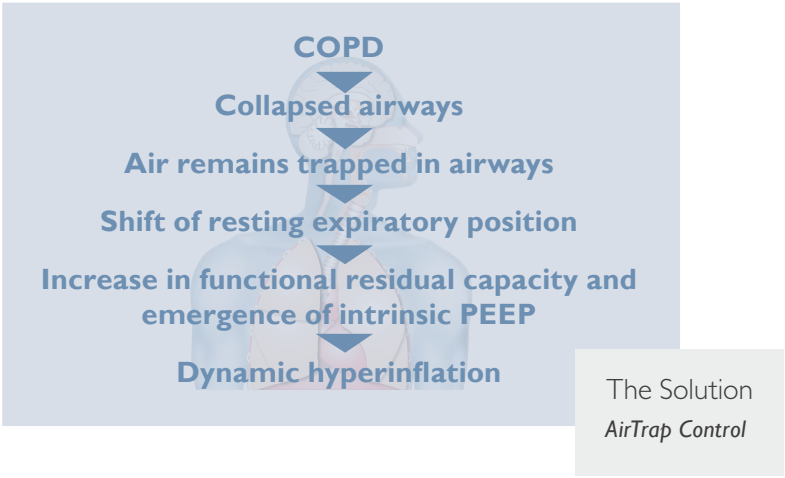


Figure 20
Use AirTrap Control when dynamic hyperinflation occurs.

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 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 limits correspond to a maximum prolongation of expiratory time of 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.

It makes sense to monitor key data in order to assess therapeutic effectiveness over the course of ventilation treatment.

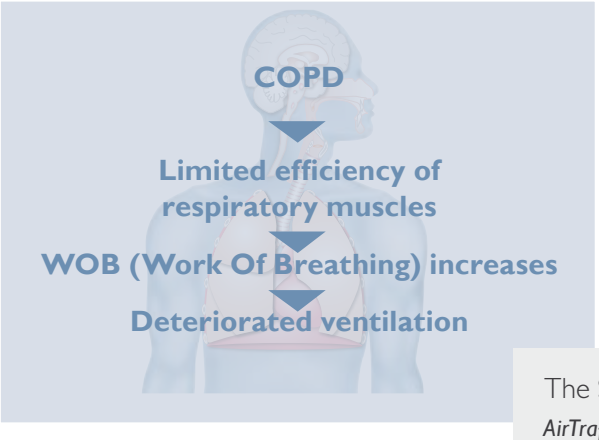


Figure 21
The effects of dynamic hyperinflation: Air trapping causes a shift in the resting expiratoy position; intrinsic PEEP develops; tidal volume is reduced. Despite elevated pressure, it is not possible to transport significantly more volume into the lungs.

Practical experience has shown that COPD patients with a significant obstructive fraction appear to benefit from AirTrap Control.

The first indication of effective ventilation with the help of AirTrap Control is a reduction in the patient's respiratory rate. The goal is to increase alveolar ventilation.

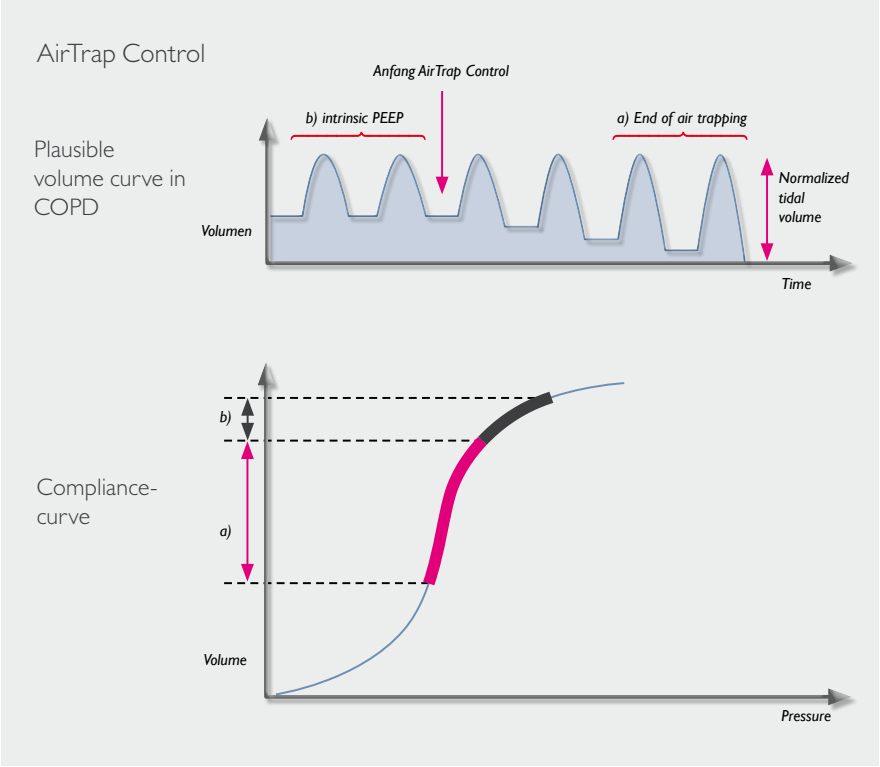


Figure 22
AirTrap Control causes intrinsic PEEP to decrease slowly

4.1.11 Trigger Lockout

The interaction of man and machine is a critical qualitative aspect in mechanical ventilation. Many different situations can influence ventilation quality and effectiveness[34]:

- Does the patient allow the machine to ventilate him or does he 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?
- Is faulty triggering of the device observed?

Dyssynchrony 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:

- leakage
- leakage compensation
- patient interface
- patient's underlying disease

Four major technical components also affect synchrony[35]:

- the triggering of the ventilator
- the phase of inspiratory curve after triggering
- the transition from inspiration to expirtion
- the end of expiration

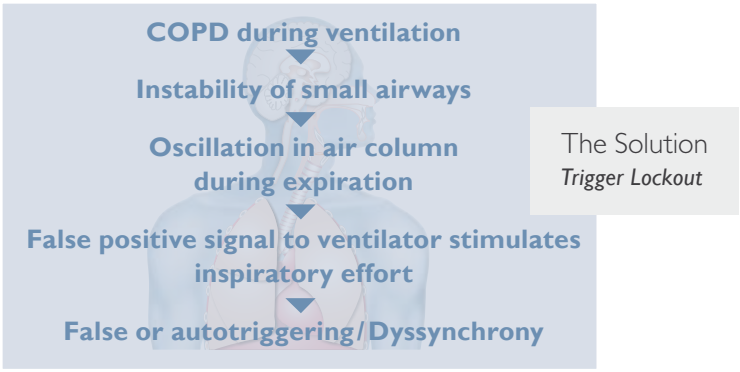


Figure 23
Autotriggerung – Because a COPD patient has an exhausted respiratory pump, he needs a sensitive trigger. Auto-triggering, a mismanagement of the ventilator, may occur with COPD patients, whose small airways are already unstable, as a result of oscillations in the air column during expiration. When a sensitive trigger is set, the device then receives a false positive signal of inspiratory effort.

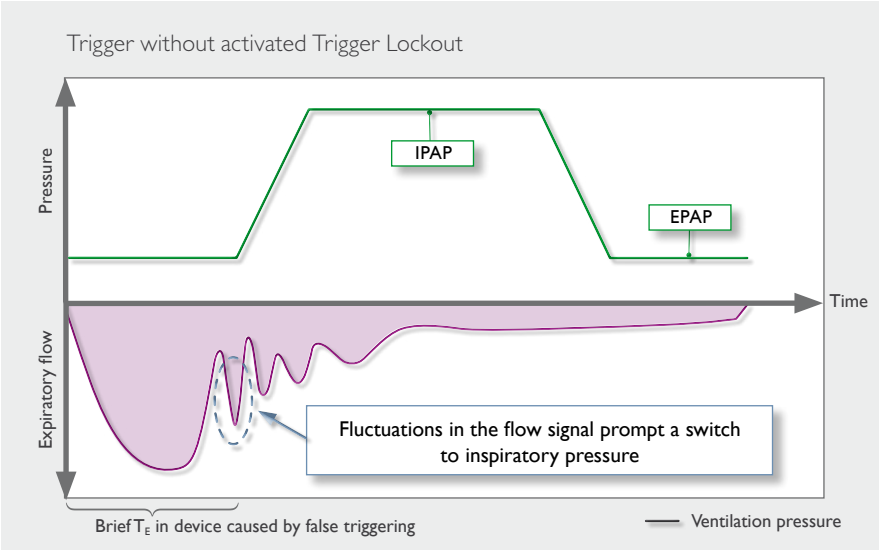


Figure 24
Assisted ventilation with false triggering caused by fluctuations in the flow curve with a sensitively set trigger without trigger lockout. The fluctuations are exaggerated in relation to real ratios. In practice, the expiratory flow is affected by the pressure increase, which is not shown for sake of simplicity.

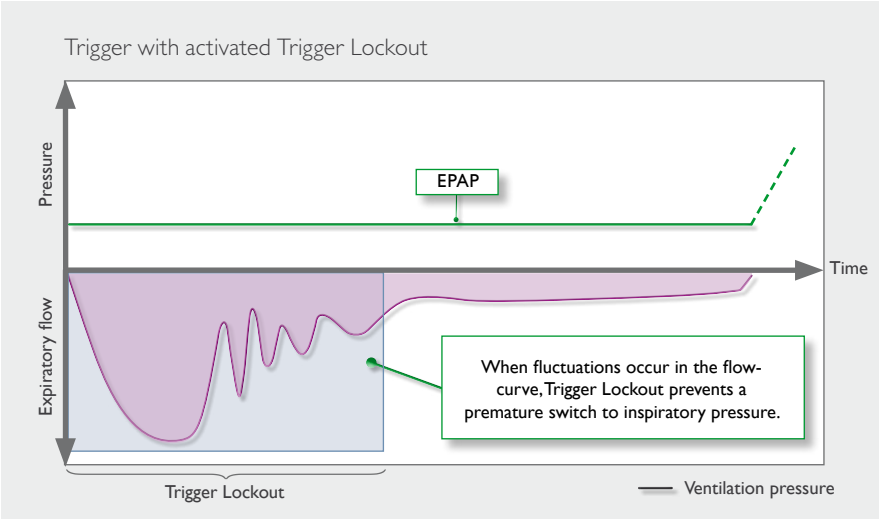


Figure 25
The setting of a suitable Trigger Lockout period prevents the ventilator from making a premature switch to inspiratory phase (see Figure 24).

The phenomenon of auto-triggering is often observed during the ventilation of COPD patients. It is suspected that oscillations occur in the air column during expiration as a result of the instability of the airways. When the trigger has a very sensitive setting, the oscillations can cause a false positive signal for the start of patient's inspiration. Oscillations which occur close to ventilation equipment (e.g., rubber lip on mask) are relevant in clinical routine.

The reduction in trigger sensitivity is the less favorable alternative for these patients because the change would increase their Work Of Breathing.

Trigger lockout, however, is an effective way to prevent faulty triggering.

At an equally high trigger sensitivity in ST mode, the device blocks the inspiration trigger for a defined period until the beginning of expiration. This greatly stabilizes the patient's spontaneous breathing pattern.

First the deisred I:E ratio is selected and then a lockout time for the inspiration is chosen in an physiological time window. To start with, a trigger lockout time of 1 s or one-third of the expiration time is recommended.

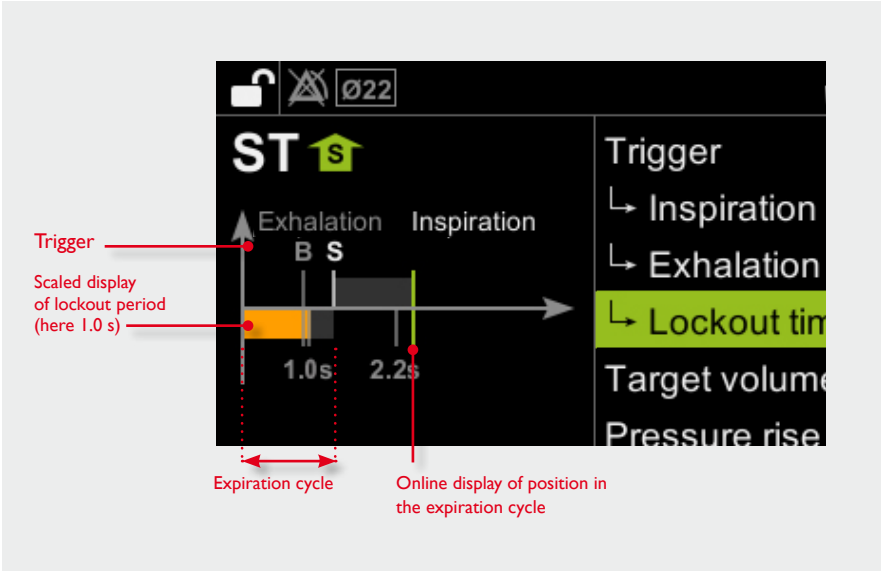
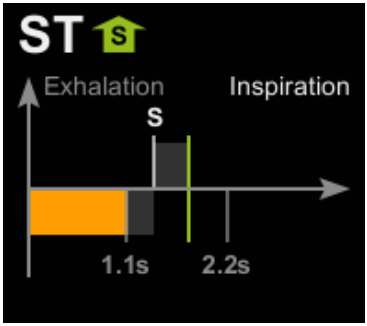
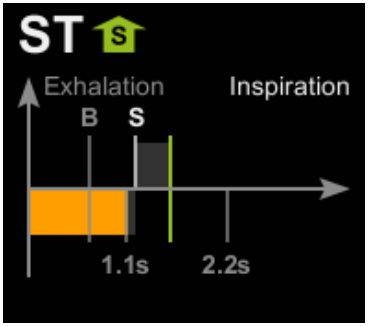


Figure 26
prisma VENT 30/40/50 operating menu:
Trigger Lockout can be set from 0.6 s to [(60/F) – Ti] (maximum 5 sec.).

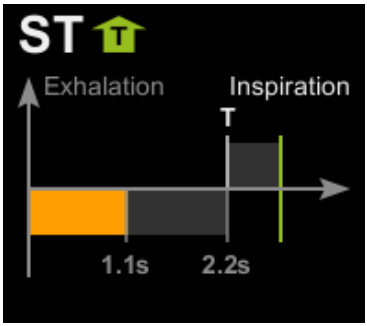
Trigger Lockout – Graphic depiction of function



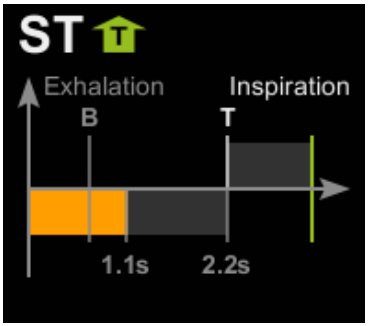
Variant A:
Spontaneous breathing



Variant B:
Spontaneous breathing with
blocked trigger impluse



Variant C:
Controlled ventilation



Variant D:
Controlled ventilation with
blocked trigger impluse

Figure 27
prisma VENT 30/40/50 – Trigger Lockout

4.1.12 Expiratory Pressure Ramp

An unrestrained expiration and a quick transition from high inspiratory ventilation pressure to expiratory pressure (PEEP) in a 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 28 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 Deutsche Atemwegsliga (German society of respiratory/pneumology experts) recommends the application of expiratory stenosis to bring about an increase in intrabronchial pressure.

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 (see flow curve in Figure 29).

The application of a slowly decreasing expiratory pressure ramp is of course possible without the ventilator's use of an increased extrinsic PEEP or EPAP. A pressure ramp is particularly effective because the coun-

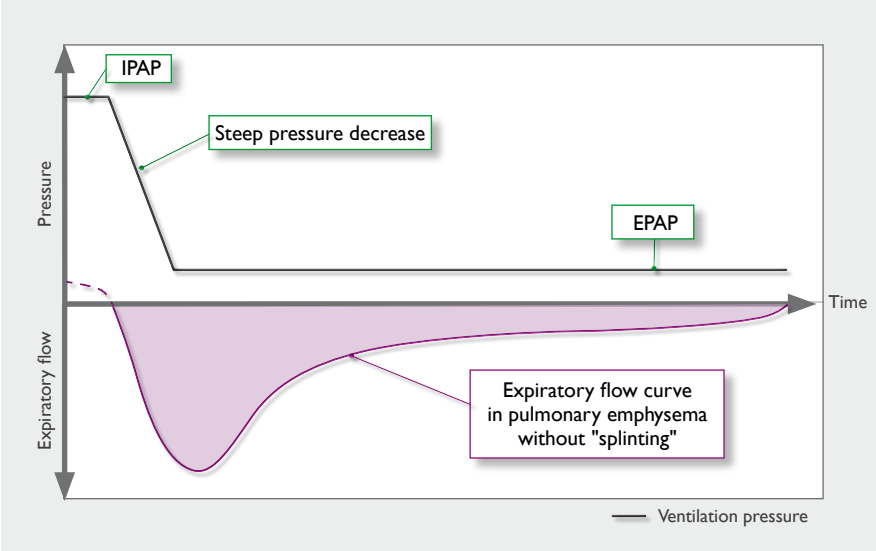


Figure 28
Expiratory flow curve during ventilation with steep pressure decrease and EPAP or PEEP ≠ 0

terpressure helps in the phase in which the flow's contribution is great and the local thoracic pressure is especially high due to 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 respiratory position can be lowered.

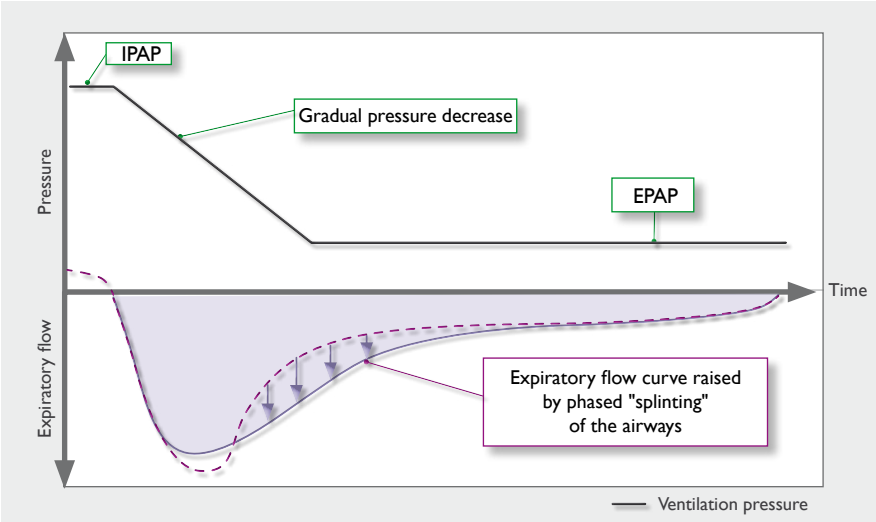
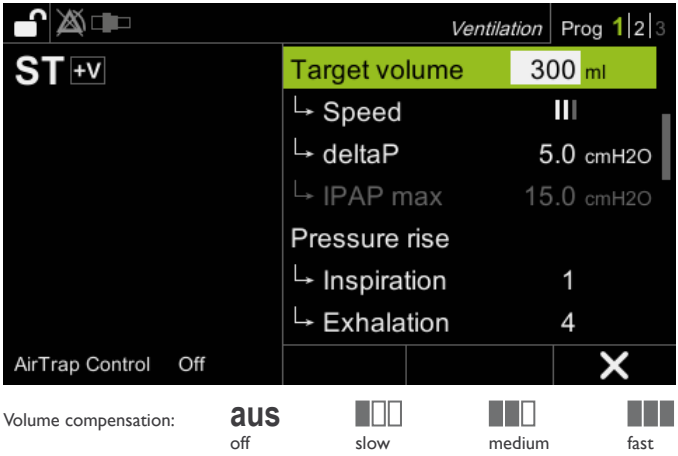


Figure 29
Effect of a flat transition between inspiratory pressure and expiratory pressure in the flow curve during expiration (dotted line: curve with steep pressure decrease; solid line: curve with flat pressure decrease). The flow remains larger on average and the expiration volume can be increased by the temporary splint.

4.1.13 Volume Compensation

Changing mechanical influences on the chest during ventilation (e.g., patient alters sleep position) can affect compliance in the lungs and chest. There is a way to compensate for lasting changes in mechanics as seen in exacerbation or in the course of a disease's progress. To that end, a certain volume must be guaranteed by pressure-controlled devices in order to provide the patient with sufficient ventilation continuously. Volume compensation should be used in narrowly defined situations. Caution is advised when treating patients with varying degrees of leakage (at night, for example). The ventilator may incorrectly reduce the pressure in this case. Pertinent information can be obtained by measuring $PaCO_2$ and bicarbonate. Moreover, a quickly changing breathing pattern (e.g., as in CSR) can overtax the target volume algorithm.

Volume Compensation – Three different speeds can be set



Slow: The device checks after eight breaths whether the target volume has been reached and changes the pressure by 0.5 hPa.

Medium: The device checks after five breaths whether the target volume has been reached and changes the pressure by 1.0 hPa.

Fast: The device checks after every breath whether the target volume has been reached and changes the pressure by1.5 hPa.

At any speed, when the pressure enters a corridor around the target volume, the device switches to exact regulation (+/- 0,1hPa / 100ml).

Figure 30
Volume compensation in prisma VENT30/40/50

4.2 Supplemental Oxygen

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

In adults a prolonged oxygen concentration of > 60 % is toxic to the lungs. Oxygen toxicity results from the production of superoxide anions and hydrogen peroxide due to hyperoxia.

Such high concentrations are not necessary for the ventilation of chronic cases in standard hospital operations.

A toxic effect from long-term administration 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[36, 37]. Therefore, the maximum allowable oxygen concentration should be very carefully observed.

The way in which oxygen is fed into the ventilator can have an effect on the quality of the ventilation. If oxygen is introduced only after the end of the inspiratory phase or to the mask, there is a danger of a faulty flow trigger and incorrect measurement of volume.

Figure 31
Rear view of prisma VENT30/40/50 – up to 15 l O_2 can be fed into the device through the O_2 supply port. The point at which the oxygen enters the system is selected so that the oxygen is registered in the flow measurement section. Then the flow trigger is not affected and the volume is correctly measured. The oxygen valve also functions as a safeguard. In the event of a device failure with ignition sparks, the valve automatically stops the oxygen feed to prevent the oxygen from entering the device's interior and starting a fire.

4.3 Humidification

With relative humidity of 100% at 37 °C, 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 point 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 o curs at a temperature of 37 °C and relative humidity of 100% (= absolute humidity of 44 mg/l).

Patients who are mechanically ventilated with invasive means 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[38]. If water vapor saturation falls below 70%, mucocilliary clearance is severely restricted. Below 50% relative humidity, ciliary activity ceases.

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 is also 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 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 based on vaporization employs a heating element to heat water (e.g., distilled water). Demineralized or boiled water are also considered sufficiently suitable[39]. The vaporization of the water yields a saturated water vapor atmosphere.

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 the prescribed cleaning procedures in order to rule out the risk of bacterial contamination.

Which patients benefit from the use of a humidifier[40]? 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 long-term non-invasively ventilated patients, however, require a humidifier.

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

Figure 32
prisma VENT40 with prismaAQUA – the humidifier prismaAQUA can be clicked easily into place on the ventilator and used with non-invasive ventilation.

4.4 Cough Management and Seccretion Mobilization with LIAM

Background

Physiology of Cough

The cough is the body's natural protective reflex to remove 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 generation.

Considering the inspiratory and expiratory flows during a cough, we see that mucous would not normally be transported out of the bronchial system, as the net effect on movement is equal to zero.

Pathophysiology of Cough

Varied pathophysiological processes lead to a restriction of and change in the cough fuction.

They include:

- **Narrowing of airway lumen**
  The 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 consistency 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 strength to generate the minimum flow of 160 to 300 liters/minute required for the normal cough function.

A disorder of the cough function leads to a variety of pathophysiological changes. The increased accumulation of secretion causes a narrowing of the airways' lumen and thereby makes ventilation difficult.

Atelectasis occurs with greater frequency. The collapse gives rise to pathological germ cells which reduce the area available for gas exchange.

The continued presence of secretions promotes increased bacterial colonization and leads to frequent viral and bacterial infection in the airways. Consequently, the patient is more susceptible to developing pneumonia.

A further consequence of secretion retention is the paralysis and destruction of the ciliary epithelium, without which 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.

When patients are unable to cough, they can be helped with manual techniques. A regular change of position, for example, promotes a more homogeneous ventilation and perfusion distribution.

Percussion, a medical manuever which involves tapping the patient on the thorax with a cupped hand, generates oscillations in the air column. In combination with the right patient position, this action prompts secretions to flow into the major airways. As a neuromuscular disease progresses, manual techniques do not suffice for effective removal of the accumulated secretion.

Under ventilation conditions too, secretion retention in neuromuscular patients should be prevented. Secretions can block the airways and jeopardize the success of mechanical ventilation[41]. 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[42] and prolongs survival[43].

Secretion Management + Cough Support

Secretion and cough management is particularly important for neuromuscular patients.

The previously mentioned **change of position** with its positive effect on homogeneous ventilation and perfusion distribution reduces the risk of infection and of poor mucocilliary clearance. This simple solution is often employed in conjunction with additional techniques.

In **secretion mobilization** a distinction is made between techniques **aimed at dissolving secretions** and those intended to **transport secretions** out of the airways.The former group includes percussion, vibration and oscillation. Secretions are mobilized either by manual tapping techniques or by means of IPV (Intrapulmonary Percussion Ventilation). These techniques exploit the physical principle that secretions are made thinner by mechanical means and then are more easily removed from the airways.

Another means of secretion mobilization is bronchoscopy. It has proven very helpful in acute situations and is possible with mask ventilation.

Secretions are transported by forced expiration. One of the oldest secretion mobilization techniques is position drainage. Gravity is utilized here to move the secretions out of the affected sections of the lungs. This method, however, is not efficient in cases of incipient respiratory failure.

Therapy is indicated from a PCF of < 270 l/min!

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

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 prior to an indication for ventilation.

Secretion management accomplishes the following:

- delays the initiation of ventilation[44]
- allows ventilation to be administered in the first place[41]
- eliminates the need for hospital stays [41, 42] and
- increases patients' life expectancy[43].

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 which integrates the secretion mobilization function in the ventilator.

Specifically, the unique cough support process LIAM (Lung Insufflation Assist Maneuver[45]) was integrated in the ventilators VENTI*logic* LS and prismaVENT 50.

The process is based on an inspiratory maneuver in which the lungs and thorax are "pre-tensed" by a deep inhalation.

How LIAM works

An inspiratory manuever is used during ventilation to overlay an additional defined mechanical breath (Δ P) on IPAP. The thoracic expansion thus generated causes an increase in Peak Cough Flow, or the maximum cough, and eases secretion elimination. The patient can then cough more effectively.

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
6. Select the number of LIAM-supported breaths

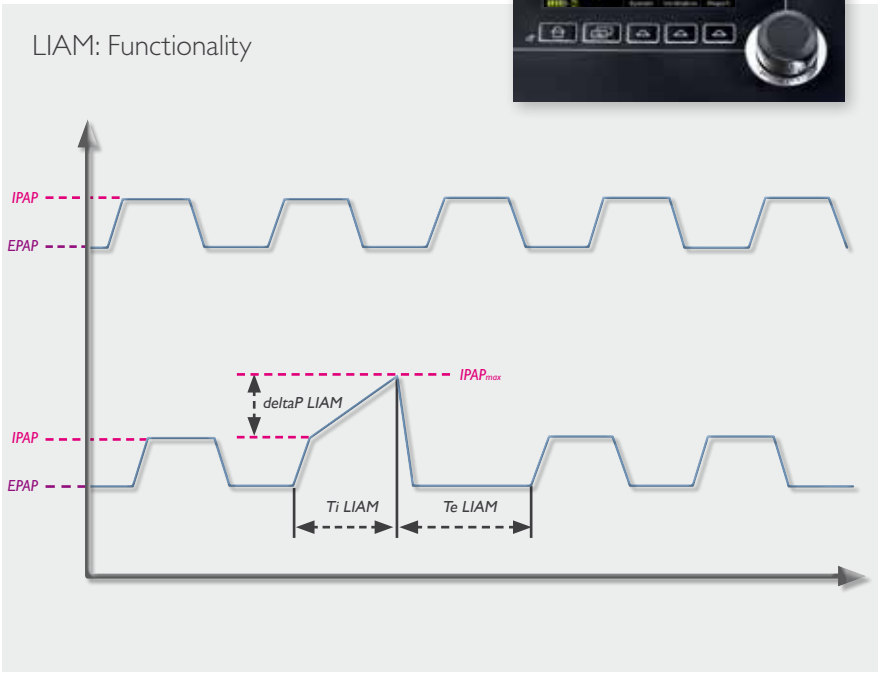


Figure 33
How LIAM works: The IPAP pressure generated by the device is overlaid with deltaP LIAM, resulting in an IPAPmax. LIAM increases Ti LIAM to the pre-set pressure level and changes Te LIAM back to EPAP at the start of the expiratory phase.

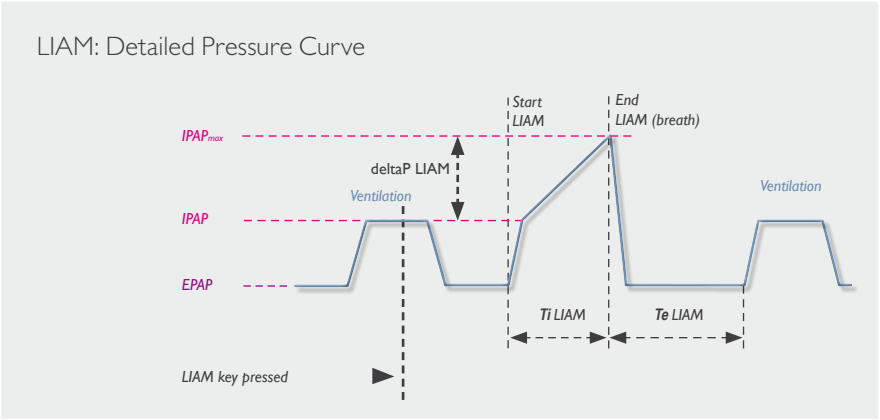


Figure 34
Pressure curve with use of LIAM

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 one minute to continuous application ∞. A setting also 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 sequential LIAM breaths to complete the maneuver successfully, up to 10 breaths can be administered.

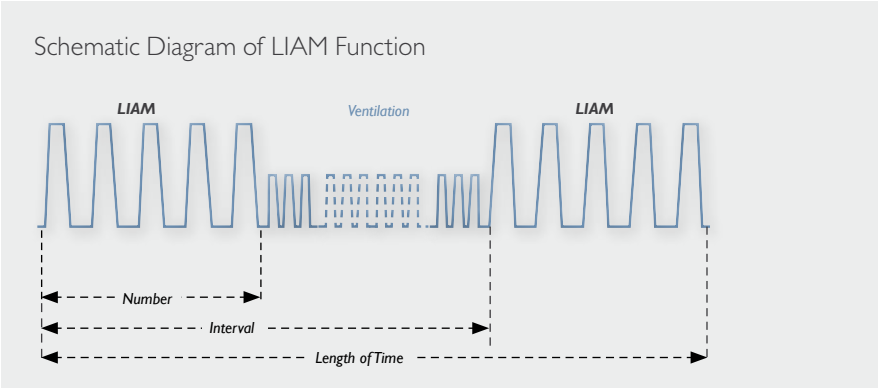


Figure 35
If LIAM is to be applied over a prolonged period, settings can be made to meet specific patient needs for the number of sequential breaths, the intervals at which they should be repeated and the duration of the application. This diagram shows two series of five sequential LIAM breaths. .

The process was developed in such a way to ensure that the set deltaP in volume compensation and deltaP LIAM have no additive effect when volume compensation 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. The differences between the LIAM and air-stacking functions are shown below in Table 6.

Comparison of Air Stacking und LIAM

Air Stacking with VCV	LIAM
⊖ possible only in VCV	⊕ can be used in all ventilation modes
⊖ breaths have to be stacked	⊕ stacking not required
⊖ the glottis has to be closed	⊕ deep insufflation is possible without glottis closure
⊕ high patient autonomy(with VCV)	⊕ limited autonomy, but caregiver can trigger function
—	⊕ adjusted expiration time

Table 6
Comparison of Air Stacking and LIAM

More benefits from LIAM:

- The patient does not have to turn off the device for secretion management, unlike with other stand-alone devices.
- The manuever takes less time than with other technologies and is thus more comfortable for the patient.
- Especially important for ALS patients is the fact that it is not necessary to close the glottis.
- The patient retains a certain degree of independence; LIAM can also 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 expiration supported by LIAM is possible.

The addition of the LIAM function is part of an integrated approach to ensure comfortable ventilation for patients with secretion retention.

LIAM is an important therapeutic expansion to ventilation functions.

4.5 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 the fit. A broad selection of standard masks from reusable types to disposable (Single-Patient-Use) offers every patient in hospital or at home the right mask. Consequently, personalized masks now play only a minor role.

Masks are available in many different sizes and shapes.

The basic differences are:

- Full face masks (sometimes with chin support)
- Nasal masks
- Nasal pillow masks and nasal prongs
- Mouthpieces
- CPAP helmet (for use in acute cases of hypoxia)

The following should be considered when fitting a mask to a patient ..

... that the right size is chosen (e.g., to accommodate an open mouth.).

... that no leakage or unequal contact pressure is detected

... that the patient or caregiver has been briefed in the handling of the mask.

It is no longer difficult to conduct an endoscopic examination while the patient is being ventilated. For that purpose, mask-specific elbows are offered which can replace conventional elbows.

Figure 36:
Endoscope adapter

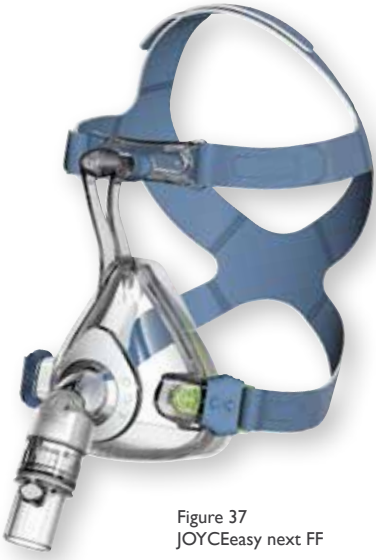


Figure 37
JOYCEeasy next FF

5 Practical Usage Tips

5.1 In Cases of Chronic Ventilatory Failure

Acclimating Chronic Stable Patients to Ventilation

The initial patient settings on the ventilator are made when the patient is awake. The choice of the initial pressure depends on the patient's underlying disease. As a rule, 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. In 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). In this case an automatic adjustment made by AirTrap Control can be helpful.

Some ventilation specialists have reported good experience with nighttime pressure regulation while the patient sleeps. If the patient cannot tolerate the high pressure regulation, he is sent home for four weeks to use a device set to his initial adaptive pressure with the objective of optimizing the pressure setting later. In cases of preexisting 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. If at the beginning the patient is allowed to trigger the device, there is a risk that he could be hyperventilated.

The choice of whether to use minimum volume or **volume compensation** 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 although the patient is underventilated. Intelligent algorithms in modern ventilators like prisma VENT50 can differentiate between leakage and volume compensation, which prevents such faulty interpretations. 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 breathing pattern. Patients with an underlying neuromuscular disease acclimate themselves relatively quickly to ventilation (two to three days); thoracic-restrictive patients generally need five to seven days, while COPD patients may require up to 14 days.

The acclimation phase, 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

The choice of mask is an important issue for chronic stable patients too. The process has already been described for cases of acute respiratory failure. In contrast to those patients, chronic stable patients are usually first fitted with a nasal mask. At high pressure > 20 hPa, however, full face masks are almost always used. They are particularly helpful in managing leakage, which often occurs.

A recent publication described the determining factors of a mask's required skin contact pressure. In order to prevent leakage during inspiration, the contact pressure ought to be about 1-1.5 hPa above inspiratory pressure, irrespective of the size of the mask cushion. Masks with a small mask cushion generate high contact pressure during ventilation[46].

Nighttime Monitoring

Today as part of the therapy settings process patents with chronic respiratory failure are diagnosed with the classic pulmonary function test and blood gas analysis and sometimes polygraphy. As far as hypercapnic insufficiency is concerned, there seems to be little difference in the effect of daytime vs. nighttime ventilation [47]. However, it has been suggested[48] 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 checked.

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 hypercapnic disorder is sleep-related hypoventilation during REM (Rapid Eye Movement) sleep. Furthermore, abnormal respiratory events frequently occur during sleep, so that ven-

tilation parameters set during the day are often therapeutically inadequate at night. Asychrony 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 also tax the cardiovascular system.

Nighttime monitoring also 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 in ST mode the patient is given controlled ventilation during the night and his respiratory pump is unloaded to the greatest extent possible[48].

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

It is particularly difficult to determine 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 also used to check whether phases of asynchrony, auto-triggering or 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 chronic stable patients with 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 means of monitoring ventilation patients at night. Supplemental video recordings are also recommended as they provide valuable information about events during the course of the night.

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

- first day: 15-20 minutes under doctor's supervision
- next several days: 4-5 hours adaption
- 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 also checked along with oximetry and/or PTc CO_2 levels and measurement of tidal volume[2]:

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

Patients may normally 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 in connection with the underlying disease.

After six weeks of ventilation at home, the patient is again examined. At that point it is often necessary to readjust the ventilation parameters. Thereafter, an annual examination of the parameters is recommended.

5.2 Pediatric Aspects of 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 non-invasive ventilation is being used with increasing frequency and success.

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

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 ventilation settings and then adjusted as needed [60].

The treating physician is advised to consider the psychological aspects of treatment and involve the child in all decisions related to his 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 how he 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.

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

Children's Acceptance of Ventilation

Ventilation of a child works 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.

With 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_2 rebreathing, the smallest possible amount of dead space should be selected.
- Special hoses 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

6 Product Solutions for Ventilation

6.1 Product Concept for Hardware shown with prisma VENT50

Figure 38
prisma VENT50, easy-to-use, high-level ventilation treatment

6.2 prisma VENT30 and prisma VENT40

Mobility and convenience at all levels

- Simplest use, thanks to intuitive menus, easy operation and quick access
- Whisper-quiet ventilation for greater comfort and well-being of patient and partner
- Suitable for invasive and non-invasive ventilation
- Includes helpful fuctions for COPD treatment: AirTrap Control, Expiratory Ramp and Trigger lockout
- With autoST mode (autoEPAP, autoF) and target volume*
- Integrated oxygen port

Figure 39
prsima VENT30 and prisma VENT40

6.3 prisma VENT50

Top-level ventilation therapy

prisma VENT50 has all the features found in prisma VENT30/40 plus the following:

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

Figure 40
prsima VENT50

* Target volume is available ony in prisma VENT40 and prisma VENT50.

6.4 VENTIlogic LS

For life supporting ventilation

- All circuit systems can be used: single and double patient circuits with patient valve and leakage circuit
- Pressure and volume-controlled ventilation modes
- Designed for mobility in hospital and at home
- Three ventilation programs can be set and saved

Figure 41
VENTIlogic LS

6.5 prismaTS and prismaTSlab

For fast and flexible
patient management

A quick overview of therapy or a detailed analysis.

The flexible and intuitively used software offers patient management dynamic report generation and device administration, including remote management.

Figure 42
prisma TS und prismaTSlab

6.6 Sleep Diagnostics with MiniScreen PRO

MiniScreen PRO gives you all the flexibility you need for services ranging from outpatient recordings to polysomnograpy in the sleep lab in compiance with AASM guidelines.

With the input from additional signals such as pressure, flow, leakage, tidal volume and CO_2, MiniScreen Pro also can provide respiratory monitoring and ventilation for treatment of Sleep-Disordered Breathing and other respiratory disorders.

Optional wireless data transmission makes diagnostics even more convenient.

Simple operation, stability and low operational and subsequent costs make MiniScreen PRO a reliable and economic partner for sleep diagnostic examinations.

Figure 43
MiniScreen PRO

7 Outlook

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

The prevalence of classic indications 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 are also being put to use for patients with periodic breathing (Cheyne-Stokes Respiration) and clinical symptoms after all drug therapies and alternative ventilation treatment methods such as anti-cyclical servoventilation have been exhausted.

The varied respiratory and ventilatory disorders are marked by different pathophysiological characteristics. It seems reasonable to respond with targeted therapeutic means. The functions AirTrap Control, Trigger Lockout and LIAM – to name just a few – make a useful contribution. Many of these innovative technologies involve biofeedback systems that continuously adapt to the needs of patients with respiratory or ventilatory failure with the goal of optimizing medical care.

Patient interface is particularly important for the compliance of the patient with his therapy. In recent years the quality of standard masks has clearly improved to the point that they can replace specially produced masks.

Most patients with chronic respiratory failure are ventilated overnight. It is therefore not surprising that during the setting phase, polygraphy or polysomnography is becoming increasingly important.

8 Glossary

aPCV / aVCV	assisted PCV / VCV
ARI	Akute Eespiratorische Insufficiency
COPD	Chronic Obstructive Pulmonary Disease
CPAP	Continuous Positive Airway Pressure
CRI	Chronic Respiratory Insufficiency
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 tTmed
T	Timed
VCV	Volume Controlled Ventilation
WOB	Work Of Breathing

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