Mechanical Ventilation

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Monitoring

- Pressure, Flow and volume in ventilator circuit
- Calculate (monitored) parameters

-Compl, Resist, RC, MAP

- Waveform analysis (Measured parameters)

-Pressure, Flow, Volume

- Loops

Pressure volume, Flow volume

PEEP

What is PEEP?

What is the goal of PEEP?

- Improve oxygenation
- Diminish the work of breathing
- Different potential effects


Open the lung?


keep the Lung
Open ?

Positive end-expiratory pressure

- Alveolar pressure at end-expiration is above atmospheric pressure : PEEP
- Extrinsic PEEP
- Auto PEEP

- Physiologic
- Optimal
- Best
- Best PEEP: Monitor Cardiac Output

  Another measure: Venous Oxygen Saturation

  If VOS decreases after PEEP applied= Drop CO

  Swan-Ganz catheter may be indicated in most patients on PEEP

Internal PEEP

External PEEP

Positive end-expiratory pressure

- CLINICAL USES:
- Reduce toxic levels of FiO2 (ARDS not pneumonia)
- Low-volume ventilation
- Obstructive lung disease (Extrinsic=Occult PEEP)

CLINICAL MISUSES:

- Reducing Lung Edema
- Routine PEEP
- Mediastinal Bleeding after CABG

PEEP

- What are the secondary effects of PEEP?
  - Barotrauma
  - Diminish cardiac output
  - Regional hypoperfusion
  - NaCl retention
  - Augmentation of I.C.P.?
  - Paradoxal hypoxemia

PEEP

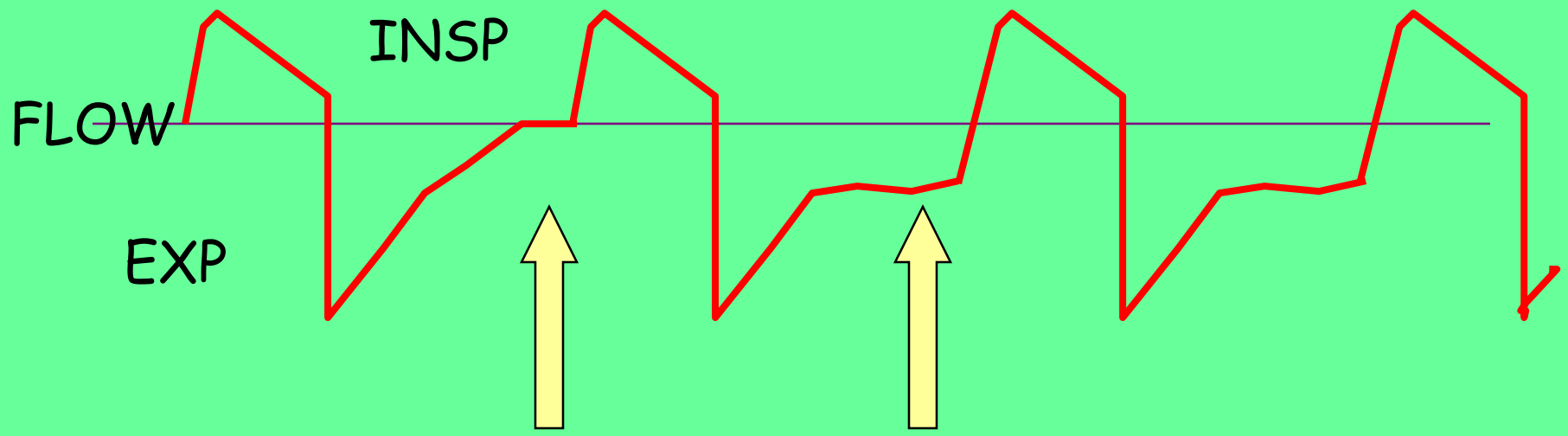
- Contraindication:
  - Barotrauma
  - Airway trauma
  - Hemodynamic instability
  - I.C.P.?
  - Bronchospasm?

Auto-PEEP

Auto-PEEP or Intrinsic PEEP

- Adverse effects:
  - Predisposes to barotrauma
  - Predisposes hemodynamic compromises
  - Diminishes the efficiency of the force generated by respiratory muscles
  - Augments the work of breathing
  - Augments the effort to trigger the ventilator

What is “Auto-Peep”?



Obstructive lung disease (pursed lip)

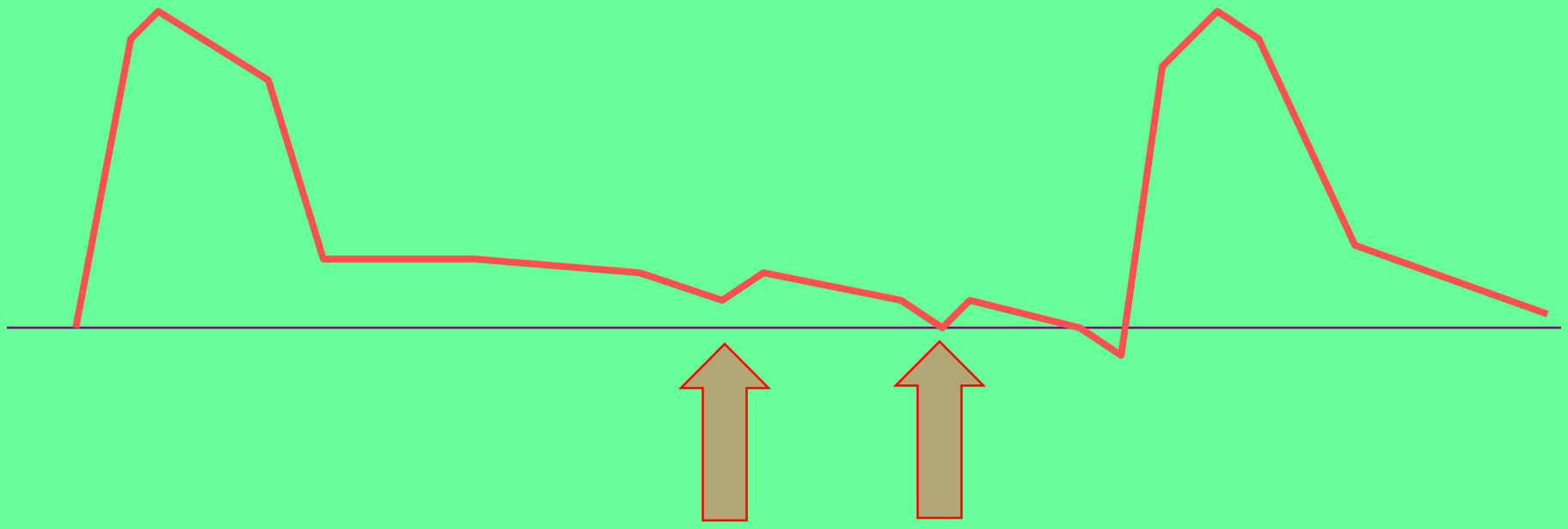
Rapid breathing (breath stacking)

Forced exhalation (anxiety)

What is “Auto-Peep”?

EXP
PAUSE

Another clue to Auto-PEEP



Failed Inspiratory Efforts

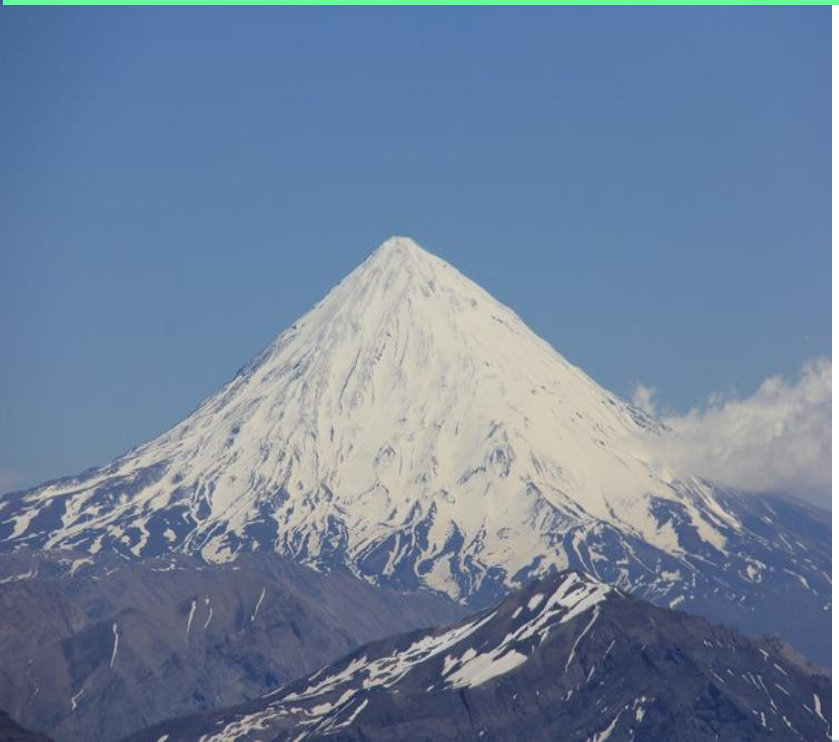
CPAP

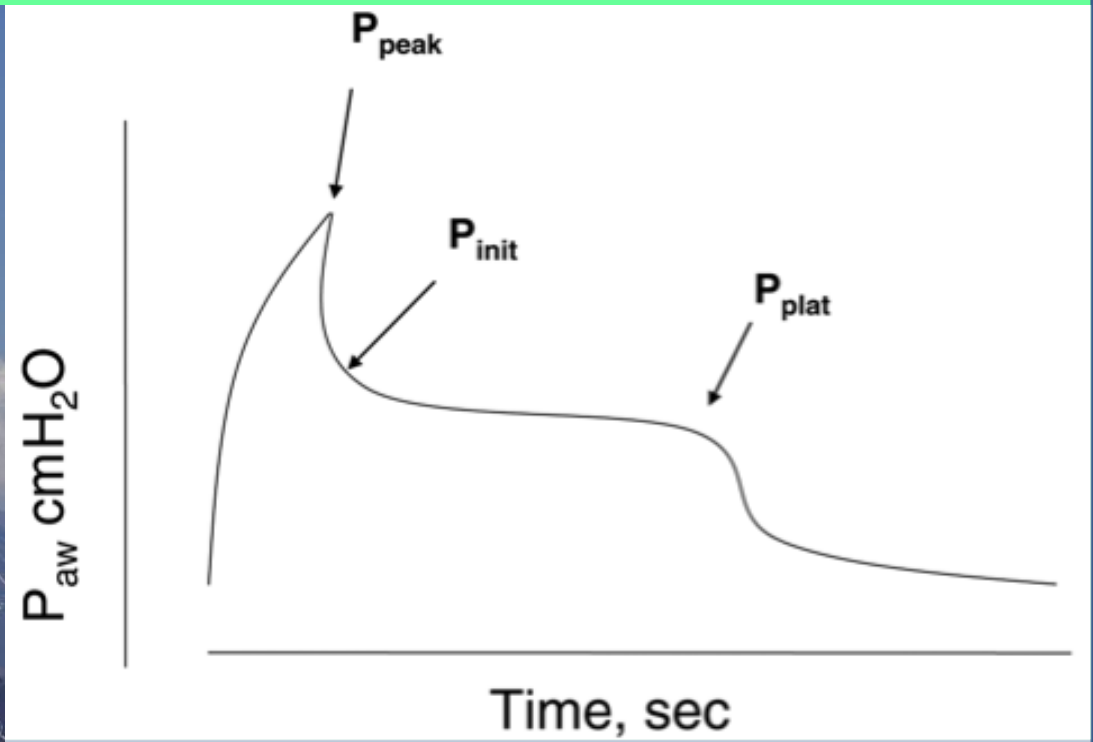
- Definition
  - Continuous positive airway pressure
  - Application of constant positive pressure throughout the spontaneous ventilatory cycle
- No mechanical inspiratory assistance is provided
  - Requires active spontaneous respiratory drive
- Same physiologic effects as PEEP

Peak Pressure (Ppeak)

- Ppeak = Pplat + Pres

Should not exceed 50cmH2O?





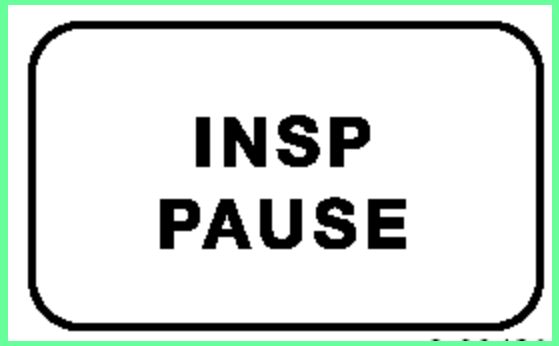
Compliance pressure (Pplat)

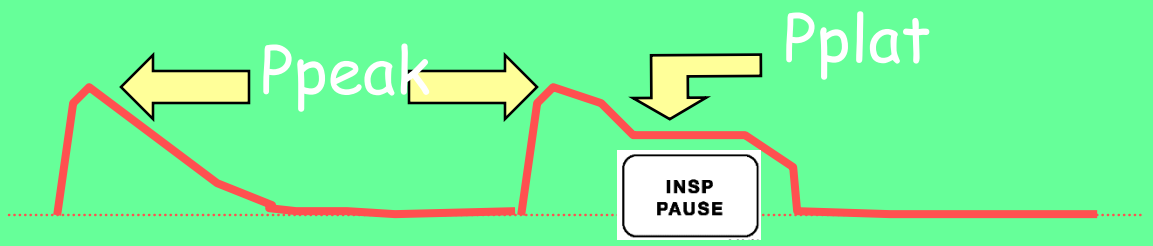
- Represent the static end inspiratory recoil pressure of the respiratory system, lung and chest wall respectively

- Measures the static compliance or elastance
- End inspiratory hold, important in lung protective strategy

Plateau Airway Pressure

- Normally Pplat = Ppeak – 5to10 mmHg
  - In what situations isn't that the case?
- Why are we more interested clinically in Pplat?

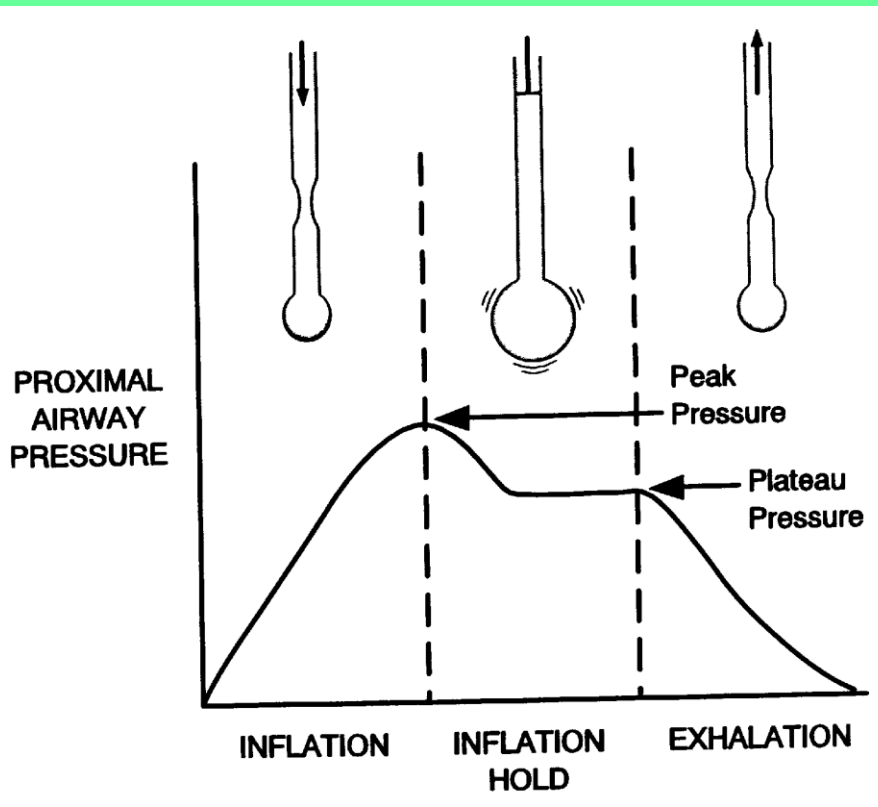




Puts a pause in the inspiratory cycle – no flow – measures pressure

Pplat

- Measured by occluding the ventilator 3-5 sec at the end of inspiration
- Should not exceed 30 cmH2O



T plat

T plat> 0.5 seconds improves oxygenation, but it may require patient sedation and leads to hemodynamic unstability

Monitoring Lung Mechanics

Use of Airway Pressures

Pk increased Pl unchanged

Tracheal tube obstruction

Airway obstruction from secretions

Acute bronchospasm

Rx: Suctioning and Bronchodilators

Use of Airway Pressures

Pk and Pl are both increased

Pneumothorax

Lobar atelectasis

Acute pulmonary edema

Worsening pneumonia

ARDS

COPD with tachypnea and Auto-PEEP

Increased abdominal pressure

Asynchronous breathing

Use of Airway Pressures

Decreased Pk

System air leak: Tubing disconnection, cuff leak

Rx: Manual inflation, listen for leak

Hyperventilation: Enough negative intrathoracic pressure to pull air into lungs may drop Pk.

Lung Compliance, Chest Wall Compliance

Total Compliance

$$C_T\ (L/cm\ H_2O) = \Delta V\ (L)/\Delta P\ (cm\ H_2O) \qquad (1)$$

The C_T of lung plus chest wall is related to the individual compliance of the lungs (C_L) and chest wall (C_{CW}) according to the following expression:

$$1/C_T = 1/C_L + 1/C_{CW} \qquad (2)$$
$$[\text{or } C_T = (C_L)(C_{CW})/C_L + C_{CW}]$$

Normaly , C l and C cw each equal 0.2 L/ cmH20

Compliance

Static Compliance (Cstat)

Distensibility of Lungs and Chest wall

Cstat = Vt/Pl

Normal C stat: 50-80 ml/cm of water

Provides objective measure of severity of illness in a pulmonary disorder

Dynamic Compliance

Cdyn: Vt/Pk

*Subtract PEEP from Pl or Pk for compliance measurement

Use Exhaled tidal volume for calculations

- To determine

  C l: dV, transpulmonary pressure gradient(P A- P pl)

  C cw: dV, transmural pressure gradient(P pl- P ambient)

  C t: dV, transthoracic pressure gradient(P A- P ambient), which can be done dynamically or statically.

The peak transthoracic pressure value is due to the pressure required to overcome both elastic and airway resistance.

Platue pressure is due to pressure required to gas distribution from stiff to more compliant alveoli(so it is less than peak pressure)

Static compliance

Vt /P plat-PEEP

Dynamic compliance

Vt / P peak-PEEP

Therefore C static is greater than C dynamic

Airway Resistance

For air to flow into the lungs, ΔP (pressure gradient) must also be developed to overcome the nonelastic airway resistance of the lungs to airflow. The relationship between ΔP and the rate of airflow ($\dot{V}$) is known as airway resistance (R):

$$R\ (\text{cm } H_2O / L / \text{sec}) = \frac{\Delta P\ (\text{cm } H_2O)}{\Delta \dot{V}\ (L / \text{sec})} \qquad (4)$$

Table 1 Monitoring strategies and targets for lung and diaphragm-protective ventilation

Parameter	Use	Advantages	Disadvantages	Suggested targets for lung and diaphragm-protective ventilation
Tidal volume (V_T)	Indirect surrogate marker of risk of ventilator-induced lung injury *Expired* tidal volume may be used to detect volumes delivered above set volume in volume-controlled mode	Readily available	Strain is quantified by V_T/EELV (end-expiratory lung volume), thus V_T alone is not a precise measure of lung strain Does not reflect lung stress and does not correct for "baby lung" size	V_T 4–8 ml/PBW
Airway driving pressure (ΔPaw)	Monitor lung stress and strain resulting from inflation with tidal volume	Readily available	Does not reflect regional lung stress when respiratory effort is high Overestimates the transpulmonary pressure (P_L) if chest wall elastance is increased and in the presence of expiratory muscle activity	ΔPaw < 15 cmH_2O
Paw and flow waveforms	Detect patient-ventilator dyssynchronies	Readily available Readily detects flow starvation, breath stacking, and premature cycling dyssynchronies	Some dyssynchronies may not be immediately evident without close inspection and additional monitoring of effort	Maintain patient-ventilator synchrony
Airway occlusion pressure ($P_{0.1}$)	Monitor respiratory drive and detect presence of low or high respiratory effort	Non-invasive Automated measurement available on most ventilators	Elevated respiratory drive does not always result in elevated respiratory effort (i.e., in the presence of respiratory muscle weakness or short inspiratory time)	$P_{0.1}$ 1–4 cmH_2O
Airway pressure swing during a whole breath occlusion (ΔPocc)	Assess for excessive respiratory effort and tidal lung stress	Non-invasive Easily measured at the bedside Can predict respiratory muscle effort (Pmus) and transpulmonary pressure swing ($\Delta P_{L,dyn}$) Detect apnea, auto-triggering Differentiate different forms of dyssynchrony	Though sensitive and specific for high respiratory effort and dynamic lung stress, the technique is not sufficiently accurate to replace direct measurement	Predicted Pmus 5–10 cmH_2O (ΔPocc 8–20 cmH_2O) Predicted $\Delta P_{L,dyn}$ < 15–20 cmH_2O
Esophageal pressure (Pes) and transpulmonary pressure (P_L)	Directly measure and monitor respiratory effort and tidal lung stress	Minimally invasive Provides gold standard information about lung stress (ΔP_L) and respiratory effort (ΔPes, PTPes)	Requires equipment and training Balloon must be calibrated before each measurement Absolute values of Pes of unclear utility	ΔPes 3–15 cmH_2O (diaphragm protective) $\Delta P_{L,dyn}$ < 15–20 cmH_2O (lung protective)
Transdiaphragmatic pressure swing (ΔPdi) and gastric pressure swing (ΔPga)	Directly measure and monitor diaphragmatic effort and expiratory effort	Minimally invasive Provides direct measurement of diaphragmatic effort Provides information about expiratory muscle activity	Requires equipment and training Balloon must be calibrated before each measurement No calibration for Pga Difficult to assess post-inspiratory effort (eccentric loading)	ΔPdi–15 cmH_2O

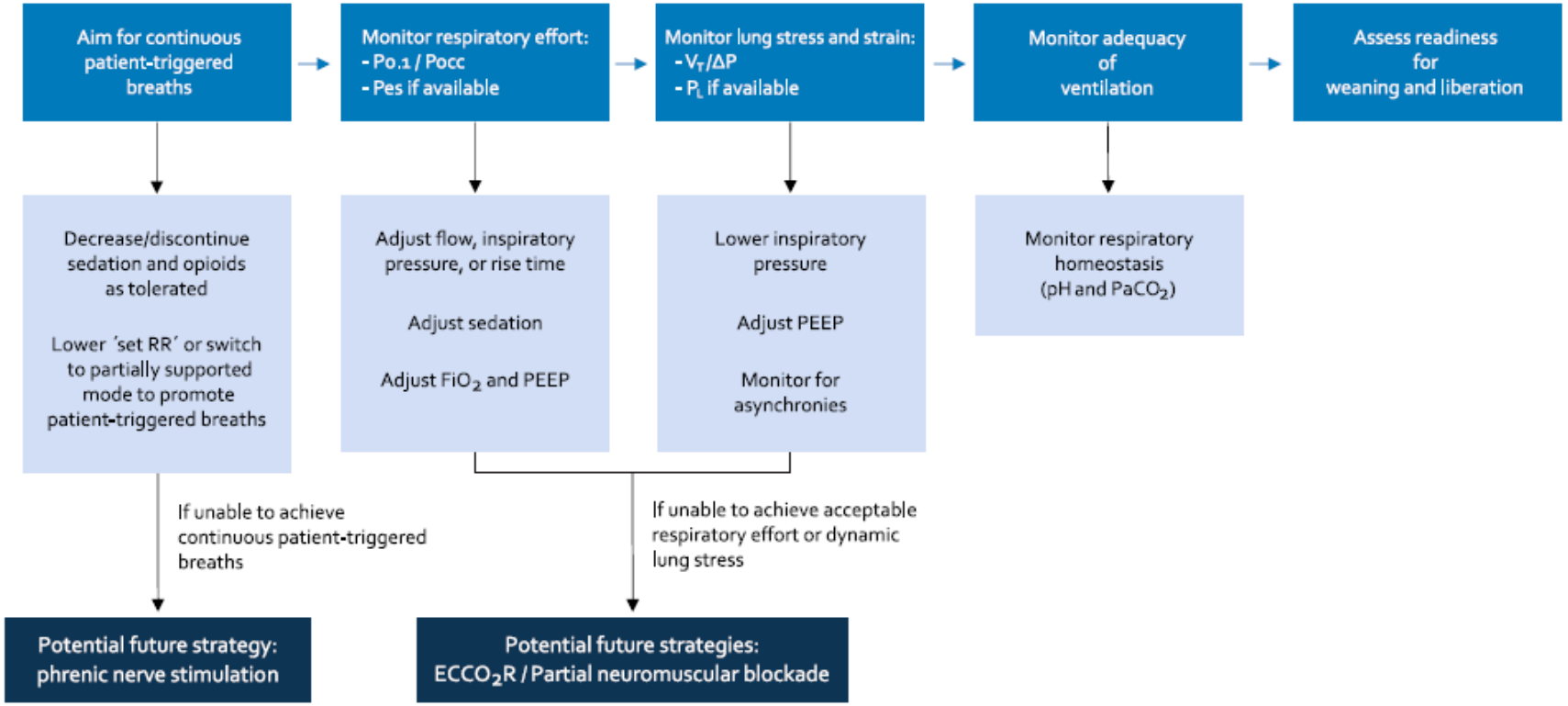


Fig. 3 Clinical-physiological pathway for achieving lung and diaphragm-protective ventilation targets. It should be stressed that at each step clinical evaluation of the patient, including signs of high breathing effort, agitation, and over-sedation is of major importance and should be interpreted together with clinical-physiological measurements as outlined in this pathway. ΔP: change in airway pressure during inspiration; $P_{0.1}$: decrease in airway pressure during the first 100 ms of inspiratory effort against an occluded airway; $PaCO_2$: arterial carbon dioxide tension; PEEP: positive end-expiratory pressure; Pes: esophageal pressure; P_L: transpulmonary pressure; Pocc: airway pressure deflection during a whole breath occlusion; RR: respiratory rate; V_T: tidal volume

Dyssynchrony

- Dyssynchronies may occur during inspiration (flow starvation,short cycles, prolonged insufflation and reverse triggering), during expiration (auto-triggering, ineffective effort) or both during inspiration and expiration (reverse triggering and double triggering).

Reverse triggering

- A diaphragmatic contraction triggered by mechanical inflation, is common in fully sedated patients (in whom drive is abolished).
- Reverse triggering can induce breath stacking resulting in excessive tidal volumes and high dynamic lung stress, and it may create eccentric diaphragm loading conditions with resultant muscle injury.
- When necessary to avoid breath stacking, reverse triggering can be abolished by neuromuscular blocking agents.
- Alternatively, the development of reverse triggering may indicate that sedation should be stopped to allow the patient to take control of ventilation

- In patients with relatively high respiratory drive and a low respiratory system time constant, the neural inspiration time may exceed the mechanical inflation (*premature cycling*). In such cases, the contraction of the inspiratory muscles continues during mechanical expiration and the diaphragm is forced to contract while lengthening (eccentric contraction).
- In volume-targeted modes, unmet high demands appear as ‘flow-starvation’, a downward curvature of inspiratory Paw, and the patient may experience dyspnea and distress, which can be resolved by increasing inspiratory flow rate using a decelerating flow pattern.
- Strong inspiratory efforts may result in *double-triggering*, *breath stacking* and, therefore, delivery of high Vt

- A better match of mechanical and neural inspiratory time can be achieved by increasing ventilator inspiratory time and using a decelerating flow pattern in volume-assist control mode, by decreasing the cycling-off criterion in pressure support mode, or using proportional modes of assist.
- Importantly, in patients with high respiratory drive, modification of inspiratory time may not suffice to resolve dyssynchrony.
- Increasing the level of assist to match the patient's demands should be considered, but, if that results in an injurious high ventilation, other means to decrease the patient's respiratory drive, such as sedation, may be required.

Auto-triggering

- The delivery of a ventilator-assisted breath in the absence of patient effort.
- Auto-triggering due to strong cardiac oscillations transmitted to the Paw or airflow signal is more likely to occur when the respiratory system time constant is low, such as in ARDS. Air leaks and moisture in the ventilator circuit are also common causes of auto-triggering

Ineffective triggering

- is generally the consequence of

1-weak inspiratory efforts, either from low respiratory drive due to sedation, metabolic alkalosis or excessive ventilatory assist

2-diaphragm weakness. When the respiratory system time constant is high, (i.e., COPD), ventilator over-assistance results in delayed cycling, dynamic hyperinflation, and increased iPEEP, predisposing to ineffective triggering. Decreasing the level of assist can therefore alleviate ineffective efforts.

MAP

- Affected by PIP, PEEP, total cycle time and RR.
- MAP= ½(PIP-PEEP)X(Ti/TCT)+PEEP
- To assess the benefit and side effects of PPV

RC

- Clinical application
- Fast alveoli: short time constant(fast filling)
- Pulmonary fibrosis(low comp and low Res)

- Slow alveoli: long time constant(slow filling)
- Asthma(high comp and high Res)

Flow Patterns

- Laminar: with flow less than critical velocity
- Turbulant: with flow more than critical velocity
- Orifice :at severe constriction such as a nearly closed larynx or a kinked endotracheal tube

Types of flow

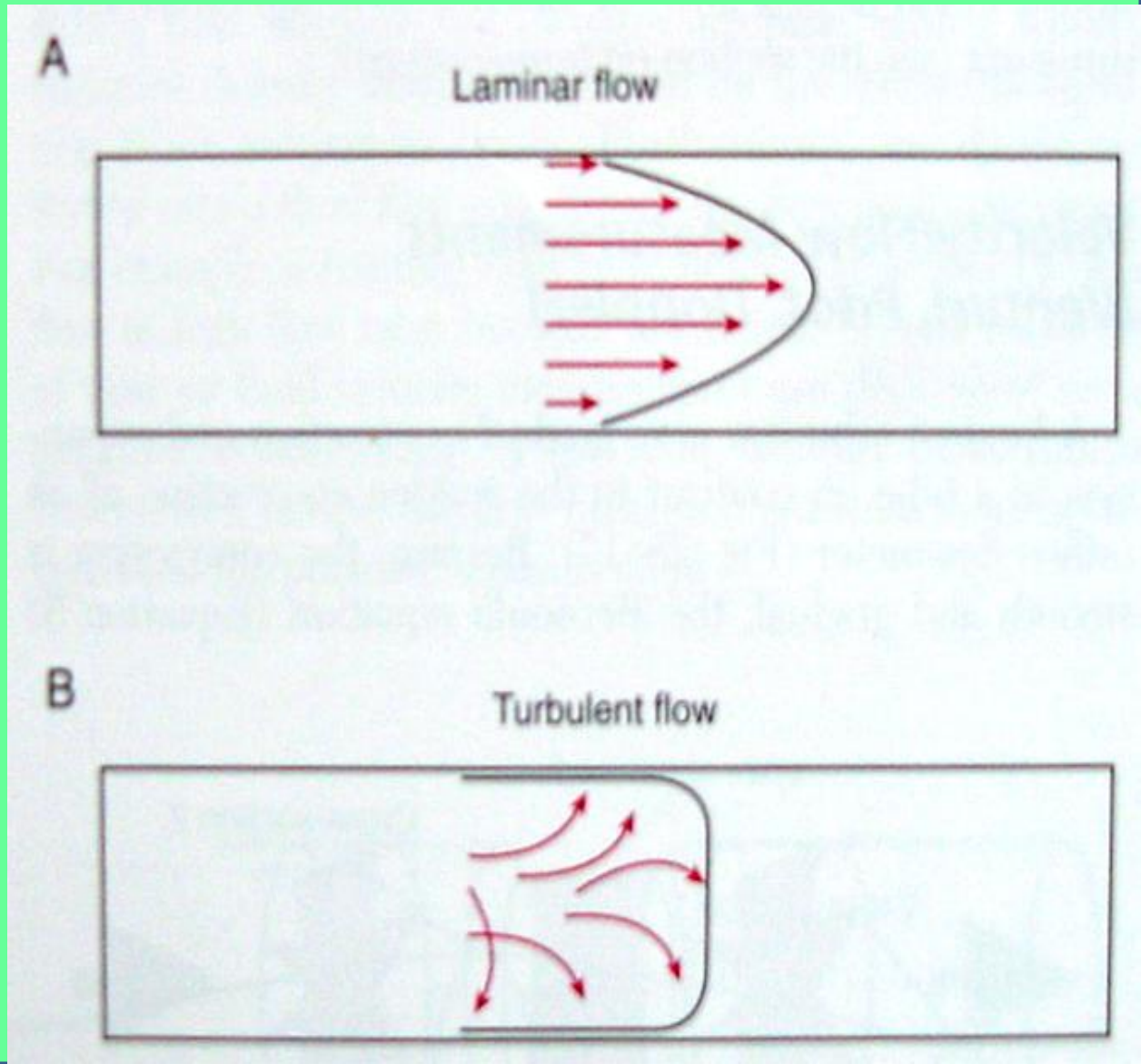
1-Laminar

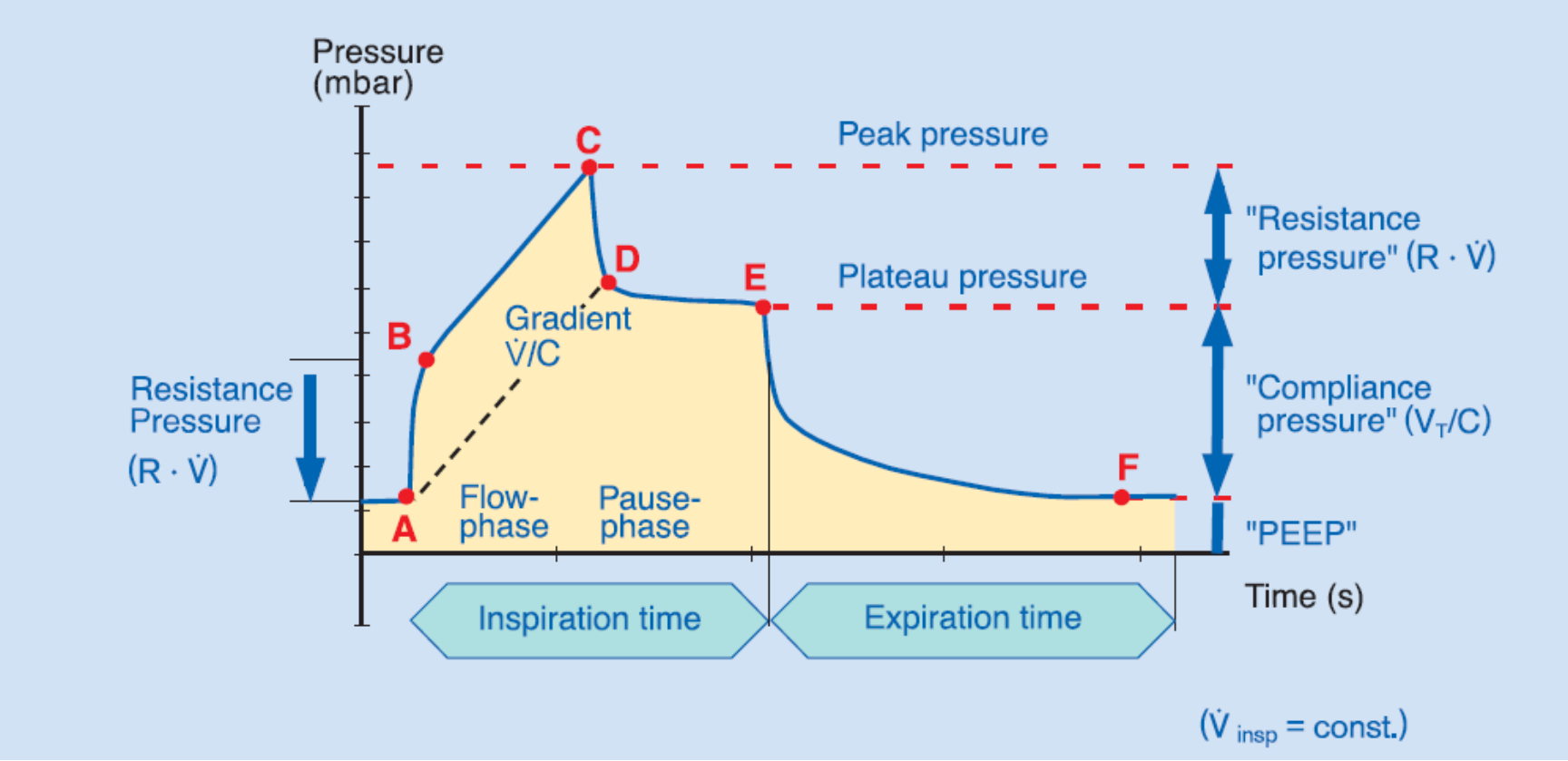
$Q=\pi pr4/8\eta l$

Hagen-poiseuille

2-Turbulent

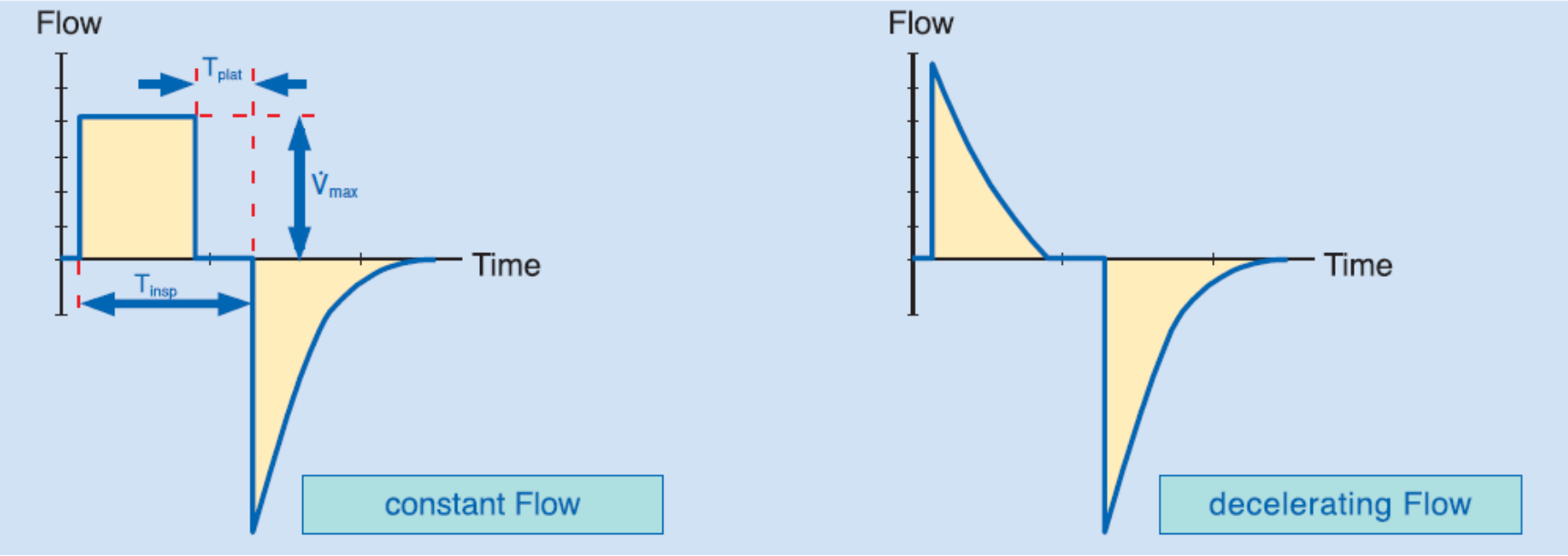
$Q2=4\pi 2pr5/\rho fl$

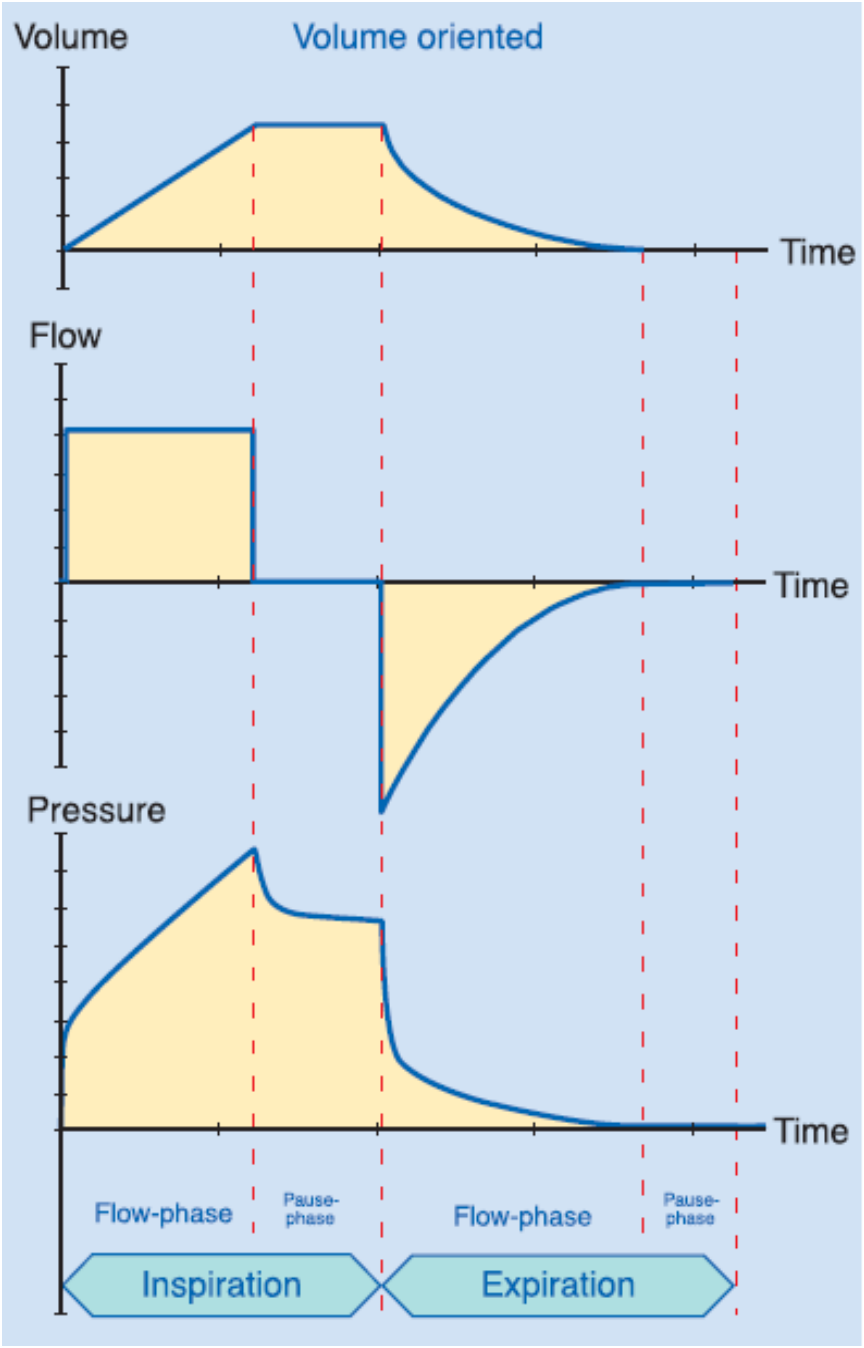


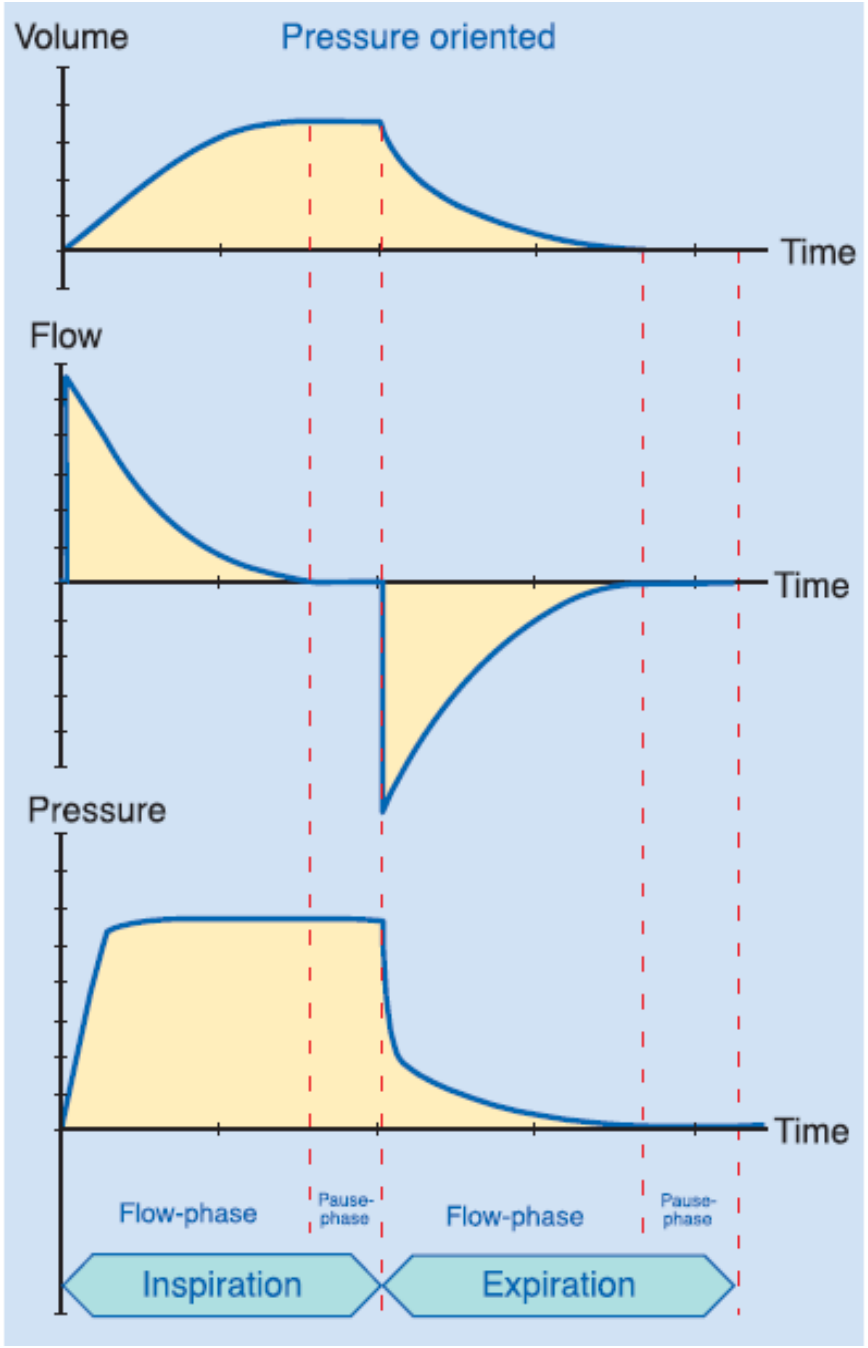


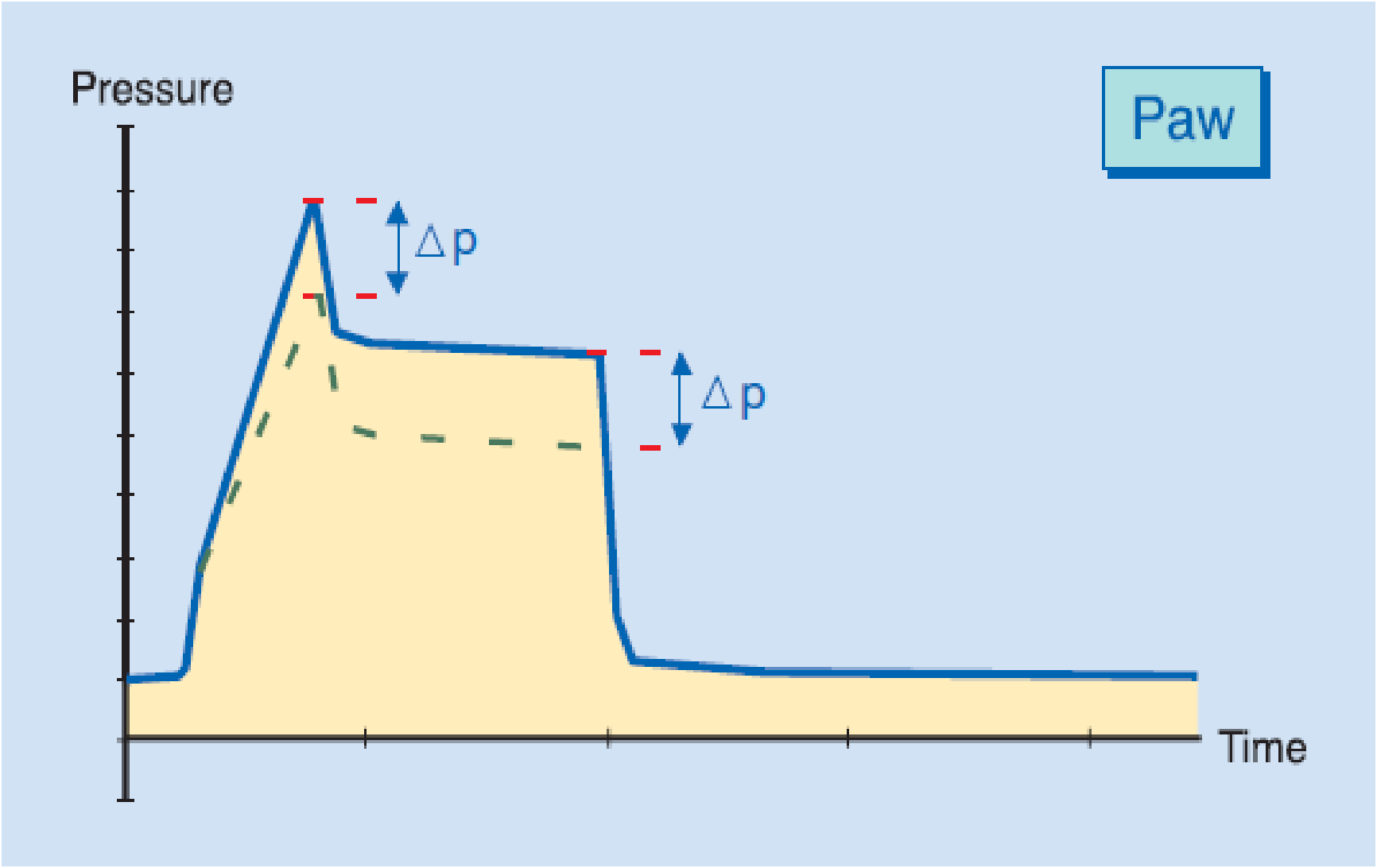
At the beginning of inspiration the pressure between points A and B increases dramatically on account of the resistances in the system. The level of the pressure at break point B is equivalent to the product of resistance R and flow. This relationship, as well as the following examples, is only valid if there is no intrinsic PEEP. The higher the selected Flow or overall resistance R, the greater the pressure rise up to point B. Reduced inspiratory flow and low resistance values lead to a low pressure at point B.

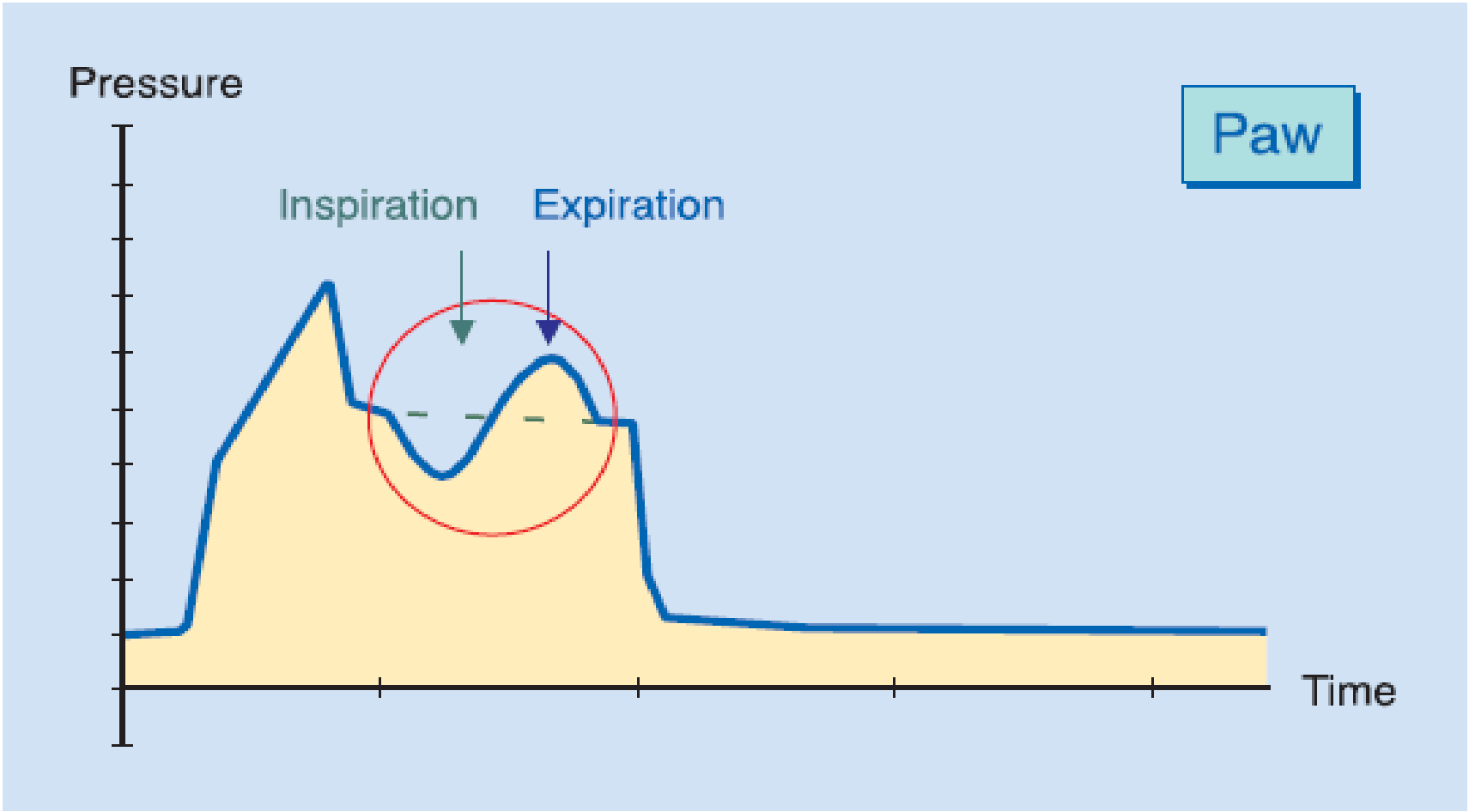
Flow time


Flow
T_{plat}
$\dot{V}_{max}$
Time
T_{insp}
constant Flow
Flow
Time
decelerating Flow

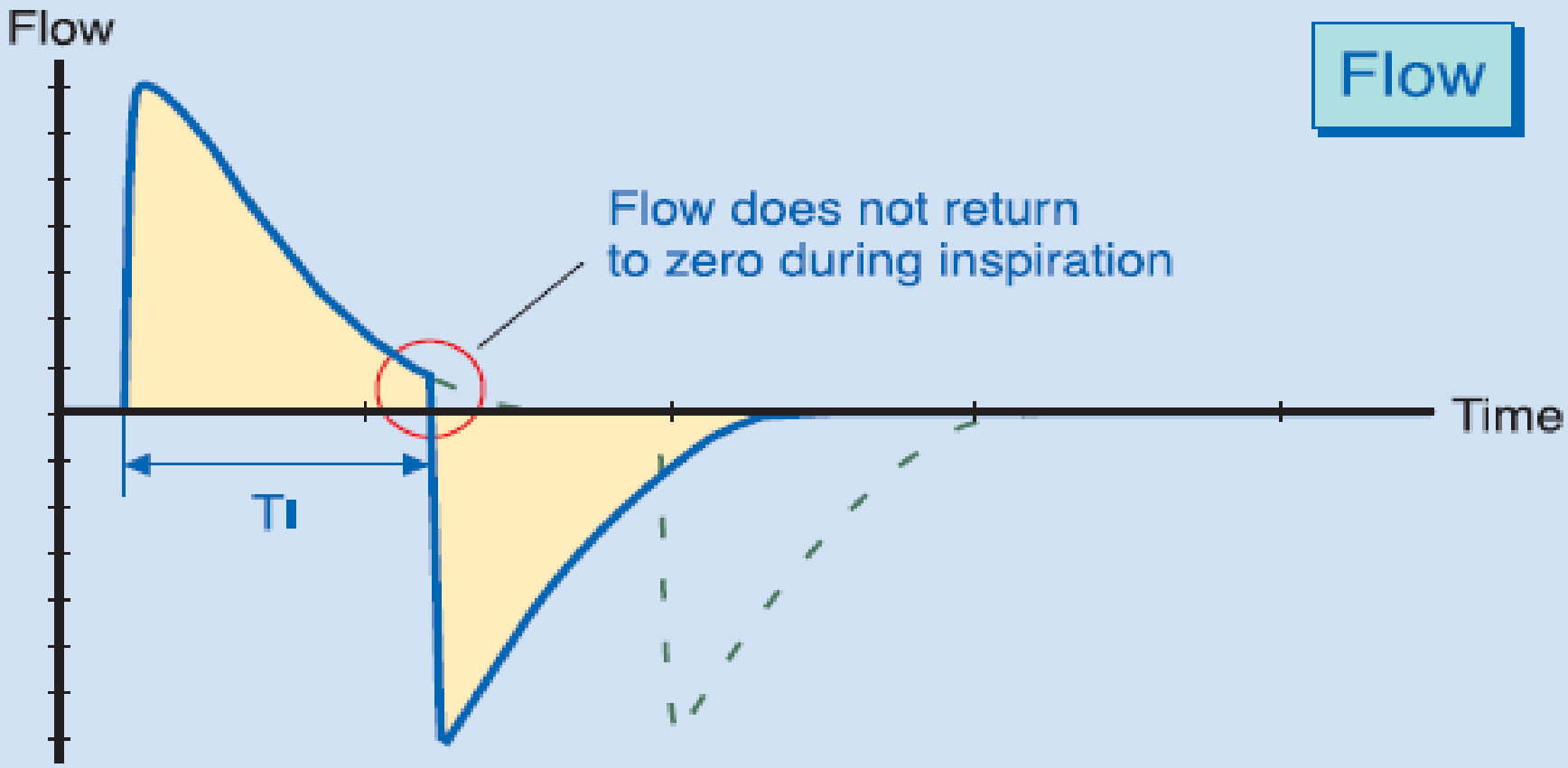

Volume
Volume oriented
Time
Flow
Time
Pressure
Time
Flow-phase
Pause-phase
Flow-phase
Pause-phase
Inspiration
Expiration


Volume
Pressure oriented
Time
Flow
Time
Pressure
Time
Flow-phase
Pause-phase
Flow-phase
Pause-phase
Inspiration
Expiration

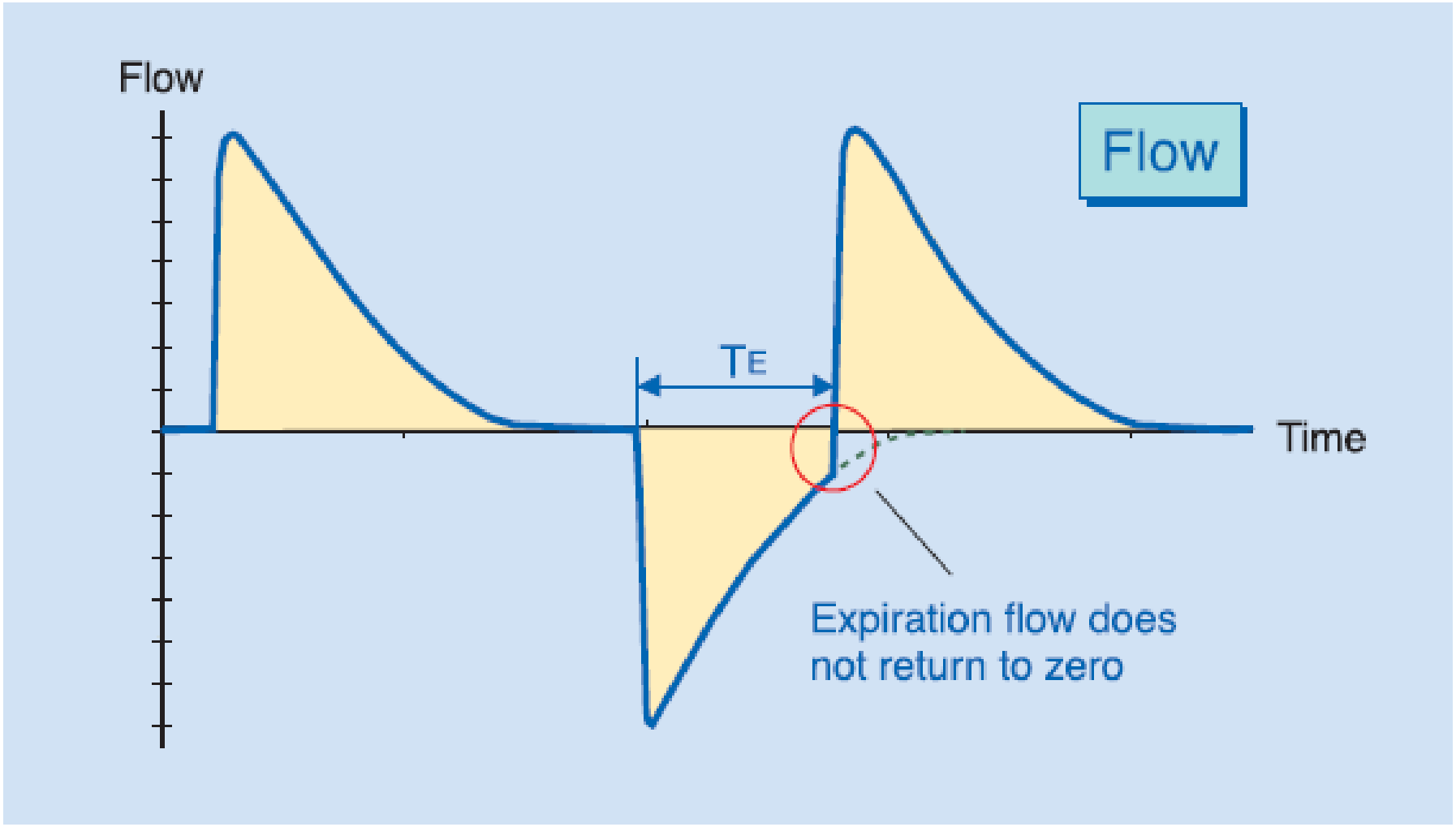

Pressure
Paw
Δp
Δp
Time



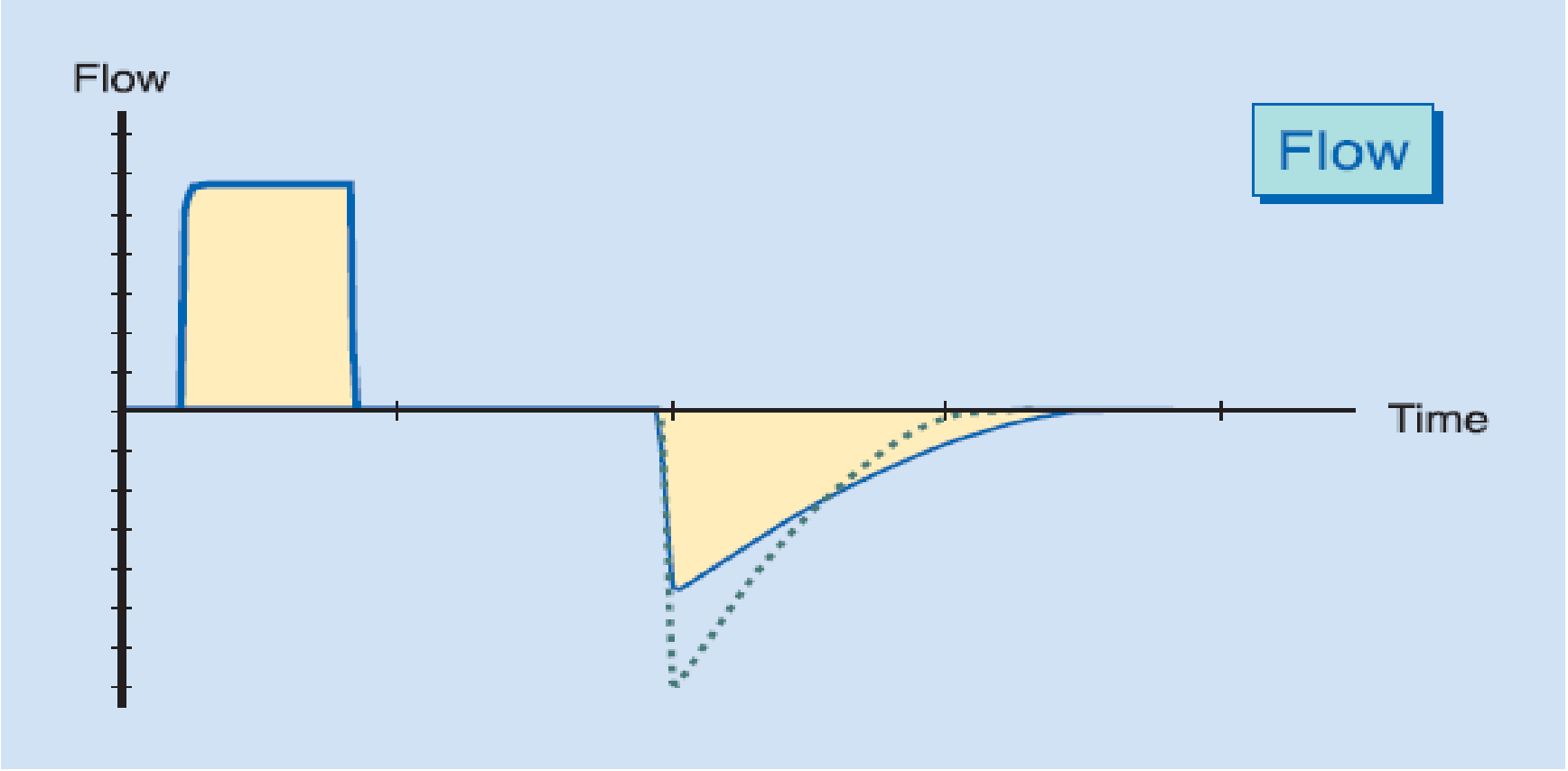
Spontaneous breathing



The flow curve in the case of insufficient inspiration time

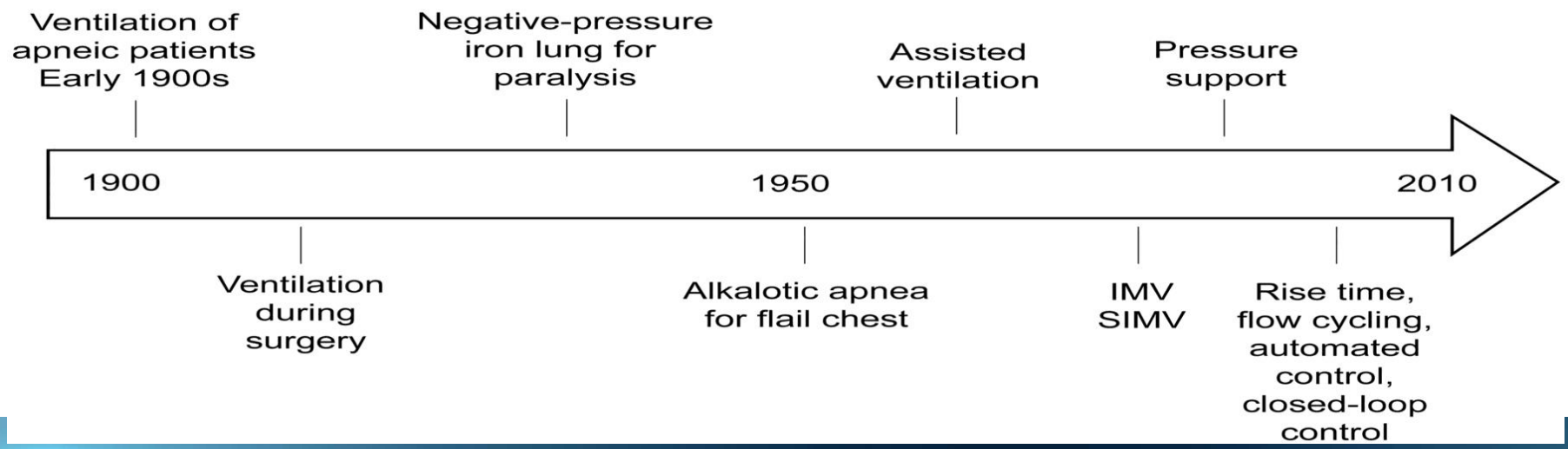


The flow curve in the case of insufficient expiration time



Flow curve in the case of increased expiratory resistances

A more gentle expiratory flow curve indicates increased expiratory resistances which may be caused by expiratory filters which have become damp or blocked as a result of nebulization.


Ventilation of apneic patients Early 1900s
Negative-pressure iron lung for paralysis
Assisted ventilation
Pressure support
1900
1950
2010
Ventilation during surgery
Alkalotic apnea for flail chest
IMV SIMV
Rise time, flow cycling, automated control, closed-loop control


Thank You