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Mechanical Ventilation

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Abstract

Exponential function defines the situation where the rate of change in a function is proportional to the size of the variable. For example; while expiratory flow is highest at the beginning during a passive expiration, the lung volume decreases as expiry progresses as it approaches functional residual capacity. Time Constant refers to the rate of change in the function. While short-time functions change quickly, long-time functions change slowly. Pressure gradient is required for a flow to occur and a displacement of volume in mechanical ventilation. The movement equation reflects the pressure change that occurs in the airway opening during breathing. According to equality, the lung is equally expanded in all directions (isotropic expansion). Airway resistance is influenced by current, breath volume and airway dimensions. Total respiratory system compliance is the sum of the compliance of the lung and rib cage.

Keywords: Exponential functions; Resistance; Compliance

Exponential Functions and Time Constants in Mechanical Ventilation

Exponential function defines the situation where the rate of change in a function is proportional to the size of the variable. For example; while expiratory flow is highest at the beginning during a passive expiration, the lung volume decreases as expiry progresses as it approaches functional residual capacity [1].

Rising Exponential Function

Indicates the rise in variable as a function of time

Pressure-time, volume-time. Inspiratory volume occurring in constant pressure modes is also a function of time; inspiratory volume-time waveform is an example of rising exponential function. The rate of change in the function is constant and proportional to the size of the variable. Constant is usually the final value of the variable.

dy/dt = (1/π) y son- y π: Time constant of the system

According to this equation, the y value of the variable will approach the end value of y over time and the change value will be zero at the end of the function. In other words, while the change is greatest at the beginning of the event, it will decrease gradually.

y = y end (1-e-t/π)

y = the value of the variable at time t

e = basal value of natural logarithm (2,71,828)

y end = the last value of y

t = time since the start of the event

π = time constant of the system

Declining Exponential Function

The decline in the variable is expressed as a function of time

Flow-time, pressure-time, volume-time. An example of the exponential function that decreases with the extinction of the lungs in passive expiration is the decrease.

In the exponential descending function, the y value is zero. The rate of change of the function is proportional to the size of the variable.

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$dy/dt = (1/\pi)\ y$

According to this formula, the value of the y function approaches zero over time and the rate of change in y decreases to zero. Since the function Y is the largest at the beginning of the event (y0), the rate of change is greatest at the beginning, and least at the end.

In both exponential function types, the behavior of the function would be linear if the rate of change was of the same value in each time slot and the function would end in a time constant. But in exponential functions, the rate of change is not always constant over time.

Time Constant

It refers to the rate of change in the function. While short-time functions change quickly, long-time functions change slowly.

Exponential functions do not end even at infinite time constant. Short time constant functions reach the same end value earlier, while long time constant reaches later.

In mechanical ventilation, an event is assumed to be completed within 3 time constants. The normal time constant in the adult respiratory system is 0.79 seconds.

The value of a time constant is measured by the product of compliance and resistance.

$\pi = C \times R = (ml/cmH2O) \times (cmH2O/mlsec^{-1})$

Expiratory time should be longer than 3 times constant to avoid air confinement. End-inspiratory flow should be slow for better ventilation of lung areas with different time constant.

Respiratory Movement Equation

Pressure gradient is required for a flow to occur and a displacement of volume in mechanical ventilation. For a volume to displace, the elastic forces of the lung and rib cage must be defeated. The pressure gradient must exceed the resistive forces of the respiratory system for gas flow to form. At any time of the inspiration, the airway opening pressure (Pawo) should balance with forces opposing the expansion of the lung and chest wall.

Powers against Expiration

- elastic withdrawal pressure (P elastic)

- pressure related to the resistance that resists flow (P resistance)

- pressure to overcome inertia (P inertans)

Pawo = P elastic + P resistance + P inertness

While P inertness can be neglected in conventional ventilation, it becomes important in high frequency ventilation.

Movement Equation in Conventional Ventilation

Pawo = P elastic + P resistance

P elastic = elastance × volume

P resistance = resistance × current

Pawo = (elastance × volume) + (resistance × current)

Pawo = (volume/compliance) + (resistance × current)

$Pawo = (ml/ml\ cmH2O) + [(cmH2O/mlsn^{-1}) \times mlsn^{-1}]$

The movement equation reflects the pressure change that occurs in the airway opening during breathing. According to equality, the lung is equally expanded in all directions (isotropic expansion).

PAO = V/C + V ° R + V °° I - Pmus

PAO: pressure in the airway opening (mouth or ET tube)

V: lung volume

C: compliance of the respiratory system

V °: gas flow

R: airway resistance

V °°: convective gas acceleration

I: impedance

Pmus: pressure created by respiratory muscles

Volume/compliance

Shows the pressure required to overcome elastic forces above functional residual capacity

Resistance x current

It is the product of maximum airway resistance (Rmax) with inspiratory flow. Indicates the pressure required to overcome resistive forces.

Setting the Pressure Control

Elastance-related component of motion equation (V/C)

If pressure is set as a function of time, the volume of dispersed volume is affected by compliance changes. Pressure is independent, volume dependent variable. In pressure-controlled ventilation, the inspiratory volume-time curve changes exponentially with the function of time and compliance. Because the expiration is passive, the expiratory waveform reflects the elastic and resistive properties of the respiratory system [2].

Resistance related component of motion equation (R x A)

If the pressure is set as a function of time, the current formed is the function of the resistance. Pressure is independent flow dependent variable. The inspiratory flow-time curve varies exponentially depending on the current, which is a function of time and resistance.

In summary, in pressure controlled modes, the adjusted pressure is reached regardless of the elastic and resistive powers of the respiratory system and remains constant during the inspiratory period. Tidal volume and current change exponentially as a function of compliance and resistance, respectively.

If the resistive item is examined: P = RxA…. A = P/R

If current is set as a function of time, the pressure changes as a function of the resistance. Flow is independent pressure dependent variable. In flow-controlled ventilation, the inspiratory pressure-time curve changes with time and pressure, which is the function of resistance. Volume increases over time, but is not directly related to current. However, since the volume is an integral of the current and the current is the derivative of the volume, the volume and the current are in an indirect relationship.

Regardless of the elastic and resistive powers of the respiratory system in flow-controlled ventilation, the adjusted current will occur and remain constant throughout the inspiratory. Pressure and tidal volume will change as a function of compliance and resistance,

respectively.

Modern ventilators can operate with current or pressure control. Constant flow (square flow) and constant pressure (square pressure) models are used most frequently.

Various current models are created by the microprocessors of the ventilators. They can create decreasing ramp (descending ramp), increasing ramp (ascending ramp) and sinusoidal current models. These current models are used in various volume loop modes. The decreasing ramp flow pattern improves the distribution of ventilation in the lungs with heterogeneous time constant.

Setting the Volume Control

Pawo = Elastic element + Resistive element = (V/C) + (RxA)

P = V/C V = PxC

If volume is set as a function of time, the pressure becomes dependent on compliance. Volume Independent pressure dependent variable. In order for the ventilator to really operate with volume control, the volume must be measured directly. Many ventilators cannot directly measure volume; calculates the amount of current over a certain time and uses the volume as a limiting variable. What does it mean; when the set volume is reached, the inspirium is terminated. Volume cycle ventilation, which is the tidal volume set by the end of the inspiration, is actually flow-controlled [3].

Constant Current

Pressure-time curve

The rise in peak pressure in inspiration and the decrease in end-expiratory pressure in expiration depend on elastic and resistance-related properties.

Volume-time curve

Reaching inspiratory tidal volume and decreasing expiratory volume depends on features related to compliance and resistance.

Flow-time curve

Reaching the peak inspiratory flow is not fixed and depends on elastic and resistive properties. The drop in end-expiratory flow depends on the same properties.

Volume-pressure cycle

The changes reflect an increase in peak inspiratory pressure, not an increase in inspiratory tidal volume. Return to expiratory volume and end-expiratory pressure depends on elastic and resistive properties.

Flow-volume cycle

Peak inspiratory flow and increase in inspiratory tidal volume reflect elastic and resistive properties. End-expiratory flow and return to expiratory volume also depend on these properties.

Constant pressure

Pressure-time curve: Reaching peak pressure is not fixed; Reaching peak pressure and end-expiratory pressure depends on elastic and resistive properties.

Volume-time curve: Reaching inspiratory tidal volume and descending to expiratory volume depends on elastic and resistive properties.

Current-time curve: Decreasing current and return to end-expiratory flow depend on elastic and resistive properties.

Volume-pressure cycle: Inspiratory tidal reflects changes in volume, while it does not reflect an increase in peak pressure. Return to expiratory volume and end-expiratory pressure depends on elastic and resistive properties.

Current-volume cycle: Descending current and increase in inspiratory tidal volume, end-expiratory flow, and return to expiratory volume are dependent on elastic and resistive properties.

Resistance indices are generally calculated by pressure-time curves. It is defined as the pressure change that occurs against the gas flow. Airway resistance is influenced by current, breath volume and airway dimensions.

Since the rate of decrease of flow in constant pressure mode is a function of resistance, current-time waveform gives information about resistance. In this mode, since the current is variable and the pressure is constant, the resistance cannot be measured with the current cut technique. Resistive properties in constant pressure mode can be examined in current-time and volume-time waves.

Constant current mode resistance is generally measured by the "current cut" technique. In this technique, the current is interrupted at the end of the inspirium and the pressure is kept constant for a certain time (pause time). Properties related to resistance in constant current mode can be examined from the pressure-time waveform.

Resistance (R)

$R = \Delta P/A = cmH2O/mlsn^{-1}$

Resistance indices are generally calculated by pressure-time curves. It is defined as the pressure change that occurs against the gas flow. Airway resistance is influenced by current, breath volume and airway dimensions [4,5].

Since the rate of decrease of flow in constant pressure mode is a function of resistance, current-time waveform gives information about resistance. In this mode, since the current is variable and the pressure is constant, the resistance cannot be measured with the current cut technique. Resistive properties in constant pressure mode can be examined in flow-time and volume-time waves.

In constant current, an exponential pressure increase first occurs depending on the current and resistance. Then, a linear pressure increase occurs due to the constant current until the peak pressure is reached. This piece reflects the elastic properties of the respiratory system. With the equalization of the pressures in the airways and the breathing circuit, a plateau is formed during the pause time, since the pressure was balanced during this period, the current was cutoff.

Inspiratory resistance: RI = (Ppeak - Pplato) /peak inspiratory flow

Expiratory resistance: Re = (Pplato - PEEPtotal) /current at the beginning of expiration

Requirements for valid resistance measurement

- passive tidal volume (inspiratory and expiratory)

- constant current during inspirium

- Pplato should be measured with at least 1 sec end-expiratory plateau and should be stable by playing around 0.5 cmH_2O in two measurements made every 10 millisecond apart.

The decrease from peak pressure to plateau pressure should be examined: With this examination, the maximum resistance index (Rmax) and the minimum resistance index (Rmin) can be calculated.

- peak pressure reflects dynamic pressure.

- Pz is the pressure that occurs at the moment when the current is interrupted.

- Plateau pressure refers to static airway pressure and is considered to best reflect alveolar pressure.

Rmax reflects the resistance created by the endotracheal tube, respiratory circuit, pulmonary and thorax tissue at the maximum lung volume.

Rmax = (Ppeak-Pplato) /peak inspiratory flow

Rmin = (Ppeak-Pz) /peak inspiratory flow

Pz is the pressure at the moment when the expiratory valve closes and the flow stops. Rmin is the component of Rmax and reflects only the resistance of the airways. The difference between Pz and Pplato reflects the gas distribution in alveolar units with different time constants; despite the cessation of flow in large airways, there is still current in lung zones with different time constants. This phenomenon is called "Pendelluft" (air hang). The greater the difference between Rmin and Rmax, the greater the time constant difference between zones. The difference between Pz and Pplato in ARDS usually ranges from 10% to 20%.

Compliance (C)

It reflects the elastic properties of the respiratory system.

C = ΔV/ΔP = ml/cmH2O

Total respiratory system compliance (Crs) is the sum of the compliance of the lung and rib cage.

1/Crs = 1/C pulmonary + 1/C chest wall

As soon as there is no current activity at the end of the total static compliance inspiratory and expiratory, the total dynamic compliance is monitored during the active inspiratory [4]. Compliance can be observed from flow-time, pressure-time waves and volume-pressure cycle.

Chest wall compliance (Ccw):

Esophageal pressure reflects pleural pressure; Ccw = Tidal volume/Pesophagus

The patient must be completely passive in order to calculate Ccw. Ccw is the main parameter in the heasplantation of the total respiratory work with the Campbell diagram. Ccw is estimated at 4% of the tidal capacity for every centimeter pressure increase in esophageal pressure. Its normal value is approximately 200 ml/cmH2O.

Lung compliance (Ppulm)

It is the breath volume formed against transpulmonary pressure change. Cpulm can be measured in active and passive breaths.

Cpulm = tidal volume/ (Pplato - Pesophagus)

Transpulmonary pressure = Palveolar –Pplevra

Pplato alveolar pressure reflects the esophageal pleural pressure

Total static compliance

Csttot reflects the pressure required to overcome the elliptical forces of the respiratory system at zero current and in any breath volume.

Requirements for a valid measurement

- passive breath volume (inspiratory and expiratory)

- correction for the compressor volume of the breathing circuit

- Pplato should be measured with an end-expiratory pause of at least 1 sec and should be stable with 0.5 cmH2O play in two measurements made every 10 milliseconds.

Csttot is the breath volume that changes relative to the airway pressure (Pplato - PEEPtotal) in the static state (no current) and the breath volume should be corrected for the volume to the compress.

Csttot = Tidal volume/ (Pplato - PEEPtotal)

It reflects the elastic properties of the lung. Causes that increase the closure of the lungs or reduce their volume reduce static compliance.

Cst is 60-100 ml/cmH2O (1 ml/1 cmH2O/kg) in adult.

In constant flow modes with pause time, Cst can be estimated from the output slope of the volume-pressure cycle (from start to Pplato).

Since static compliance is affected by conditions such as the patient's structure, relaxation, lung volume, and flow, dynamic values that are constantly monitored are more appropriate.

Dynamic Compliance

Gas flow creates a resistance in a dynamic event. The volume-pressure relationship in a dynamic event still depends on the resistance [6]. Therefore, the definition of dynamic properties is more suitable. In normal flows (50-80 L/min), the dynamic properties are 10% to 20% lower than static compliance. It reflects the sum of the pressures necessary to overcome the parenchymal compliance elements of the lung and the airway resistance formed by the current during a breath volume application. Therefore, it reflects the features of the respiratory system related to compliance and resistance.

dyn.ch. = tidal volume/ (Ppeak - PEEP)

Its normal value is 50-80 ml/cmH2O in adult, whereas it is 5-6 ml/cmH2O in newborn.

The difference between static and dynamic compliance can be used as an index of the current-resistance properties of the respiratory system.

Respiratory Work (WOB)

The work is the displacement of a mass with the applied force. In the respiratory system, power pressure and displacement are considered as volume. A person who breathes from a completely blocked endotracheal tube will not do any work at all, as it will not create any tidal volume [7], only exerting effort. Microprocessors can calculate WOB and digitize it.

W = F × D (Work = Force × distance) = Newton × meter

In the respiratory system;

Power = pressure × area

W = Pressure × Area × Distance

Area × distance = volume

W = Pressure × volume

Since the pressure in the respiratory activity constantly changes in size and direction, the formula becomes:

$W = \int P.\, dV$

According to this equation, the area inside the Pressure-volume cycle gives the work of breathing.

In order for the displacement of the volume in the movement equation of the respiratory system, the guiding force (patient or ventilator) must overcome the elastic and resistive elements of the respiratory system.

WOB = WOB elastic + WOB resistive

If there is spontaneous breathing while under the ventilator support;

WOB total = WOB patient + WOB ventilator

WOB Elastic

It is the work done by the patient and ventilator to defeat elastic forces. It is evaluated by the slope of the volume-pressure cycle. It cannot be easily calculated at the bedside; Complications of the lung and chest wall need to be calculated. This requires assessment of intrapleural pressure with esophageal pressure. The patient must be completely loose. Intraabdominal pressure changes affect the elasticity of the chest wall compliance [8]. The elastic work of the ventilator can be easily found when the patient is completely passive. Area = base × (height/2)

ABCA area = breath volume × Ppeak/2 = 880 × 23/2 = 10120 cmH2O.ml

The business unit is Joule/L or Joule/min. Need correction; It is converted to cmH2O.ml kg.m. Joule is converted in Kg.m. 1 cmH2O. ml = 10-5 kg.m. In 1 kg.m = 0, 1 joule.

As a result 10120 cmH2O.ml = 10.12 × 10-2 kg.m

To get the WOBelastik as joule/l, the joule value is reflected in the liter value; 0.01 joule/0.880L = 0.011 joule/l. This account is valid for passive patients.

WOB Resistive

This is the work done by the patient and the ventilator to overcome the resistive (nonelastic) powers of the respiratory system. The curve in the inspiratory arm of the volume-pressure curve is formed by constant current [8].

Calculation of WOB Total

It has 2 types.

1. Planimetrically from the pressure-volume curve

2. By electronic integration of the pressure-volume product

Planimetric measurement of the volume-pressure cycle using the Campbell diagram: Defined by Campbell in 1958. Ccw and Cpulmonary are taken into consideration. While Cww patient is passive, Cpulmonary patient is measured while breathing spontaneously. This planimetric calculation is based on the footprint of the cycle, tidal volume and peak pressure. The measured area is converted to work with the formula [8].

Pressure-Flow Integration

Ptotal = Pelastic + Prezistif

WOB total = WOB elastic + WOB resistive

$WOB = \int P.\, dV$

Airway resistance is defined as the relationship between pressure gradient (ppeak - Pplato) and flow. Resistance and respiratory work relationship; WOB resistif = $\int$ (Ppeak –Pplato) V˙dt

In this equation, the current is expressed as volume (V˙dt) as a function of time.

WOB elastic = WOB lung + WOB cw

The elastic properties of the lung are defined by the pressure gradient (Pplato - Pesophagus) and tidal volume relationship. W Lung = $\int$ (Pplato –Pesophagus) V˙dt

The elastic properties of the rib cage are defined by the relationship between pressure gradient (Pesophagus - Patmosphere) and tidal volume. WOB cw = $\int$ (Poseophagus - Patmosphere) V˙dt

WOB elastic = $\int$ (Pplato - Poseophagus) V˙dt + $\int$ (Poseophagus - Patmosphere) V˙dt

WOB total = $\int$ (Ppeak –Pplato) V˙dt + $\int$ (Pplato –Pozophagus) V˙dt + $\int$ (Poseophagus - Patmosphere) V˙dt

OtoPEEP (intrinsic PEEP): It refers to the positive pressure that remains involuntarily in the lungs at the end of expiration [9].

Determining factors

1. Tidal volume

2. Expiratory time constant

3. Short expiration time

Oto PEEP results in less calculation of static compliance. Since the functional residual capacity will only increase after the auto PEEP is exceeded, auto PEEP should be taken into account when setting PEEP.

If the expiratory flow does not decrease to zero before the new inspirium starts in the current-time wave, it means that there is oto PEEP. In this waveform, auto PEEP can be detected but cannot be calculated. Oto PEEP can only be measured on the pressure waveform during expiratory hold in a patient who has been completely relaxed. Requires esophageal pressure measurement in a patient breathing spontaneously.

Oto PEEP can work in two ways;

1. Auto peEP without dynamic hyperinflation: occurs in active expiration. Strong expiratory muscle activity lasts until the end of expiration.

2. Dynamic hyperinflational auto PEEP: This occurs during passive expiration. The resulting dynamic hyperinflation is equal to oto PEEP. It may occur with or without expiratory flow limitation.

It increases the breathing work in the spontaneously breathing patient because it increases the auto PEEP trigger threshold. The effect of oto PEEP on respiratory work can be reduced by applying

PEEP at a value lower than Oto PEEP.

Oto PEEP can be reduced by approaches that allow normal expiration;

1. Bronchodilator

2. Wide endotracheal tube

3. Reducing minute ventilation

4. Shortening the duty-cycle

5. Reduction of breathing depth

6. Increasing the flow (increasing the flow decreases the required inspiratory time, thus increasing the expiratory time)

Bias Current

It is the gas flow created by the ventilator in the breathing circuit and is associated with current triggering. With the onset of expiration, a constant current is supplied to the breathing circuit by the ventilator. This current continues through the expiratory phase unless the expiratory patient flow is interrupted. With the onset of the next inspiratory (patient-triggered or mandatory) bias current is interrupted [7,8].

This flow is given during pressure and flow triggering. The current is automatically selected according to the patient:

- 32 ml/sec (2 L/min) for adult

- 16 ml/sec (1 L/min) for the child

- 8 ml/sec (0.5 L/min) for neonates

Constant current generating ventilators

The pressure that occurs because it creates a constant current regardless of the physical condition of the respiratory system depends on the elastic and resistive properties of the respiratory system. The inspiratory arm of the current-time waveform is square. The initial inspiratory part of the pressure-time waveform rises exponentially, and then increases linearly up to the peak pressure.

Constant pressure generating ventilators

Due to the constant pressure application, the flow pattern varies according to the characteristics of the respiratory system. The inspiratory arm of the current-time waveform descends exponentially towards zero. The press-time wave is square.

Duty Cycle

Defines the inspiratory period of a breath or defined as Ti/T total. The I/E ratio is another form of expression. Its normal duration is 0.2 s to 0.4 s. Over 0.5 means reverse rate ventilation.

Dynamic airway compression

These are cases where pleural pressure is higher than atmospheric pressure (such as forced expiration, cough). In a demanding expiration, the pressure around the airlines is higher than the pressure inside. In other words, intrapleural pressure will narrow or close the airways.

Dynamic hyperinflation

These are cases where the end-expiratory lung volume exceeds functional residual capacity. It may be due to early expiration of expiration or slow lung discharge rate.

Dynamic state

Defines the activity that occurs during the movement of the lungs and thorax. For example, the measurement of the volume-pressure relationship during the active phase of the breathing cycle is dynamic.

Functional Residual Capacity (FRC)

It is known as static equilibrium volume. It is the volume remaining in the long after normal passive expiration. FRK has decreased in ARDS and PEEP is used to reach the point that will form FRK [10].

Hysteresis

It is a loop characteristic. Inspiratory and expiratory follow different arms, but merge at the end of the cycle. This phenomenon is common in elastic structures. In the volume-pressure cycle, the area occupied by the cycle is called hysteresis. It is often referred to as elastic hysteresis and reflects the work of respiration. Loops are expressed by the magnitude of hysteresis. Increased hysteresis is associated with loss of lung volume.

Increasing hysteresis means that the volume increase is low despite the pressure increase. This indicates that compliance is decreasing; resistance is increasing, thus increasing respiratory work.

Impedance

Defines all of the respiratory system's limiting forces working against inspiratory.

- elastic recovery forces of the lung and thorax

- resistance to flow

Inertia

Describes the state of forces that oppose the movement of a system. It is determined by the mass of the system and its distribution along the axis of motion. The respiratory system has low intensity because there is practically no resistance to movement. Inertia of movement of the respiratory system is neglected.

Infection point

Defined as the point at which the slope of the curve changes suddenly in any cycle. In the static volume-pressure cycle, the infection point in the lower part of the inspirym arm indicates the reopening of the units that were closed in expiry. The static pressure-volume cycle is quite different from the dynamic volume-pressure cycle. The static state is required to eliminate resistance related elements from the volume-pressure relationship.

Small airlines are closed at the pressure below the infection point, and the lung is overstressed above the deflection point.

The dynamic pressure-volume cycle also reflects resistive elements. Increased inspiratory resistance may make the inspiratory arm more prone to bending the neck, which may cause misinterpretation of this point as an infection point due to alveolar collapse.

Inspiratory occlusion test: Used to measure total static compliance. At the end of the inspiration, the current is interrupted and constant pressure is maintained during the pause.

For reliable test;

1. current-time wave: current must be zero during pause, occlusion must be at the end of inspiratory

2. pressure-time: leakage must be below 20%

3. flow-pressure: flow and volume must remain at zero throughout the pause

Leakage

It is the percentage expression of the difference between inspired and expired tidal volumes. Generally, under 20% leakages is insignificant, more should be the reason. The measured leakage percentage is often erroneous as breath volumes are constantly changing in spontaneous breathing. The stop of the volume-time curve before the next breath indicates leakage. The failure of a loop to return to its origin is related to leakage or air confinement.

Overdistension

It refers to the inflation of the lungs above its optimal compliance. Overdistension can be detected in the volume-pressure cycle. A deflection point appears in the last third of the cycle.

Graphs and their use in mechanical ventilation:

- 4 parameters: pressure, volume, flow, time

- 3 curves: flow-time, volume-time, pressure-time

- 2 cycles: flow-volume, volume-pressure

Cycle time and respiratory rate relationship:

Tc (time cycle) = 60 $secmin^{-1}$/frequency

Tc = Tinspirator + Tekspiratuar

Ti = Tidal volume/flow rate

Example: Tidal volume = 750 ml frequency = 12/min flow rate (v) = 30 L/min (500 ml/s)

Tc = 60/12 = 5 sec

Ti = 750 ml/500 $mlsec^{-1}$ = 1.5 sec

Te = 5 - 1.5 = 3.5 sec

Increasing the flow rate to reach the same tidal volume shortens the inspiratory time and causes the expiratory time to extend for the same breathing frequency.

Effect of resistance and compliance changes: The gas flow in inspiration and expiration is responsible for the formation of resistance in the airways. Molecular friction activity causes pressure to build up. This pressure is found by multiplying the airway resistance (Raw) by the gas flow rate. The pressure required to overcome the airway resistance when there is current in the airlines is called airway resistance pressure (Praw).

Praw = flow rate x Raw = (L/sec) × (cmH2O/L/sec) = cmH2O

When the gas molecules reach the alveoli, trying to distribute the tidal volume despite the elastic recovery forces of the alveoli results in a pressure buildup. This pressure is called alveolar pressure (Pa). Pa can be detected in the inspiratory hold or pause; it is determined by the name Pplato or Pstatik. This pressure can be calculated from the compliance of the tidal volume and respiratory system.

Pplato = Tidal volume/Crs = ml/ (ml/cmH_2O) = cmH_2O

Ppeak can be calculated from Praw and Pplato.

Ppeak = Praw + Pplato

Starting the inspiration: It depends on the trigger mechanism. In controlled modes or devices with backup function, the inspirium is started by the ventilator (time triggered) when the predetermined time has passed. Mechanical assist is triggered by the patient in assisted modes or SIMV mode (patient triggered).

Inspiratory: Mechanical breathing dissipates. The flow, volume and pressure characteristics of respiration depend on factors such as resistance, compliance, flow type-size, and tidal volume.

Termination of inspiration: The parameter set by the clinician to terminate the inspection is called the cycling mechanism. Breathing can be volume, pressure, time or flow cycles.

Start of expiration: It starts with opening the expiratory valve. In case of inspiratory hold or pause, exhalation valve is not opened even if the inspiratory flow is interrupted.

Expression: It is passive. The characteristics of the expiration are determined by the resistance of the airway and its equipment and the restoring forces (compliance) of the lungs.

Controlled Mode Ventilation

1. Inspiratory and expiratory procedures are responsible for the termination of inspiration and expiration.

2. Negative deflection occurs only in the flow-time curve. The flow sensor detects inspiratory flow as positive and expiratory flow as negative deflection [11].

1. Square stream shape shows constant current

2. When the current is constant, the volume delivered is rectilinear (ascending straight line)

3. The initial pressure increase is the pressure required to overcome the airway resistance (Praw). The pressure increase after this point depends on lung compliance and the tidal volume delivered.

4. At the end of the inspiration, gas flow stops, all tidal volume is given and Ppeak is reached.

5. Flow-time curve: With the start of the inspiration, the current rapidly reached its peak value and remained constant for the specified inspiratory time.

Pressure Controlled Ventilation

1. Inspiratory termination ends when the inspiratory time is up.

2. Current goes to zero before inspiration ends. The pressure remains constant during the set inspiratory period. Since the pressure-controlled mode is a time-cycle mode, the current will decrease to zero just before or after the inspiratory time runs out. The tidal volume delivered depends on the characteristics of the lung [12].

Functional residual capacity

It is the lung volume at the moment when the forces that force the lungs to come back and the forces of the chest wall trying to keep the lungs open are in balance. The pressure at this point is called distending pressure.

Pressure-volume cycle

Cdinamik toatal = ΔTidal volume/ΔPressure

Expiratory infection: sudden alveolar degree

Inspiratory infection: sudden alveolar recurrence

It is generally elliptical. The upper point (x) of the ellipse gives the total dynamic compliance. It is found by dividing the volume at this point by pressure (Tidal volume/Ppeak) (if there is no PEEP).

Expiratory and inspiratory infection points: Each cycle has at least two infection points (inspiratory and expiratory), but can be more.

A decrease in FRK causes a decrease in distending pressure. The reduction in distending pressure decreases functional residual capacity and compliance, thereby increasing respiratory work.

Airway obstructions reduce maximal expiratory flow. Insufficient expiratory time and early closure of small airways cause air arrest.

Mean airway pressure (Pmean)

In normal spontaneous breathing, intrathoracic pressure is negative throughout the breathing cycle. Intrapleural pressure ranges from -5 (in expiratory) and -8 (in inspirium) cmH_2O. Alveolar pressure varies between +1 (in expiratory) and -1 (in inspirium) cmH2O. The decrease of intrapleural pressure during inhalation facilitates lung inflation and venous return. The maximal static transpulmonary pressure (= intraalveolar pressure - intrplevral pressure) that may occur during spontaneous inspirium is 35 cmH2O.

In positive pressure ventilation, intrathoracic pressure increases in inspirium and decreases in expiratory. If the expiration time is too short or if the alveolar pressure is too high in the expiration, venous return decreases.

Pmean is the average pressure applied to the airways during the ventilatory cycle. It relates to the size and duration of the pressure applied.

Influencing factors

1. Inspiratory pressure level: The mean pressure increases as the peak pressure increases. In volume cycle ventilation, the peak pressure adjusted tidal volume, inspiratory flow, resistance, compliance and PEEP affect. Peak pressure is set in ventilation with pressure cycle.

2. Expiratory pressure level: The airway pressure during expiration is determined by PEEP.

3. I/E ratio: Mean pressure increases as inspiratory time increases. End-inspiratory hold means increases pressure.

4. If inspiratory pressure is kept constant and inspiratory flow is increased, mean pressure increases.

5. Square wave current creates more mean pressure than descending ramp currents.

Shunt (perfusion-free ventilation): Total shunt = capillary shunt + anatomical shunt

Blood passing through the capillary shunt nonventile area (atelectasis, ARDS, Pneumonia)

Anatomical shunt

Blood bypassing the lungs (congenital heart disease, deep bronchial veins pouring into pulmonary veins, thabesian circulation of the heart)

Inspiratory pressure that exceeds the opening pressure of the alveolar opening in the inspiration and the application of more pressure than the closing pressure of the alveoli at the end of expiration reduces the shunts by preventing the collapse of the alveoli. However, overdistation in a region may direct the blood flow to nonventile areas from here and cause the shunt rate to increase. Positive pressure ventilation can reduce the rate of capillary shunt while increasing pulmonary vascular resistance and increasing blood flow through anatomical shunts. Mean pressure should be kept as low as possible in the presence of anatomic right-left shunt.

As with airway diseases, poor distribution of ventilation causes the relative shunt effect:

1. Some alveoli are relatively less ventilated than perfusion (shunt-like effect and low V/P ratio)

2. Some alveoli are more ventilated than perfusion (dead space effect, high V/P ratio)

Ventilation: It is the inward-outward movement of gas within the lungs.

Minute ventilation (VE) = tidal volume (Vt) × frequency

VE = dead space ventilation (VD) + alveolar ventilation (VA)

VD / Vt ~ 1/3

- Dead space: ventilation without perfusion

- Anatomical dead space: volume of transmitting airways (~ 2 ml/kg)

- Alveolar dead space: Conditions that reduce pulmonary perfusion

- Mechanical dead space: This is the breathing volume of the breathing circuit, increasing the anatomical dead space. Since the anatomical dead space is constant, low tidal volume increases the dead space fraction and reduces alveolar ventilation. That is, a decrease in tidal volume increases the dead space ratio (VD/Vt). Increased dead space fraction in this case requires more minute ventilation for continuity of alveolar ventilation.

The required ventilation level depends on the intended PaCO2, alveolar ventilation and CO2 production.

PaCO2 α CO2 production (VCO2)/VA

PaCO2 = (VCO2 × 0.863) / (VE x [1 - VD/Vt])

Mechanical ventilation can produce alveolar dead space effect with alveolar overdistension. Mechanical ventilation can increase anatomical dead space by creating distention in the airways.

Alveolar overdistension causes barotrauma. Alveolar rupture air enters the bronchovascular sheath;

1. Pulmonary interstitial emphysema

2. Systemic air embolism

3. Pneumomediastinum

4. A pneumothorax

5. Pneumopericardium

6. Pnomoretroperitoniu

7. Pneumoperitoneum

8. It can cause subcutaneous emphysema.

Ventilator Induced Lung Injury

High peak inflation volumes (volutrauma) cause alveolar

overdistension. Alveolar over distension is associated with high alveolar pressure. Since localized overdistations are difficult to monitor, overdistation is estimated from peak alveolar pressure (end-inspiratory plateau pressure). Pplato should be kept below 30 cmH2O, and alveolar overdistension can be minimalized with low tidal volume (6 ml/kg).

VILI may also occur due to alveolar collapse (atelectasis). The pressure on the contact surfaces of open and collapsed alveoli can reach 100 cmH2O. Cyclic opening and closing of the alveoli is harmful and can be prevented by providing optimal recruitment and avoiding degree growth.

Alveolar overdistention and degree enhances inflammation in the lungs (biotrauma). Inflammatory mediators (cytokines, chemokines) can switch to the pulmonary circulation and cause SIRS.

Ventilator Induced Pneumonia

The agent is usually Gram - bacteria originating from the oropharynx and gastrointestinal system. Frequent replacement of ventilator circuits does not affect VIP development, while ulcer prophylaxis, subglottic aspiration, cuff pressure (20 to 25 mmHg) that prevents aspiration, and elevation of the head reduce VIP frequency.

Hyperventilation

Barotrauma causes VLI, causing overdistension. Overdistation lowers cardiac output. Respiratory alkalosis hypokalemia caused by hyperventilation causes lowering of ionized calcium and separation of oxygen from hemoglobin (left shift). Hyperventilation lowers arterial carbon dioxide pressure in COPD and causes alkalinity of blood pH.

Hypoventilation

50 to 70 mmHg arterial partial oxygen pressure and blood pH above 7.2 are generally well tolerated.

Oxygen toxicity

FiO2 values higher than 0.6 for a period longer than 48 hours should be avoided. The lowest FiO2 (<0.6) that will hold PaO2 between 60 to 80 mmHg should be targeted. The oxygen-related sebaceous radicals cause ultrastructural changes in the lung tissue.

High PaO2 values increase partial arterial carbon dioxide pressure due to Halden effect (carbon dioxide is separated from hemoglobin), blood flow to low ventilated areas due to inhibition of hypoxic pulmonary vasoconstriction and inhibition of ventilation.

Cardiac Effects

Increased mean airway pressure and lung compliance and hypovolemia lowers cardiac output. Increasing intrathoracic pressure decreases venous return and reduces right heart filling pressure. The increased alveolar pressure in positive pressure ventilation increases the transpulmonary pressure, thereby increasing pulmonary vascular resistance, thereby decreasing left ventricular filling pressure and increasing right ventricular after load, thereby assisting interventricular septal cipher. It decreases. In positive pressure ventilation, decreasing cardiac output and atrial natriuretic peptide and increasing antitidiuretic hormone decrease urine flow. Cuff leak and aerophagia cause meteorism.

Nutrition

In malnutrition, respiratory muscles break down, the risk of pulmonary infection and edema increases. In overfeeding, the need for ventilation increases.

Excessive daytime insomnia and cognitive impairment cause delirium, patient ventilator desynchronization and sedation-related ventilator dependence.

Causes of patient ventilator desynchronization:

- Poor trigger sensitivity

- Incorrect inspiratory flow and time setting

- Inappropriate tidal volume

- Inappropriate mode

- Otopeep

Barotrauma

Tearing of the alveolar capillary membrane causes dissection of air in the facial plane and accumulation in the pleural and other cavities. Although barotrauma is thought to be related to high alveolar pressure, it should not be forgotten that the volume and pressure causing barotrauma are patient-specific.

Volutrauma

Early ARDS-like table occurs.

- Alveolo-capillary membrane permeability increases

- Pulmonary edema development increases

- Neutrophils and proteins accumulate in alveoli

- Surfactant construction decreases

- Hyalen membrane development increases

Pulmonary compliance decreases, it called on to be the primary cause of overdistension. The risk of VILI increases

when the plateau pressure is above 30 cmH2O.

If there is overdistation in a lung unit, the transpulmonary pressure is high (intra-alveolar pressure-non-alveolar pressure). As the difference between alveolar and pleural pressures increases, distension of the lungs increases. The chest wall plays an important role in determining alveolar overdistension pressures. As the chest wall compliance increases, the transpulmonary pressure increases and the volume of the lungs increases for the given unit pressure.

Transpulmonary pressure = alveolar pressure - pleural pressure

When the chest wall is solid (abdominal distension excess fluid resuscitation, deformity, etc.), transpulmonary pressure drops. So that a higher pressure is combined with an alveolar less alveolar distension. The solid chest wall does not allow overdistation, protecting the lungs from VILI. In these cases, plateau pressures of more than 30 cmH2O can be applied with less risk.

Atelectotrauma

Cyclic opening and closing of unstable atelectatic alveoli is the cause of VILI. It is the stress that occurs between alveoli, the mechanism of which is stable and not. Stress pressure exceeding 100 cmH2O occurs on the surface of the alveolar when tension pressure of 30 cmH2O is applied.

Biotrauma

Overdistension, cyclic opening and closings would be attempted by the release of proinflammatory mediators. As a result, pulmonary edema, neutrophil accumulation increases, and vascular smooth

muscles relax. PEEP protects from inflammatory damage. With proper PEEP and low tidal volume, bacterial translocation from the lungs is reduced.

High vascular volume, rapid respiratory rate, and high body temperature also increase the risk of VILI. To protect from VILI, the tidal volume should be 4 to 8 ml/kg, the plateau pressure should be less than 30 cmH2O and the degree should be prevented by PEEP (10 to 15 cmH2O).

How the Ventilator Works

1. Control variables: ventilator manipulates a control variable to create inspiration; pressure, time, volume or flow.

- Pressure controller: pressure waveform is unaffected by resistance and compliance changes

- Volume controller: it can measure tidal volume and this measurement is used to control the volume waveform

- Current controller: the measured volume is used as the feedback signal and the volume waveform remains constant

- Time controller: control variables are only inspiratory and expiratory times [13].

2. Phase variables: These variables are used to start the phases of the ventilatory cycle: triggering, limit and cycle

- triggering: inspirium is started. It may be time or patient triggered.

In triggering time, inspirium is started with the intervals determined by the clinician. Patient triggering may be pressure or flow triggered. If the patient's spontaneous effort reduces the airway pressure below the specified level in pressure triggering and the patient draws the specified inspiratory flow in triggering the trigger, it triggers the ventilator to begin inspiration.

- Limit: This variable can be any pressure, volume, or flow that cannot be exceeded during the inspiratory period. When the limit is reached, the inspiration does not have to end.

Cycle

This variable can be a pressure, volume, flow or time that terminates inspiratory. The first ventilators were typically pressure cycled. Pressure support ventilation is current loop. Volume control ventilation is time or volume cycled. Pressure control ventilation is time cycled.

1. Variables controlling the expiratory phase are called baseline variables and can be PEEP or CPAP.

2. Ventilator system: Since the patient is given gas, they definitely have a pneumatic component. The first ventilators were pneumatic ventilators, that is, they used gas pressure to power the ventilator and ventilate the patient. The new ventilators are electronically controlled with a microprocessor.

- Pneumatic system: Responsible for delivering the gas mixture to the patient. Room air and 100% oxygen typically come to the ventilator at 50 Lb/in2 pressure. The ventilator lowers this pressure and mixes it with air according to the predicted FiO2. The exhalation valve is closed for the purpose of insufflation of the lungs during inspiratory and is responsible for the control of PEEP. Traditional exhalation valves are completely closed on the inspiratory tract. In new ventilators, an active exhalation valve operates during pressure-controlled ventilation: if the set pressure is exceeded during the inspiratoryum, the exhalation valve opens.

Pneumatic systems can be single or double circuit. While the gas that supplies power and goes to the patient is the same in single circuits, they are separated in double circuits.

- Electronic system: Most new ventilators are microprocessor controlled. The microprocessor controls expiratory and inspiratory valves. It controls and manages current information (volume, pressure, current) from the monitor system of the ventilator. Controls alarms.

3. Movement equality and patient-ventilator relationship: Patient-ventilator relationship is defined by equality of motion; The pressure (Pt) required to deliver a volume of gas to the lungs is determined by the elastic (Pe) and resistive (Pr) properties of the respiratory system.

Pt = Pe + Pr the elastic properties of the respiratory system are determined by compliance and tidal volume, resistive properties by flow and airway resistance (R).

$Pe = Vt/C$

$Pr = R \times A$

$Pt = (Vt/C) + (R \times A)$

That is, the pressure required to exhale; tidal volume is correct with current and resistance and inversely proportional to compliance.

The pressure required to deliver a breath is created by the pressure applied to the proximal airways and the pressure created by the respiratory muscles.

Pt = Phavyolu + Pkas

In volume-controlled ventilation, the tidal volume and flow delivered to the patient are constant. If the patient performs breathing effort (Pkas) in volume-controlled ventilation, the proximal airway pressure decreases (most often patient-ventilator incompatibility) but the flow does not increase.

In pressure-controlled ventilation, the proximal airway pressure is constant, and patient-ventilator synchronization improves by increasing the flow and volume of the patient's inspiratory effort.

2. Traditional modes: The relationship between the breath type and the phase variable is called the mode. The pale type can be mandatory or spontaneous:

- Continuous Mandatory Ventilation (CMV)

- Continuous Spontaneous Ventilation (CSV)

- Synchronized Intermittent Mandatory Ventilation (SIMV)

Volume, Pressure-controlled ventilation

In volume-controlled ventilation, the ventilator actually controls the inspiratory flow. In pressure-controlled ventilation, inspiratory flow decreases as the alveolar pressure approaches proximal airway pressure during the inspiratory period.

Volume Control

Continuous Mandatory ventilation (continuous mandatory breathing) (CMV)

In this mode, the minimal breathing rate is set by the clinician.

Although the patient may trigger the ventilator, the breath delivered is a pressure or volume controlled breath. CMV is often referred to as assist/control (A/C) ventilation.

Continuous Spontaneous Ventilation (continuous spontaneous ventilation (CSV)

All breaths are triggered by the patient. The most frequently used forms are Pressure Support Ventilation (PSV) and continuous positive airway pressure. In CPAP application, ventilator performance is better in triggering current.

In PSV, the ventilator triggered by the patient's inspiratory effort creates current with the set pressure. PSV is a current loop mode. The patient determines the respiratory rate, inspiratory time and tidal volume. Secondary cycle mechanisms in PSV are pressure and time. In other words, when the determined flow is reached, the pressure is reached and the inspiratory time is up, the expiratory phase is passed. New ventilators have a backup function, which means that if there is apnea in PSV, they switch to controlled mode (VC or PC CMV).

The current through which the ventilator goes to the expiratory phase in PSV:

- Fixed absolute amount of current

- May be a current based on peak inspiratory flow

- The peak inspiratory flow and the elapsed inspiratory time can be based on time.

The new ventilators allow the patient to adjust the current and the rise-time at which the inspiratory end is appropriate. Rise-time is the time to reach the pressure support level set at the beginning of the inspiratory.

Synchronized Intermittent Mandatory Ventilation

Volume or pressure controlled breaths are given with the set frequency. Between these mandatory breaths, the patient breathes spontaneously. Mandatory breaths are given synchronously with the patient's breath effort. If inspiratory effort is not detected, mandatory breaths are given after the time matching the set frequency. In SIMV, spontaneous breaths can be pressure supported.

Dual Control Modes

Mechanical ventilators control either gas flow (volume control) or airway pressure (pressure control) for the inspiratory period. Dual control modes are designed to combine the constant minute ventilation of volume control ventilation and the fast variable current advantages of pressure control ventilation [14]. All dual control modes provide pressure controlled breath using pressure limit and descending flow model. The volume produced varies depending on the patient's effort and pulmonary impedance. In the dual control mode, unlike the pressure control mode, the output is changed according to the measured input (volume). Dual control modes can be patient or time triggered, current or time cycled.

Dual Control modes allow you to set the volume target while the ventilator is pressure controlled breathing. In dual-in-breath mode, the ventilator switches from volume control to volume control, according to the patient's inspiratory effort and ability to reach the set minimum tidal volume, within the same breath. While the device is working in Pressure Support (PS) or Pressure Control (PC) mode in breathless dual control mode, with the operation of the feedback ring, it decreases or increases the pressure limit to provide the tidal volume set by the clinician.

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