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Weaning

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Summary

When the patient is able to leave the ventilator safely, the ventilator support is reduced, and the respiratory work is returned to the patient. Failure is defined as the fact that the minute ventilation required during mechanical ventilation is not achieved by the patient while in spontaneous breathing. The balance between ventilatory requirement and ventilatory capacity determines the weaning process. Patients who underwent mechanical ventilation for less than 24-48 hours constitute 2-4% of the total mechanical ventilation time, and in these patients, the weaning process can be completed in less than 1 hour. Patients undergoing mechanical ventilation for more than 7 days, 60-70% of the total mechanical ventilation time and weaning in these patients can be completed within days or even weeks [1].

To prolong mechanical ventilation unnecessarily [1,2]

1. Lung damage due to ventilator
2. Nosocomial pneumonia
3. Unnecessary sedation
4. Damages due to endotracheal tube

Early cessation of mechanical ventilation and early extubation [2]

1. Failure of the respiratory muscles
2. Gas exchange deterioration
3. Failure to maintain airway clearance

Weaning process

Patient's ventilatory support and airway needs should be evaluated frequently. Factors that can cause ventilatory support dependence should be constantly reviewed. Ventilatory support strategies should minimize the workload of the respiratory muscle while ensuring the patient's maximal comfort. Cases requiring long-term mechanical ventilation should be taken to specialized units in this regard [2].

Conditions that increase the ventilatory requirement

- Fire
- Infection
- Increased breathing effort
- Metabolic acidosis
- Overfeeding with carbohydrates
- Liver diseases
- Other causes that increase the metabolic rate

Causes that reduce ventilatory capacity [3]

- Weakness of the respiratory muscles
- Hypokalemia
- Hypophosphatemia
- Decreased breathing effort
- Bronchospasm
- Secretion accumulation
- Low compliance
- Neuromuscular weakness
- Malnutrition
- Small endotracheal tube
- Resistance increase

In the first stage of weaning, spontaneous breathing effort is initiated.

Spontaneous non-assisted pale includes two separate requirements

1. Ability to initiate rhythmic inspiratory effort (brain and nervous system)
2. Strength and endurance of the muscles that will provide tidal ventilation

Spontaneous breathing exists but acceptable minute ventilation does not occur

a. Insufficient tidal volume

b. Inability to trigger

c. Patient-ventilator dissynchrony

There may be no spontaneous breathing rhythm (primary post-ventilation apnea)

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Primary post-ventilation apnea[4]

In patients without central or peripheral nervous system disease, the most common cause of apnea developing after mechanical ventilation (unless neuromuscular blocker is applied) is central nervous system depression.

The cause of this depression may be sedatives, opioids, or metabolic alkalosis. While normal spontaneous breathing rhythm occurs in mechanical ventilation for less than 72 hours, the infusion of sedative and opioid agents occurs within 30-60 minutes after discontinuation, especially under certain conditions, this period may last for days.

1. Biological advanced age
2. Liver and kidney failure
3. Long-term infusion of benzodiazepines opioids
4. Poor neurological reserve (dementia, encephalitis)
5. Hypothermia
6. Electrolyte abnormalities (especially sodium)

Inadequate tidal volume formation in assist modes [5]

Most cases tolerate the transition to mandatory non-compensable triggered modes, such as pressure support, by reducing mandatory breaths after rotating respiratory effort. The reason for not being able to tolerate the mode may be low respiratory system compliance and patient's inconvenience. These cases need high inflation pressure to reach the desired tidal volume. Pulmonary receptors stretched with high inflation pressures and peak currents reduce neuronal inspiratory time and cough, thereby reducing inspiratory time and volume. Increased respiratory effort due to insufficient tidal volume causes an increase in expiratory muscle activity, further reducing the compliance of the respiratory system. In this case, decreasing the inflation pressure or increasing the ramp time and decreasing the peak inspiratory flow may stabilize the patient in pressure support ventilation. In cases where insufficient tidal volume occurs, assist-controlled modes are used, which are gradually reduced by mandatory tidal ventilation and switched to assist modes.

Secondary apnea [6]

While the central center is awake, the tidal volume is controlled while the sleeper is controlled. If the incoming inputs are ineffective due to sedation and central nervous system dysfunction, apnea occurs. Assist modes with back-up function switch to controlled or assist-controlled modes in the event of apnea, protecting them from severe hypoxia (apnea ventilation). Recurrent apneas suppress the patient's spontaneous efforts, as this will cause the patient to remain in more apnea ventilation.

Maneuvers that reduce secondary apnea [7]

Reducing system gain by reducing inflation pressure. Shortening the central nervous system response time by avoiding nighttime use of drugs that depress respiration and using nighttime respiratory stimulants. Shortening the circulation time with inno-dilators (milrinon). The second stage of weaning is the transfer of respiratory work from the ventilator to the patient after normal ventilatory rhythm is achieved. In patients who have undergone mechanical ventilation for less than 3-4 days, this process can be completed within minutes, since no significant respiratory muscle dysfunction has yet developed. Factors that increase respiratory work and whether the patient can tolerate this work should be examined in patients with long-term mechanical ventilation.

Determinants of the pale work [8]

- Elastic properties of the respiratory system (elastance)
- Resistance to inspiratory gas flow
- Lungs and inertia of the rib cage
- Friction due to tissue deformation
- Tidal volume
- Respiratory rate

Increased Elastance (pressure against unit volume) [9]

- As the lung volume approaches functional residual capacity (expiratory)
- As the stiffness of the chest wall increases
- As the end-expiratory lung volume increases
- As the surfactant activity decreases
- The mass and elasticity of the chest wall and abdomen and dysfunction of the chest and abdominal muscles due to pain and coordination during inspiration also affect elastane.
- Pulmonary fibrosis, which is difficult to correct, and treatable alveolar consolidation also affect elastane

Pulmonary edema [10]

It can occur due to 3 factors:

- Increase in capillary hydrostatic pressure
  - Liquid loading
  - Diastolic heart failure
  - Mitral valve dysfunction
  - Hypertension
- Decrease in capillary reflectance coefficient (sepsis pancreatitis, transfusion, pulmonary inflammation)
- Decrease in plasma oncotic pressure

Pulmonary edema increases pulmonary elastance, regardless of its etiology. Conservative fluid therapy and negative fluid balance reduce mechanical ventilation time. If pulmonary vascular congestion is thought to increase pulmonary elastance, cardiac function should be corrected, and diuretics should be used to reduce extravascular fluid volume. β2 agonists (salbutamol) reduce extravascular fluid by up-regulating epithelial basal sodium pump [11].

Pain

Reflex contraction of the expiratory muscles during inspiratory increases the elastance of the respiratory system due to pain. Carbon dioxide production increases due to the activated sympathetic system. Raising the head significantly improves pulmonary mechanics (elastance decreases) in cases of abdominal distention and cases with abdominal weight. Alveolar collapse, which reduces respiratory compliance (increases elastance), can be corrected by recruitment

maneuvers. Awake and mildly sedated weaning cases cannot tolerate recruitment maneuvers.

Therefore, alveolar collapse should be protected:

- An appropriate PEEP level should be used
- The clearance of secretions should be ensured
- The correct posture should be given, and abdominal distension should be treated [12]

Inertia

The power required to overcome inertia is proportional to the weight of the lungs. Normally, while each lung is 150 grams, 1000 grams exceeds in ARDS cases. High breath rates and high inspiratory flow are required to ensure the movement and acceleration of the lungs with increased weight. Assuming that the lung weight is increased 5 times and the breath rate, and the peak airway flow increase 2 times, the use of force for inaction increases 20 times (5 x 2 x2): this power requirement significantly affects weaning.

Respiratory rate and tidal volume

Alveolar ventilation = frequency x (tidal volume - dead space volume)

Increased dead space increases respiratory work as it does not participate in gas exchange. Since the tidal volume and the breathing frequency are inversely related to a fixed minute volume (V = Vt X fr), the breathing work associated with resistance increases while the breathing work associated with resistance increases while the breathing frequency increases (inspiratory time decreases). Total breathing work, which is the sum of resistive and elastic breathing jobs, is normally at a minimum of 12-15 breaths per minute for adults. Increased resistance requires the respiratory frequency to be lowered in terms of respiratory work. Elastance increase requires increasing respiratory frequency in terms of respiratory work [13].

Increasing metabolic demand increases the need for ventilation [1,2]

- Over nutrition
- Systemic inflammation
- Hyperthyroidism
- Hyperpyrexia
- catecholamines
- Some medicines (theophylline)
- Seizure, mania, hyperactive delirium
- Respiratory section (RQ = VCO2 / VO2): It refers to the mole of carbon dioxide formed per mole of oxygen used. In carbohydrate metabolism, 1 mole of carbon dioxide is formed in each mole of oxygen use.

  RQ = 1 for carbohydrate metabolism

  RQ = 0.6 for oils
- Providing nutrition with a high carbohydrate ratio increases the need for ventilation due to more carbon dioxide production
- Agitation and anxiety
- In intensive care patients, 83% of agitation and delirium develop after sedation ceases.

Sleep disturbance occurs in intensive care [1]

- Disturbance of normal sleep-wake rhythm
- Loss of circadian rhythm of melatonin production
- Decreased REM sleep
- Stressful intensive care environment
- Sedative agents applied

Complications of agitation and delirium

- Prolonged weaning
- Need more sedatives
- Self extubation
- Bad cooperation
- Increased energy use

Risk factors for delirium [1-3]

- Old age
- Cognitive state
- Severity of the disease
- Sedative and opioid application
- Drug discontinuation (alcohol, smoking, sedative, analgesic)
- Dehydration
- Hearing impairment
- Sepsis
- Pain
- Hypoxia
- Liver and kidney failure
- Poor glycemia control
- Sleep disorders

The patient with respiratory failure has pressure and volume load on the respiratory muscles

Volume load can be estimated [1]

From the minute ventilation requirement of the patient

From the rate of ventilation lost due to dead space

$Vd / Vt = (PaCO_2 - PECO_2) / PaCO_2$

From ventilation required to meet oxygen use and carbon dioxide production (metabolic demand)

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Muscle function [1,2,12]

Causes such as electrolyte retort, endocrine disorders, drugs, etc. may affect Novartis during mechanical ventilation. Two major events that affect the respiratory muscles that cause the need for mechanical ventilation may occur during the critical illness process:

1. Ventilator-induced diaphragmatic dysfunction (VIDD)
2. Critical illness myopathy (CIM))

To avoid VIDD, trigger assist modes should be used rather than controlled modes. Other than VIDD, systemic inflammation (sepsis, pancreatitis) causes a marked reduction in diaphragm strength. Hyperoxia is another cause of diaphragm dysfunction. Inhibition of hyperoxia and VIDD-induced diaphragm dysfunction by free radical purifiers suggests oxidative damage as a mechanism of damage. Acute necrotizing myopathy, an exaggerated form of myopathy in which the concentration of creatine kinase is elevated, may be observed in patients taking high doses of corticosteroids.

Myopathies have been described under the term CIM. Clinical distinction between variants of CIM is not possible.

- They all have loose novelties.
- Deep tendon reflexes may be normal or decreased
- There is usually no pain.
- Plasma creatine kinase may be normal

Needle electromyography cannot distinguish CIP-CIM in unconscious or sedated patients; novelization is required. CIM is more common than CIP.

- Novelization
- Brain-vault novelization of muscle function due to some problems
- The problem may be related to novelization (intrinsic fatigue)
- The problem may be related to neuromuscular junction (neuromuscular transmission fatigue)

Neural fatigue [3,4,13]

a. Spinal fatigue: the number of motor units activated, and the rate of motor neuron firing is low

b. Central fatigue: function of complex emotional factors and central respiratory effort is insufficient

Intrinsic fatigue is divided into low and high frequency on the basis of the warning frequency that causes contractile insufficiency. High-frequency fatigue occurs quickly and returns quickly. Deficiency in t-tubule depolarization is due to decrease in calcium release from sarcoplasmic reticulum and inability in excitation-contraction mating. Low-frequency fatigue develops slowly and returns slowly. Intracellular lactic acidosis and increased inorganic phosphate concentration are linked to inhibition of actin-myosin interaction. Can be reduced by prior administration of N-acetylcysteine.

Strength is determined by the warning frequency. The continuation of the contraction is inversely proportional to the warning frequency. Spinal fatigue is the reflex reduction of the action potential frequency generated by the anterior neck cells of the spinal cord to prevent complete contraction failure. Power plant capacity is involuntarily reduced engine effort. A significant portion of the power loss of the

diaphragm under long-term inspiratory load is caused by central mechanisms. Central and spinal fatigue protect the cardiovascular system from the development of intrinsic fatigue in the muscle in cases of respiratory load exceeding its ability to provide a casing substrate. For example: in stable congestive heart failure with little exercise tolerance, intrinsic diaphragmatic fatigue does not develop despite functional limitation

Improvement of respiratory novelization [2-4]

- Non-exercise approaches that improve the physical performance of the muscle:
- Increasing muscle mass (anabolic steroids, human chorionic gonadotropin and luteinizing hormone for men, insulin, insulin like growth factor Nov.)
- Increasing contractility (methylxanthines, amphetamines, beta adrenergic agonists)
- Increasing oxygen delivery (blood transfusion))
- Physiological approaches (reducing stress by music and hypnotism, motivation)
- The level of testosterone decreases in patients undergoing long-term mechanical ventilation. Mortality was found to be increased in intensive care patients undergoing androgenic and growth hormone therapy, especially due to impaired glycemic control.
- Methylxanthines and especially aminophylline are bronchodilators, as well as increase diaphragm contractility

Maneuvers to evaluate ventilatory novelization [1]

PImax: the patient's maximum inspiratory pressure against the closed inspiratory valve is the negative pressure measured during inspiratory exertion. It should be at least-20-30 cm H_2O to separate it from the ventilator

Another maneuver is a simple vital capacity maneuver. Vital capacity should not be below 1 litre

Maneuver P0, 1: 100 of inspiratory effort against closed circuit. it is inspiratory pressure that occurs in milliseconds. This measurement reflects 2 features:

- inspiratory effort

- ventilatory novelization

Pressure-triggered assist mode in PSV mode; end-of-expiration occlusion maneuver applied by ventilator for measurement of P0. 1

The vertical shaded area shows the first 100 ms of inspiratory exertion in the occlusion

The horizontal shaded area also shows the corresponding pressure drop

The initial moment of occlusion is determined on the V'aw curve, where it cuts the V'aw bascule.

Reading 1 cmH_2O for Paw first at this point

Second Paw value should be read After 100 ms

In this example -2 cmH_2O stop.

P0. 1 is defined as the difference between the first and second paw values, where 3 cmH_2O stop

Low P0, 1 may reflect two states:

- Novelization of muscle
- Low respiratory effort

Low P0, 1 can be interpreted positively if it reflects good patient comfort. However, if it reflects depressed breathing effort, it is interpreted negatively.

High P0, 1 may reflect two states: strong novelties and strong inspiratory effort

High P0, 1 is interpreted as positive if it reflects robust patient effort

Evaluation of load-capacity balance [1,2]

Crop index (Compliance, rate, oxygenation, pressure)):

CROP = [dynamic complement x PImax x (PaO_2/PAO_2)] / frequency

PImax; maximum inspiratory pressure

If CROP > 13, separation from the ventilator is likely to succeed

Evaluation of load-capacity balance [2,11]

Pressure - time multiplication (PTP): this parameter relates only to the applied pressure and the duration of this application.

For example:

Inspiratory muscles provided a pressure drop of 6 cmH_2O lasting 1 second, in this case:

PTP = 6 x 1 = 6 cm H_2O.sn it will be.

If the same effort lasts 1.5 seconds, PTP = 6 x 1.5 = 9 cmH_2O.sn sure.

Thus, measurement of the mechanical performance of respiratory muscles is obtained, both independent of the resulting volume change and related to the duration of the effort.

Pressure - Time Index (PTI): evaluates the need for ventilatory pressure against the ability of ventilatory muscles to create pressure

PTI = (PTP / respiratory cycle time) / PImax

PTI < 0,15 indicates excessive load and inadequacy of respiratory muscles.

Criteria for successful weaning [1,2]

- Ventilatory capacity:
- And< 10 l / min and spontaneous and= mechanical and
- Vt> 5 ml / kg
- Vital capacity> 10 ml / kg
- Spontaneous breathing frequency< 25 / min
- Maximum negative inspiratory pressure (NIP)< -25 cmH2O
- Maximum voluntary ventilation> 2x mechanical and

Ventilatory requirements [1,2]

FiO_2< 0.5 to PaO_2> 60 mmHg

Vd / Vt< 0.5

D $(A-a)O_2$>300-350 torr at 100% Fio_2

Intrapulmonary shunt rate(Qs/Qt)< 20 %

Combined indicators [1,2]

f / Vt< 100

CROP 13 ml / breathing / min

Rapid superficial respiration index (RSBI) = number of respiration (/min) / Vt (l)

>100 min/l patient can leave ventilator

Over 120 still require mechanical ventilation

Spontaneous breathing trials (SBT))

The most direct method of evaluating the load-capacity relationship is to determine whether the patient can tolerate spontaneous breathing trial (SBT) for a period of 30-120 minutes. There's no point doing more than one SBT a day. If the first spontaneous breathing trial has failed, the factors that increase respiratory work and the factors that may cause neuromuscular weakness should be reviewed. Tracheostomy should be evaluated after two or 3 failures. The decrease in respiratory work, the need for sedation, the frequency of ventilator-induced pneumonia, and the duration of mechanical ventilation made early tracheostomy popular.

During the time between daily spontaneous breathing trials, trigger modes should be used that will not cause novelization. Modes that automatically adjust the pressure support level based on patient effort (ASV, SmartCare) provide faster weaning than traditional doctor-directed weaning. SBT successful:

1. Gas exchange:
   a. $SpO_2 \geq 85\text{-}90\ \%$
   b. $PaO_2 \geq 50\text{-}60$ mmHg
   c. pH ≥ 7.32
   d. Paco2 increase < 10 mm Hg
2. Hemodynamic stability:
   a. Heart rate < 120-140 / min, change < 20 %
   b. Systolic blood pressure < 180 mmHg and > 90 mmHg, change < 20 %
   c. No vasopressor requirement
3. Stable ventilatory pattern:
   a. Respiratory count ≤ 30-35 / min
   b. Respiratory rate of change < 50 %

SBT fail

- Deterioration of Mental condition (somnolence, coma, agitation, anxiety)
- Deterioration of patient comfort
- Sweat
- Signs of increased respiratory work (accessory novelistic activity, thoracoabdominal paradox)

The early phase (screening) of SBT should be closely monitored. Severe respiratory muscle loads may occur during this period. If the continuation of SBT after the early phase is decided, this period should be at least 30 min and at most 120 min. (2,3)

Management of the patient who does not tolerate SBTS

- Factors that will cause ventilator dependence are reviewed
- Respiratory loads are reduced by correcting respiratory mechanics (reduction of edema, correction of airway function)
- Reduced metabolic need (use of oxygen, formation of carbon dioxide)
- The ability of the patient is strengthened (nutrition, fluid status, electrolyte balance, toxic drugs)
- Oxygen delivery is evaluated (cardiac flow, hemoglobin concentration, oxygen saturation). Intensive care patients can tolerate hemoglobin concentrations of up to 8 g/dL.
- Heart failure may precipitate due to decreased intrathoracic pressure and increased Left Ventricular afterload with the patient's separation from the ventilator.

Ventilatory management of the case that failed in SBT trials 2 aims

1. Normalizing respiratory load
2. Improving patient comfort by optimizing patient-ventilator synchronization

Overload causes further muscle failure, while reducing overload causes atrophy of novices. The best approach in these patients is to ensure that the patient does close to normal breathing work. The main goal should be to reduce total respiratory work. Patient-ventilator sync needs to be corrected in order to normalize the breathing pattern as much as possible

The patient ventilator relationship is related to the following functions:

- Assisted breathing triggering method
- Current distribution process
- Breath termination process

A responsive trigger system must be used to correct the patient-ventilator relationship and reduce additional breathing loads, and additional PEEP must be added When the pressure to reach the trigger threshold is increased by the internal PEEP

- Inspiratory flow rates must be used to respond to patient demand.
- Proper ventilation cycle times should be used to prevent air confinement
- November puts the muscle load on the ventilator in a similar way to the normal load that helps each breath and effort with the support of ventilation.

Two new approaches to correct patient-ventilator synchronization

Tracheal pressure target (automatic tube compensation): this approach essentially eliminates endotracheal tube RESIATANCE. Continuous stable airway pressure is achieved in the trachea by automatic tube compensation

Adjusting the time of pressure increase during pressure-controlled breathing: adjusting the speed of reaching the target pressure without

affecting the adjusted target pressure. The purpose of this speed adaptation is to provide a smooth square shaped current wave. Patient comfort improves as the highest tidal volume occurs in this current form [2,3,14].

Parameters that determine the outcome after the patient leaves the ventilator [1-3]

Threshold values of measurements performed while patient is in ventilator:

- Minute volume (l / min) 10-15
- PImax (cm H_2O) -20, -30
- P0, 1 / NIF (cm H_2O) 0.3
- Item 13

Threshold values of measurements made 1-2 minutes after the patient breathes spontaneously:

- Respiratory frequency (/min) 30-38
- Tidal volume (ml) 325-408 (4-6 ml / min)
- Frequency / tidal volume (faint/L) 60-105

After the patient tolerates SBT, attention is paid to whether the patient can tolerate extubation

Is the patient's cough enough?

What is the need for Aspirin?

Peak cough current should be > 160 L / min in patients without neuromuscular and spinal damage

The patient's aspiration need should be aspiration ≥ 2 extubation and decannulation

Criteria

1. patient not needing artificial airway to maintain upper airway opening
2. the patient has neurological function that can protect the airway
3. having laryngeal function to clear pulmonary secretions
4. the patient has sufficient strength for spontaneous breathing

5 to 10% of intubation-induced airway stenosis may occur

Post-extubation stridor is more common in women, in those with large Lumen tubes and in those with more than 36 hours of enubes.

If there are fewer than 110 ml leaks in cuff leakage test in patients who have been intubated longer than 36 hours, corticosteroids can be given every 4-6 hours starting 12-24 hours before extubation. Nebulized adrenaline may reduce the need for reintubation in patients who develop post-extubation stridor. Reintubation risk increases 3-fold in those with GCS below 8. Peak Expiratory Flow rate below 60 L / min increases the risk of reintubation 5.1-fold. The risk of reintubation increases 5.3-fold as novelization decreases muscle endurance in anemia. Rapid superficial inhalation index (RSBI = frequency/Tidal haim) is high risk of reintubation in those below 100

Telling the presence of sticky secretion by the nurse increases the risk of reintubation by 8.7 times. Reintubation risk increases 3-fold in sputum production of more than 2.5 ml / hour. If more than one aspiration is required every two hours, the risk of reintubation increases 16-fold. Diuresis, seen in the recovery phase of critical disease, is a sign of the transition of fluid between tissue to capillaries due to returning capillary reflectance. In these cases, 6 hours of urine is more than 500 ml. In cases with negative fluid balance in the last 24 hours, the risk of reintubation decreases [1,2,3,13].

IMV (Intermittent challenging breathing)

As the patient's spontaneous frequency increases, the ventilator frequency decreases. $PaCO_2$< 45 mmHg and respiratory number < 30 remain, mechanical ventilator frequency drops to 1-2 / min If the IMV frequency drops to 1-2/min, blood gas values are also sufficient, the case can be separated from the device.

PSV (pressure support ventilation) [1,2,13]

SIMV + PSV (synchronized intermittent mandatory ventilation + PSV)

VS (volume support): PSV where minimal tidal volume is guaranteed. The PS level is automatically adjusted to reach the tidal volume set by the clinician. Vaps (Pa) (Volume assured pressure support) PSV where minimal tidal volume is guaranteed. In order to reach the tidal volume, set by the clinician, if necessary, the current is added at the end of the ethyl alcohol.

MMV (mandatory minute ventilation)

Simv, where minute ventilation is guaranteed. Additional demanding machine solutions are added if necessary, according to the volume of minutes the clinician has set.

Aprv (airway pressure release ventilation) pressure difference between inflationary and draining. As the start decreases differently, more spontaneous breathing is needed

ASV (adaptive support ventilation) It is the mechanical ventilation mode in which the ventilator provides the gas flow with the positive pressure value set by the physician. In devices with Feedback rings, the inspiratory flow is thought to be at 25% of the initial value, and the device passes into the expiratory phase and the airway pressure drops to its initial value.:

Net pressure= PSV pressure - alveolar pressure

As the inspiratory current continues, the increased pressure in the alveoli causes the net pressure to drop.

the decreasing net pressure causes the inspiratory current to decrease as the inspirium progresses. When the inspiratory current drops to ¼ of the initial value, the inspiratory current is cut off.

Current in PSV where the ventilator goes into expiratory phase [1,2]

- The fix can be an absolute amount of current
- Peak may be a current based on inspiratory current
- Peak inspiratory current and passing inspiratory current can be based on time.

The new ventilators allow the patient to adjust the current and rise-time at which the appropriate inspirium will end. Rise-time is the time to reach the pressure support level set at the start of the inspiration. In this mode, the physician only determines the inspiratory pressure

required for the gas flow. The patient determines the physiological mechanisms and the need and strength of ventilation and the rate of inspiratory flow, the duration of the inspirium and the frequency. Tidal volume is a function of Net pressure and inspiratory time determined by the patient. Adjusted inspiratory pressure, complement, resistance, the strength of the patient's breathing effort, the desired tidal volume and minute ventilation varies according to. Generally, 25-30 cm H_2O pressure is started or tidal volume 6-10 ml/kg to reach the value of the pressure adjustment is made. Pressure adjustments are made until the appropriate breathing pattern is established and the number of breaths is below 20/min. Getting more tidal volumes with lower peak airway pressure reflects.

References

1. Alía I, Esteban A (2000) Weaning from mechanical ventilation. *Crit Care* 4: 72-80. [Crossref]
2. El-Khatib MF, Bou-Khalil P (2008) Clinical review: Liberation from mechanical ventilation. *Crit Care* 12: 221. [Crossref]
3. Hsieh MH, Hsieh MJ, Cheng AC, Chen CM, Hsieh CC, et al. (2019) Predicting weaning difficulty for planned extubation patients with an artificial neural network. *Medicine (Baltimore)* 98: e17392. [Crossref]
4. Muñoz V, Calvo L, Ramírez MF, Arias M, Villota M, et al. (2014) Ventilatory weaning practices in intensive care units in the city of Cali. *Rev Bras Ter Intensiva* 26: 137-142 [Crossref]
5. Carlucci A, Ceriana P, Prinianakis G, Fanfulla F, Colombo R, et al. (2009) Determinants of weaning success in patients with prolonged mechanical ventilation. *Crit Care* 13: R97. [Crossref]
6. Bornitz F, Ewert R, Knaak C, Magnet FS, Windisch W, et al. (2020) Weaning from Invasive Ventilation in Specialist Centers Following Primary Weaning Failure: A Prospective Multi-Center Study of Weanability in Patients Receiving Prolonged Domiciliary Ventilation. *Dtsch Arztebl Int* 117: 205-210. [Crossref]
7. Nemer SN, Barbas CSV, Caldeira JB, Cárias TC, Santos RG, et al. (2009) A new integrative weaning index of discontinuation from mechanical ventilation. *Crit Care* 13: R152. [Crossref]
8. De Medeiros Silva CS, Timenetsky KT, Taniguchi C, Calegaro S, Azevedo CSA, et al. (2012) Low mechanical ventilation times and reintubation rates associated with a specific weaning protocol in an intensive care unit setting: a retrospective study. *Clinics (Sao Paulo)* 67: 995-1000 [Crossref]
9. Heunks LM, Van der Hoeven JG (2010) Clinical review: The ABC of weaning failure - a structured approach. *Crit Care* 14: 245. [Crossref]
10. Lago AF, Gastaldi AC, Mazzoni AAS, Tanaka VB, Siansi VC, et al. (2019) Comparison of International Consensus Conference guidelines and WIND classification for weaning from mechanical ventilation in Brazilian critically ill patients: A retrospective cohort study. Medicine (Baltimore) 98: e17534. [Crossref]
11. Ladeira MT, Vital FMR, Andriolo RB, Andriolo BNG, Atallah AN, et al. (2014) Pressure support versus T-tube for weaning from mechanical ventilation in adults. *Cochrane Database Syst Rev* 5: CD006056.[Crossref]
12. Zein H, Baratloo A, Negida A, Safari S (2016) Ventilator Weaning and Spontaneous Breathing Trials; an Educational Review Emerg (Tehran). *Spring* 4: 65-71. [Crossref]
13. Tseng CC, Huang KT, Chen YC, Wang CC, Liu SF, et al. (2012) Factors Predicting Ventilator Dependence in Patients with Ventilator-Associated Pneumonia. *Scientific World Journal* 547241. [Crossref]
14. Vassilakopoulos T, Zakynthinos S, Roussos C (2006) Bench-to-bedside review: Weaning failure – should we rest the respiratory muscles with controlled mechanical ventilation? *Crit Care* 10: 204. [Crossref]