CACCN Dynamics September 2013
Halifax, NS
Mechanical Ventilation Workshop
Saturday Sept. 21

Kathy Johnson RRT

Coordinator Simulation Program IWK

Clinical Leader IWK Life Flight team

Noel Pendergast RRT MPH

Faculty, Respiratory Therapy

Dalhousie School of Health Sciences

Agenda

- 0800-1000
  - Welcome / Introductions
  - Lecture/Group discussions
- ***1000-1030***
  - ***Coffee break***
- 1030-1200
  - Lecture / Group discussions
- 1200-1230
  - Test yourself quiz
- ***1230-1300 Lunch***
- 1300-1315
  - Orientation to case study stations
- 1315-1345
  - Station #1
- 1345-1415
  - Station #2
- 1415-1445
  - Station #3
- ***1445-1500 Break***
- 1500-1530
  - Station #4
- 1530-1600 Wrap up

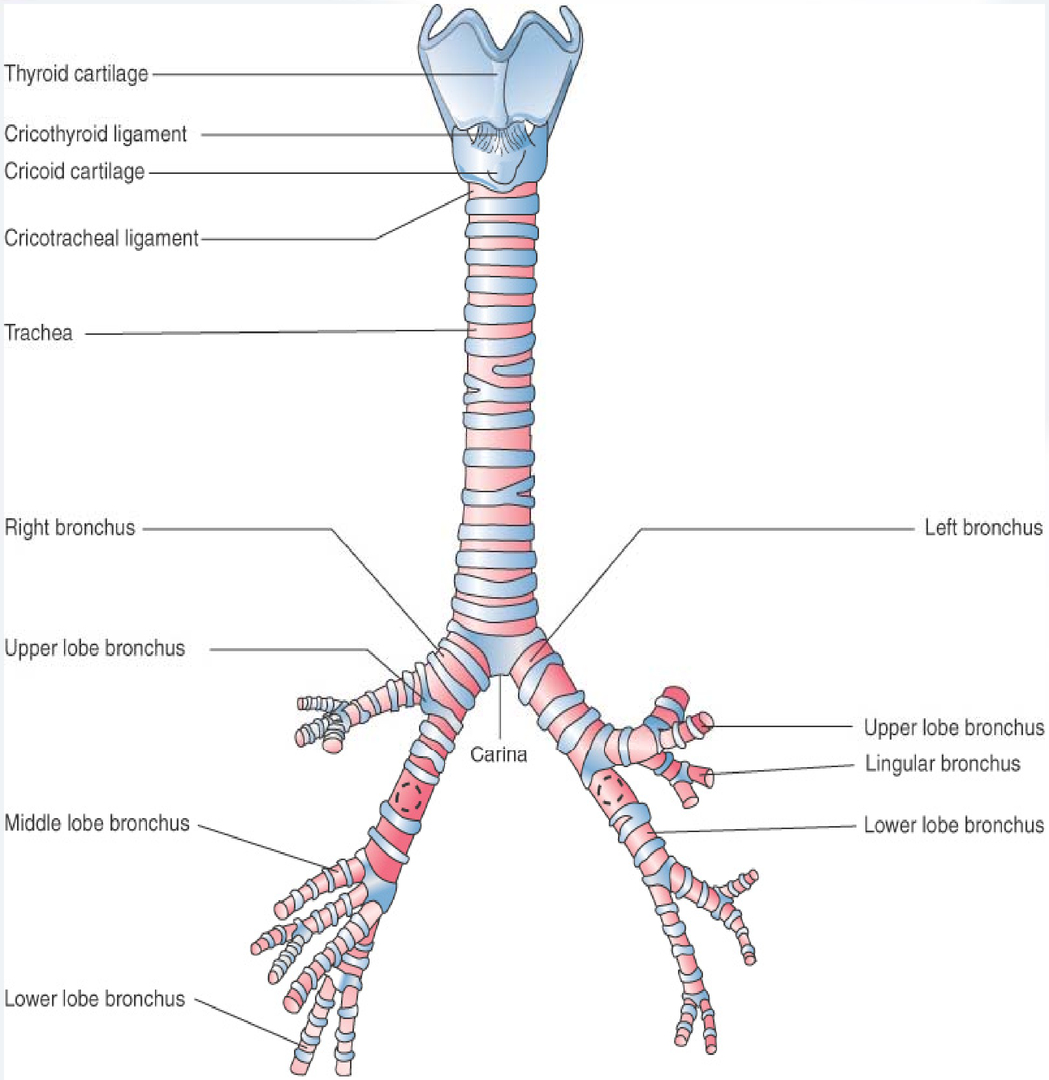
Objectives

1. Review respiratory physiology and pathophysiology
2. Review basic ventilator types, modes and waveforms
3. Discuss appropriate action when dealing with ventilator alarm situations
4. Discuss advanced mode of positive pressure ventilation (PPV) and their application with hypoxemic and hypercapnic respiratory failure
5. Apply learning to clinical problems with interactive case studies.

Respiratory Physiology

- Conducting Airway Zone vs Respiratory Gas Exchange Zone
- Alveolar-Capillary (A-C) membrane
- Ventilation: Perfusion relationship
- $\uparrow$ Dead-space (Vd/Vt)
- $\uparrow$ Shunt (Qs/Qt)

Respiratory Physiology



(From Hicks GH: Cardiopulmonary anatomy and physiology, Philadelphia, 2000, WB Saunders.)

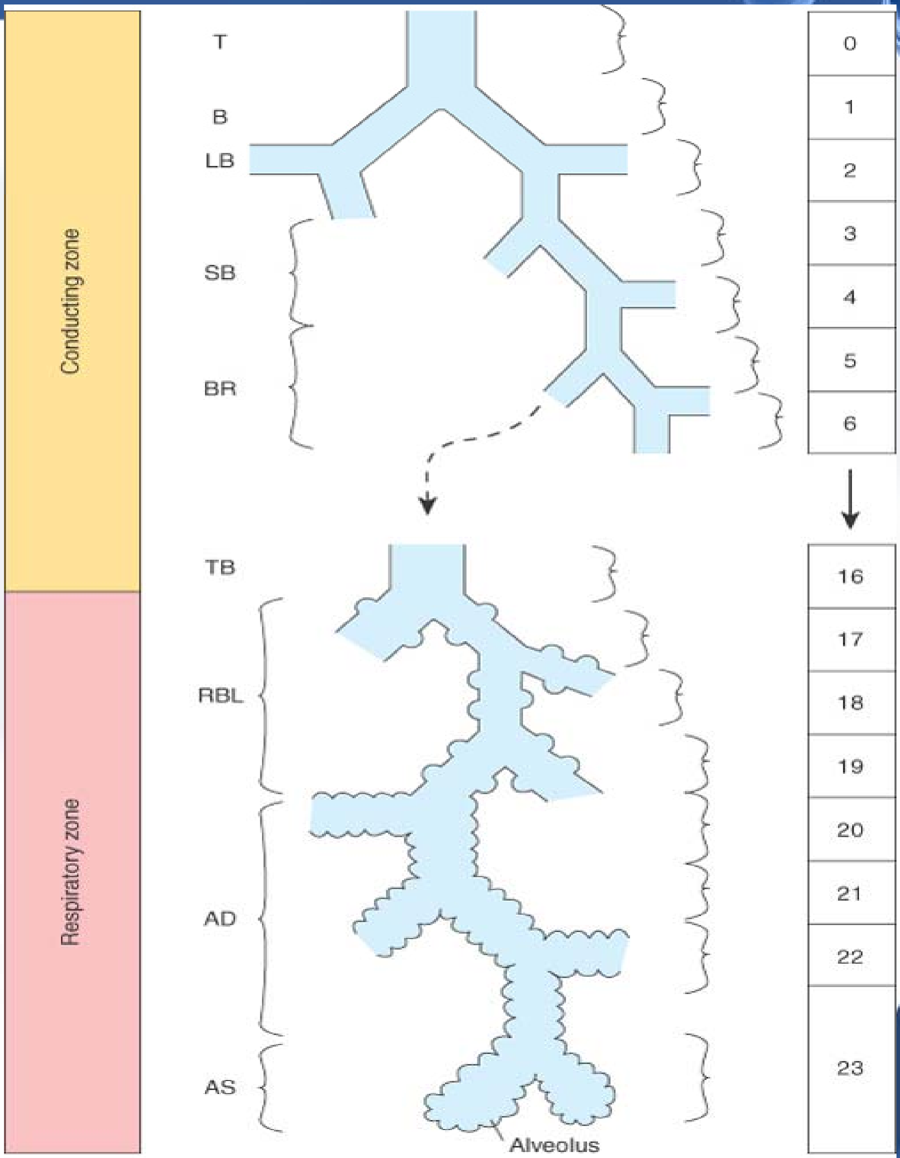
- Conducting or **Airway** Zone
- Trachea> Bronchi > Bronchioles
- Resistance (Raw) of the lungs
-

Respiratory Gas Exchange Zone

Respiratory bronchioles >

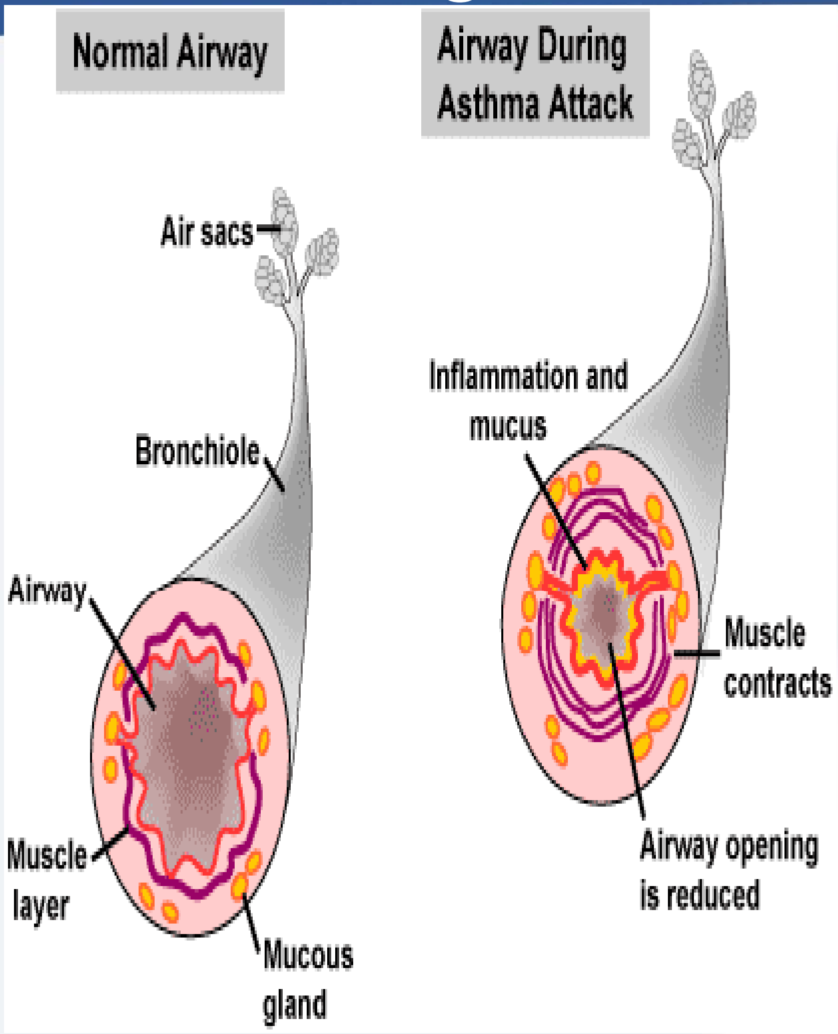
Alveolar ducts >

Alveolar sacs

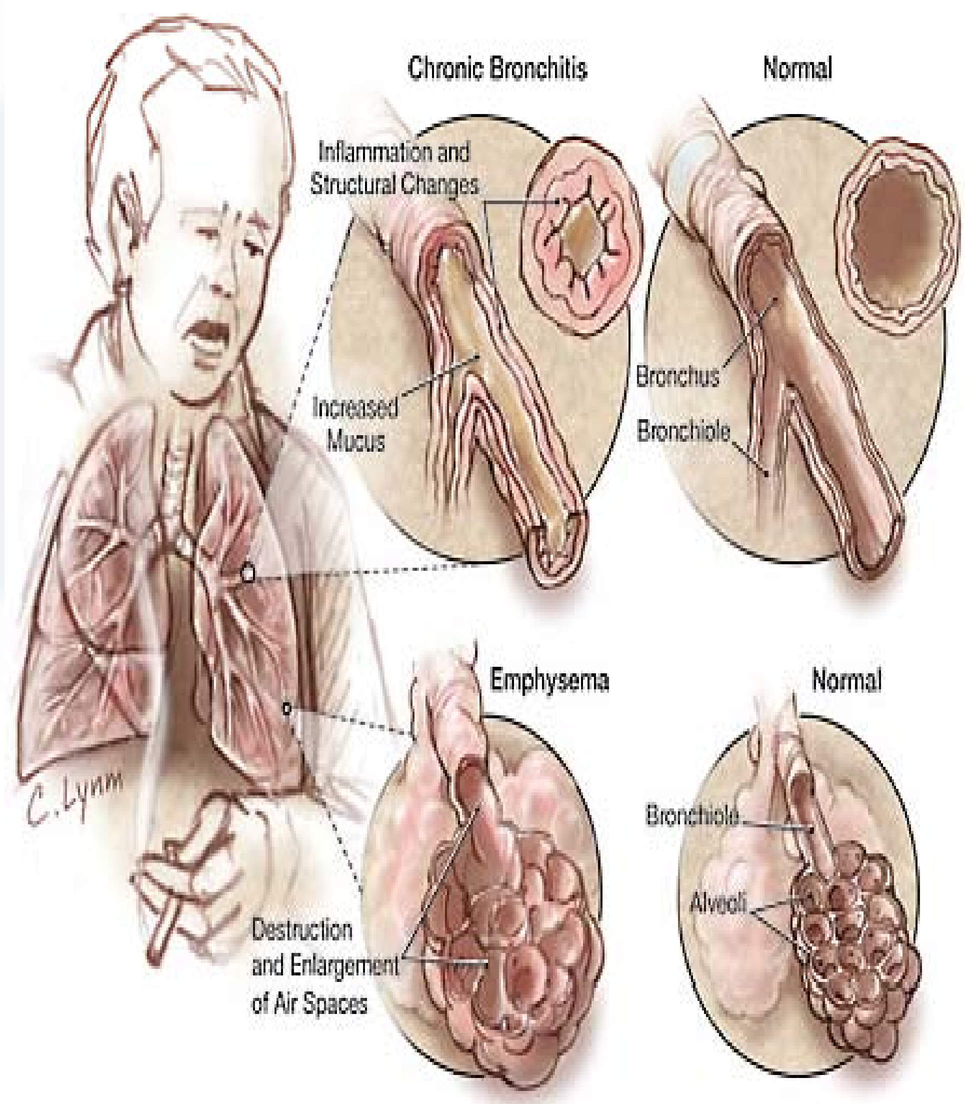


(From Hicks GH: Cardiopulmonary anatomy and physiology, Philadelphia, 2000, WB Saunders.)

Respiratory Diseases that effect the Conducting Zone



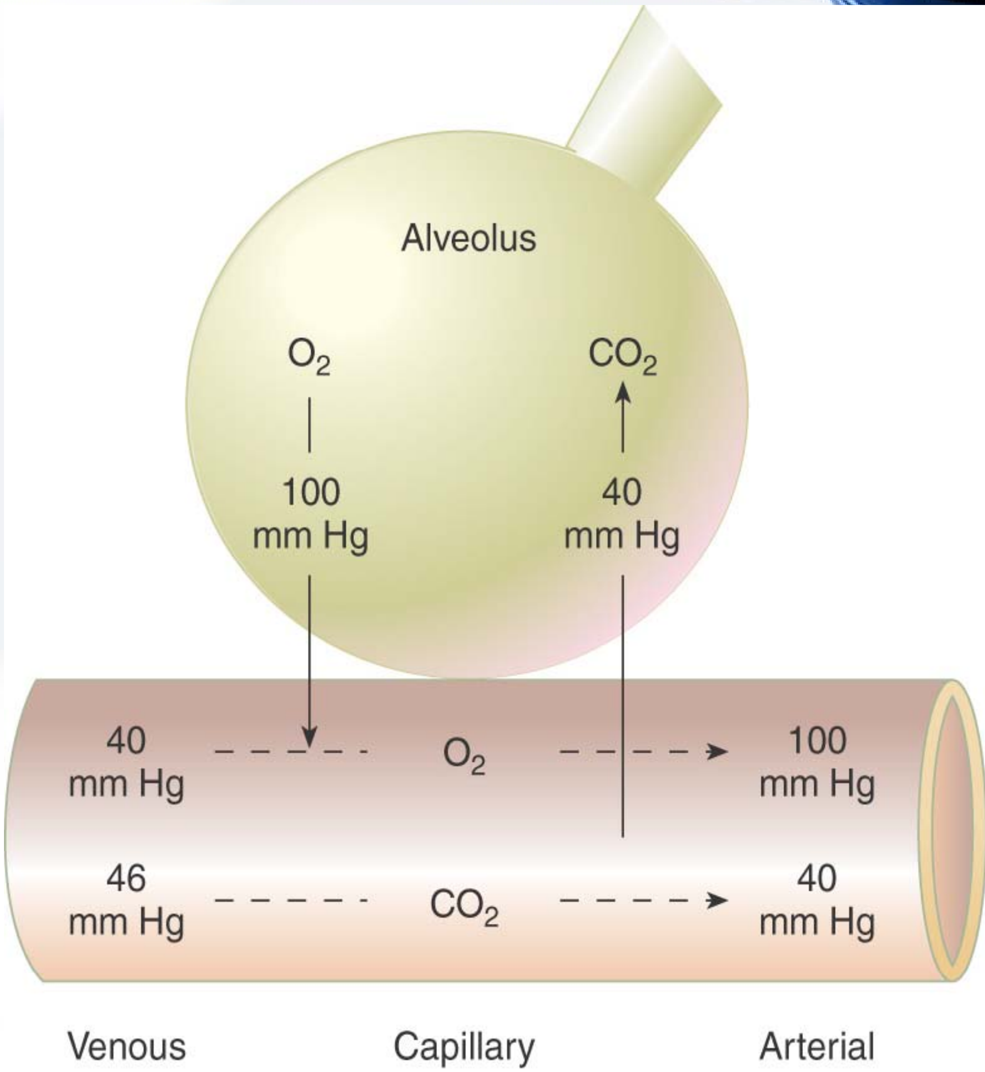
ASTHMA



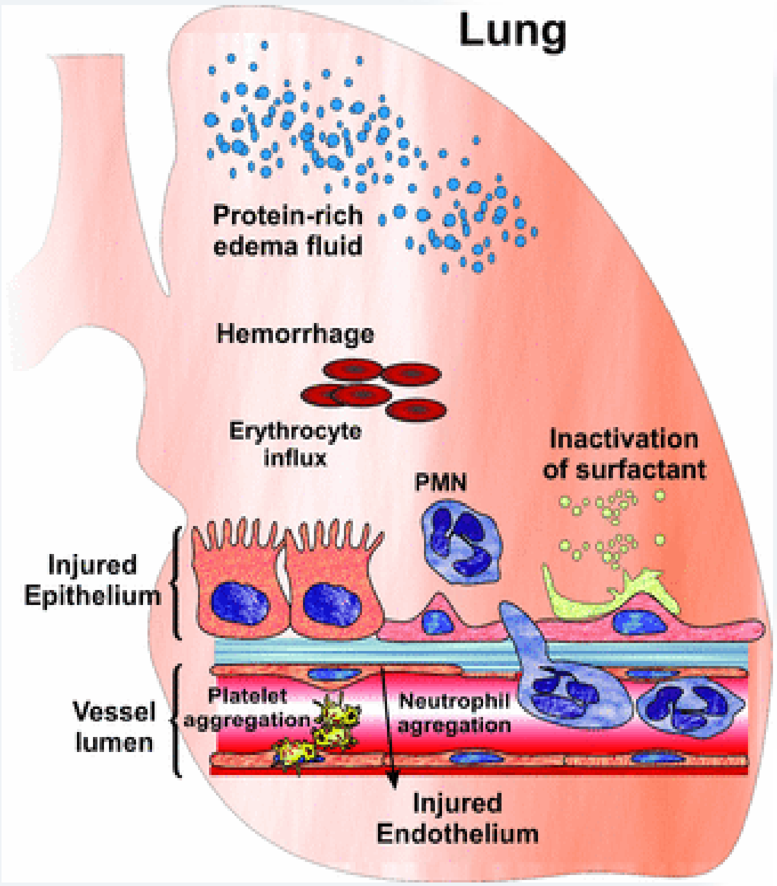
COPD

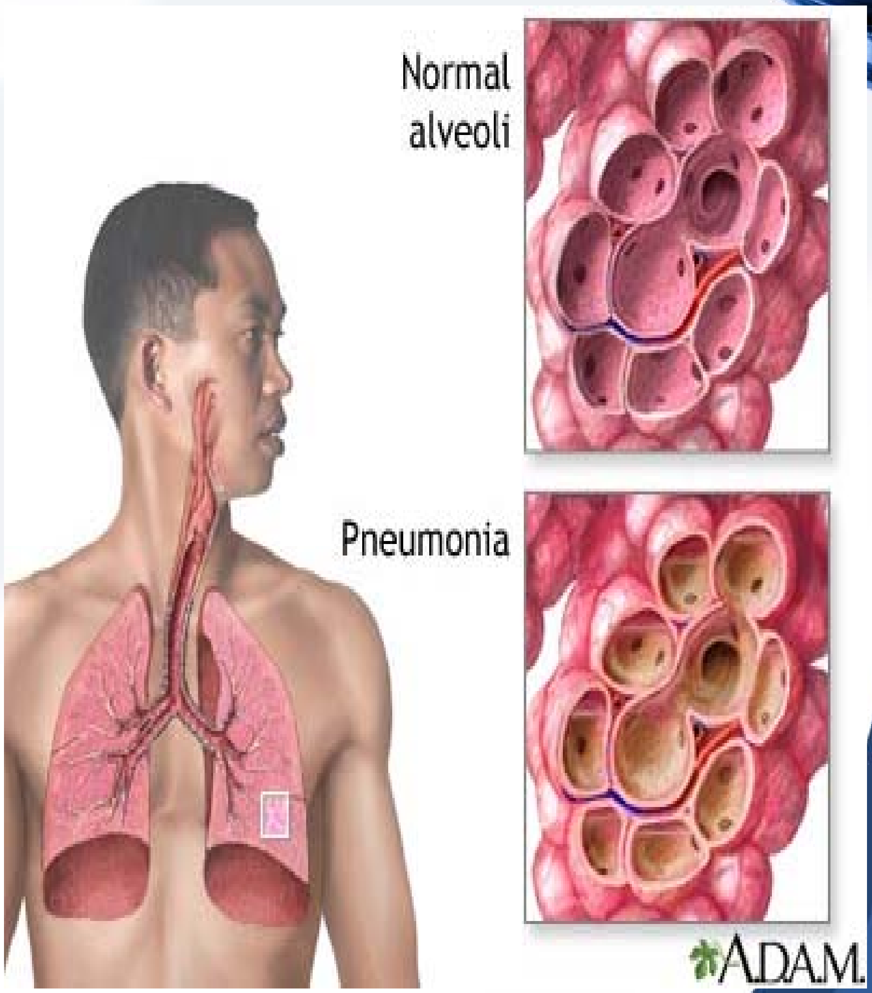
Respiratory Gas Exchange Zone

- Diffusion of gases across the Alveolar-Capillary membrane
- ***"A-C membrane"***
- **0_2** and **$C0_2$** go down their pressure gradients to exchange across the membrane



Respiratory diseases that effect the Respiratory Zone

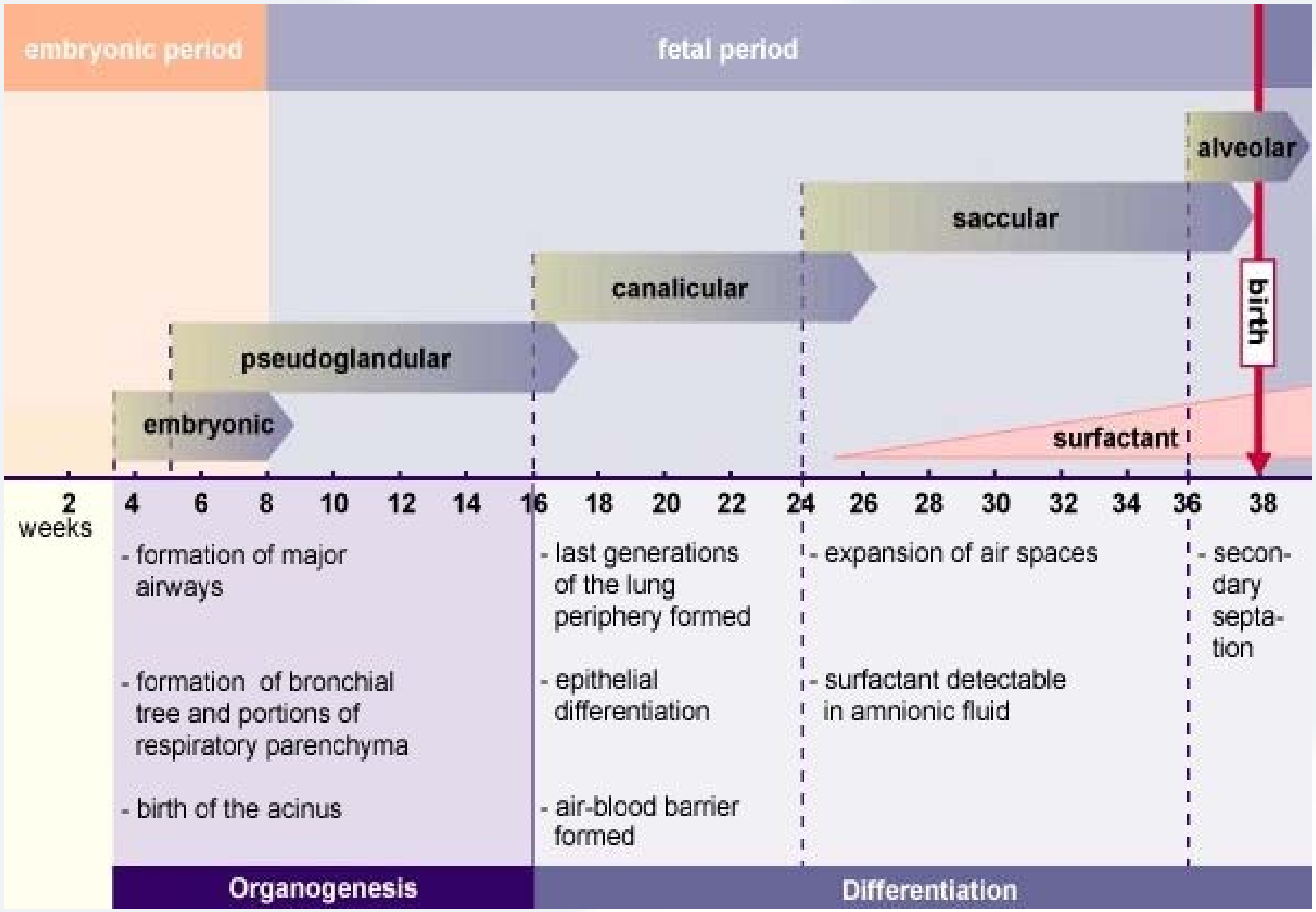




ARDS

PNEUMONIA

Respiratory Physiology – Lung Development



Respiratory Failure – Types

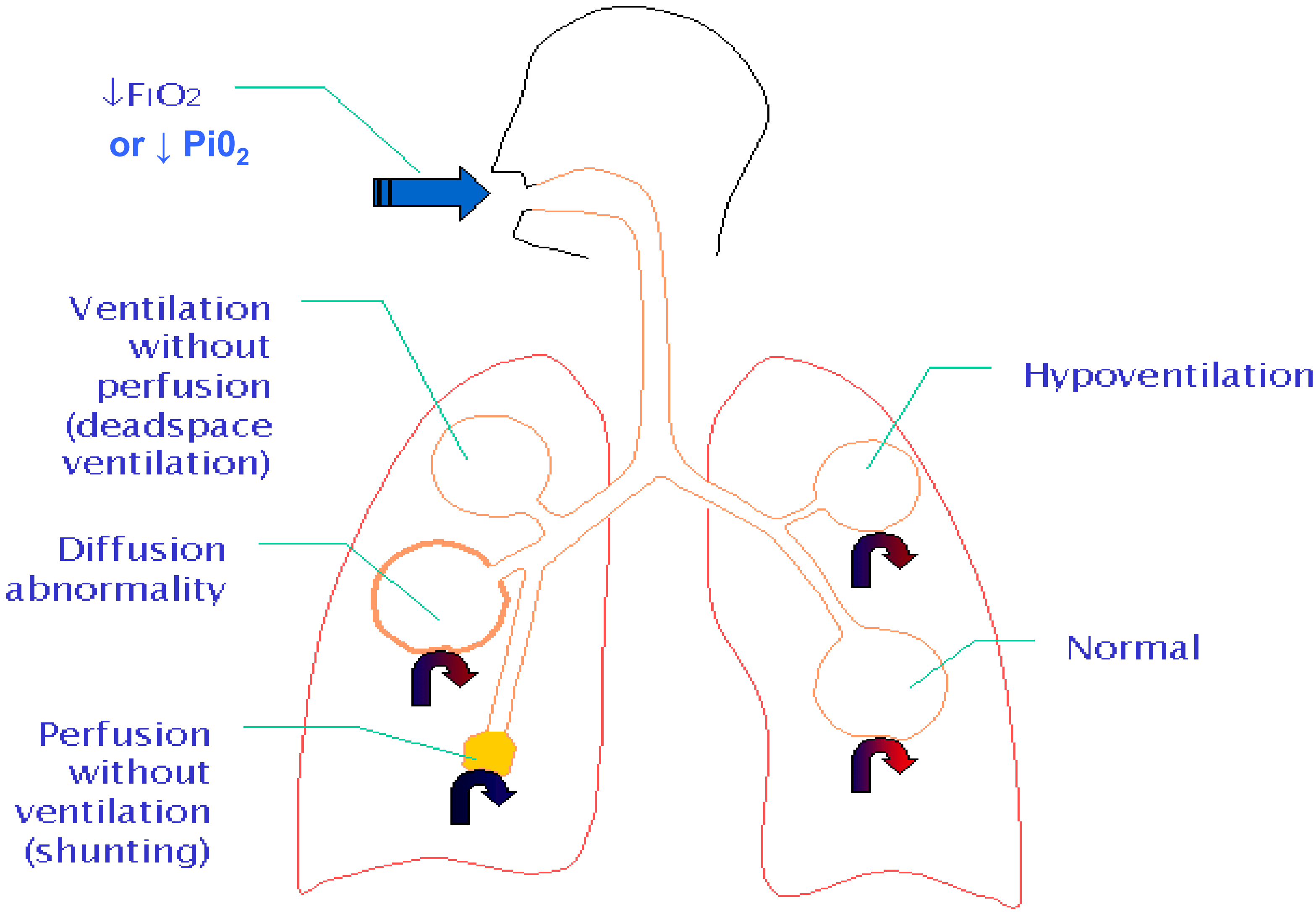
Type I

- **Hypoxemic**
- **Normal or ↓ $C0_2$**

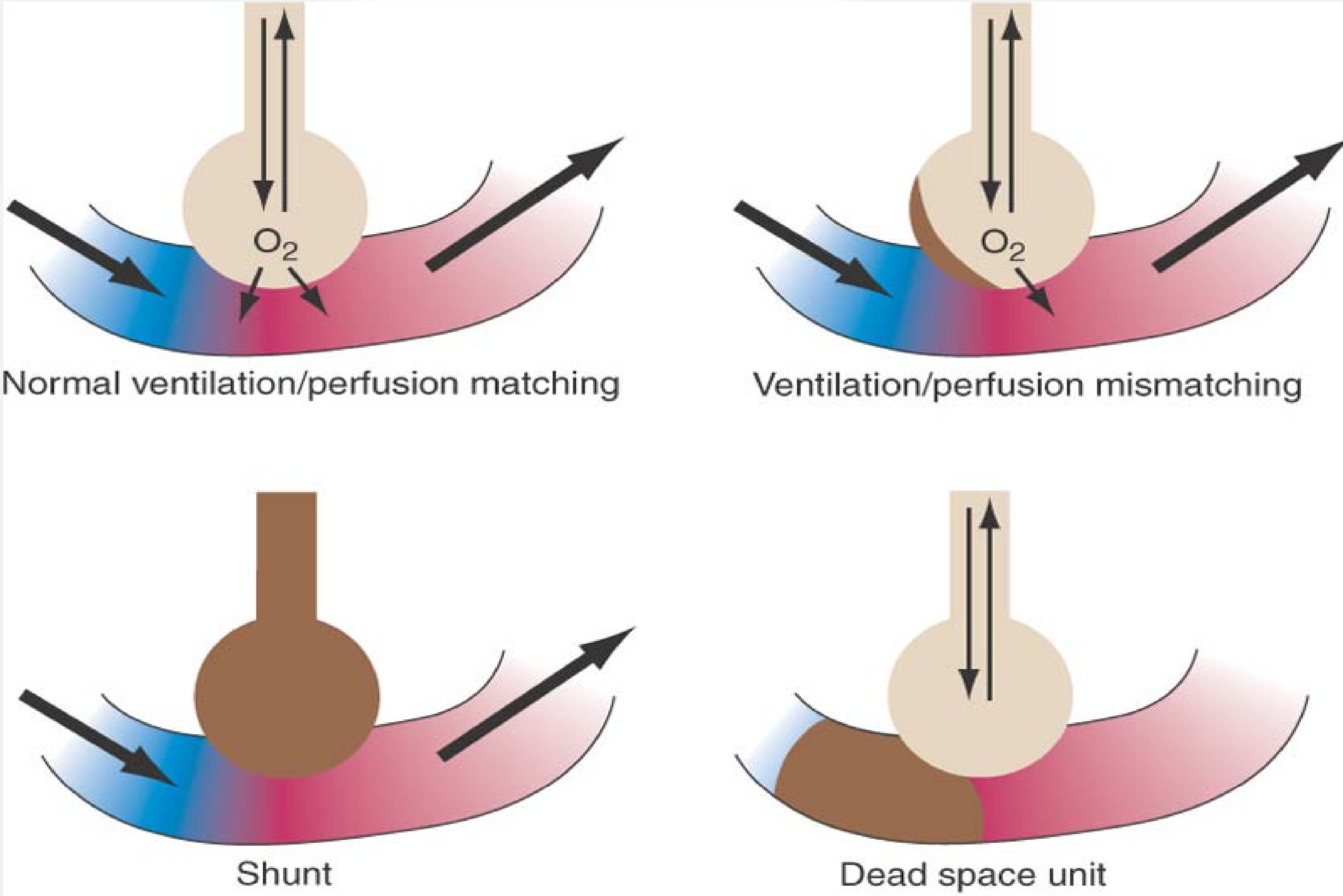
Type II

- **Hypercapnic**
- **↓ Ventilation**
- **Often with ↓ 0_2 as well**

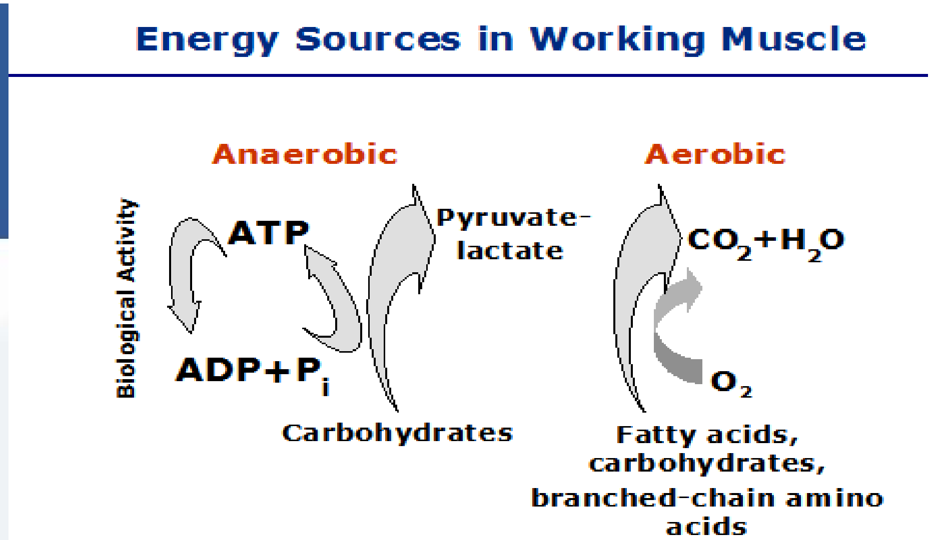
Causes of Respiratory Failure



Causes of Respiratory Failure; V/Q mismatch



Type I Hypoxemic Respiratory Failure



- Why do we need **OXYGEN?**
- Energy contained in carbohydrates, lipids and proteins is converted - ***IN PRESENCE OF O_2*** – into Adenosine Tri-Phosphate (ATP)
- **ATP** can be produced **WITHOUT 02 (anaerobic)**
- AEROBIC conditions produces approximately **19 X the ATP**!!
- Which method is more **EFFECTIVE** and **EFFICIENT**?

Hypoxemia caused by ↓P_i02

- Causes a reduced **ALVEOLAR pressure of 0_2 (P_A0_2)**
- Most common cause is **HIGH ALTITUDE** (ex. Mountains, airplanes)
- These conditions cause a **DECREASED barometric pressure (P_B)** so the partial pressure of 0_2 is also reduced.
- **Fi02 is still 0.21!!**

Most airplane cabins are pressurized to ~8000 ft. altitude

Hypoxemia

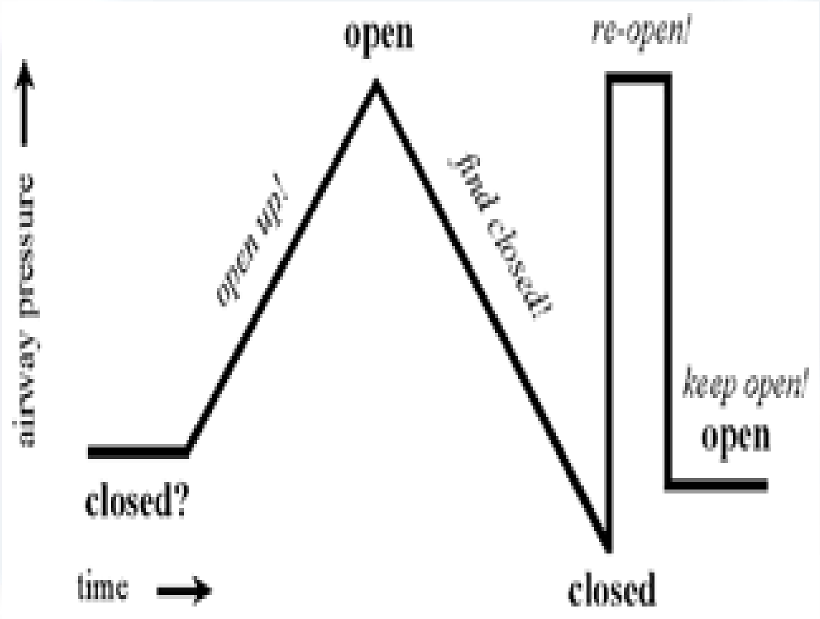
- Also may be caused by:
  - **Hypoventilation**
    - **caused by fatigue, muscle weakness, brain injury**
  - **SHUNT**
    - **Alveolar collapse (atelectasis), alveolar filling (pneumonia or pulmonary edema)**
    - **Doesn't usually respond to 0_2 therapy – *'refractory'***
  - **V/Q mismatch**
    - **Most common cause of hypoxemia**

RESP Failure / Hypoxemia - #1 treatment is 0_2 therapy!

- **↓'ed P_i02** and **HYPOVENTILATION** GENERALLY **RESPOND TO OXYGEN THERAPY**
- **Shunt** and ***V/Q mismatch*** may **NOT** respond to 02 therapy
  - ***"refractory hypoxemia"***
  - **May require mechanical ventilation with positive pressure +/- ventilation**
  - **PEEP +/- PPV**

Hypoxemic Respiratory Failure Ventilation management

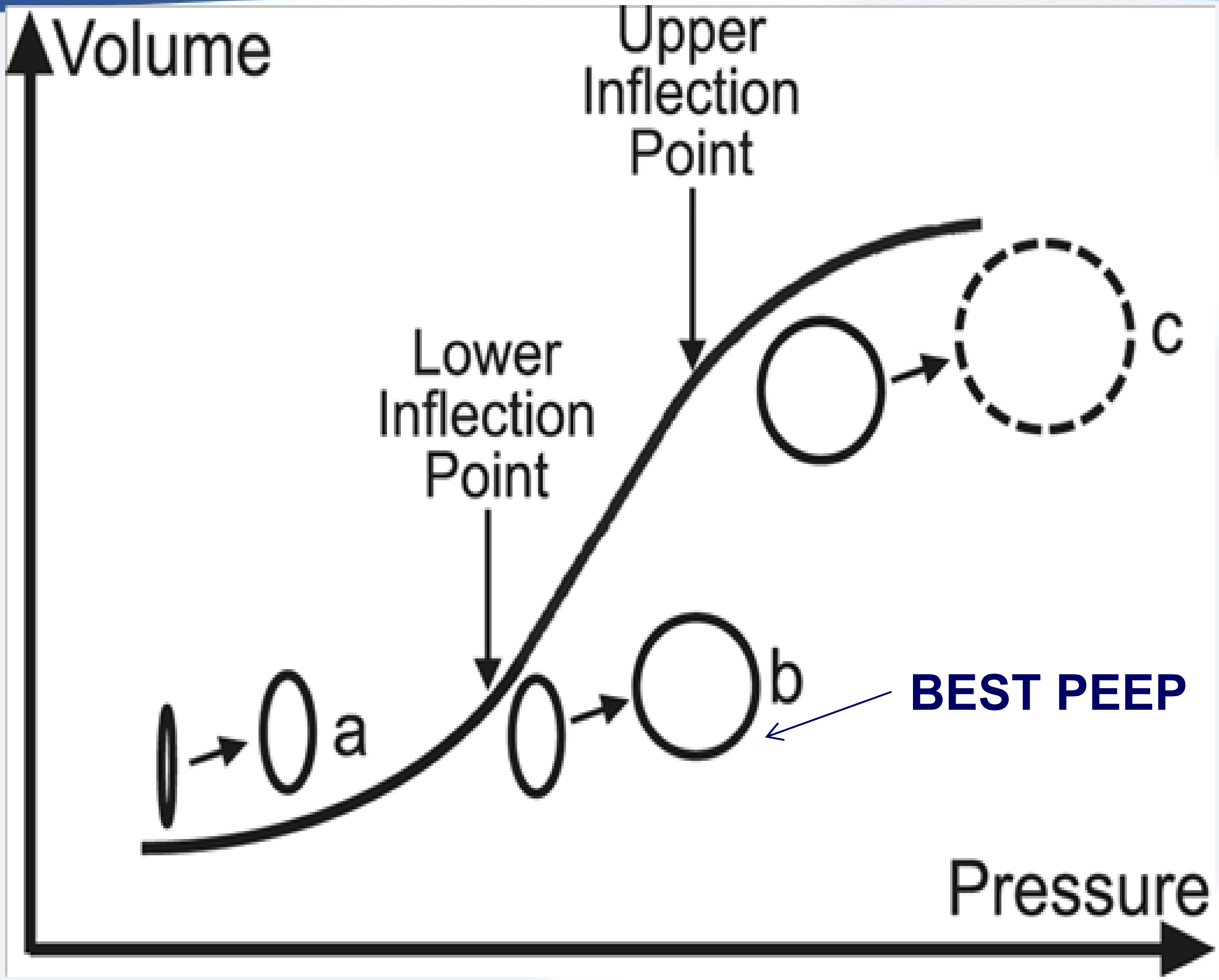
- Some common forms of ***HYPOXEMIC RESP failure*** are refractory to 0_2 therapy
- Require Positive Pressure to OPEN and RECRUIT alveoli
- ***"OPEN LUNG CONCEPT"***



The goal is to find the critical opening pressure of the alveoli and KEEP them OPEN!

Papadakos and Lachmann.
Crit Care Clin (2007)

Open Lung Concept



BEST PEEP

Miller, Russell. *Chest* (2012)

Ventilation strategies that apply the Open Lung Concept

- Higher PEEPs
- APRV
- HFO
- Lung Recruitment Maneuvers (LRM)

Lower PEEP/higher FiO2

FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7
PEEP	5	5	8	8	10	10	10	12

FiO_2	0.7	0.8	0.9	0.9	0.9	1.0
PEEP	14	14	14	16	18	18-24

Higher PEEP/lower FiO2

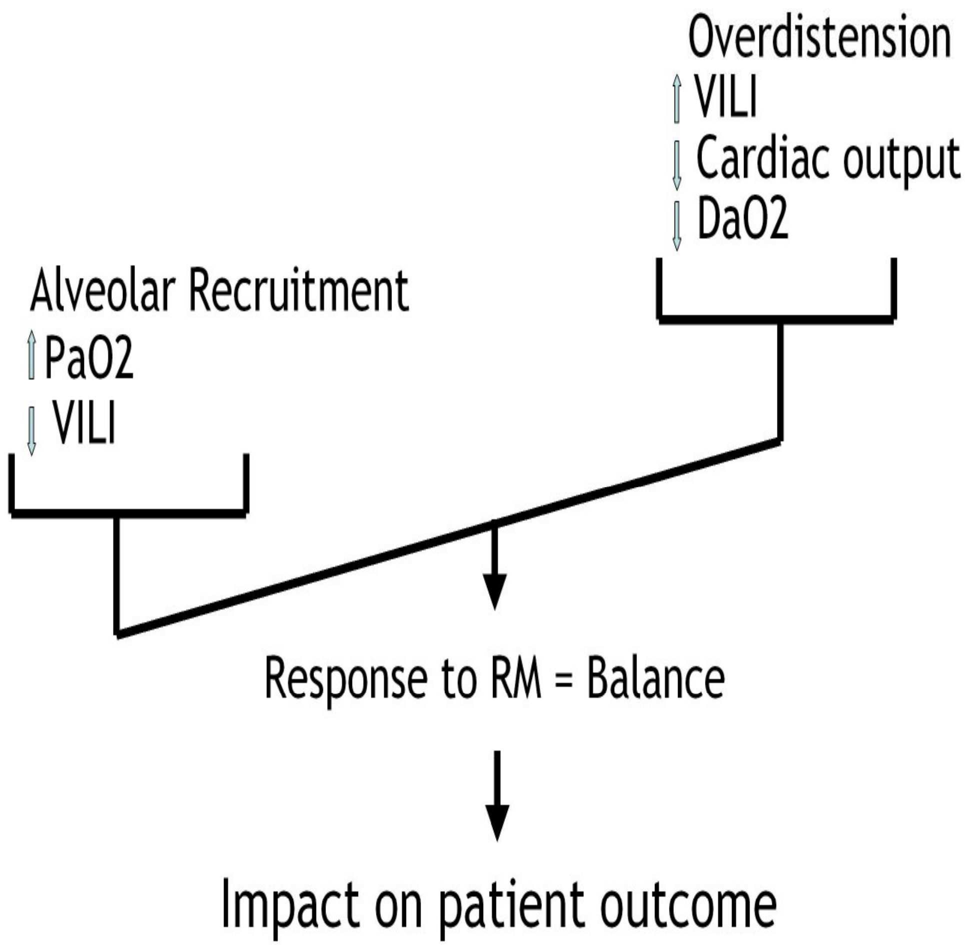
FiO_2	0.3	0.3	0.3	0.3	0.3	0.4	0.4	0.5
PEEP	5	8	10	12	14	14	16	16

FiO_2	0.5	0.5-0.8	0.8	0.9	1.0	1.0
PEEP	18	20	22	22	22	24

Lung Recruitment Maneuver

- Attempts to OPEN the lungs and improve oxygenation
- Typical is 30 cmH20 PEEP x 30 sec., or 40 cmH20 PEEP x 40 sec.
- Evidence shows ↑ oxygenation, but no Δ survival

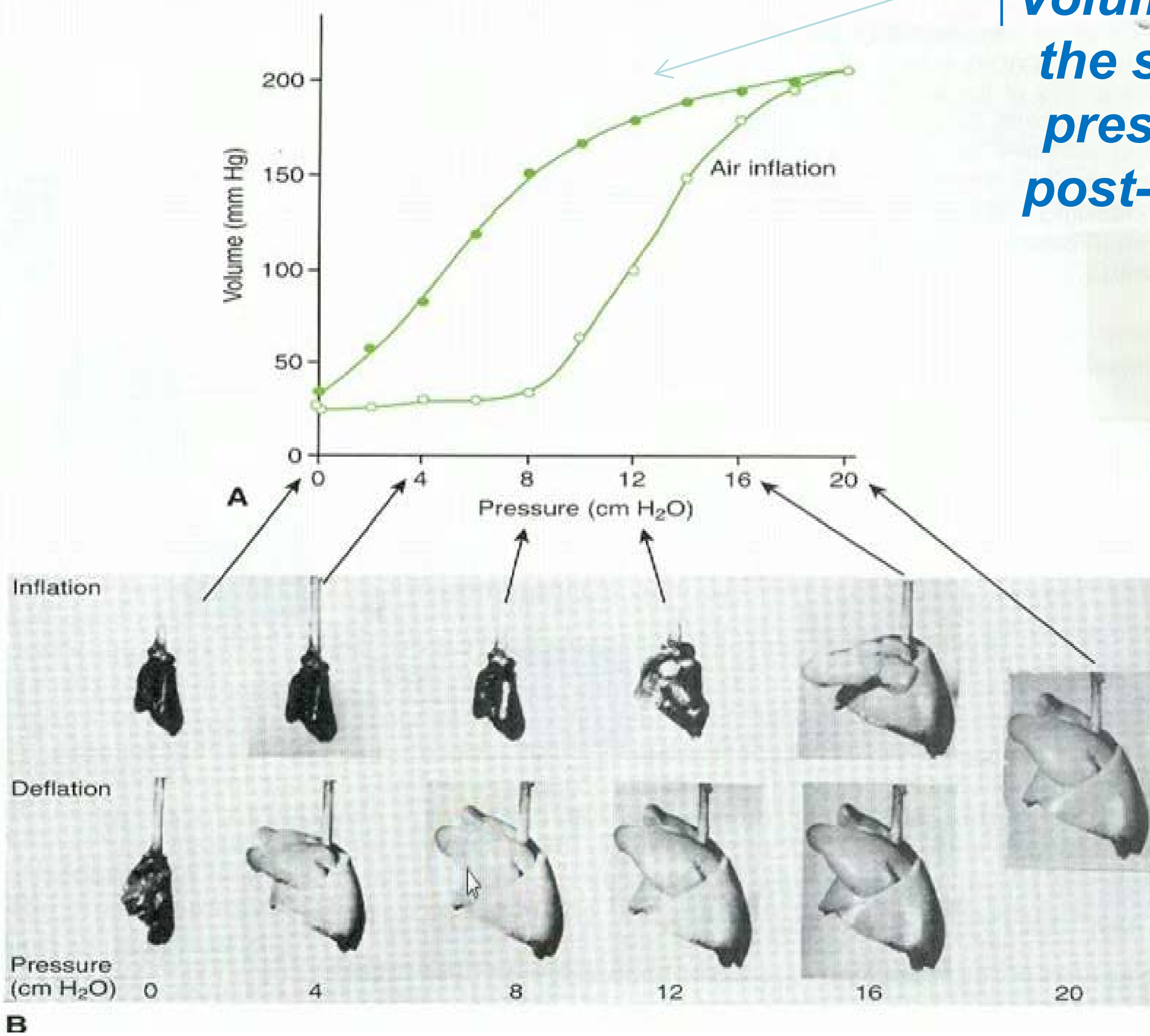
Esan *et al Chest* (2010)



Guerin *et al. Annals of Intensive Care* (2011)

Lung inflation following
Recruitment Maneuver

↑Volume at the same pressure post-LRM

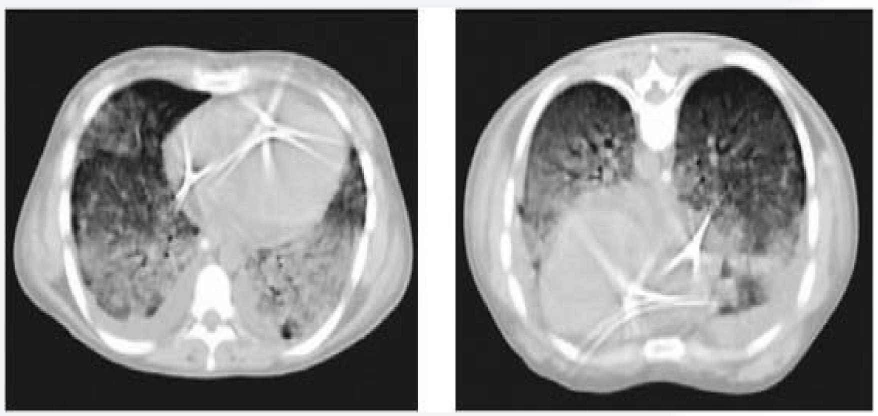


PERMISSIVE HYPERCAPNIA

- ALLOWS AN ↑'ed C02
- LIMITS A/W PRESSURES
- C02 >50 mmHg
- pH >7.20
- MAINTAIN OXYGENATION

- TIDAL VOLUMES **4-6 MLS/KG**
- REDUCES RISK OF VOLUTRAUMA and BAROTRAUMA
- ANIMAL STUDIES SHOWED NO SIGNIFICANT DEFICIT WITH C02 500 !!
- BE CAREFUL WITH *CEREBRAL TRAUMA* AND CARDIOVASCULAR INSTABILITY

PRONE POSITIONING



Raoof *et al* ***Chest*** (2010)

- FOUND TO ↑ OXYGENATION IN 75% OF CASES;
  - *No sig. Δ mortality*
  - ↑ V/Q MATCHING
- ***MAKES BASIC CARE OF PATIENT DIFFICULT***
- ↑ risk of accidental extubation

Type II Hypercapnic Respiratory Failure

- **Caused by ↓'ed Ventilation**
- **Acute Ventilatory Failure**
  - ↑**$PaC02$ (>45 mmHg)** for a short time
  - pH is ↓'ed **< 7.35** – **acidosis**
- **Chronic Ventilatory Failure**
  - ↑**$PaC02$** over a long period of time
  - **pH is usually normal: 7.35-7.40**
  - Metabolic compensation has occurred over time

How does the body compensate for an ↑PaC02?

HYPERCAPNIA – causes

- **Hypoventilation**
  - Drug overdose
  - Brain injury
  - Fatigue / tired
- **V/Q mismatch**
  - $C0_2$ is not eliminated from alveoli
- **↑'ed Dead Space**
  - More ventilation to areas with no perfusion
  - $C0_2$ is not eliminated

- **$C0_2$ elimination MUST equal $C0_2$ production**
- ***PaC02 = $C0_2$ production / ALV. Ventilation***
- **If C02 production increases, ALV. Ventilation must increase the SAME proportion – otherwise, $PaC0_2$ will ↑!!**

Hypercapnia – treatment

- **If patient does not ↑ ventilation may require mechanical ventilation (PPV)**
  - **Patient may NOT be able to compensate for hypoventilation due to fatigue / tired or muscle/nerve dysfunction**
- **Monitor ABG's for effectiveness**
  - **What do you look at on the ABG's to determine if the patient's ventilation is effective?**

VENTILATION vs *RESPIRATION*

- Important to remember what ***"VENTILATION"*** means;
  - ***gas IN and OUT***
- Different from ***"RESPIRATION"***
  - ***Internal vs external respiration***
- *What does a machine called a **"VENTILATOR"** achieve?*
- *What does a machine have to achieve to be accurately referred to as a **"RESPIRATOR"**?*

VENTILATOR

RESPIRATOR

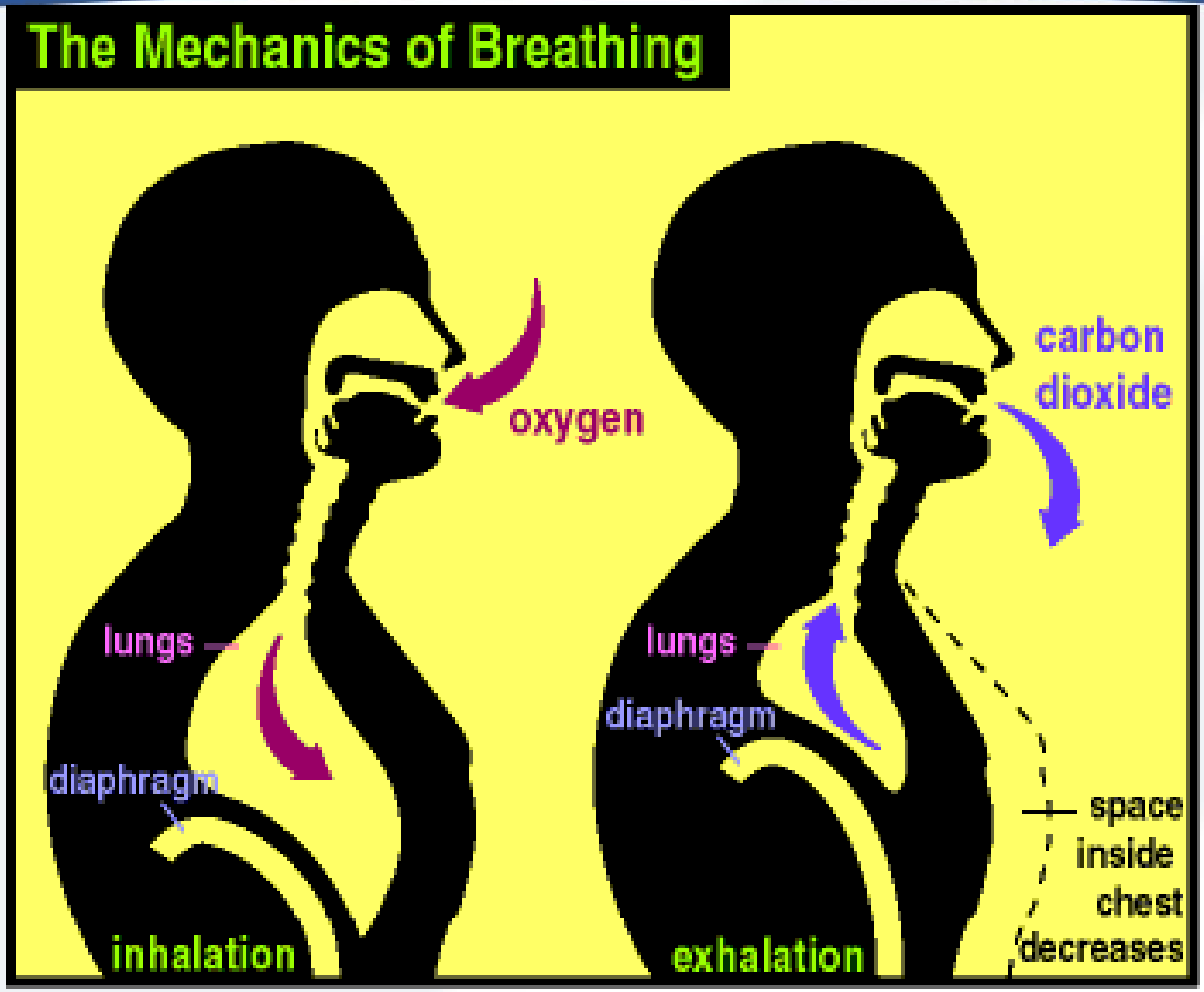
Normal SPONTANEOUS ventilation – *INSPIRATION*

1. Diaphragm and EXT. intercostals contract
2. ↑'s VOLUME of thorax
3. ↓'s PRESSURE in thorax
   alveolar pressure or P_A; ***BOYLE's LAW***
4. Creates a ***PRESSURE GRADIENT*** (Δ P)
   P_{Mouth} (0 cmH20) > P_A (–3 cmH20)
5. Gas flows from ***MOUTH*** > ***ALVEOLI***

Normal SPONTANEOUS ventilation – *EXPIRATION*

- As **ALVEOLI** fill from gas flow > P_{Alv} ↑'s
- When P_{Alv} reaches 0 – gas flow **STOPS**
  - No longer Δ P: $P_{Alv}=0$, $P_{Mouth}=0$
- END of INSPIRATION
- **Muscles RELAX – Lung tissue RECOIL**
- Volume in thorax ↓'s
- P_{Alv} ↑'s
- New Pressure gradient: P_{Alv} (+3 cmH20) > P_{Mouth}
- Gas flows from ***ALVEOLI*** > ***MOUTH*** = EXP.
- Flow continues until $P_{Alv} = 0$

Mechanics of SPONTANEOUS ventilation



Negative pressure on INSP **Positive pressure on EXP**

Mechanics of SPONTANEOUS ventilation (Pilbeam 2010)

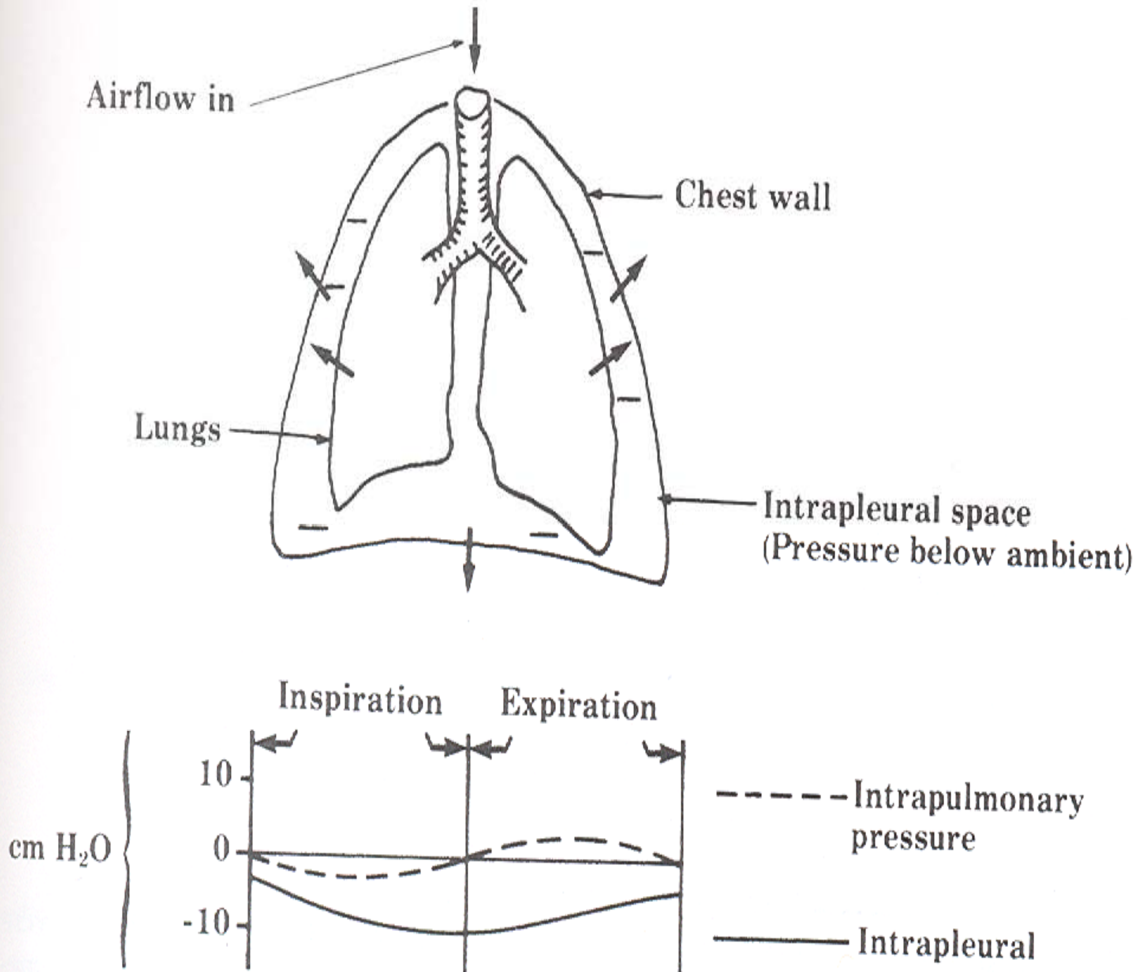


Figure 3-2. The mechanics of spontaneous ventilation and the resulting pressure waves showing approximately normal values (see text).

Indications for Mechanical Ventilation

- *Hypoxemic Resp. Failure*
- Pa02 < 60 mmHg or Sp02< 90% on>50% Fi02
- Typical causes:

V/Q mismatch, R>L shunt, diffusion defect, alveolar hypoventilation

- *Hypercapnic Resp. Failure*
- Acute ↑ PaC02 >50 mmHg or acutely above normal with pH < 7.30 (COPD)
- Typical causes:

pump failure (drive, muscles, WOB), ↑ C02 production, ↑ deadspace

Some history of mechanical ventilation

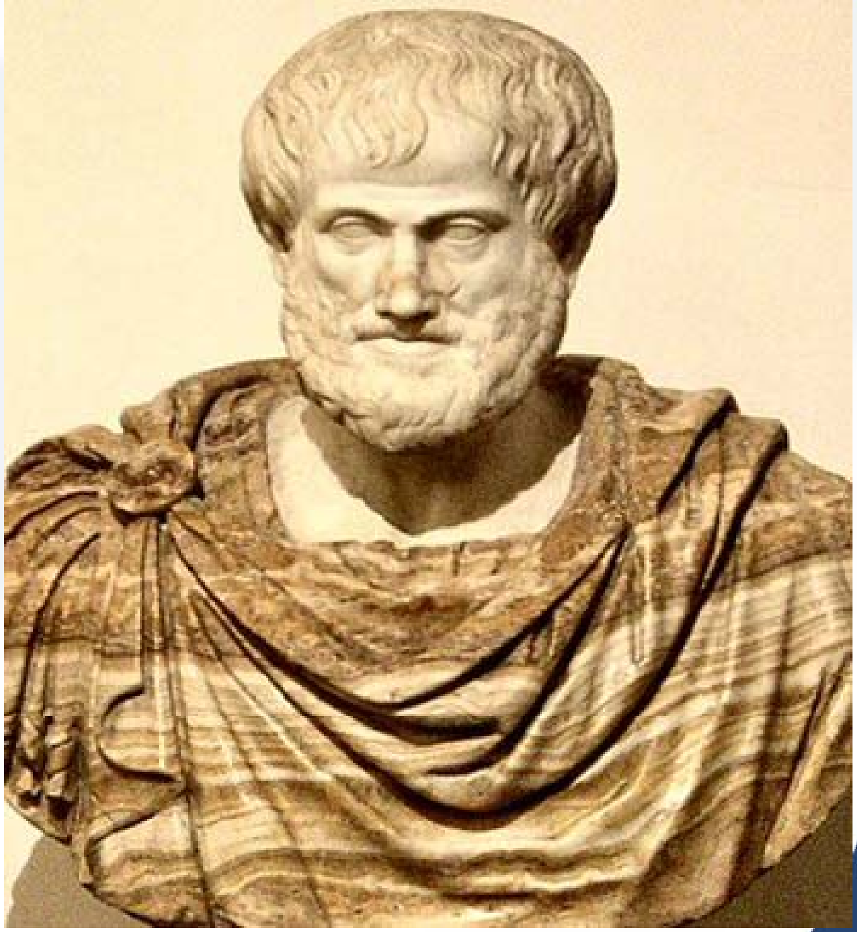
HIPPOCRATES – 'the Father of Medicine' *circa 370 B.C.*

"Treatis on Air"

" one should induce a cannula into the trachea along the jawbone so that air can be drawn into the lungs"

ARISTOTLE
circa 322 B.C.

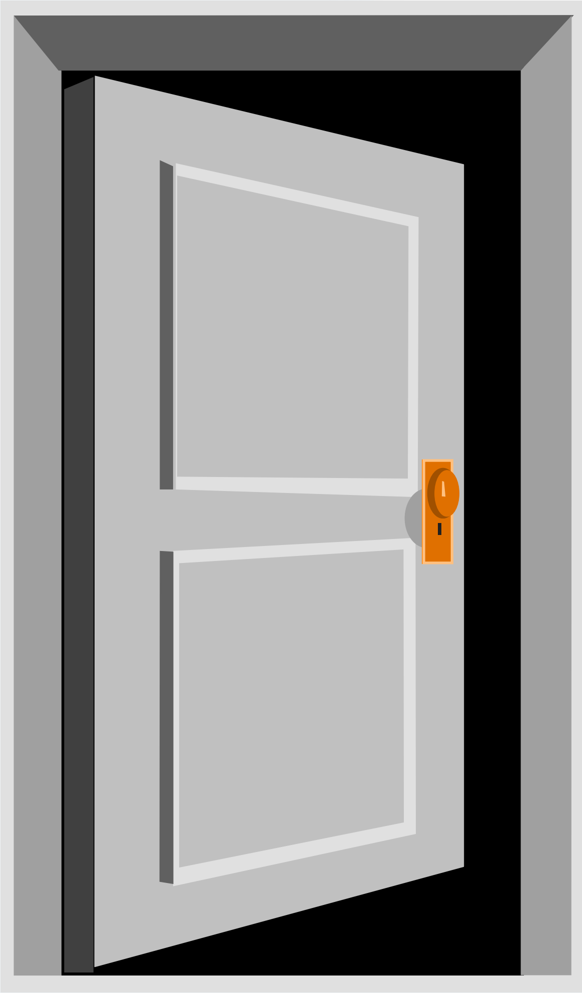
- demonstrated animals required air to breath
- when animals were placed in closed boxes….they died!!
- initially thought to be secondary to the inability to cool themselves



GALEN
2nd Century

- Performed extensive studies on animal anatomy
- presumed humans to be similar
- for centuries it was believed humans had a segmented sternum similar to an apes!!

DARK AGES
for the next 1300 years



- RELIGIOUS AND POLITICAL BELIEFS PREVENTED HUMAN DISSECTIONS

- GALEN'S 2nd CENT. DISCOVERIES WERE FOLLOWED FOR MOST OF THIS TIME

16 th CENTURY

RESEARCH BEGAN ONCE AGAIN ON THE HUMAN BODY

MANY RESEARCHERS BEGAN TO WORK ON EXPLAINING HUMAN PHYSIOLOGY

PARACELSUS & VESALIUS

- During the 16th century credited with being the first to manually ventilate with bellows and tubes inserted into patients airways

- Vesalius restarted practice of human dissection

Foot-powered resuscitator

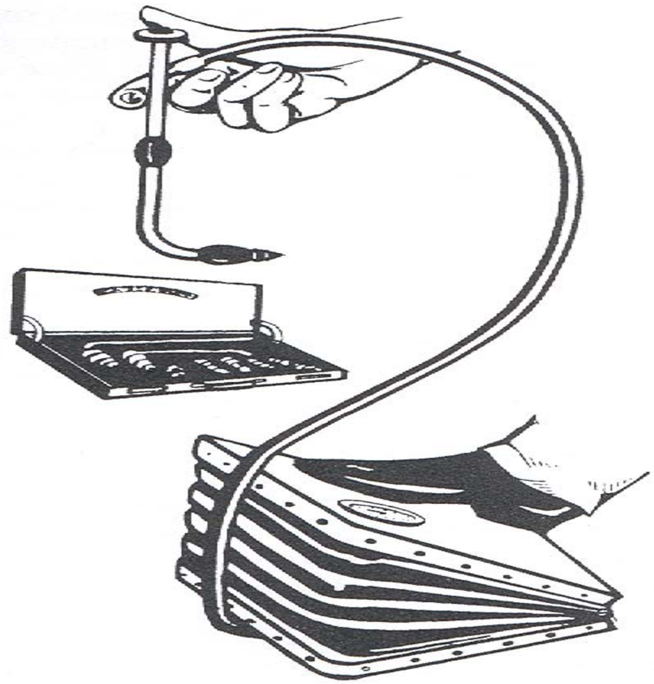


Figure 1-5. A ventilating device similar to the original Fell apparatus. It incorporated O'Dwyer's laryngeal tube (1888). The airway was attached through a flexible tube to a foot bellows. The external end of the endotracheal tube had two branches, one connected to the bellows and one to the operator's thumb. During inspiration the operator covered one branch with his thumb, forcing the air from the bellows into the lungs. During expiration the thumb was removed and the accumulated air from the lungs was allowed to pass into the room. (Redrawn from Mushin WL, Rendell-Baker L, Thompson PW, et al: *Automatic ventilation of the lungs,* ed 2, Oxford, England, 1969, Blackwell Scientific Publications, p 186. Used by permission.)

IRON LUNG TECHNOLOGY

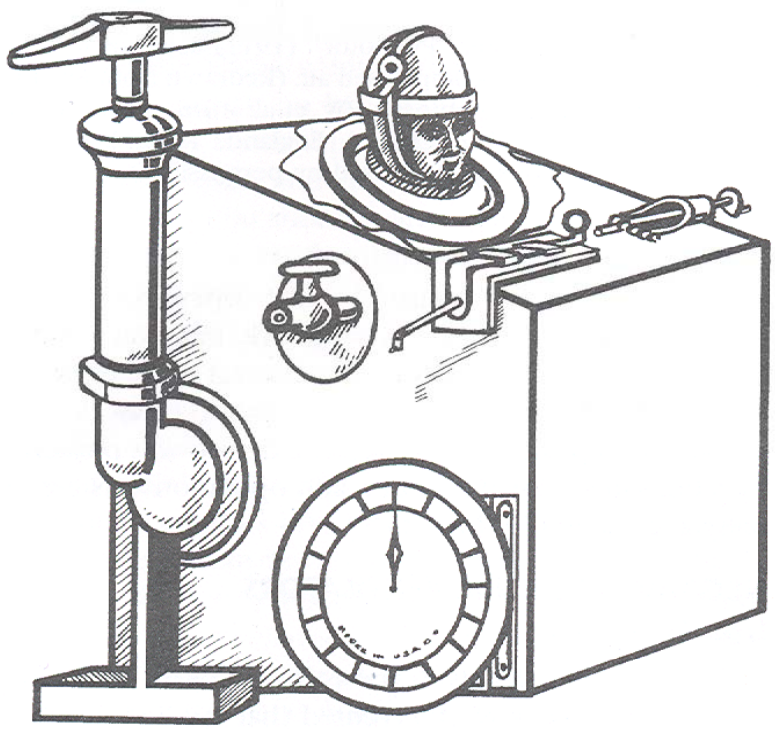


Figure 1-2. An early negative pressure device designed by Alfred F. Jones in 1864. (Redrawn from Young JA, Crocker D: *Principles and practices of inhalation therapy,* St. Louis, 1970, Mosby. Used by permission.)

Many developed from mid 1800's – early 1900's

negative pressure ventilation

chest "cuirass"

Alexander Graham Bell

Hand-powered Iron Lung

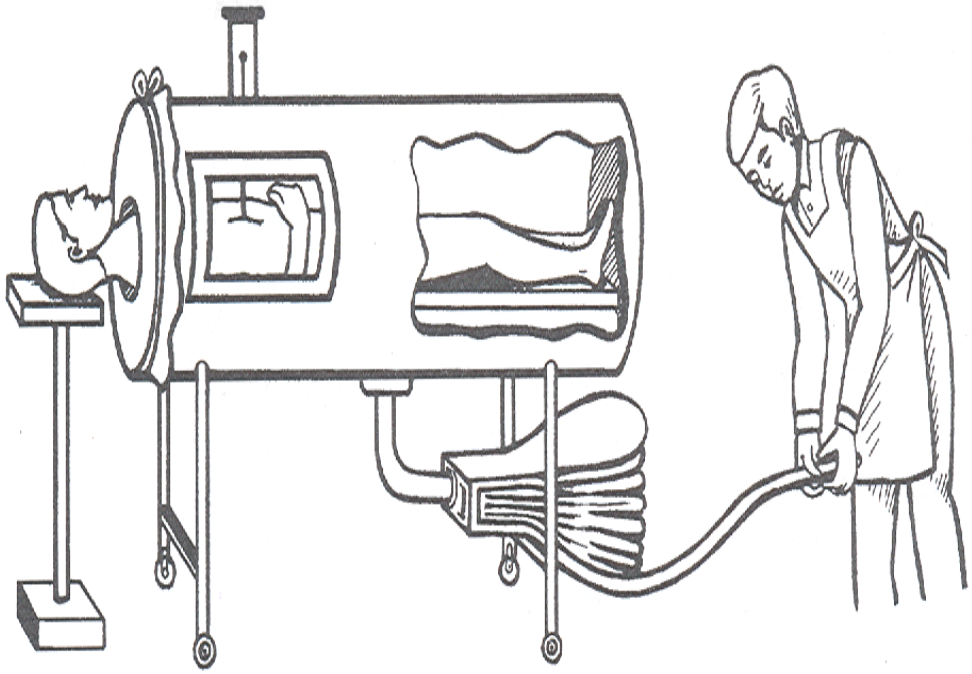
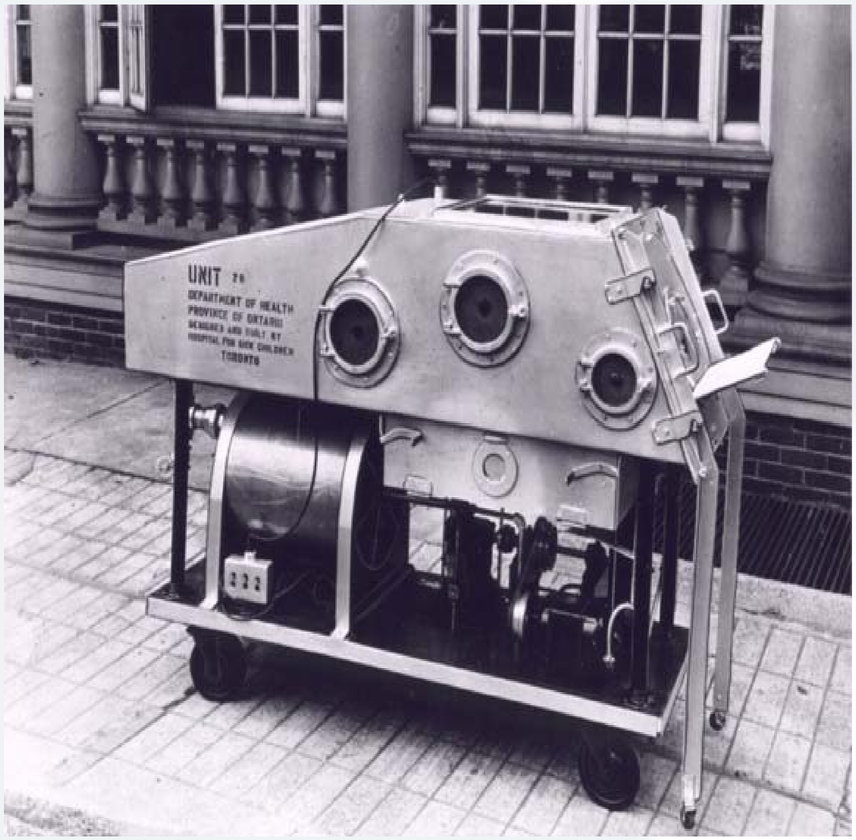


Figure 1-3. A negative pressure device designed for resuscitation by Woillez in 1876. (Redrawn from Young JA, Crocker D: *Principles and practices of inhalation therapy*, St. Louis, 1970, Mosby. Used by permission.)

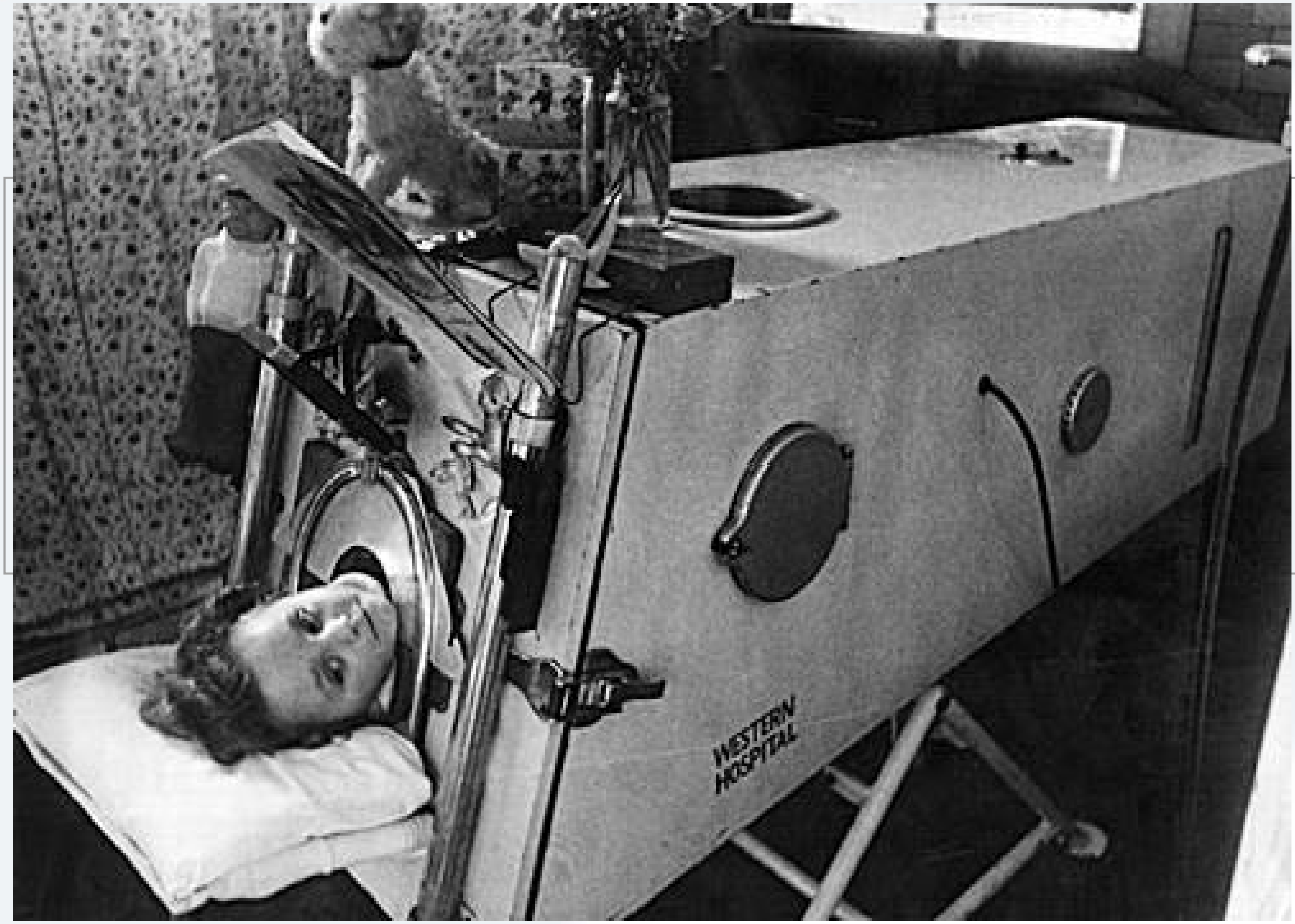
Iron Lung;

A lot MORE bulky and less-portable than current transport ventilators!!

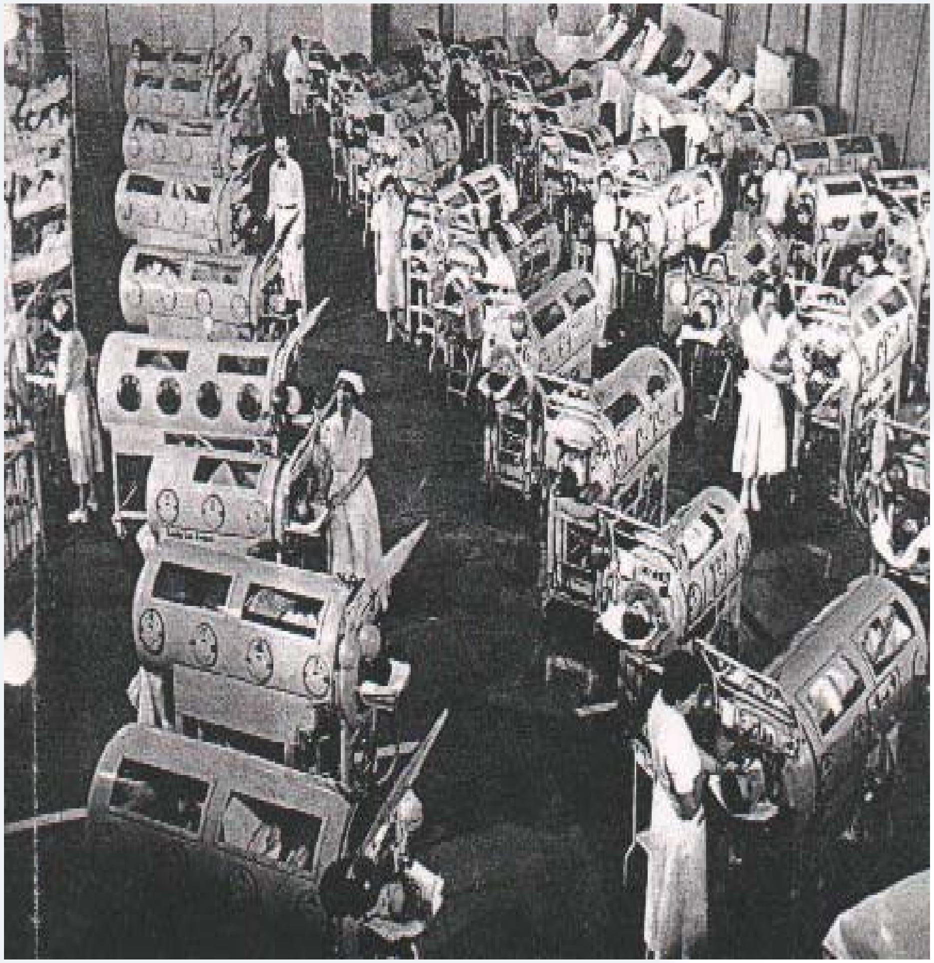


Iron Lung;

can you see how basic care of the pt. would be difficult?!



Iron Lung wards in U.S., circa 1950's



Chest cuirass;

another example of *NEGATIVE* pressure ventilation

An early *Chest Curiass*

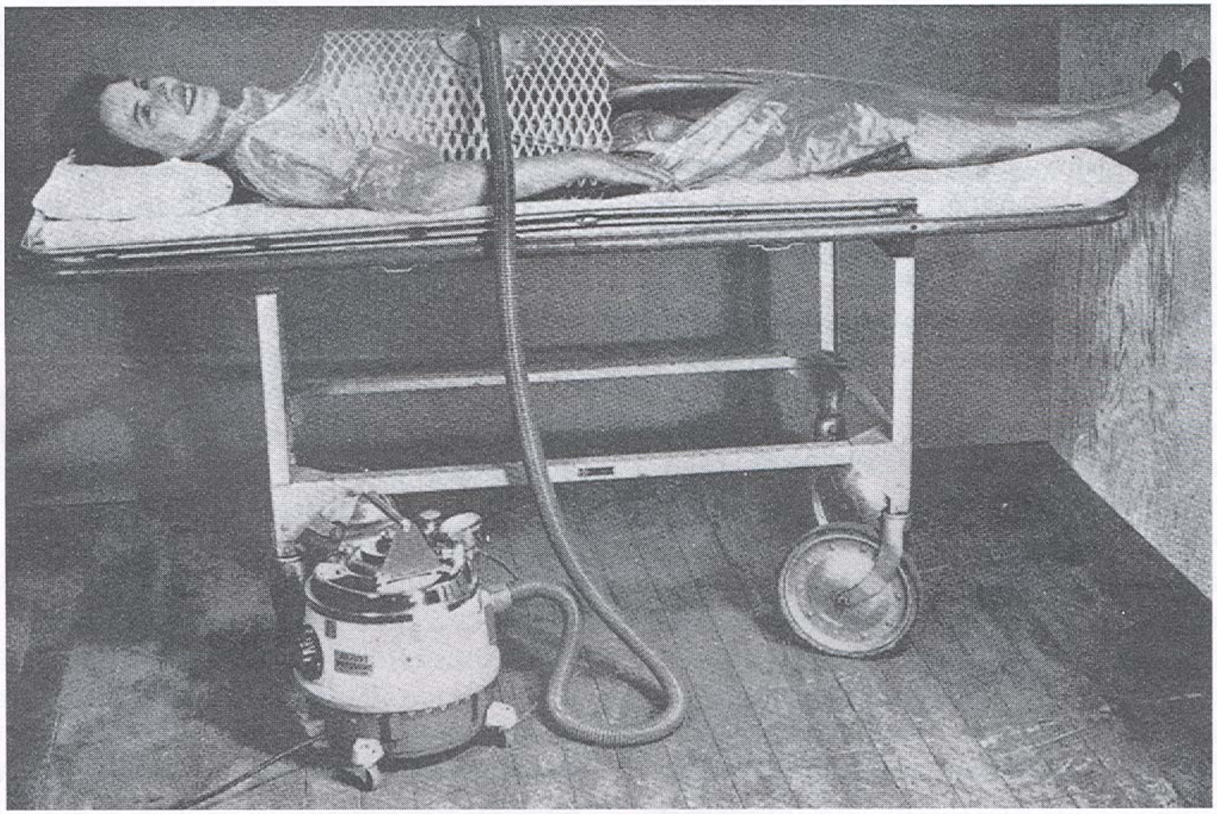


Figure 1-4. The chest respirator developed by Emerson. (Courtesy J.H. Emerson Co., Cambridge, Mass.)

MID 1800'S :ADVANCES IN SURGICAL ANESTHESIA

- POTENT **ANESTHETICS** AND **ANALGESICS** CAUSED **RESPIRATORY DEPRESSION**

- SURGEONS NEEDED METHOD OF **ASSISTING VENTILATION; or else patients would DIE!**

A negative pressure OR chamber

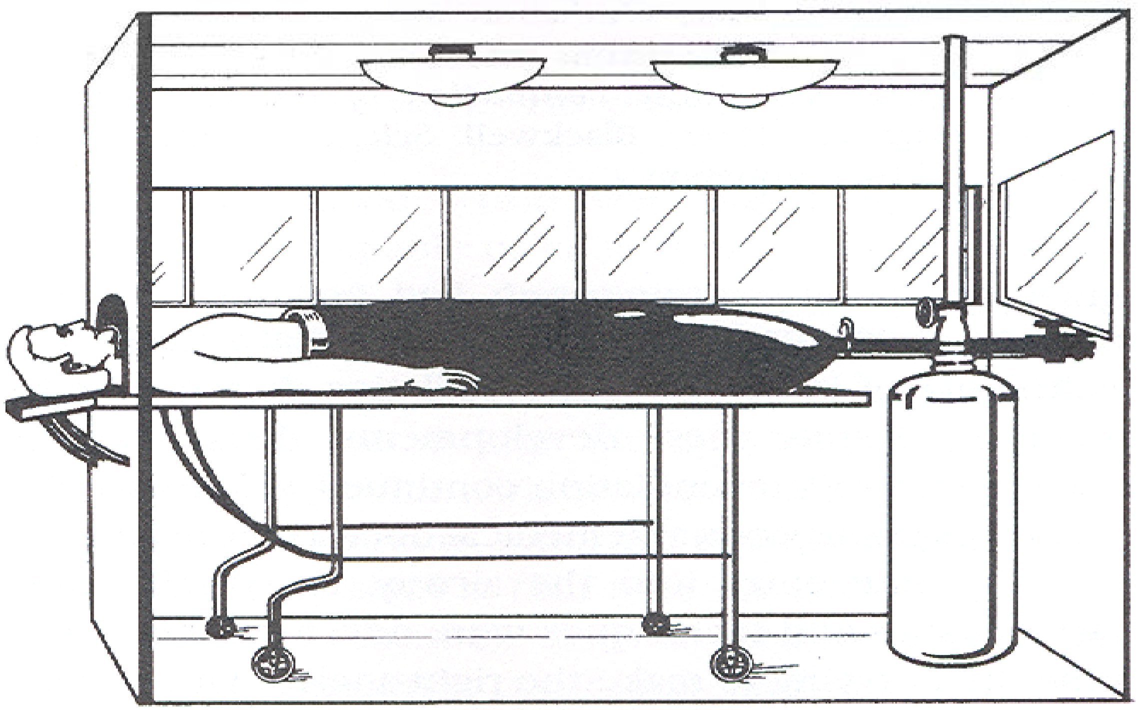


Figure 1-7. A negative pressure chamber designed by Sauerbruch in 1904 for use in thoracic surgery procedures. (Redrawn from Mushin WL, Rendell-Baker L, Thompson PW, et al: *Automatic ventilation of the lungs,* ed 2, Oxford, England, 1969, Blackwell Scientific Publications, p 188. Used by permission.)

Early 1900's positive pressure ventilator used for thoracic surgery

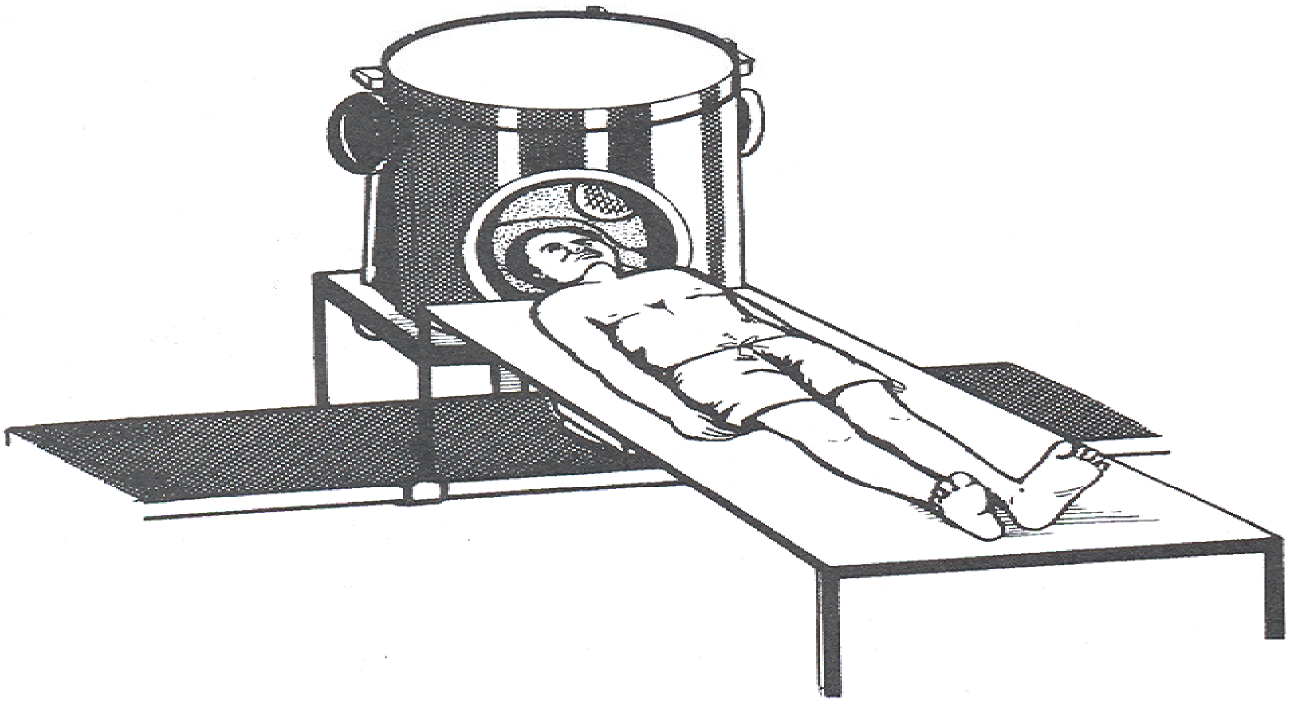


Figure 1-8. Brauer's continuous positive pressure head chamber for supporting ventilation during thoracic surgery (1905). (Redrawn from Mushin WL, Rendell-Baker L, Thompson PW, et al: *Automatic ventilation of the lungs,* ed 2, Oxford, England, 1969, Blackwell Scientific Publications, p 189. Used by permission.)

An early 1900's positive pressure resuscitator

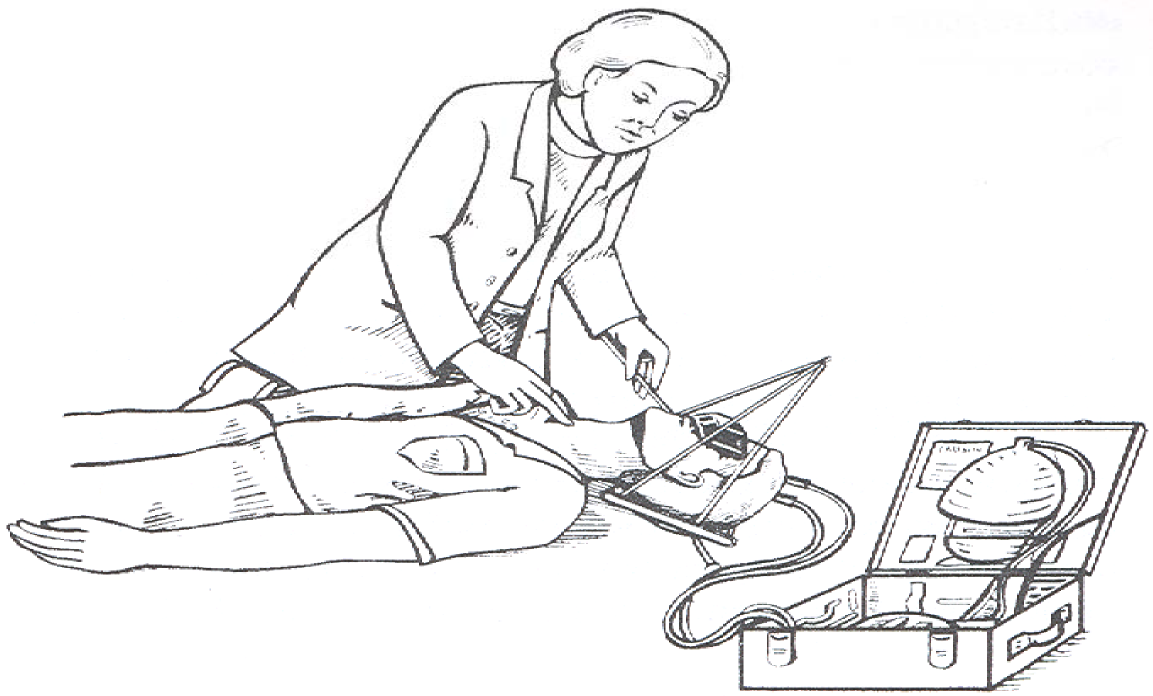
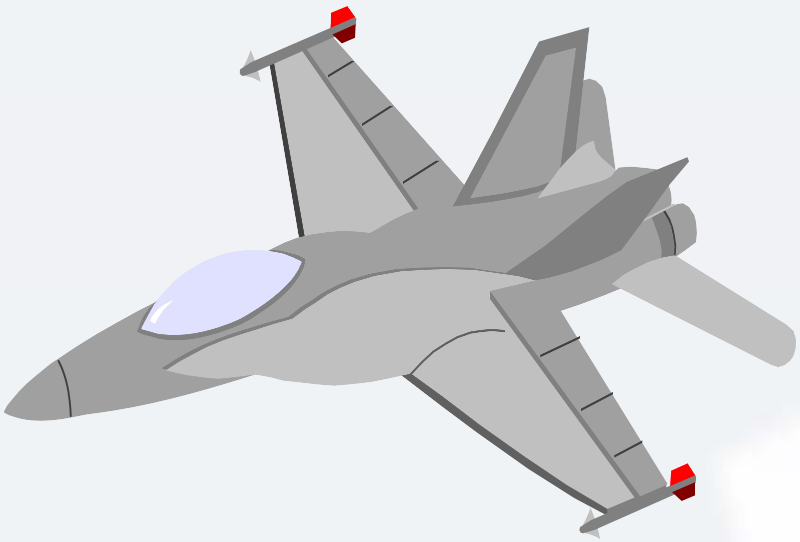


Figure 1-1. The Dräger "Pulmotor" (1911) used for resuscitation and powered by compressed air. (Redrawn from Mushin WL, Rendell-Baker L, Thompson PW, et al: *Automatic ventilation of the lungs,* ed 2, Oxford, England, 1969, Blackwell Scientific Publications, p 197. Used by permission.)

WORLD WAR II STIMULATED RESEARCH

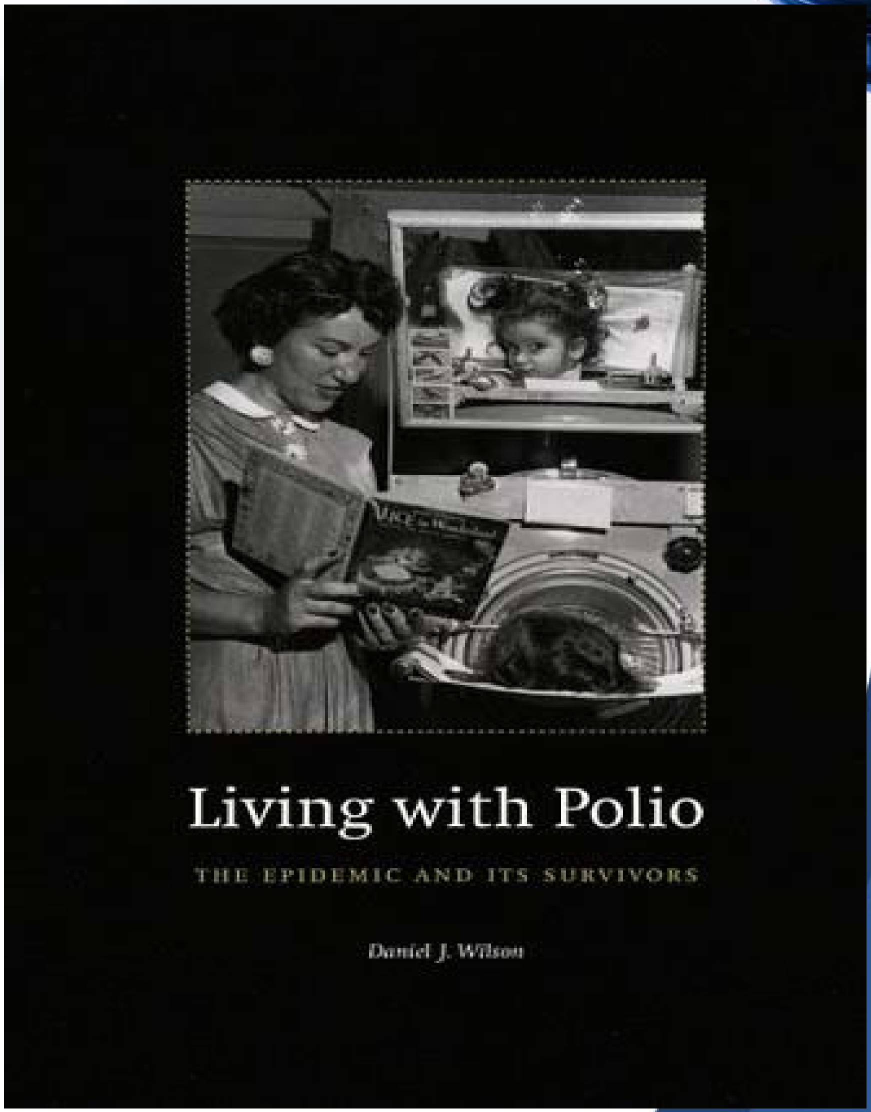


- Positive pressure breathing devices for high altitude

- Bird, Bennett and Emerson (early ventilator companies) were all researchers for the military

POLIO EPIDEMIC
DENMARK 1952

- 2722 cases > 315 required ventilation
- 27/31 first pts died due to lack of ventilatory support
- new techniques developed; i.e. cuffed ETT's and positive pressure ventilation
- *medical students bagged patients*!



Invasive vs NON-invasive

Two BREATH TYPES

- VOLUME Control
- PRESSURE Control

Patient vs. Ventilator:

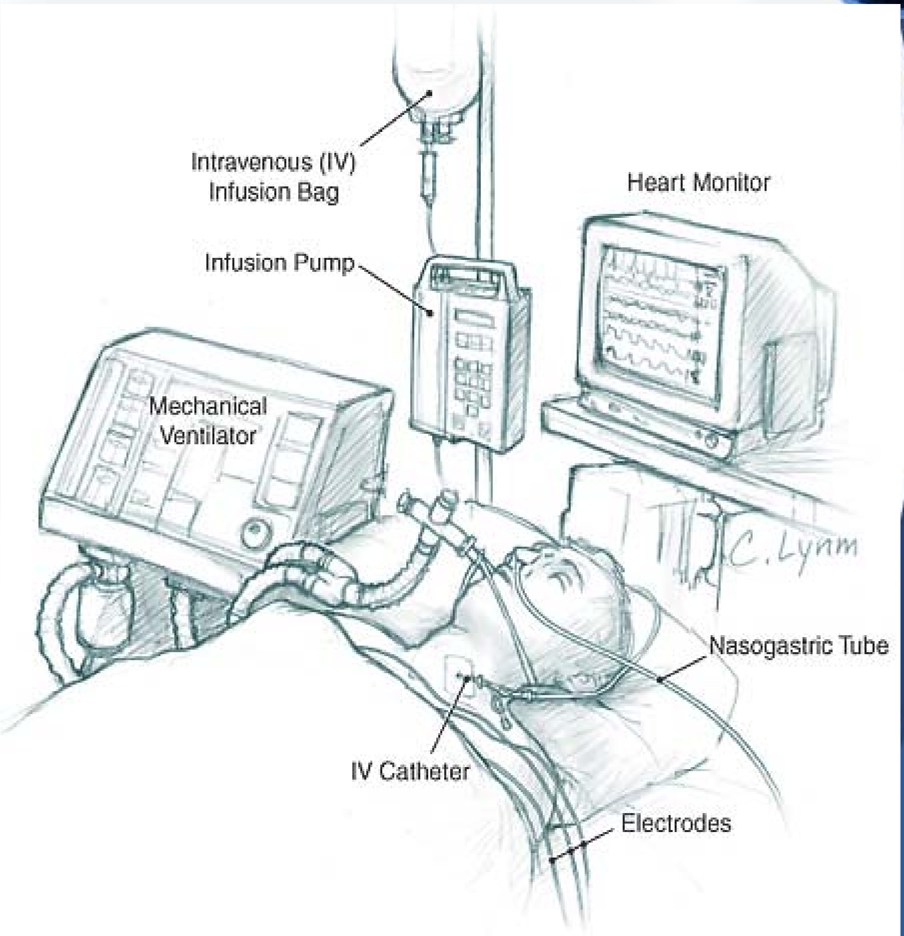
SPONTANEOUS vs MANDATORY

Invasive vs Non-Invasive

- Invasive refers to ventilation with an *ENDOTRACHEAL tube (ETT)* or *tracheal tube ('trach')*
- The *"traditional"* form of ventilation
- Has risks that go along with an 'invasive' procedure

- Non-invasive refers to ventilation provided via a *MASK* applied to the NOSE or FULL FACE
- Patient must be able to protect their a/w
- Less risk of infection and patient can still talk/eat

Invasive ventilation via *ETT* or *TRACH*



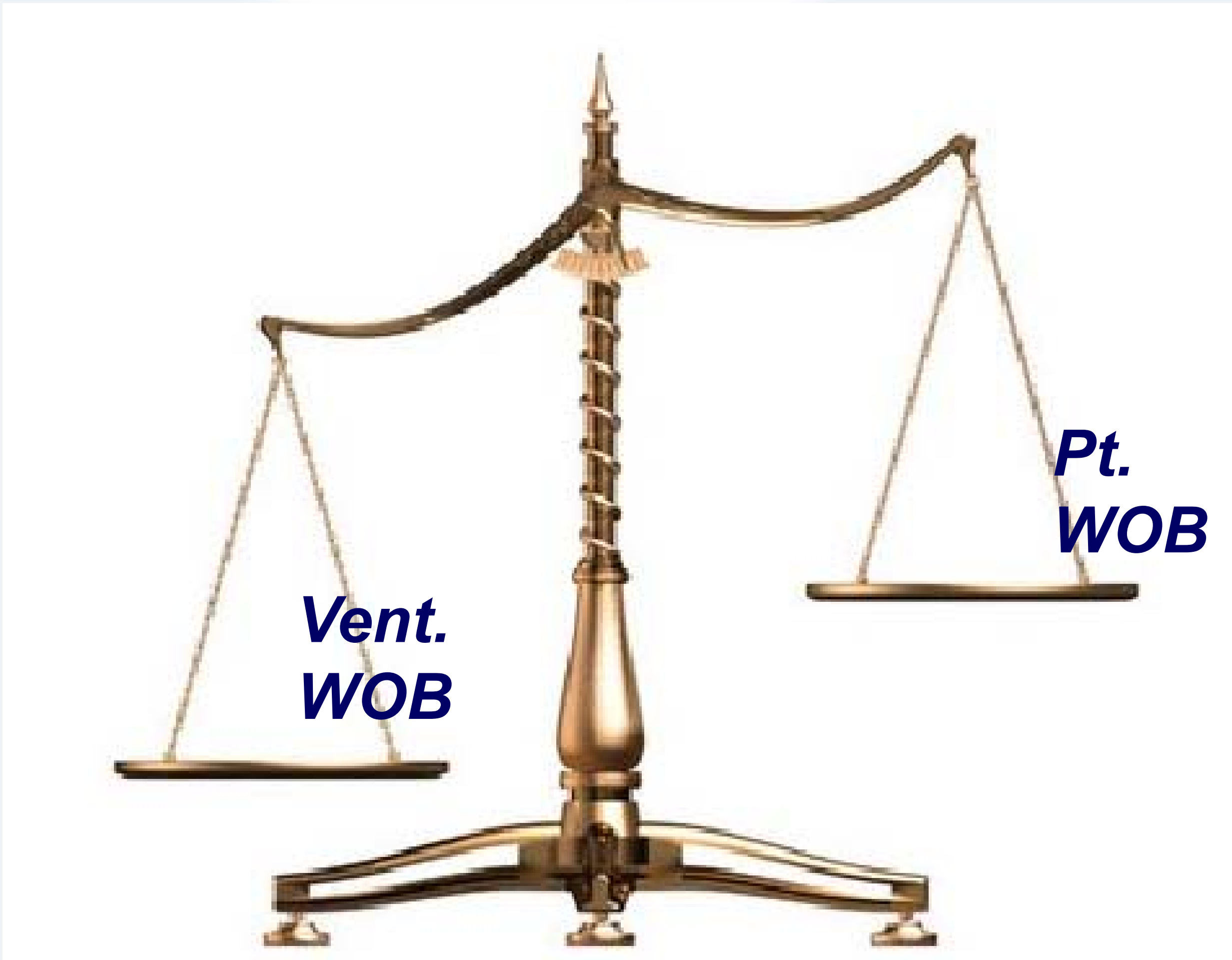
Non-invasive positive pressure ventilation (NIPPV)

FULL vs *PARTIAL* support

- FULL VENTILATORY SUPPORT involves the patient's ventilation being COMPLETELY achieved by the machine
- The patient's ***WOB*** would be zero/minimal
- Used for patients who are totally paralyzed or anesthetized

- PARTIAL VENTILATORY SUPPORT involves the **patient** doing some of the ***WOB*** and the **ventilator** doing some of the WOB
- Some combination of both
- Generally considered more "***patient-friendly***" as patient can control some of his/her own breathing

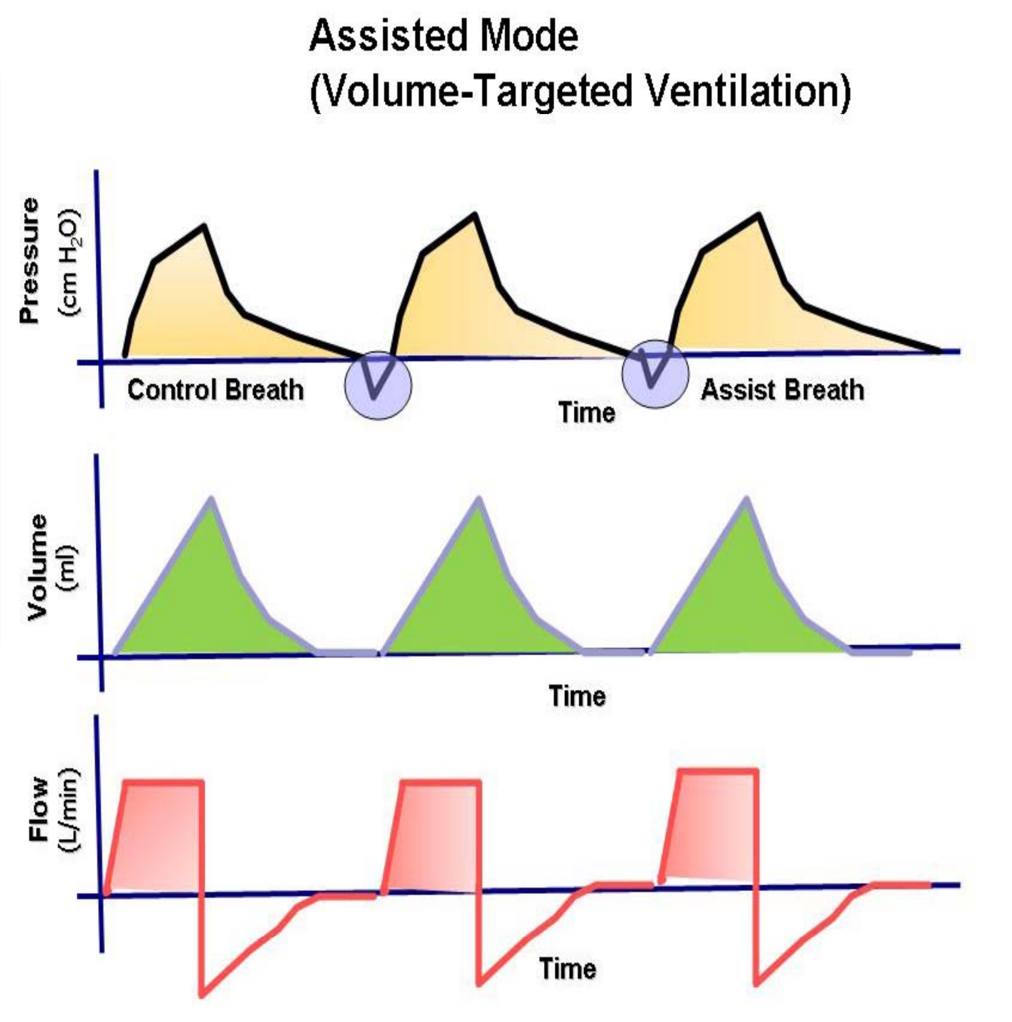
Equation of Motion; "keep the diaphragm moving"



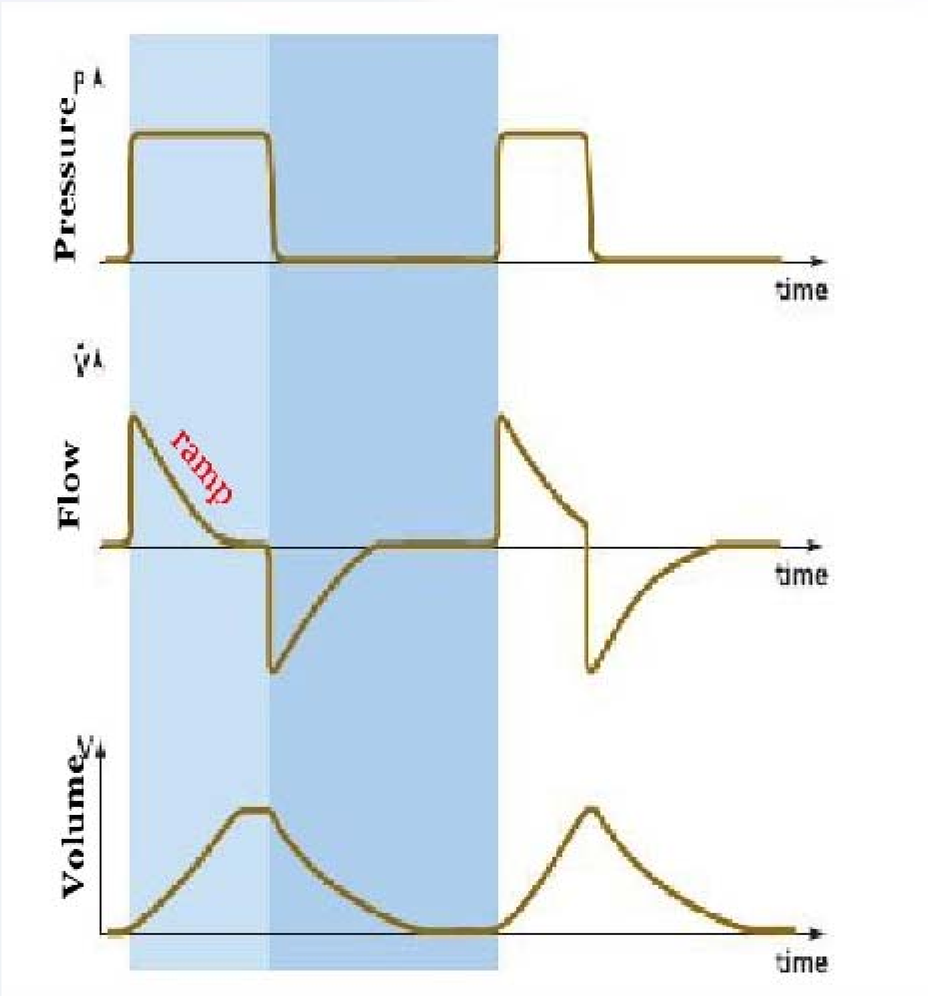
Breath Types: VOLUME CONTROL

- VENTILATOR DELIVERS SET TIDAL VOLUME (V_T) AND SET RATE (RR)

- AIRWAY PRESSURE (Paw) DETERMINED BY *PATIENT* COMPLIANCE (C_L) / Airway Resistance (Raw)



Breath types: PRESSURE CONTROL



VENTILATOR DELIVERS BREATH AT SET PRESSURE AND RATE

VT DETERMINED BY ***PATIENT*** COMPLIANCE / Raw

VENTILATOR MODES

CONTROL **(Controlled Mandatory Ventilation or CMV)**

- RARELY USED
- SET RATE DELIVERED BY VENTILATOR
- PT IS NOT ABLE TO TRIGGER FOR ADDITIONAL BREATHS

ASSIST-CONTROL **or A-C**

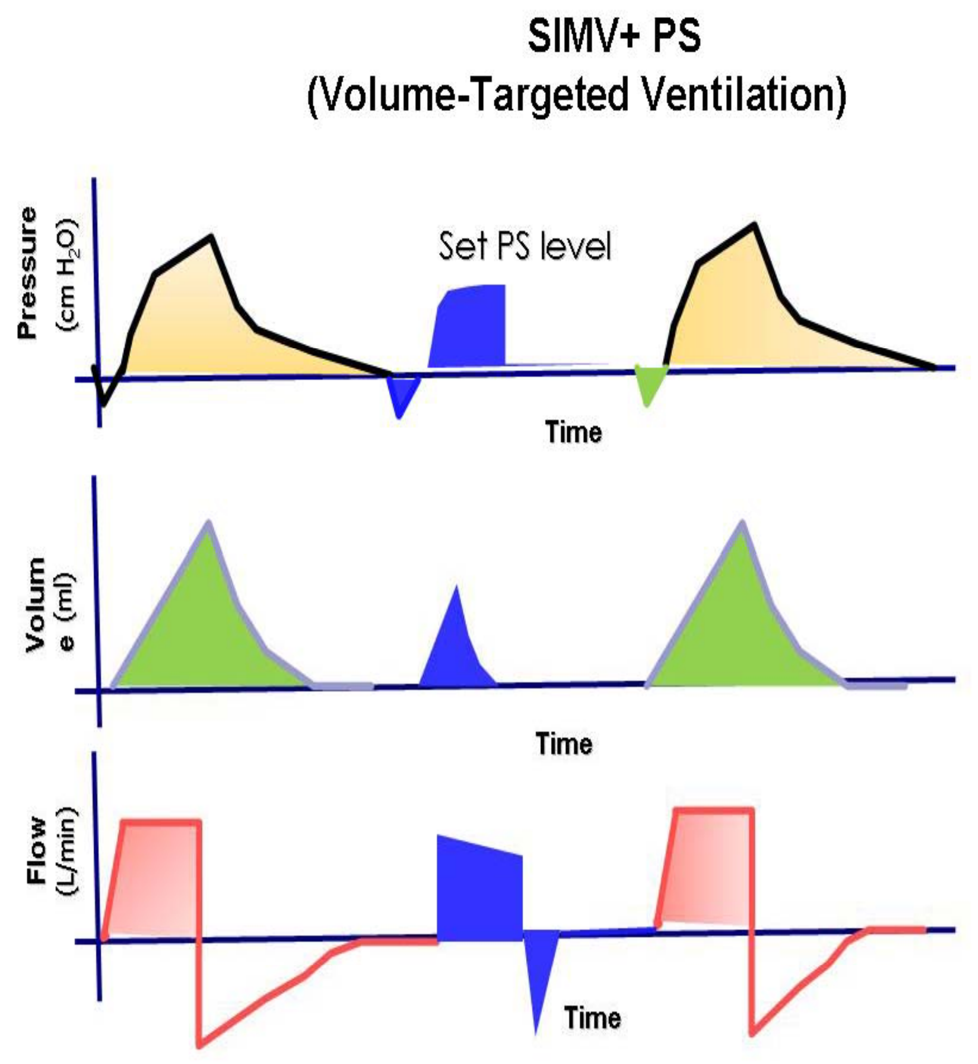
- SET RATE DELIVERED BY VENTILATOR
- PT CAN TRIGGER FOR ADDITIONAL MACHINE BREATHS
- ALL breaths are Ventilator-controlled breaths
- ***NO SPON breaths***

MODES (cont'd)

SYNCHRONIZED INTERMITTENT MANDATORY VENTILATION **(SIMV):**

Set **RR** is delivered by the ventilator but patient can breath ***SPONTANEOUSLY*** in between timed ventilato breaths

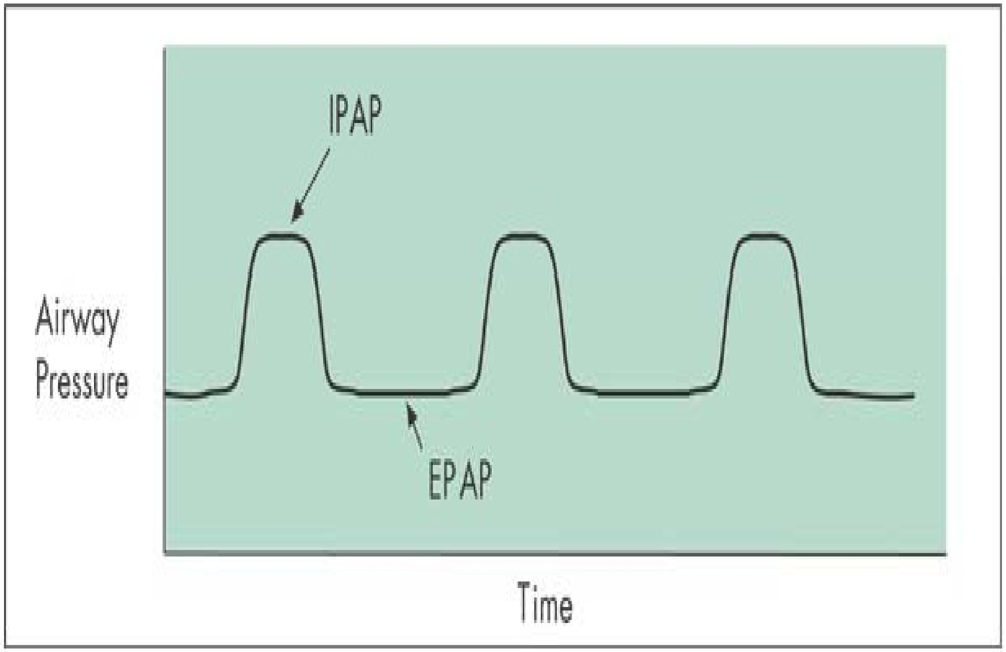
PRESSURE SUPPORT can be added to assist the **SPON** breaths



SPONTANEOUS MODES

CONTINUOUS POSITIVE AIRWAY PRESSURE **(CPAP)** :PROVIDES A POSITIVE AIRWAY PRESSURE TO SPLINT AIRWAYS OPEN AND DECREASE WORK OF BREATHING

SAME CONCEPT AS 'PEEP'

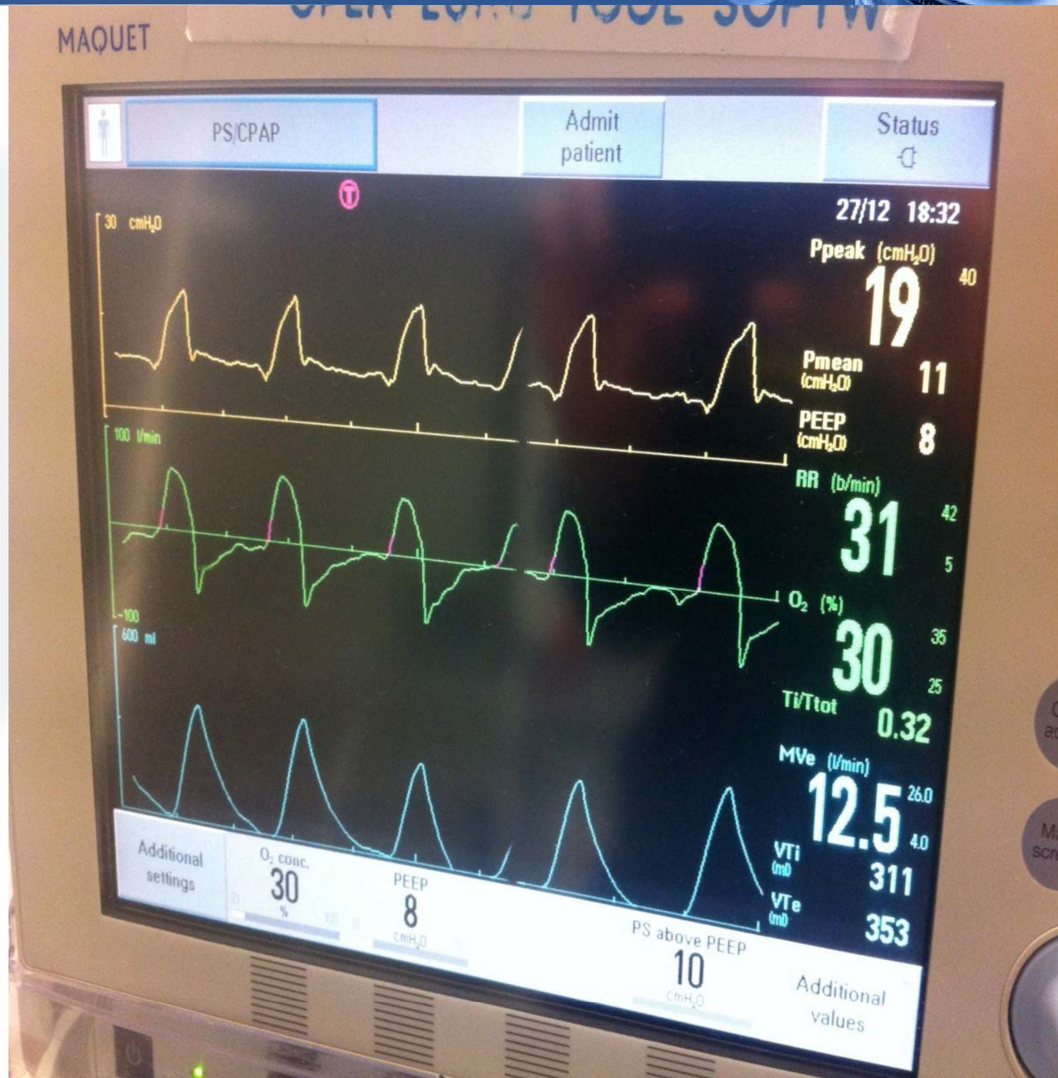


(From Pilbeam SP: *Mechanical ventilation: physiological and clinical applications*, ed 3, St Louis, 1998, Mosby.)

SPONTANEOUS MODES

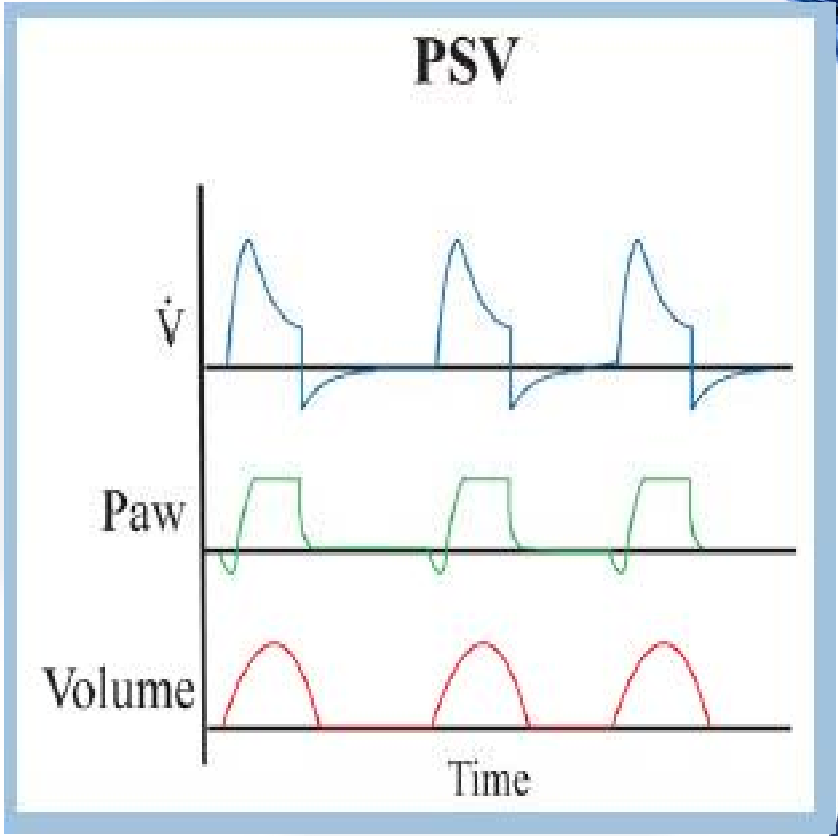
PRESSURE SUPPORT* (PSV):** ΔP/Flow to help patient overcome ***Raw of the ETT/Circuit and also ↓ ***WOB***

Not specifically a "Mode" but can be implemented with any mode the incorporates spontaneous breathing (e.g. SIMV, CPAP/SPON, APRV, MMV, etc)



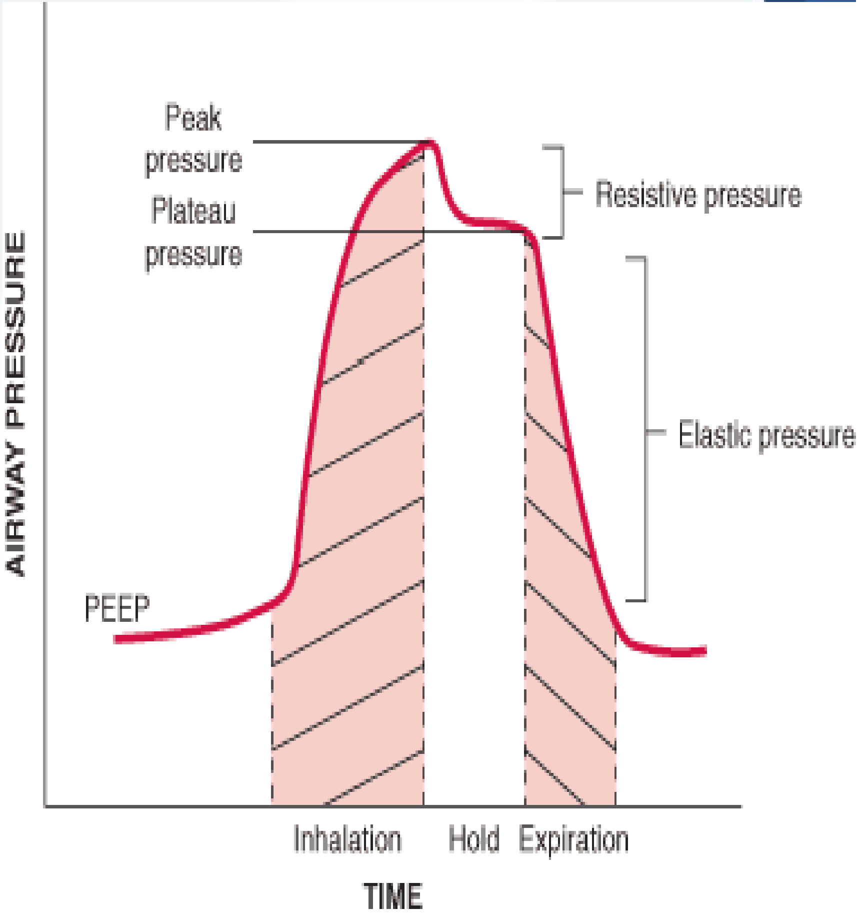
Pressure Support Ventilation (PSV)

- PT CONTROLS TIDAL VOLUME (V_T), INSPIRATORY TIME ($T_{insp.}$) AND INSPIRATORY FLOW
- *MORE COMFORTABLE* FOR PATIENT COMPARED TO THE VENTILATOR CONTROLLING VT, I-TIME AND I-FLOW!!
- *↑'ed Patient:Ventilator synchrony*



Positive End-Expiratory Pressure ("PEEP")

- OPENS CLOSED AIRWAYS AND IMPROVES LUNG VOLUMES
- ↑'s *FUNCTIONAL RESIDUAL CAPACITY* (FRC)
- ALLOWS FOR IMPROVED OXYGENATION AT A LOWER F_IO_2
- USUAL RANGE IS 5-15 cmH20



Positive End-Expiratory Pressure ("PEEP")

http://www.youtube.com/watch?v=hOa7zO1lImI

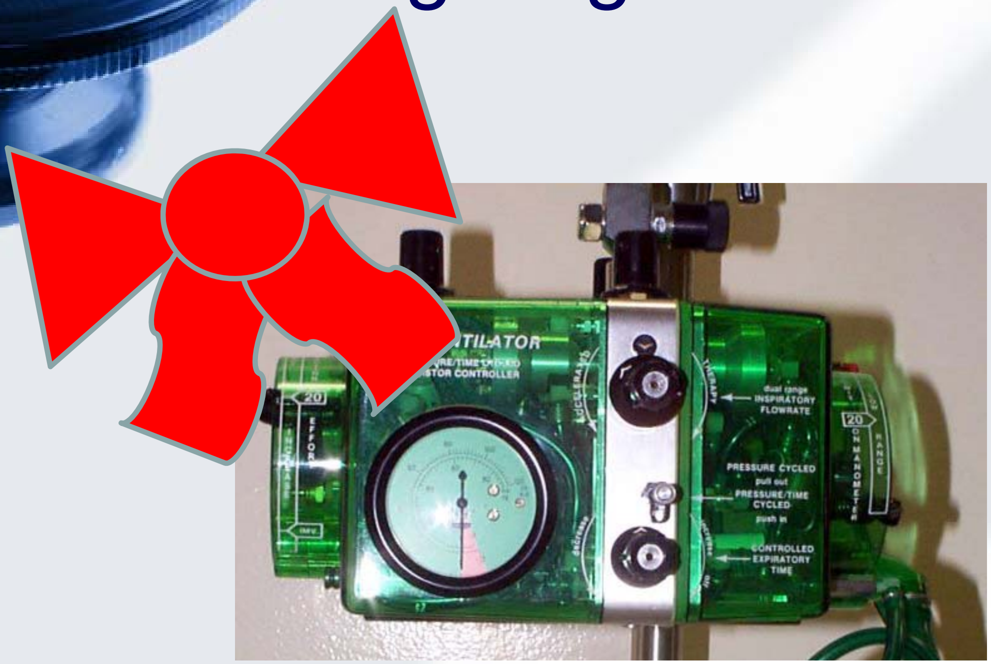
PEEP ↑'s INTRATHORACIC PRESSURE

↓*'s* ***VENOUS RETURN***

THIS EFFECT MAY BE DETRIMENTAL OR MAY BE BENEFICIAL

"OPTIMAL PEEP" : PEEP LEVEL WHICH LEADS TO IMPROVED OXYGENATION WITHOUT HEMODYNAMIC FAILURE

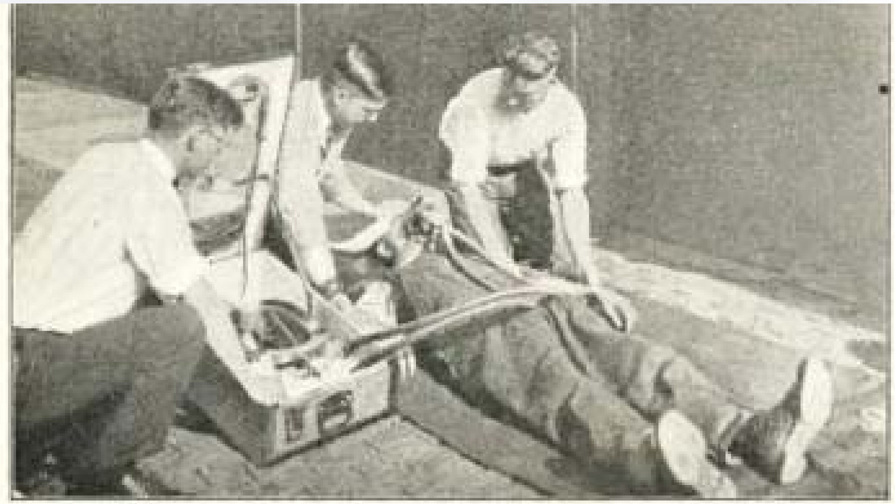
Pressure Control Ventilation
New ventilator mode or "Re-gifting"?



Disclosures

I have a bias towards pressure controlled forms of ventilation…

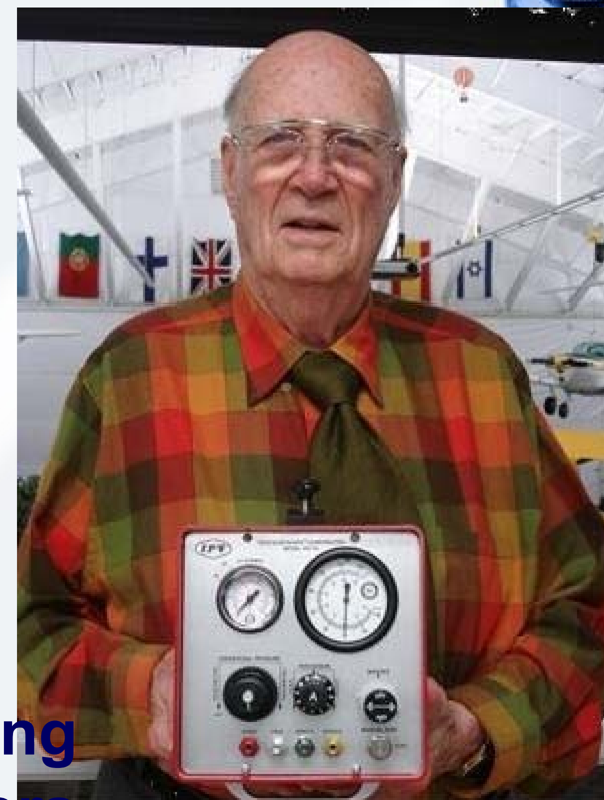
Early Positive Pressure Ventilation
The 1st Generation



BROUGHT BACK TO LIFE

The pulmotor is a machine for supplying oxygen and producing artificial respiration in persons overcome by smoke, gas, electric shock, or drowning. The pictures shows the pulmotor squad of the gas company attempting to revive a man rescued from a gas-filled building. Persons apparently dead are sometimes brought back to life by the pulmotor.

The 2nd Generation
mid 1970's – early 1980's



3 Machine parameters

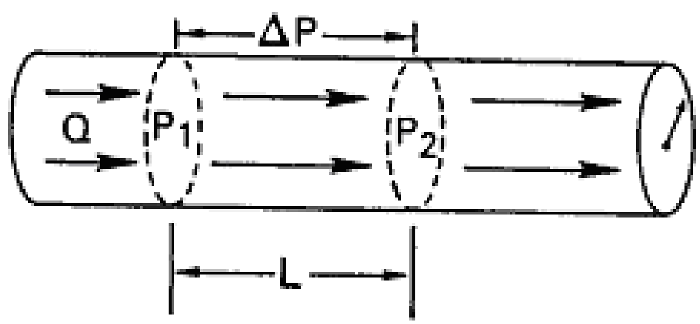
> Pressure
> Flow

2 Lung parameters

> Compliance
> Resistance
> Time

The Mechanics of Pressure Ventilation

Poiseuille's law (1938) : Properties of fluid flow through a tube



$$Q = \frac{\Delta P \, r^4 \, \pi}{\eta L \, 8}$$

Directly proportional to:

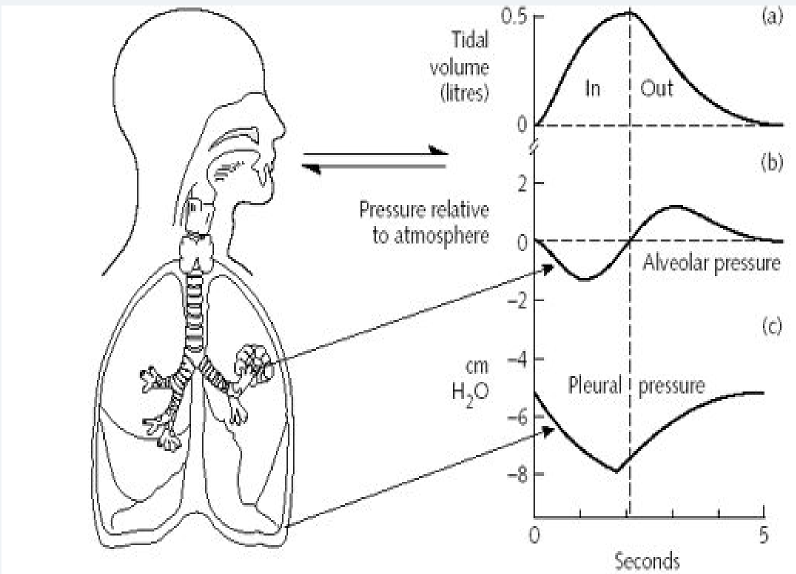
> Pressure gradient
> Radius

Indirectly proportional to:

> Length
> Viscosity

The Mechanics of Pressure Ventilation

During normal respiration:



Flow – highest at start of inspiration because △P is greatest

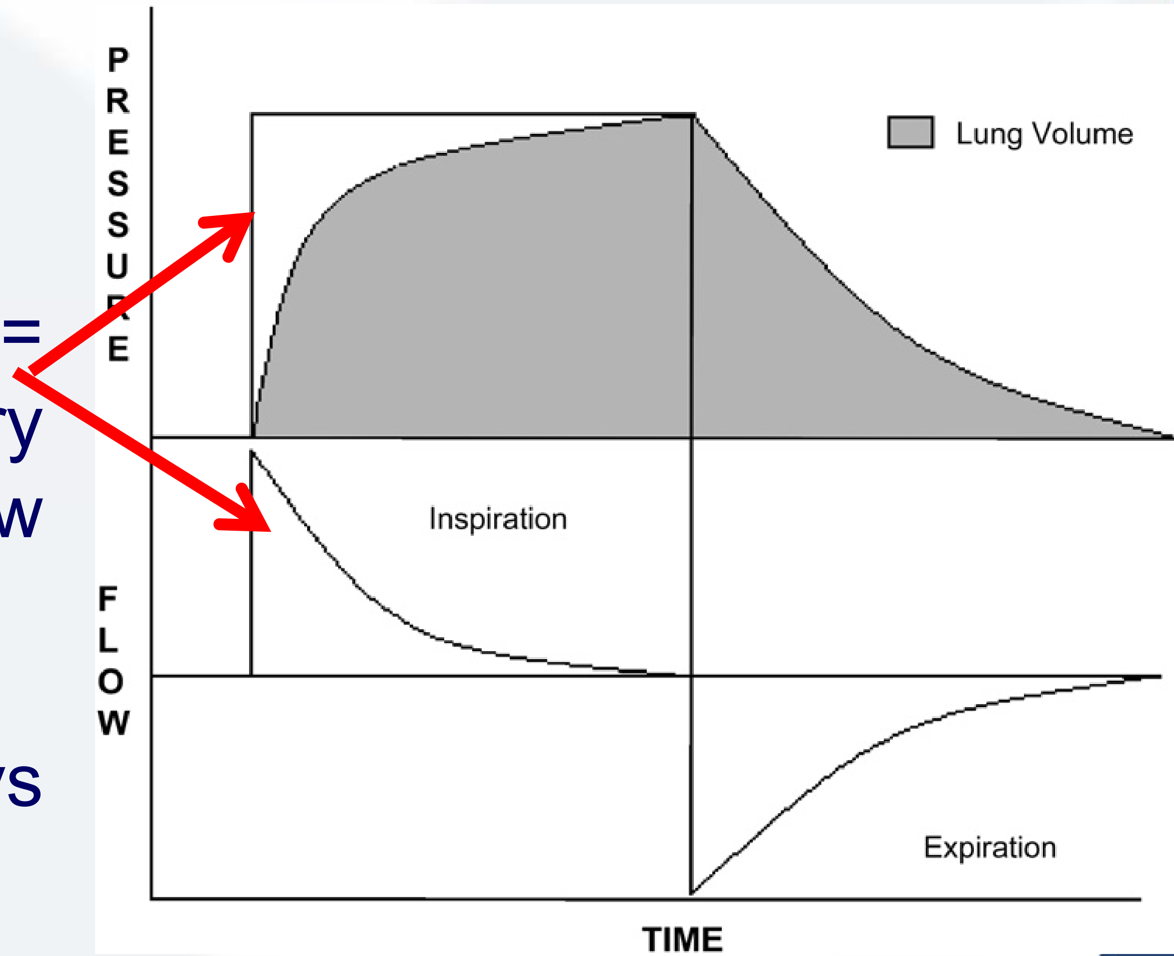
△P decreases when lungs start filling and back pressure develops

As △P decreases, flow decreases – this corresponds to smaller AW filling

The Mechanics of Pressure Ventilation

High ΔP = high inspiratory flow

Large airways



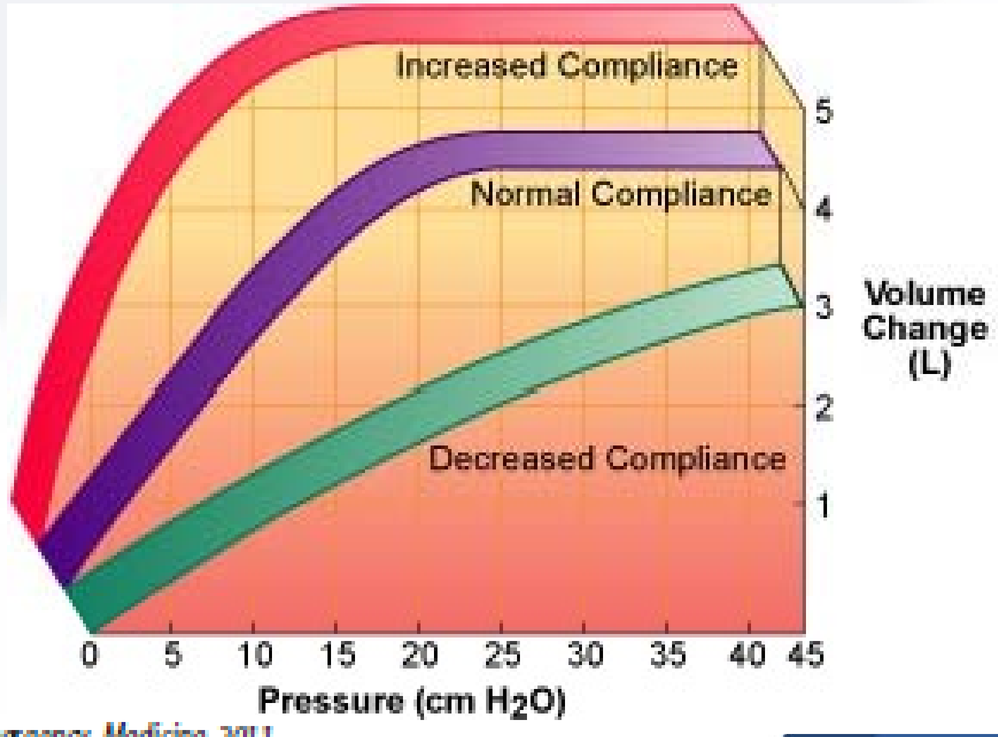
Nichols & Haranath Crit Care Clin 23 (2007) 183–199

The Mechanics of Pressure Ventilation

Relationship of pressure, flow and time to compliance and resistance

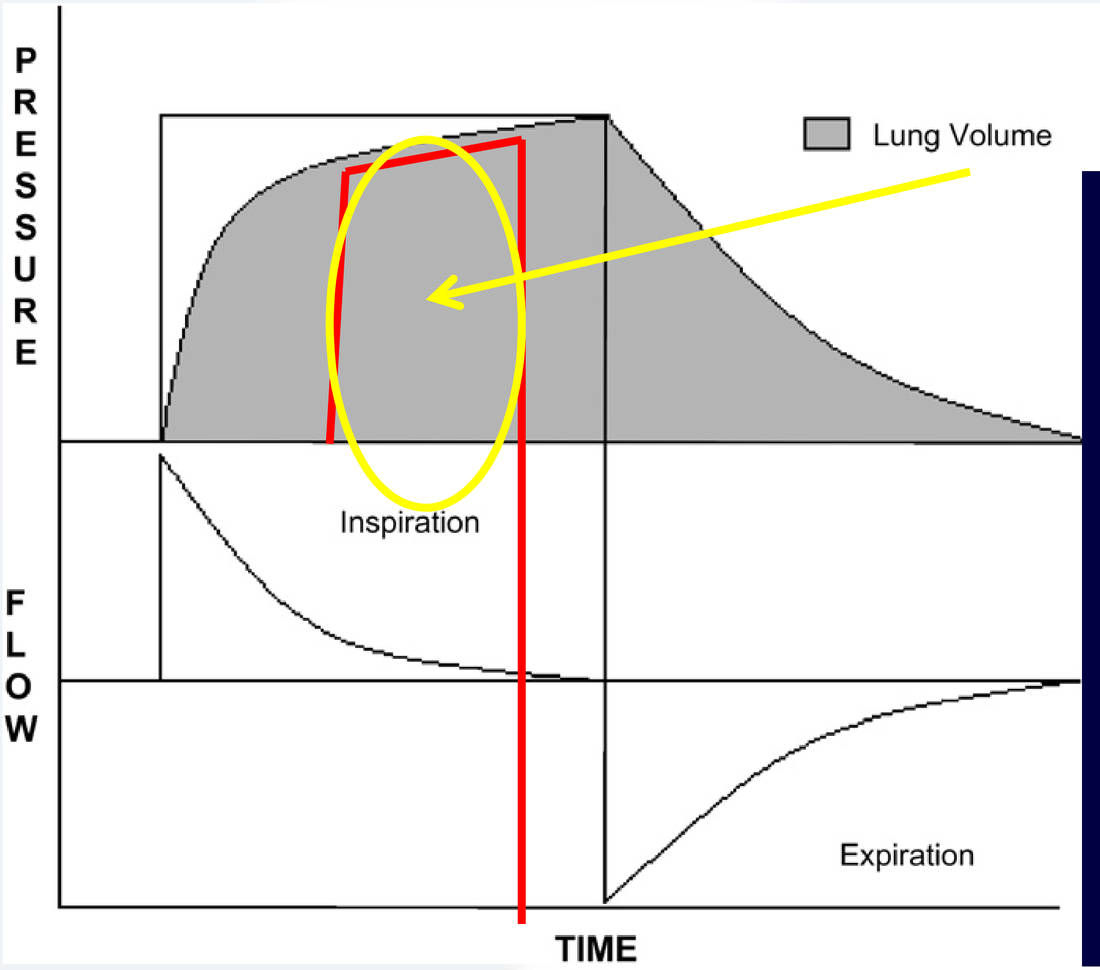
$C_L = \Delta P / \Delta V$

(flow/time)



J.-L. Vincent (ed.), *Annual Update in Intensive Care and Emergency Medicine 2011*

The Mechanics of Pressure Ventilation



Back pressure causes pressure gradient to degrade quickly, decreasing volume delivered

Nichols & Haranath Crit Care Clin 23 (2007) 183–199

The Mechanics of Pressure Ventilation

Decreased C_L (↓ Volume)

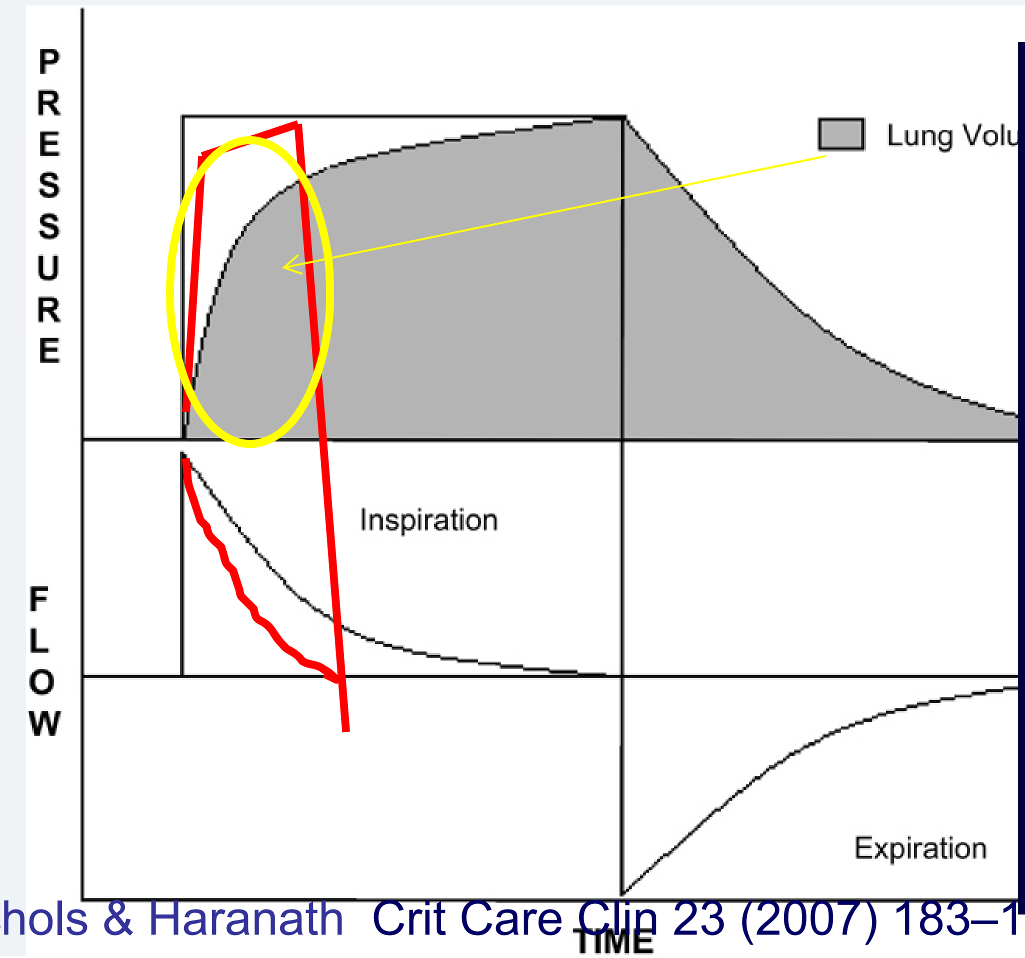
- Pulmonary Edema
- Pneumonia
- Atelectasis
- Fibrosis
- ARDS
- Abdominal pressure
- Bandages
- Burns
- Pain/discomfort

Increased C_L (↑ Volume)

- Post diuretic
- Recruitment / PEEP
- Emphysema
- Positioning
- Gastric decompression
- Pain control
- Paralysis

The Mechanics of Pressure Ventilation

$R_{AW} = \triangle P / \dot{V}$ (volume / time)



Turbulent flow through airways causes back pressure causes pressure gradient to degrade quickly, decreasing volume

Nichols & Haranath Crit Care Clin 23 (2007) 183–199

The Mechanics of Pressure Ventilation

Increased RAW
(↓ Volume)

- Bronchospasm
- Secretions
- Small ETT
- Decreased lung volumes
- Malacia/stenosis
- Kinked tubing
- Water in circuit

Decreased RAW
(↑ Volume)

- Bronchodilators
- Suctioning
- Larger ETT
- Recruitment
- PEEP
- Size of circuit (neo/pede/adult)

Types of Pressure Ventilation

Alphabet Soup:

Pressure Cycled Ventilation

Pressure Control Ventilation

Pressure Limited Ventilation

Pressure Support Ventilation

Pressure/Volume "Hybrid" Ventilation

MacIntyre, CHEST / 140 / 2 / August,2011

Types of Pressure Ventilation

Pressure Cycled Ventilation:

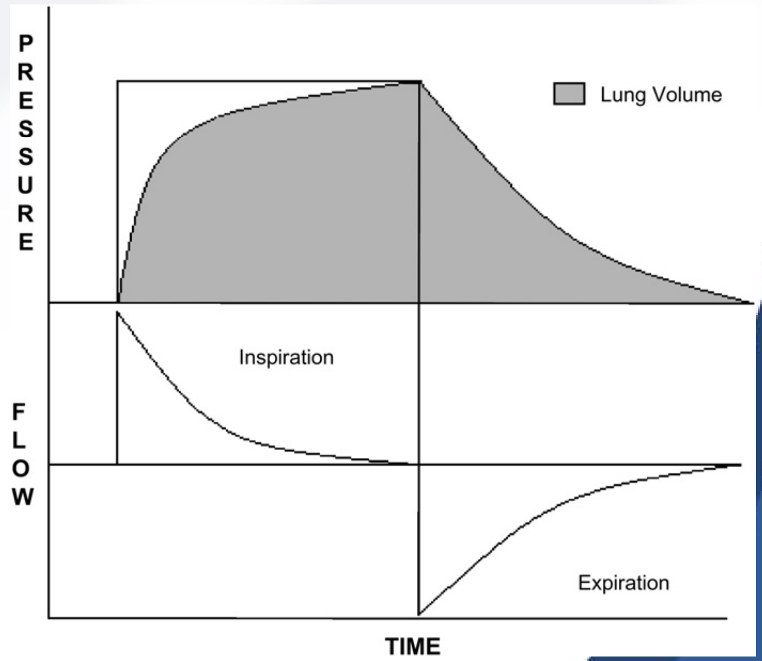
Ventilator cycles into expiration when target pressure is reached.

Flow is high at start insp.

Decelerating flow.

Inspiratory time variable

A/C or SIMV



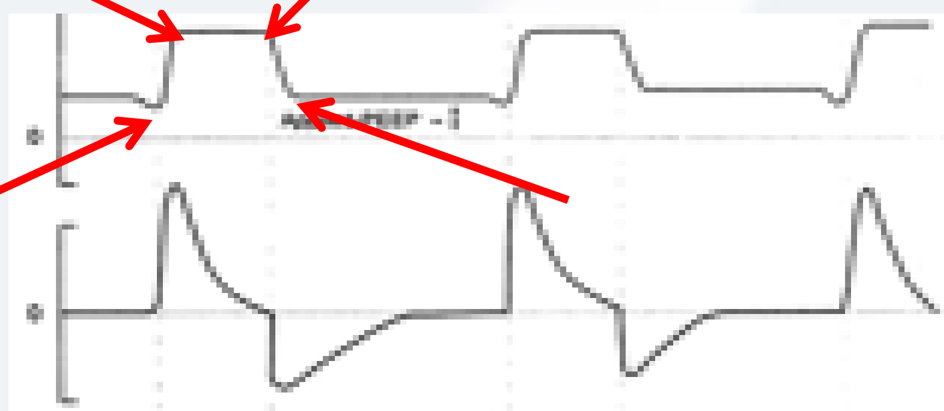
Core Topics in Mechanical Ventilation, ed. Iain Mackenzie. Cambridge University Press 2008.

Types of Pressure Ventilation

Pressure Controlled Ventilation:

Pressure is one of the phase variables (usually in combination with time)

Flow is variable (demand flow)

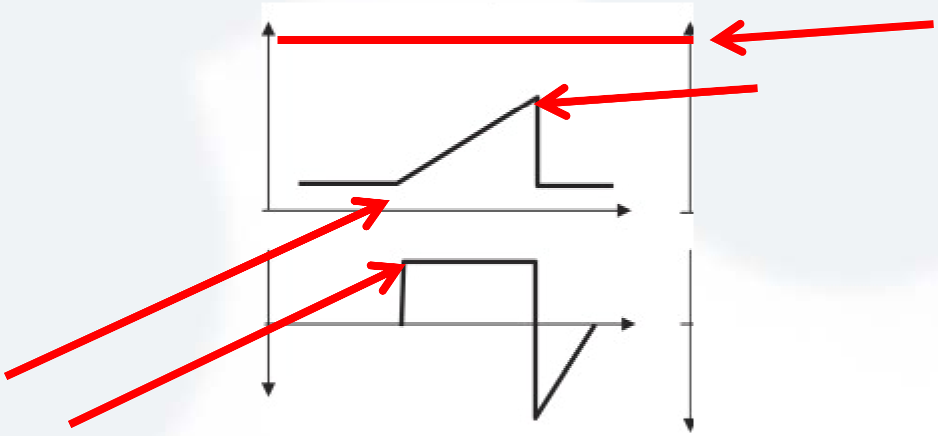


Core Topics in Mechanical Ventilation, ed. Iain Mackenzie. Cambridge University Press 2008.

Types of Pressure Ventilation

Pressure Limited Ventilation:

Volume is the controlling phase variable, but a pressure limit is set to decrease potential for increases in PIP



Core Topics in Mechanical Ventilation, ed. Iain Mackenzie. Cambridge University Press 2008.

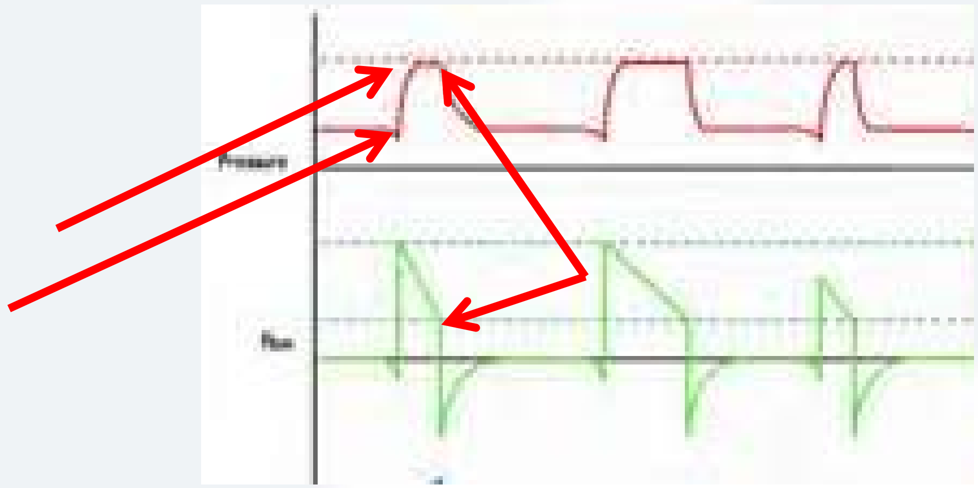
Types of Pressure Ventilation

Pressure Support Ventilation:

Applied to spontaneous breaths.

Pressure controlled. Flow cycled.

Variable inspiratory time. Variable Insp. Flow



Core Topics in Mechanical Ventilation, ed. Iain Mackenzie. Cambridge University Press 2008.

Types of Pressure Ventilation

Pressure/Volume Hybrid Ventilation: PRVC, VG

Volume is set. Pressure limit is set.

Inspiratory Time set.

Decelerating flow pattern.

Inspiratory Flow variable to achieve volume within set pressure limit and inspiratory time.

Core Topics in Mechanical Ventilation, ed. Iain Mackenzie. Cambridge University Press 2008.

Types of Pressure Ventilation

Pressure ventilation definition depend on:

- Is pressure the controlling factor or is it an overall pressure limit?
- How the vent cycles into expiration (pressure, time or flow)

Pressure ventilation characteristics:

- Tidal volume dependant on compliance and resistance
- Decelerating flow

Use of Pressure Ventilation

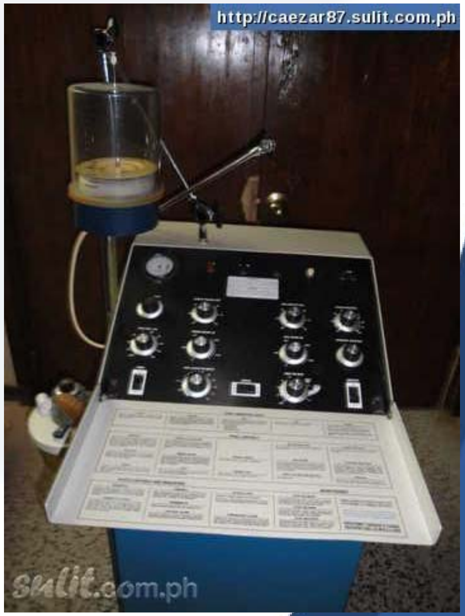
Early 1980s – 1990s 2nd generation ventilators:

Assist/Control and early IMV/SIMV modes

Volume monitoring (bellows, pneumotachs)

Integration of PEEP

“Volume” ventilation

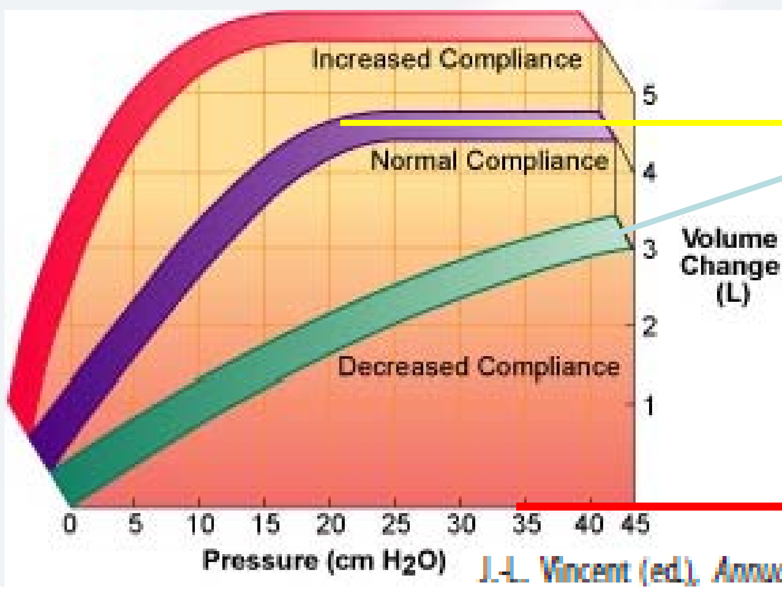


Use of Pressure Ventilation

Perceived benefits to volume ventilation:

- Consistent tidal volume – consistent ventilation

$C_L = \triangle P / \triangle V$ (flow/time)



J.-L. Vincent (ed.), *Annual Update in Intensive Care and Emergency Medicine 2011*

Use of Pressure Ventilation

Perceived benefits to volume ventilation:

- Consistent tidal volume – consistent ventilation

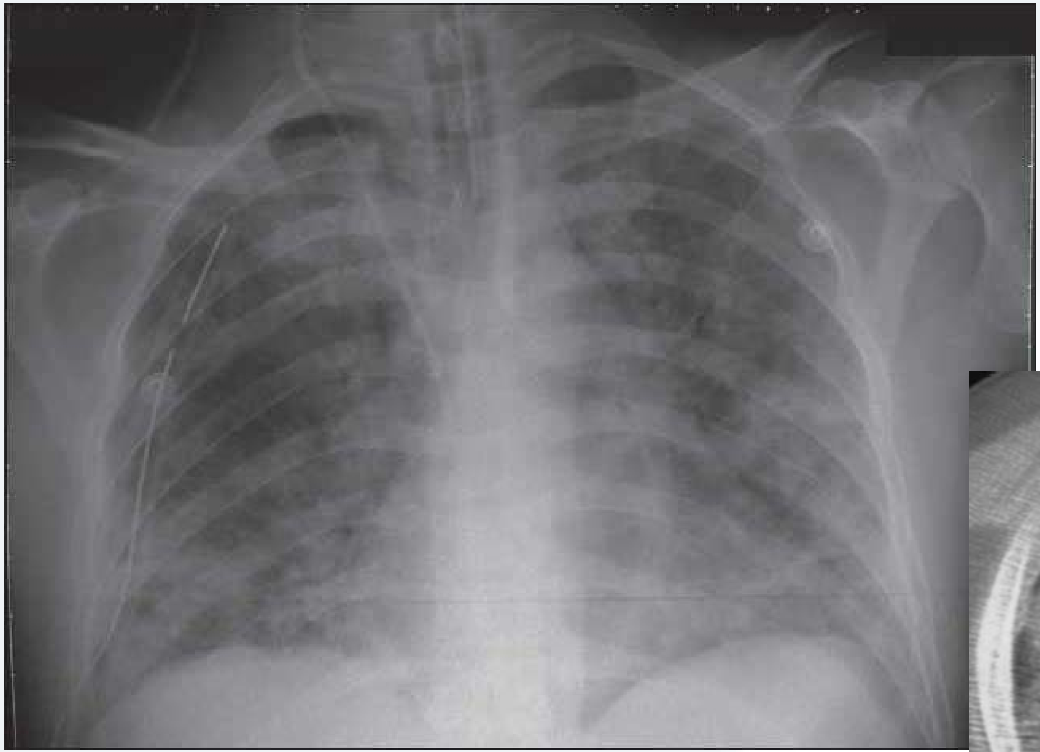
$R_{AW} = \triangle P / \triangle V$ (volume / time)

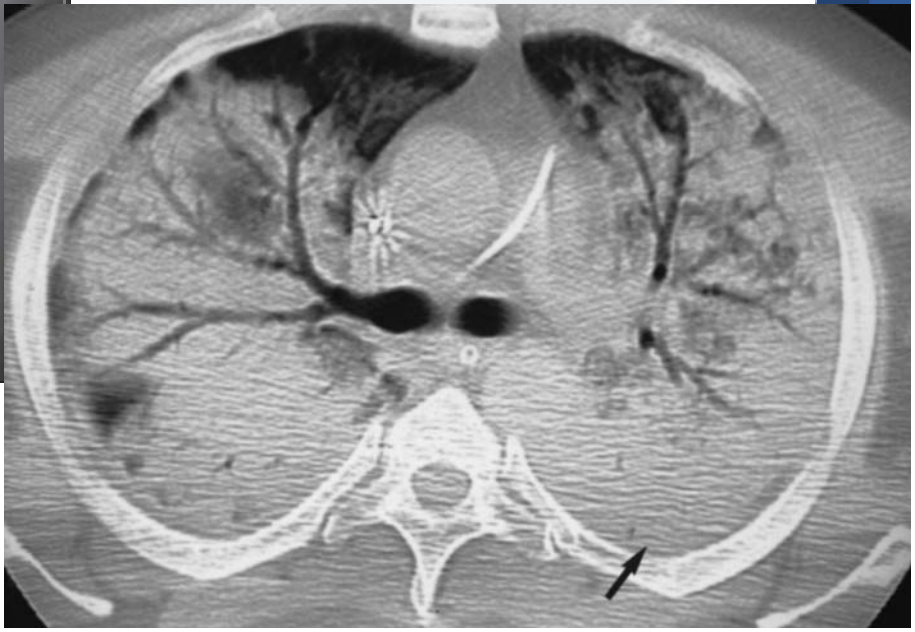
Increase PIP to overcome airway resistance

J.-L. Vincent (ed.), *Annual Update in Intensive Care and Emergency Medicine 2011*

Use of Pressure Ventilation

ARDS





http://www.ardsnet.org/

Use of Pressure Ventilation

Pressure ventilators of the day at a disadvantage:

- Limited capacity for PEEP (external)
- Limited maximum driving pressure
- Limited inspiratory time control
- Non-heated humidity
- Variable volumes, variable ABGs

Use of Pressure Ventilation

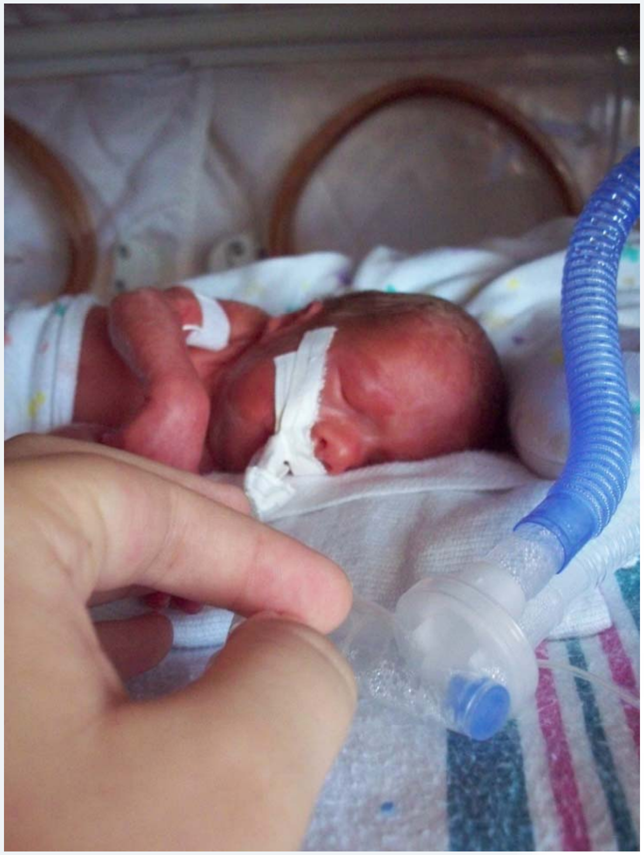
Thompson et al: Ventilator strategies in ARDS

- ARDSnet 1996-1999
- “low volume strategies”
- > 10% using PCV

CHEST / 120 / 5 / NOVEMBER, 2001

Use of Pressure Ventilation

Main area of use for pressure ventilation:



Use of Pressure Ventilation

1990s – early 2000s 3rd Generation Ventilators:

Introduction of microprocessor technology

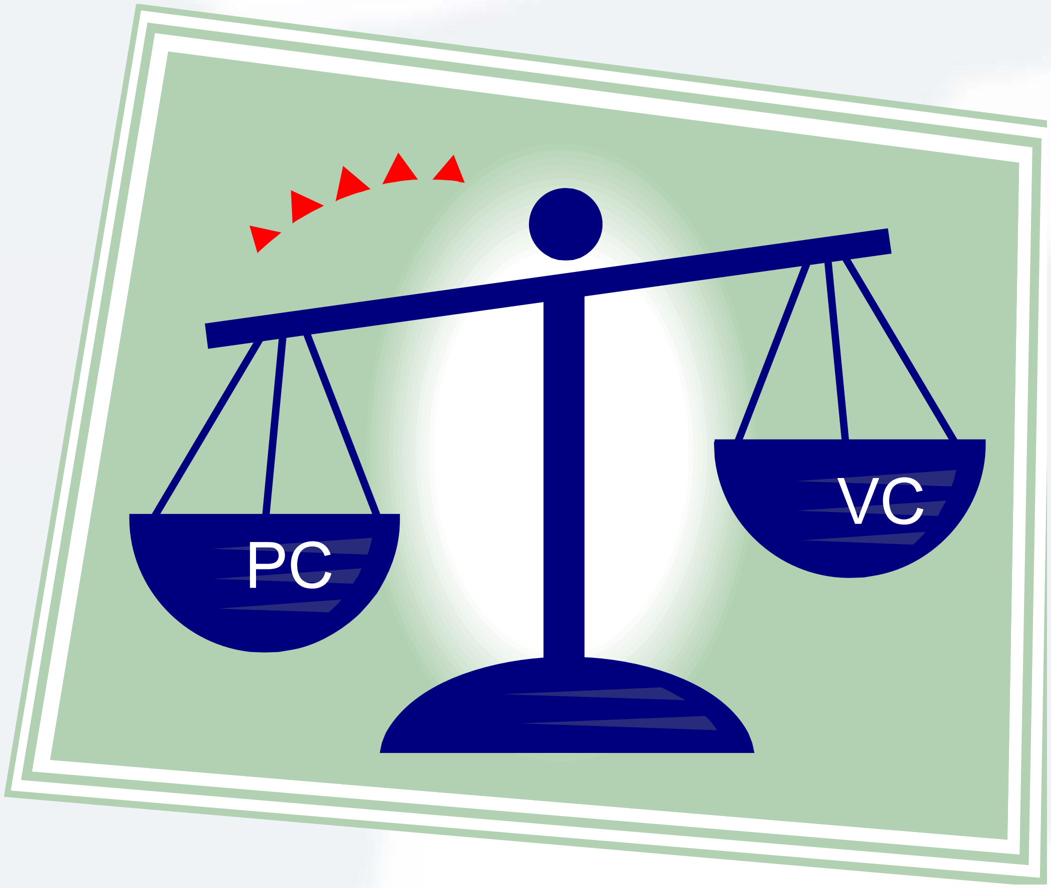
Increased responsiveness

Improved monitoring and alarms

New "modes" – pressure ventilation reintroduced as PC and PS

Renewed interest in PC ventilation

Use of Pressure Ventilation



Use of Pressure Ventilation

Davis et al: examined the hypothesis that the decelerating inspiratory flow waveform associated with PCV provides improvement in gas exchange in ALI.

• Prospective, controlled, crossover study.

•Compared PVC and VCV with decelerating flow waveform.

Improved oxygenation at lower PIP occurred with both PVC and VCV with decelerating flow waveform.

Hypothesis: the decelerating flow waveform is the key to improving oxygenation regardless of controlling variable.

Journal of Trauma-Injury Infection & Critical Care: 1996 - Volume 41 - Issue 5 - pp 808-814

Use of Pressure Ventilation

Guldager et al: PIP during PCV vs VCV

Peak inspiratory pressure was significantly lower during PCV ventilation than during VC ventilation,

Hypothesis: PCV may be superior to VC in certain patients.

However did not demonstrate improved outcome.

Guldager et al. Critical Care 1997, 1:75

Use of Pressure Ventilation

Kalet et al, 2000; WOB during PCV vs VCV

- Randomized crossover trial
- Measured lung mechanics
- PCV “significantly” decreased WOB during mechanical ventilation

Hypothesis – high inspiratory flow/demand flow decreased WOB compared to VCV fixed flow.

Kalet et al, RESPIRATORY CARE , 2000 VOL 45 NO 9

Use of Pressure Ventilation

Chiumello et al: WOB in patients on VCV vs PCV

- When the peak inspiratory flow of VCV was adjusted properly to support a given V t , there were no differences in WOB

Hypothesis: If ventilator parameters (peak flow, V t) are adjusted properly , VCV can produce patient ventilator synchrony similar to PCP.

Respir J . 2002 ; 20 (4): 925 - 933

Use of Pressure Ventilation

Mid 2000s - ??? 4th Generation Ventilators

- Dual control hybrid modes
- Wide variety of ventilators
- Pressure control available in different forms
- Volume targeting for neonates
- No standardized terminology, categorization of ventilators or modes - ***confusion***

Use of Pressure Ventilation

Meade et al: Largest trial of VCV vs PCV

- 983 patients
- Randomized to Standard VCV or PVC with "open lung" strategy (High PEEP, recruitment maneuvers, higher allowable P_{PLAT}
- No difference in all cause mortality
- Improvement in oxygenation and decreased use of adjunctive treatments in PCV group

JAMA. 2008;299(6):637-645

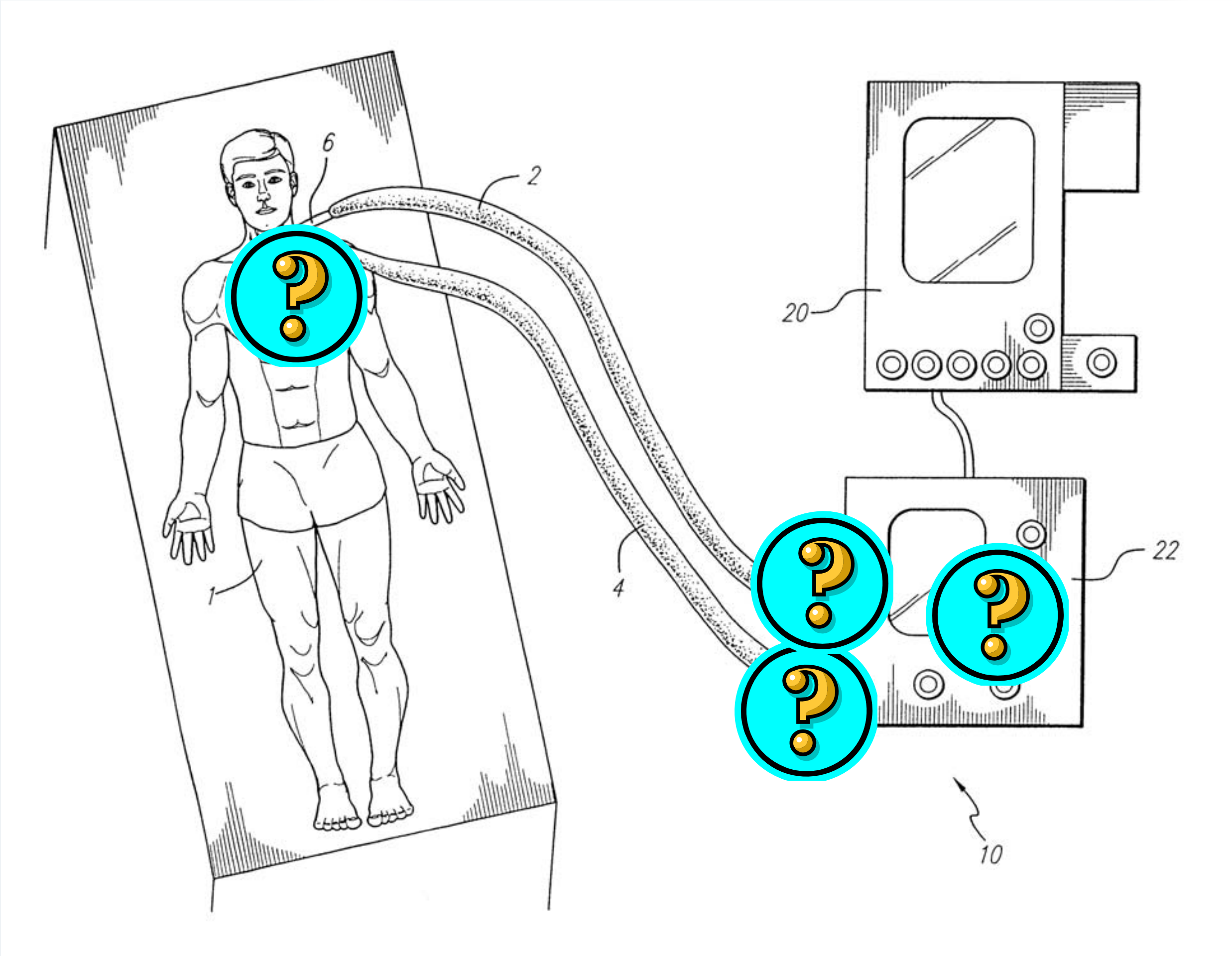
Use of Pressure Ventilation

At the end of the day, there are very few studies that directly compare flow/volume-targeted and pressure targeted strategies for lung-protective ventilation in ARDS, and most have confounding issues.

MacIntyre CHEST, 140(2) AUGUST, 2011

Use of Pressure Ventilation

Where are pressure and volume parameters measured?



Use of Pressure Ventilation

As emphasized in the 1993 American College of Chest Physicians Consensus Conference on Mechanical Ventilation;

“*although the quantitative response of a given physiologic variable may be predictable, the qualitative response is highly variable and patient specific*”

Nichols & Haranath Crit Care Clin 23 (2007) 183–199

Parting thoughts

Pressure control ventilation can be a lot of work

- *Really* need to know your vent

  > Is pressure control ventilation combined with A/C or SIMV? Are spontaneous breaths supported?

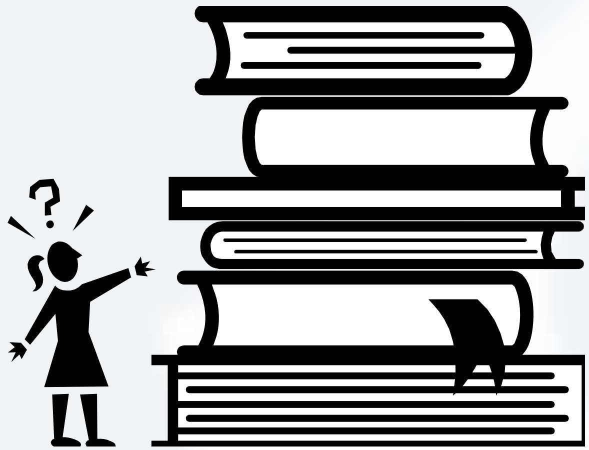
  > Are breaths pressure cycled, pressure controlled, pressure limited, flow cycled, dual control?

  > What are the flow limitations, is it demand flow, can it be changed

  > How does Inspiratory time change volumes, mean airway pressure and oxygenation?

  > Where are pressure and volume parameters measured?

Questions???



Non-Invasive Ventilation

NIV - Definition

Application of ventilatory support without an artificial airway

Interfaces can include nasal and full face masks, nasal prongs, full helmets

Equipment can range from simple flow generators to mechanical ventilators with NIV modes

Why NIV?

Avoid complications related to intubation and invasive mechanical ventilation:

- Loss of airway defense mechanisms – Aspiration during intubation, aspiration of oral secretions
- Upper airway/larygeal/tracheal trauma
- Arrythmias and hypotension
- Increased risk of Barotrauma/Volutrauma
- Direct conduit to lower airway for pathogens
- Difficult to wean/extubate patients
- Intubation inappropriate or refused

Why NIV?

Complication that occur after removal of ETT

- Hoarseness, sore throat, cough
- Upper airway obstruction
- Muscle weakness – upper airway/thoacic and diaphragm

From the patient's point of view

- Discomfort
- Decreased ability to eat and communicate
- Intubation not wanted

Why NIV?

Advantages of NIV

- Leaves upper airway intact
- Preserve airway defense mechanisms
- Allows patient to eat, drink, verbalize and expectorate
- Enhance comfort, convenience and portability
- Less cost ?????????

Types of NIV

Negative Pressure NIV

One pressure – CPAP

Two pressures – BiPAP, BiLevel, SiPAP. NIPPV

Types of NIV

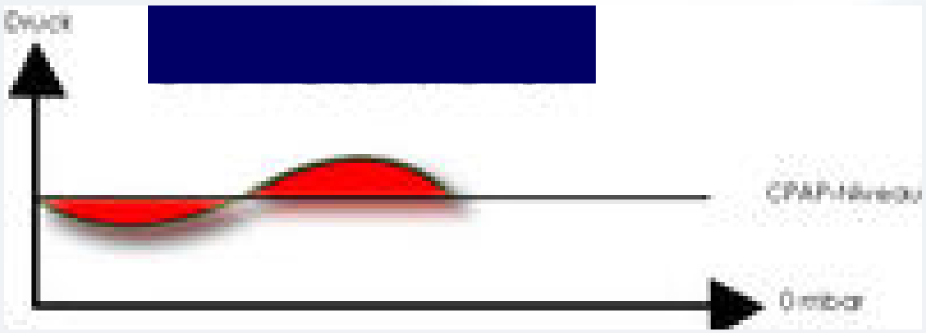
Negative Pressure Devices

- Iron lung, chest cuirass

“One Pressure” -CPAP

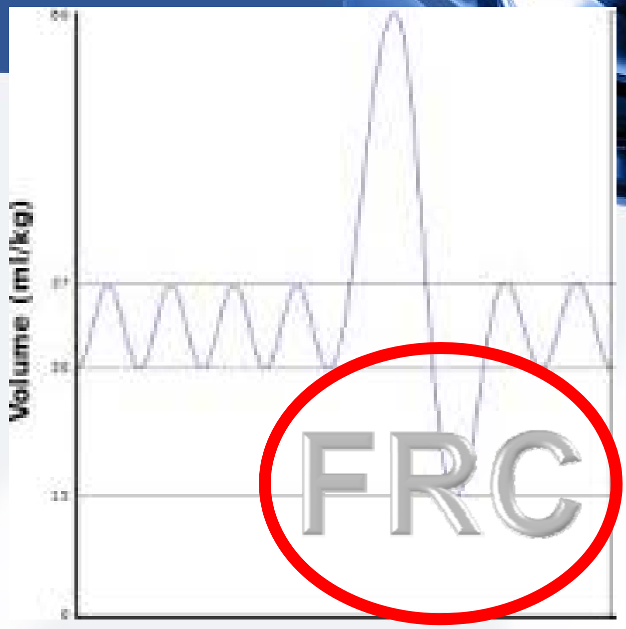
- “Continuous Positive Airway Pressure”
- Maintains upper airway patency – sleep apnea
- Reduces work of breathing
- Increases FRC – increases functional area for gas exchange.
- Can “splint” airways prone to collapse or stenosed

“One Pressure” -CPAP



- Flow must be high enough to prevent loss of continuous pressure
- Demand flow
- Assisted or triggered exhalation
- Leak compensation

Indications for CPAP



- Obstructive Sleep Apnea
- Airway malacia
- Apnea of Prematurity
- Pulmonary Edema
- RDS in neonate
- Pneumonia
- Post extubation

Best candidates for CPAP

- Conscious and co-operative
- Able to protect airway
- Intact respiratory drive
- No excessive secretions
- Sick but not moribund
- Haemodynamically stable
- Few co-morbidities
- Able to fit mask/prongs/other interface

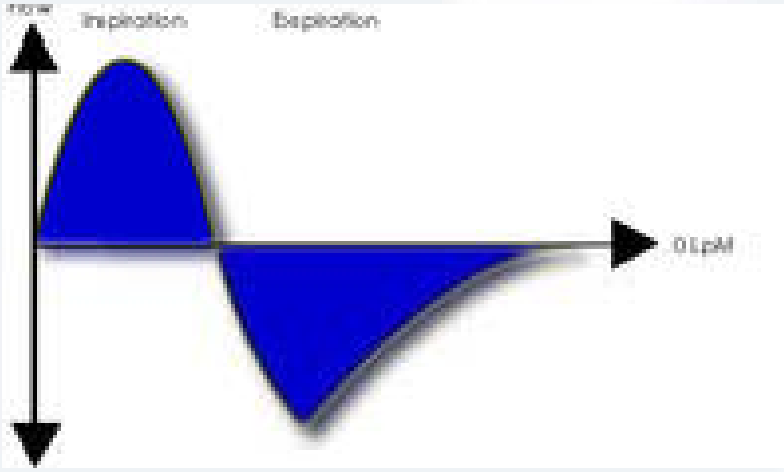
Two Pressures:
BiPAP, BiLevel, SiPAP, NIPPV

- Two levels of support pressure
- Positive pressure in inspiration and end expiration
- IP augments patients own inspiration
- EP = CPAP/PEEP to prevent loss of FRC
- Helps to reduce WOB and increase alveolar ventilation

Two Pressures:
BiPAP, BiLevel, SiPAP, NIPPV

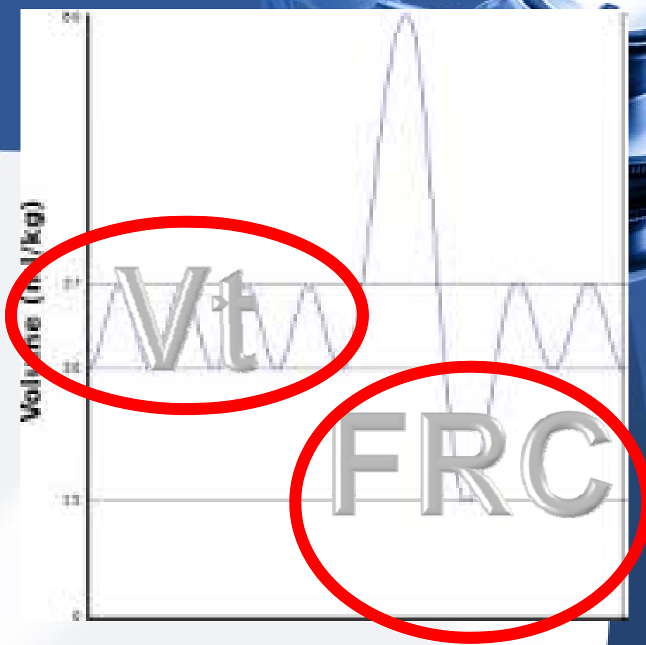
- Cycling inspiration can be patient triggered (spontaneous), timed (T) or a combination of the two (S/T)
- Cycling expiration can be by flow, pressure or time.

Two Pressures:
BiPAP, BiLevel, SiPAP, NIPPV



- Inspiratory flow capability is dependant on the device – some have features such as adjustable ramping, variable flow

Indications for BiPAP

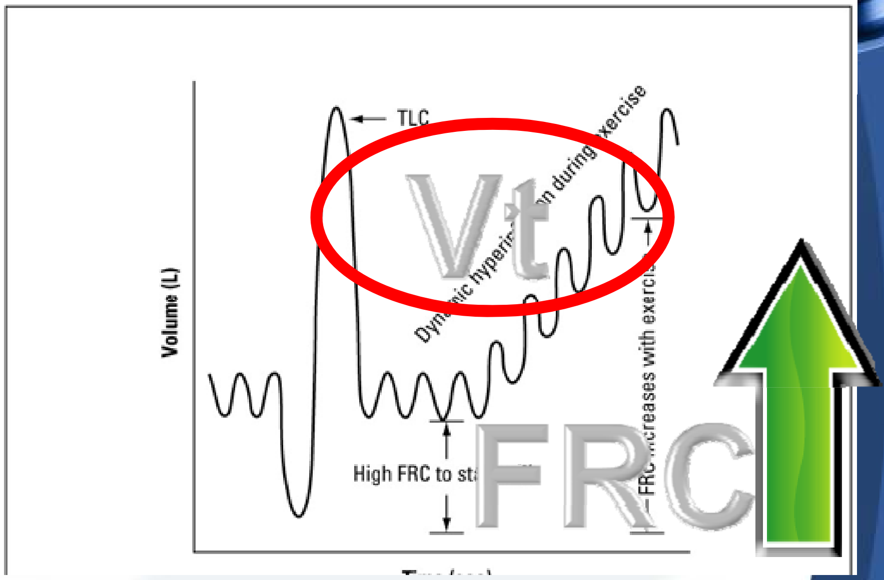


- Failure of CPAP
- Therapeutic trial
- Respiratory failure with chest wall deformity or neuromuscular disease
- Pneumonia with respiratory acidosis
- Post-extubation
- Post-op

Indications for BiPAP

Cautions

- Exacerbation of COPD
- Exacerbation of Asthma

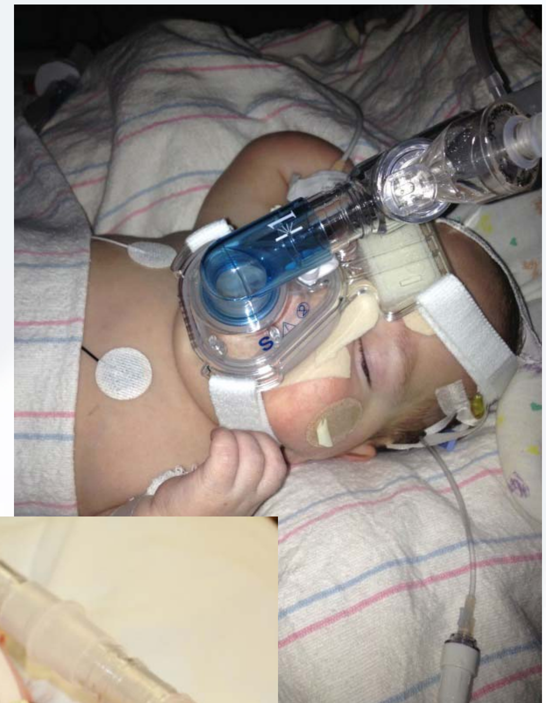


Unsuitable candidates for NIV

- Respiratory arrest
- Haemodynamically unstable
- Uncooperative
- Unable to protect airway
- Facial, oesophageal, or gastric surgery
- Craniofacial trauma or burns
- Lower airway obstruction with advanced hyperinflation
- Untreated physiological air leaks - Pneumothorax

NIV Interfaces

NIV Interfaces



NIV Failure

- In appropriate candidate for NIV
- Poor ventilation secondary to leaks
- Device not suitable for patient’s needs
- Gastric distension
- Patient intolerance
- High level of surveillance required

NIV Complications and cautions

- Gastric distension
- Skin breakdown
- Abnormal facial bone growth
- Alarm issues – must meet safety requirements for ventilatory support device

HHFNC – the new kid on the block

Normally flow delivered by NP is limited by patient discomfort, nasal mucosal damage, increased turbulent gas flow

- HHFNP came on the market in the early 2000s
- Heated, humidified gas allowed much higher flow via nasal prongs

Table 3. Variables in Evaluating Cannula Systems

Factors Directly Affecting the Volume of Oxygen Inhaled Via Cannula
Gas flow
Concentration of oxygen from the flow meter
Volume inspired
Respiratory rate
Total respiratory time
Inspiratory time
Inspiratory flow
Patient inspiratory flow pattern
Additional Factors That May Cause Air Dilution of the Inspired Oxygen Volume
Proportion of room-air versus system air (eg, open-mouth or closed-mouth inhalation)
Volume of anatomic airways serving as a gas reservoir
Proportion of dead-space gas rinsing
Secondary effect of distending expiratory threshold pressure or CPAP, which is affected by
— Gas Flow
— Resistance characteristics of the patient airways, including open mouth
— Outside diameter of cannula, compared to lumen dimensions of nares

HHFNC

HHFNC

How/Why does this work?

- Heated, humidified gas reduces resistance to air flow through upper airway
- Heated, humidified gas reduces energy cost of "conditioning" inspired gas
- High inspiratory flow washes out anatomical dead space – creates reservoir and reduces total dead space
- May have more consistent FiO2
- Reduced WOB
- Creation of positive pressure

HHFNC

Adult Literature

- Patient comfort
- Consistency of FiO_2
- PaO_2 and SpO_2 increased
- Fewer treatment failures
- Pressure generated was flow dependent

Pediatric/Neonatal Literature

- Reintubation rates in neonates equivocal – more recent literature favour HHFNC
- Pressure generated varied widely based on methodology
- Longer stay in O_2
- Not as well studied in pediatrics – some reduction in intubation for bronchiolitis

Pros and Cons

Best candidates

- Same criteria as for CPAP and BiPAP
- Minimal reduction in FRC

Cautions

- Lower airway obstruction – air trapping
- CO2 washout in patients with low respiratory drive
- Easy to think “just nasal prongs”
- Easy to ignore

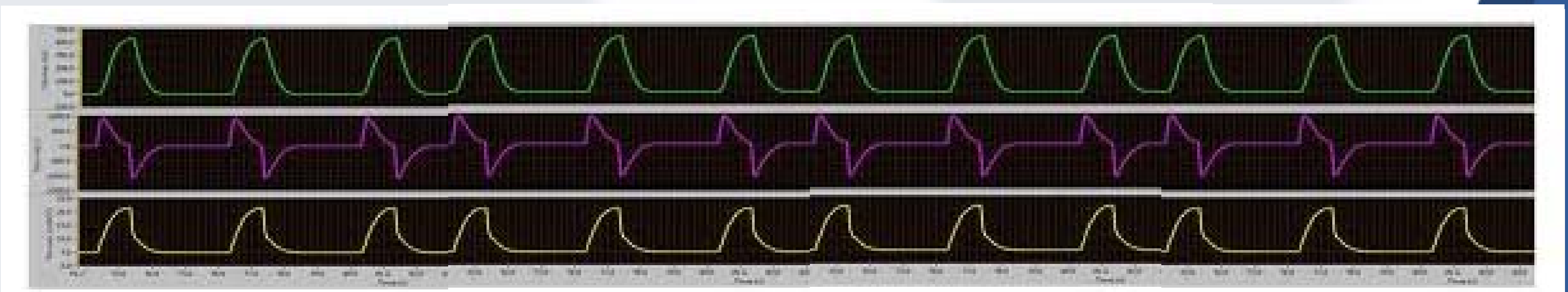
References

Pediatrics, 2013 May;131(5):e1482-90. doi: 10.1542/peds.2012-2742.

RESPIRATORY CARE • JANUARY 2013 VOL 58 NO 1

Intensive Care Med (2013) 39:247–257
DOI 10.1007/s00134-012-2743-5

Ventilator Waveforms



Purpose of Ventilator Waveforms

- Allows the clinician to see what's happening "on the inside"
- Allows the clinician to tailor ventilator settings to meet patient needs
- Allows the clinician to detect undesirable ventilatory patterns and alarm conditions
- Provides visual confirmation of the results of ventilator parameter changes

Types of Ventilator Waveforms

Waveforms that are plotted against time: SCALARS

> Pressure - Time

> Flow - Time

> Volume - Time

Waveforms where one parameter is plotted against another parameter: LOOPS & CURVES

> Flow - Volume

> Pressure - Volume

Types of Ventilator Waveforms

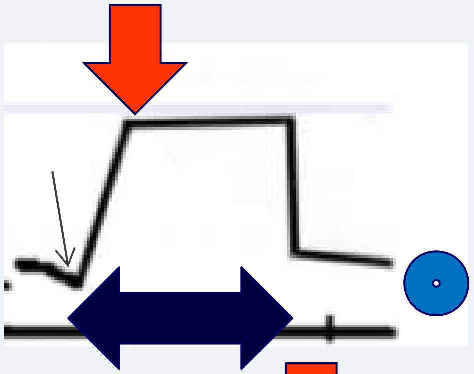
Waveforms that are plotted against **time**:

SCALARS

> Pressure - Time
> Flow - Time
> Volume – Time

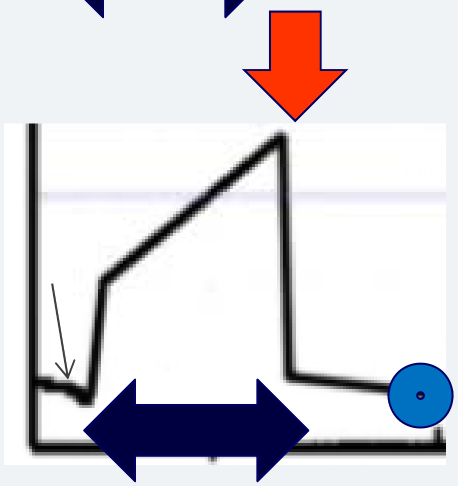
Ventilator Waveforms Plotted Against Time

Pressure – Time Waveforms



Pressure Control

Square Wave



Volume Control

Shark fin

Peak Inspiratory Pressure (PIP)

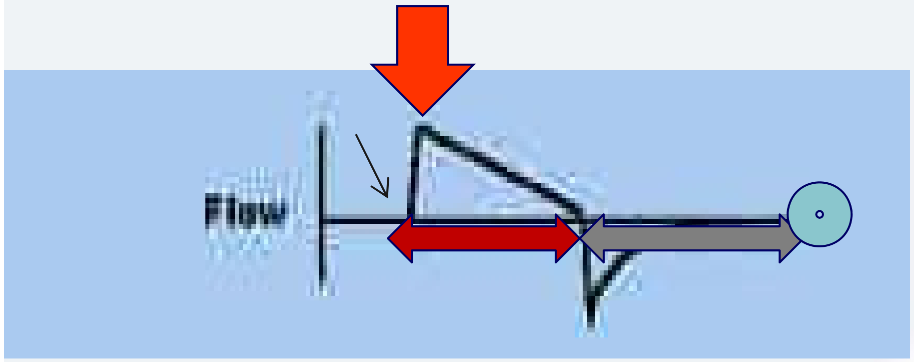
Inspiratory Time

Triggering

PEEP

Ventilator Waveforms Plotted Against Time

Flow – Time Waveforms



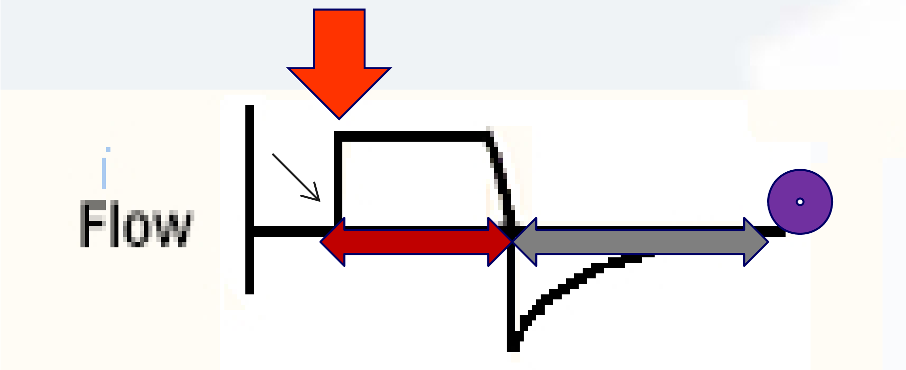
Pressure Control

Decelerating flow

Peak Inspiratory Flow(PIF)

Inspiratory Time

Expiratory Time



Volume Control

Square Wave

Triggering

End Expiratory Flow

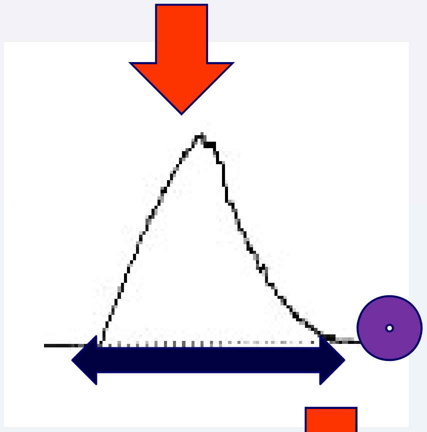
Ventilator Waveforms Plotted Against Time

Volume – Time Waveforms

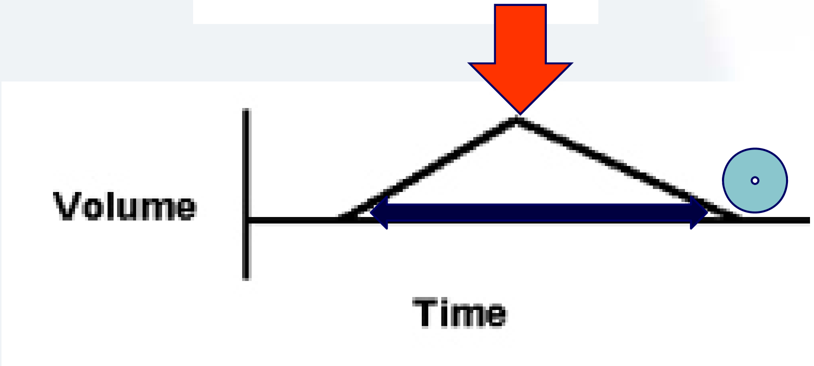
Tidal Volume

Inspiratory Time

End Expiratory Volume



Pressure Control



Volume Control

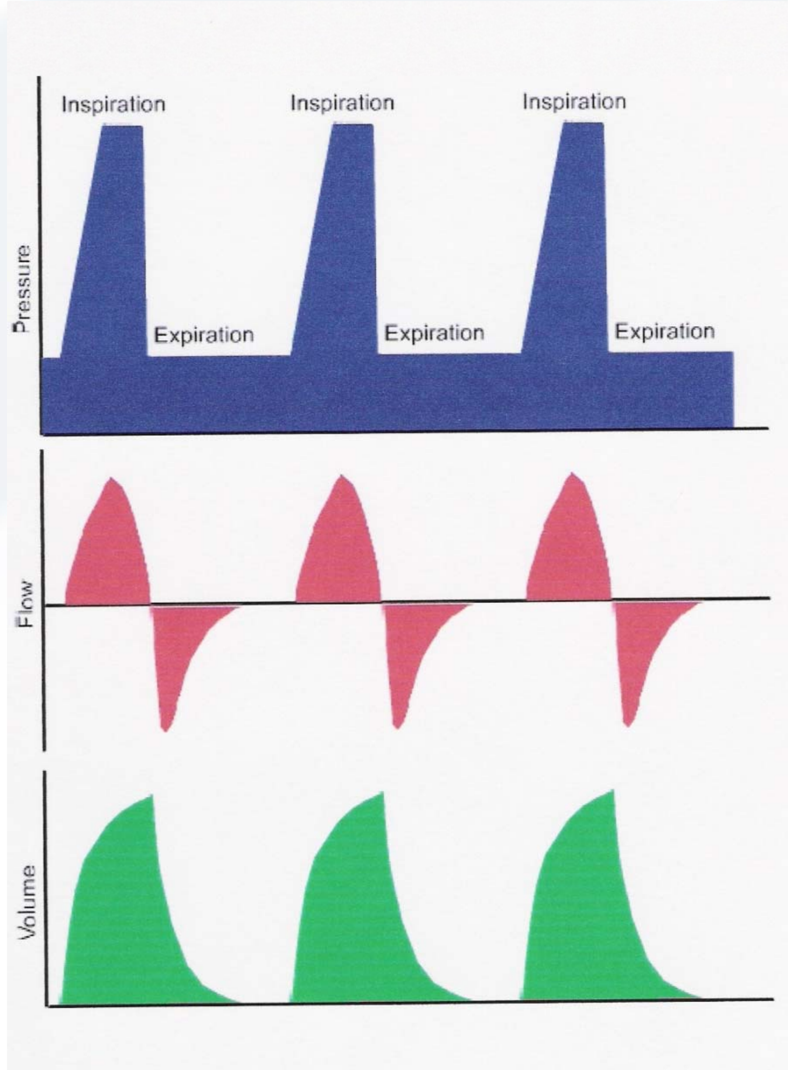
Ventilator Waveforms Plotted Against Time

Normal Scalars – PC

Pressure-Time

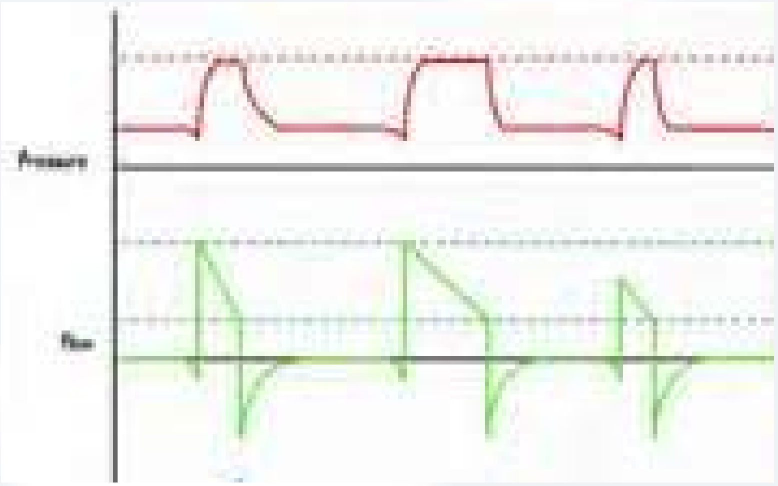
Flow-Time

Volume-Time



Ventilator Waveforms Plotted Against Time

Normal Scalars – Flow cycling



Ventilator Waveforms Plotted Against Time

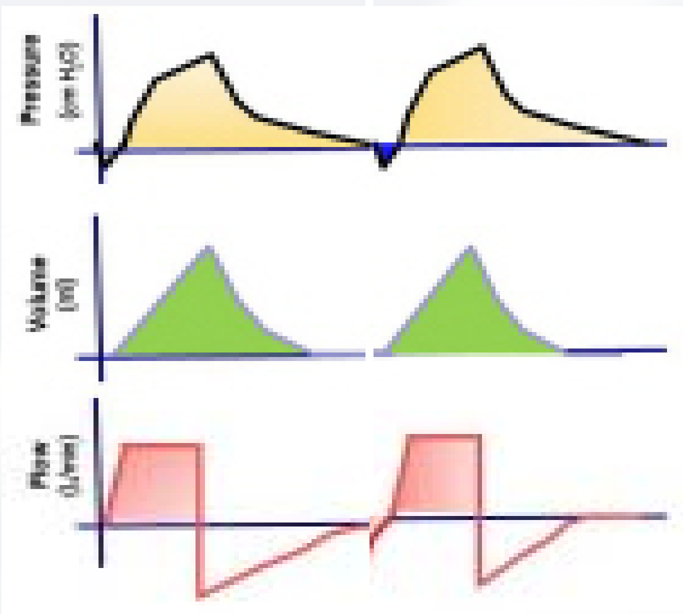
Normal Scalars – VC

Patient effort

Pressure-Time

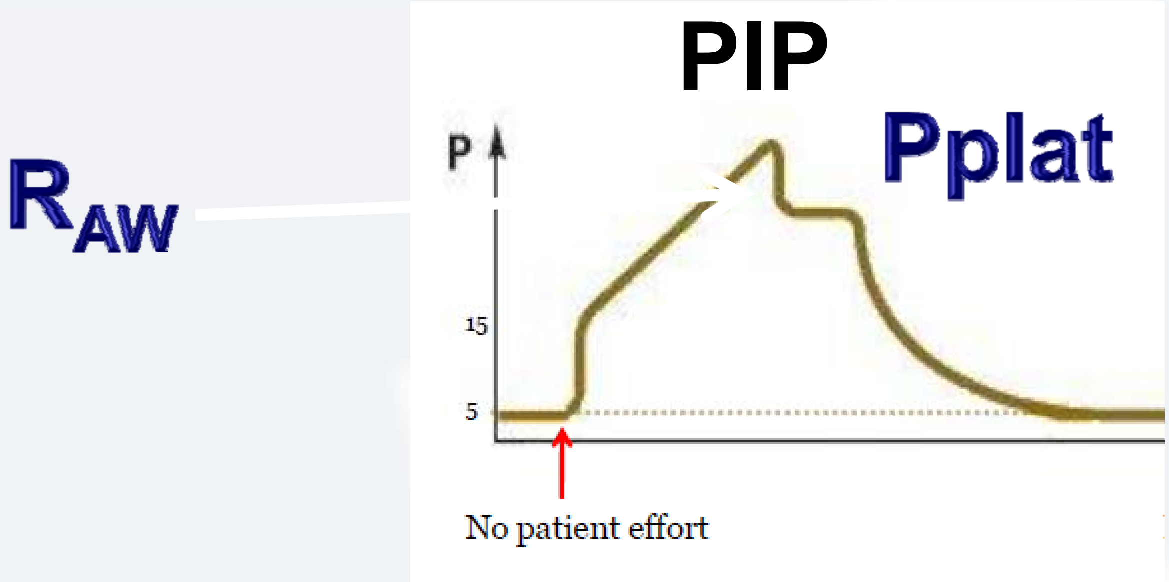
Volume-Time

Flow-Time

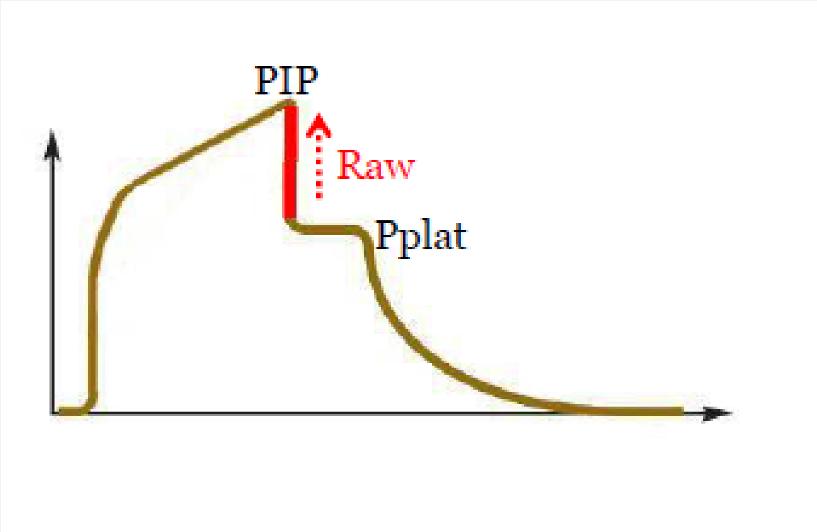


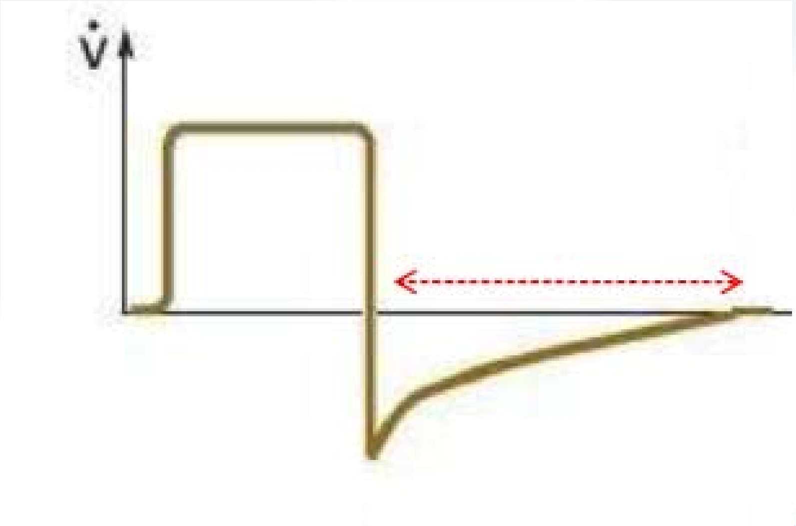
Ventilator Waveforms Plotted Against Time

Scalars – VC with inspiratory hold



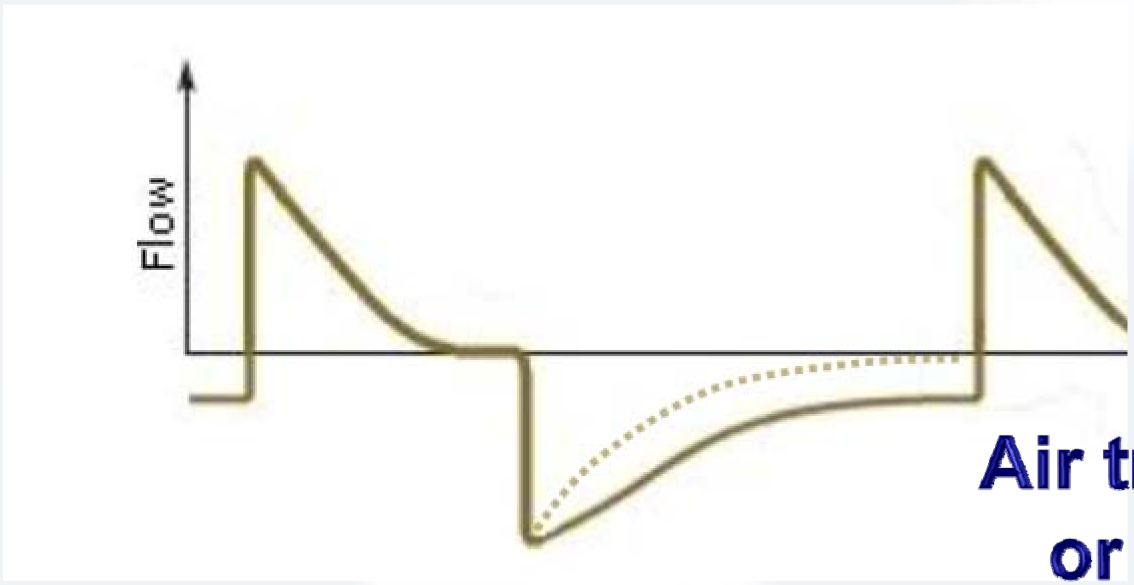
Abnormal Scalars – Volume Control





Increased airway resistance

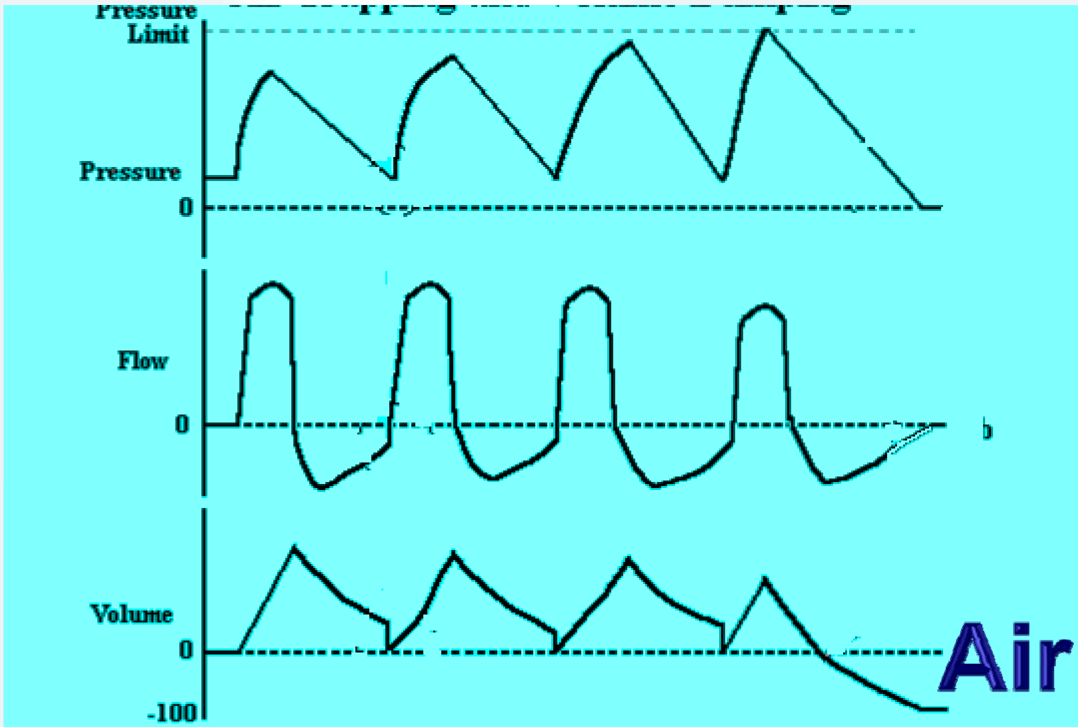
Abnormal Scalars – Pressure Control



Air trapping or leak?

Increased airway resistance

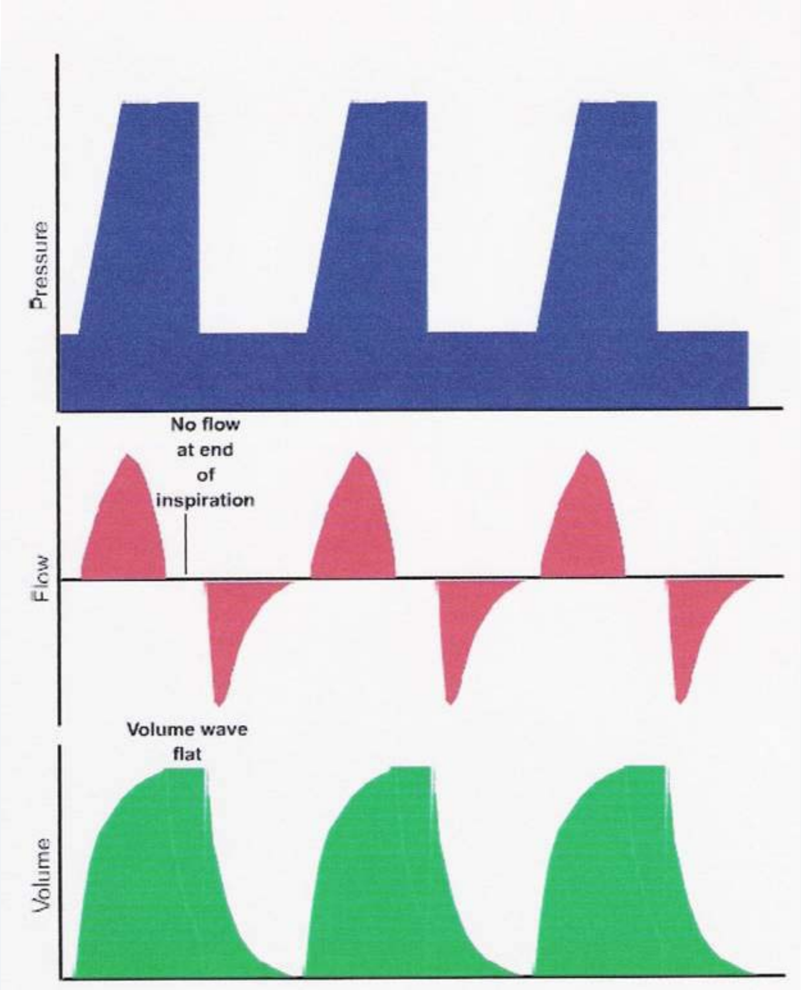
Abnormal Scalars – Pressure Control



Air trapping

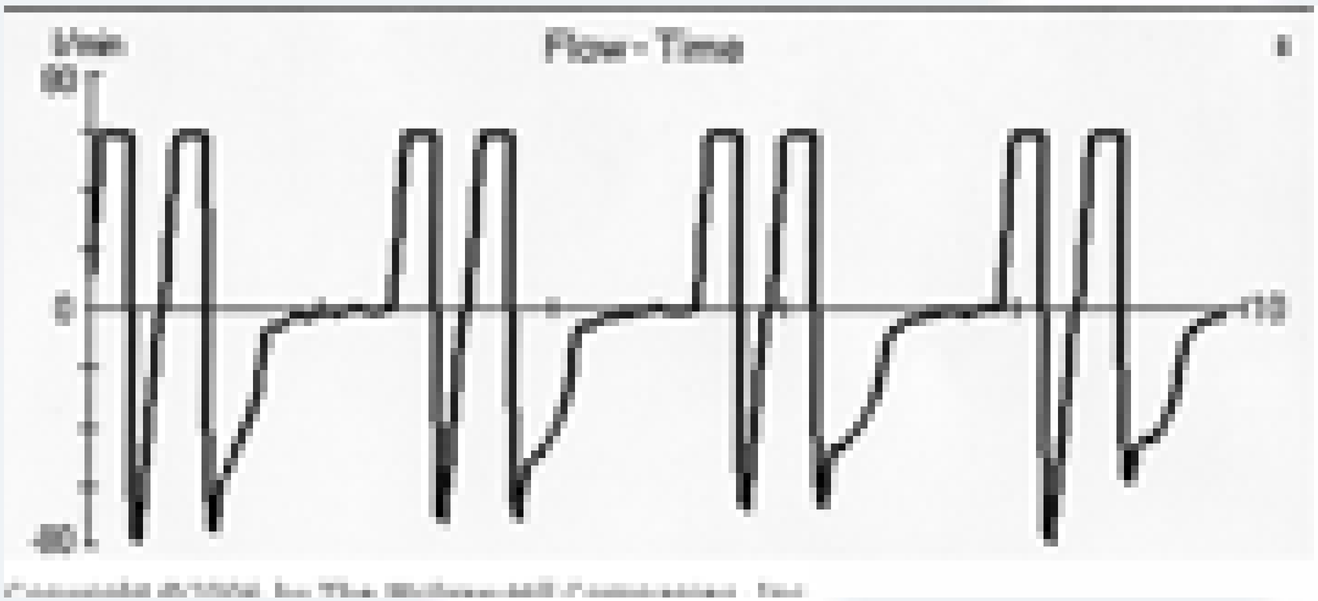
Increased airway resistance

Abnormal Scalars – Pressure Control



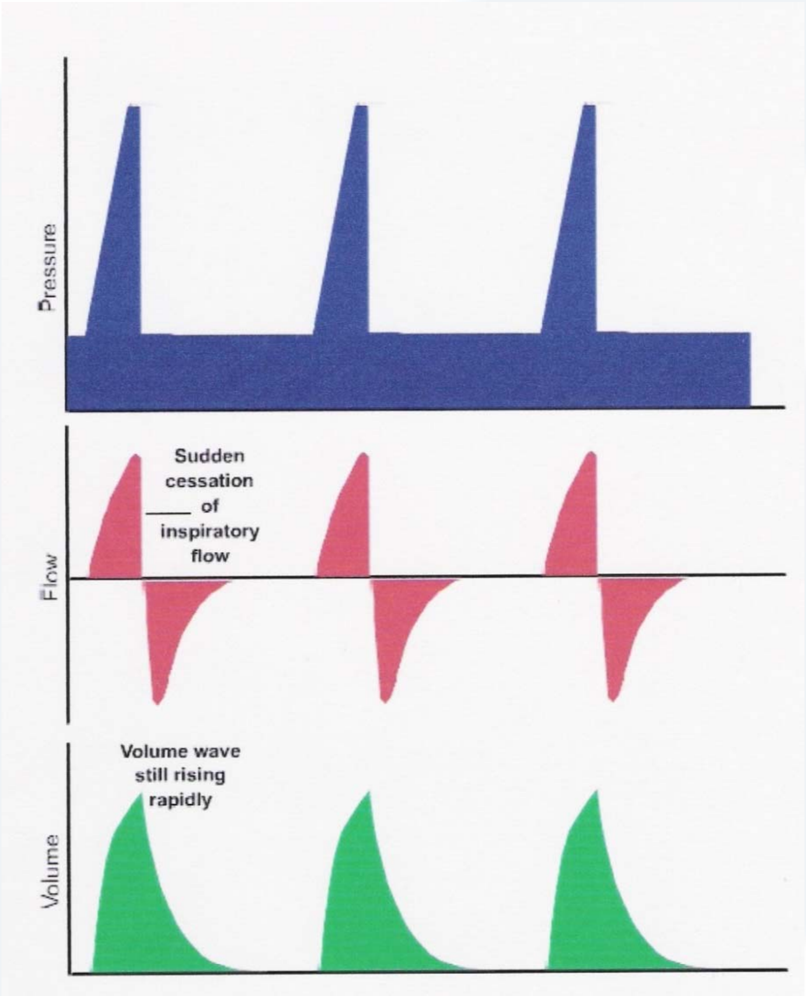
Inspiratory time too long

Abnormal Scalars – Volume Control



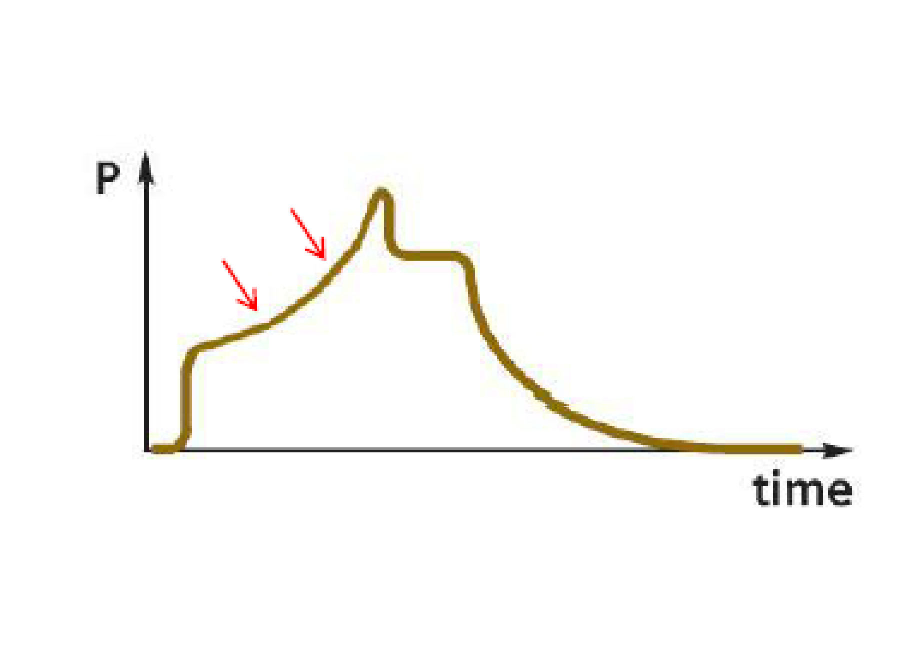
Inspiratory time too long

Abnormal Scalar – Pressure Control



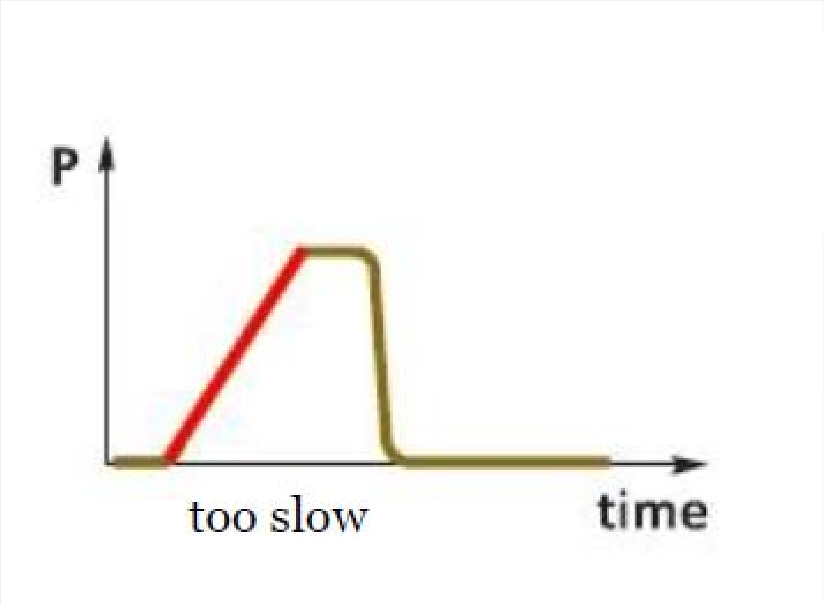
Inspiratory Time too short

Abnormal Scalars – Volume Control



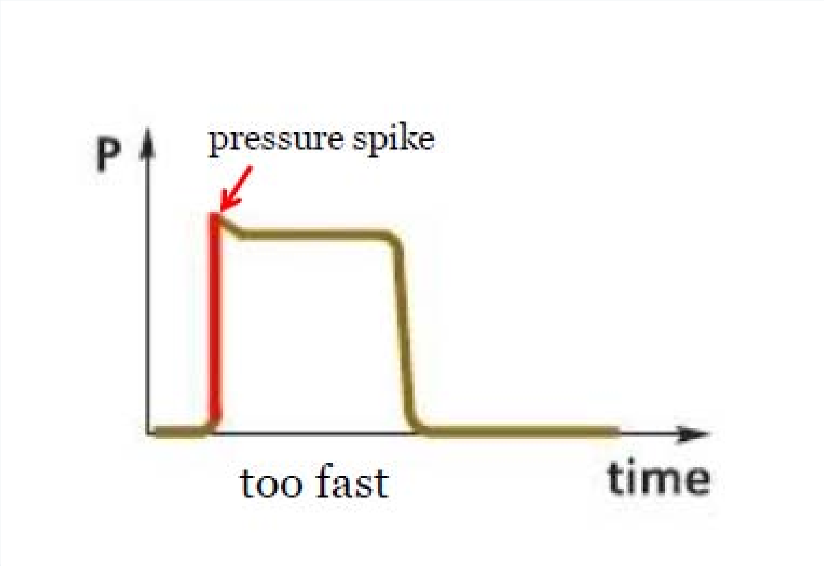
Inspiratory flow too low

Abnormal Scalars – Pressure Control



Inspiratory flow too low

Abnormal Scalars – Pressure Control



Inspiratory flow too fast

Types of Ventilator Waveforms

Waveforms where one parameter is plotted against another parameter:

LOOPS & CURVES

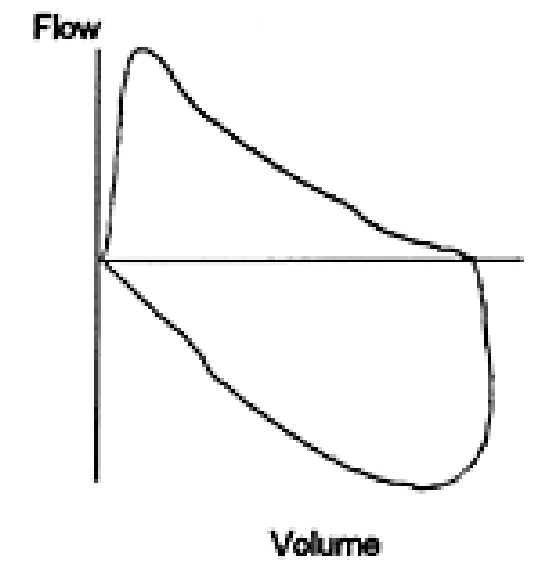
> Flow - Volume loop

> Pressure – Volume curve

Flow-Volume Loop

Normal Flow-Volume Waveform

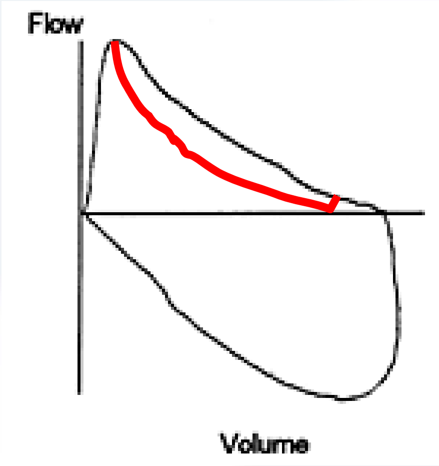
Expiration



Inspiration

Flow-Volume Loop

Increased expiratory resistance



Expiration

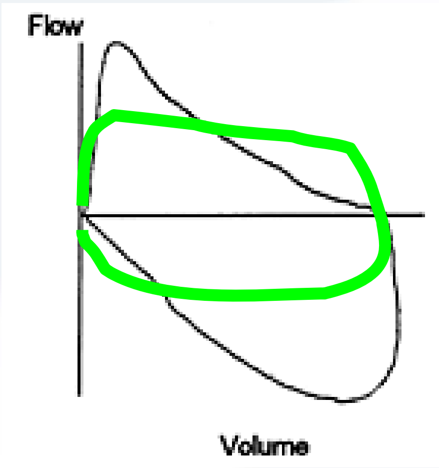
Inspiration

Flow-Volume curve changes with changes in airway resistance

Abnormal Flow-Volume Loop

Expiration

Inspiratory and Expiratory obstruction to air flow

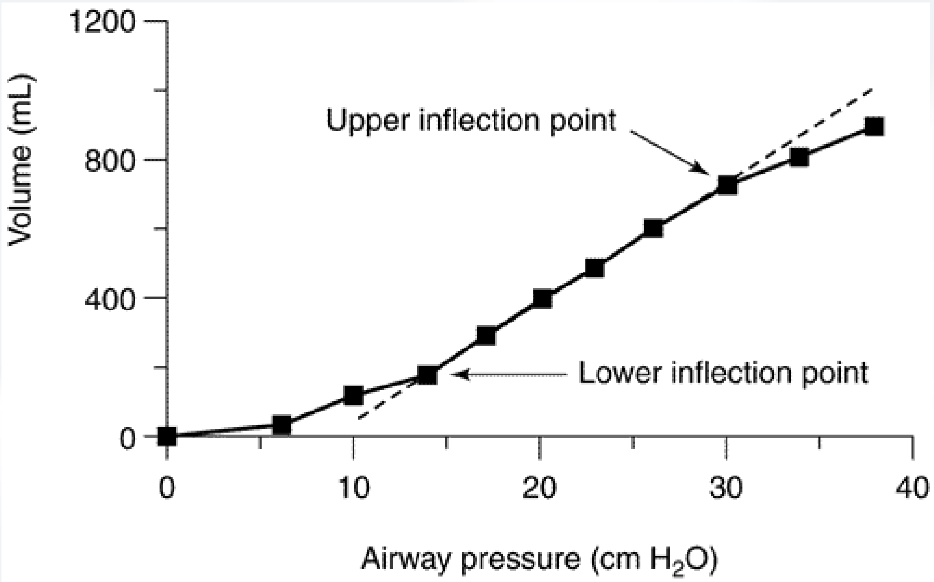


Inspiration

Flow-Volume curve changes with changes in airway resistance

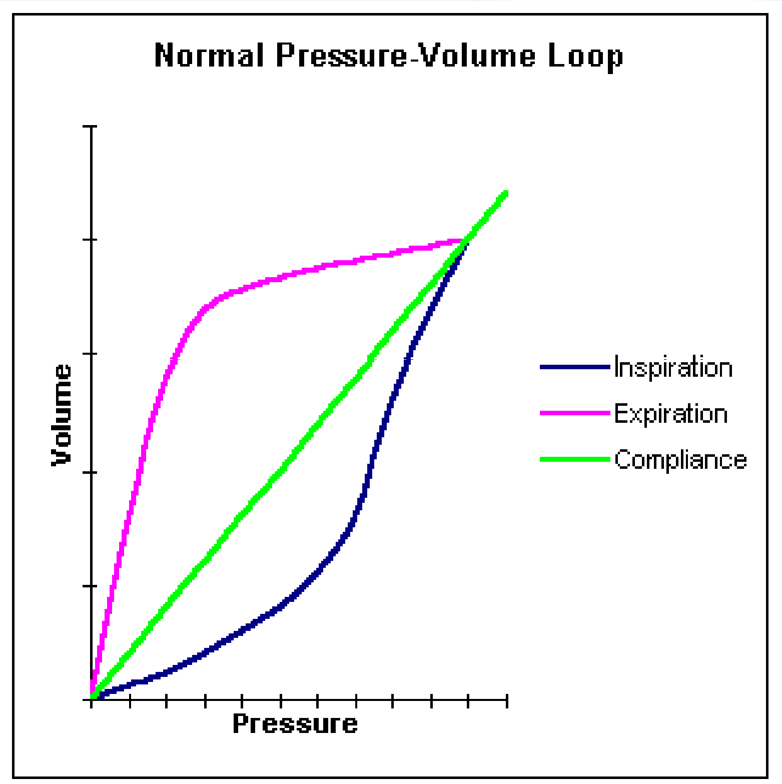
Pressure-Volume Curve

Pressure-Volume Waveform



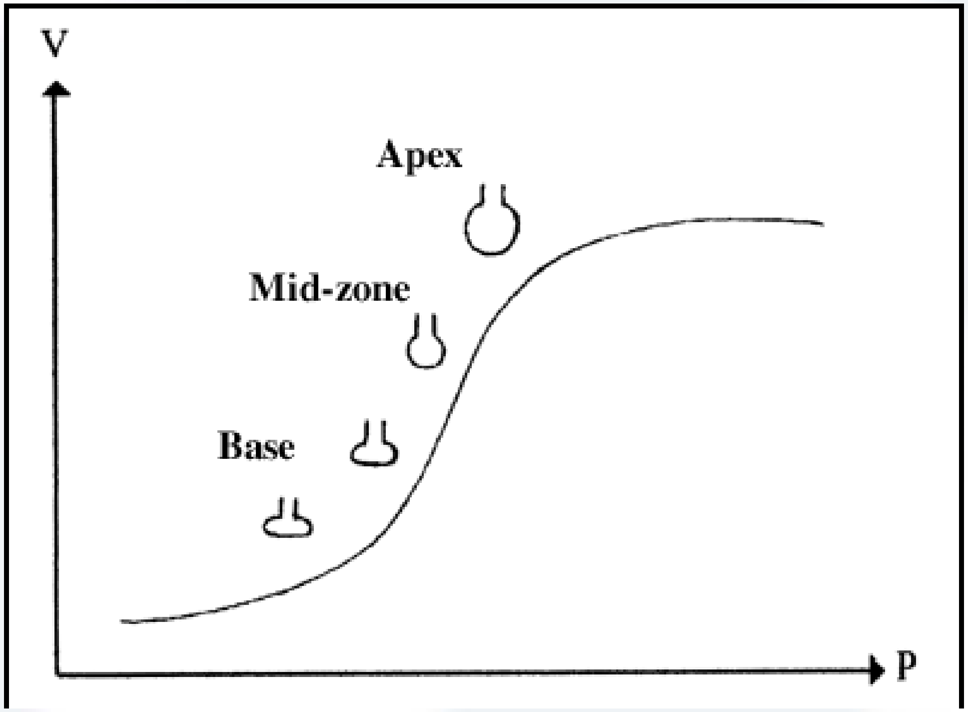
Pressure-Volume Curve

Normal Pressure-Volume Waveform



Pressure-Volume Curve

Pressure-Volume



Pressure-Volume Curve

Normal Pressure-Volume curve for a spontaneous breath

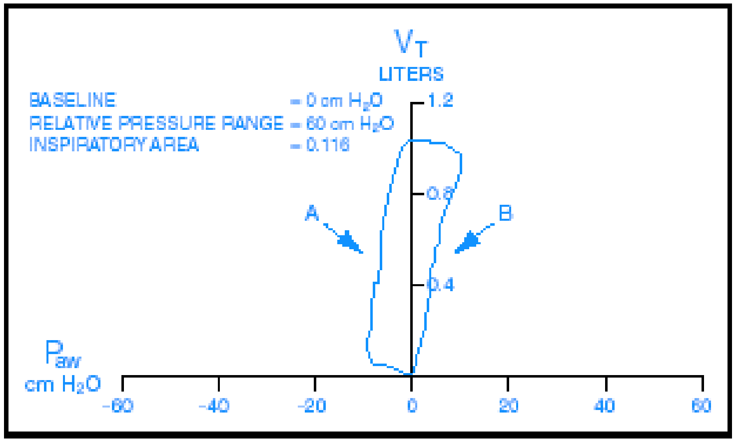
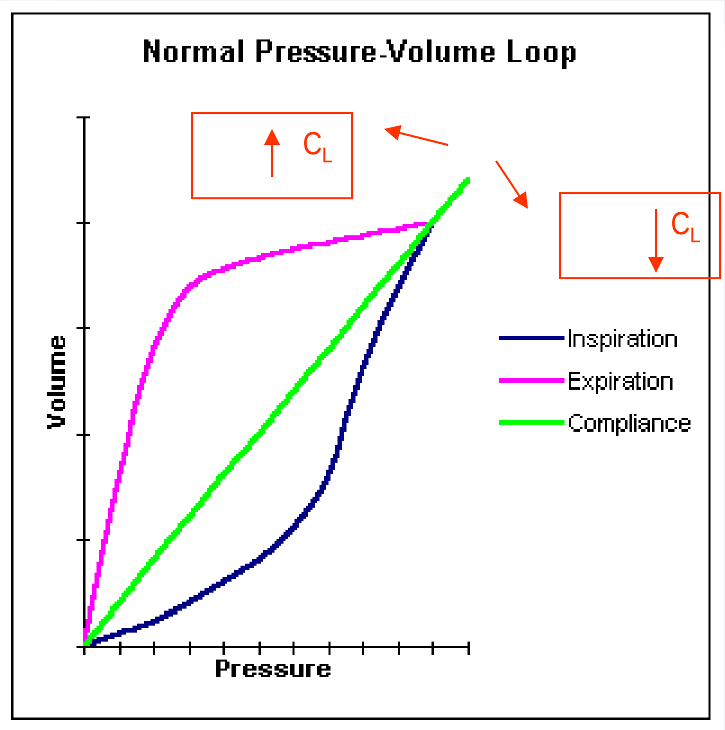


Figure 28-19 *A spontaneous pressure versus volume loop. Note how inspiration is in the subambient pressure range, while expiration is positive. The loop progresses clockwise from left to right.*

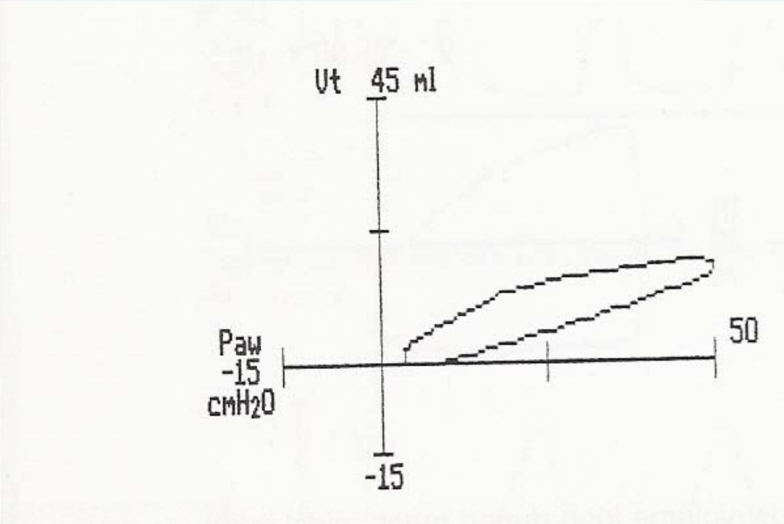
Pressure-Volume Curve



Pressure-Volume curve changes with compliance

Abnormal Pressure-Volume Curve

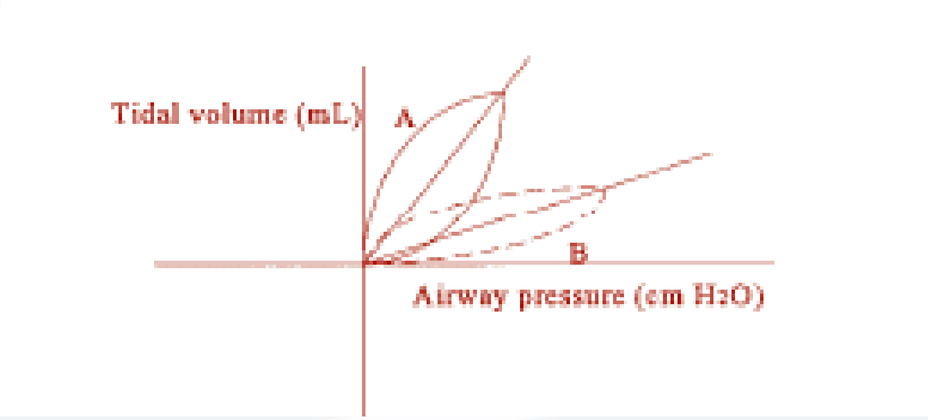
Poor Compliance



Pressure-Volume curve changes with compliance

Common Examples of Abnormal Pressure-Volume Waveforms

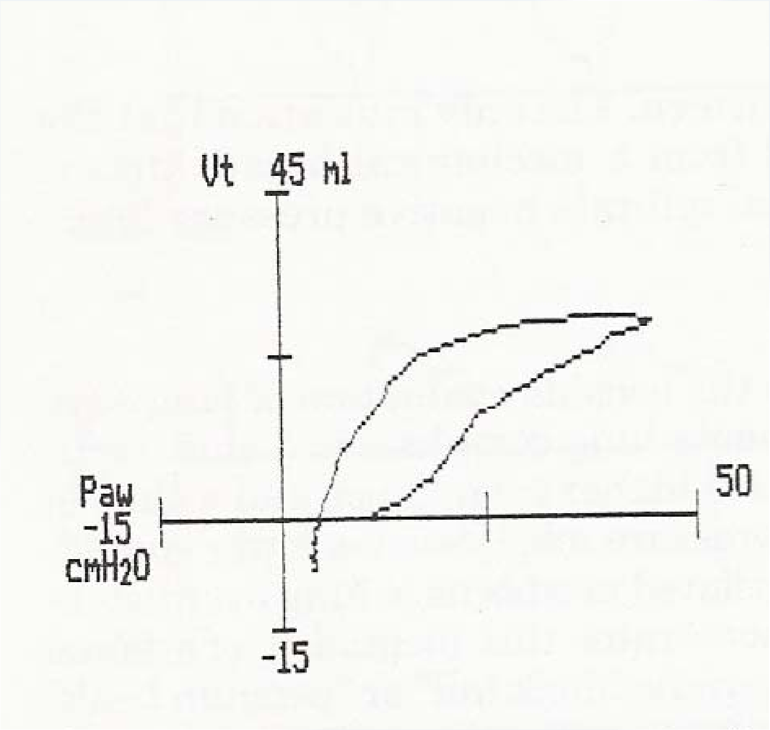
Improved Compliance



Pressure-Volume curve changes with compliance

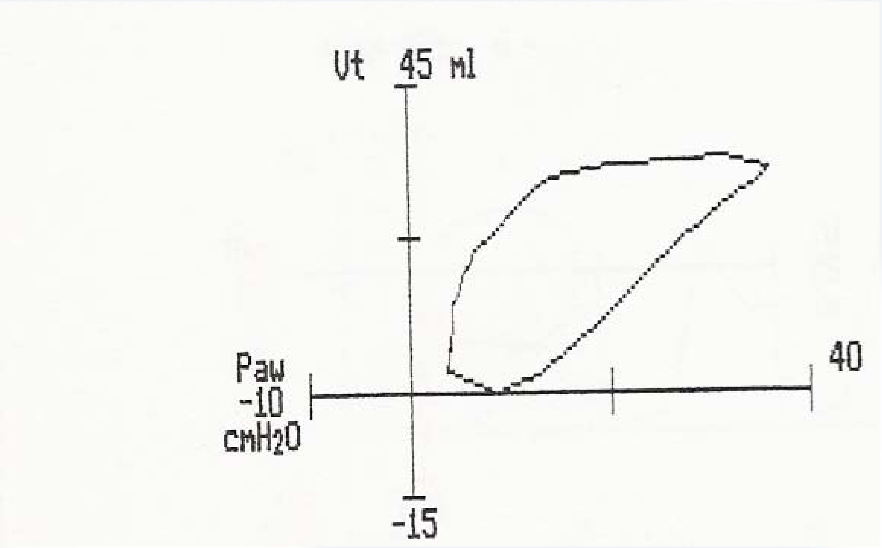
Abnormal Pressure-Volume Curve

“Beaking”



Abnormal Pressure-Volume Curve

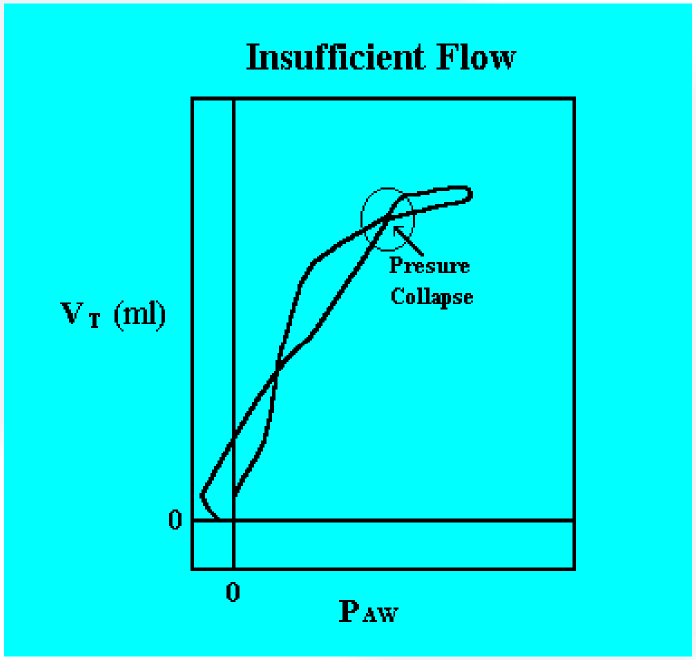
Increased expiratory resistance



Pressure-Volume curve changes with changes in airway resistance

Abnormal Pressure-Volume Curve

Inadequate inspiratory flow



Newer / Advanced modes

- Airway Pressure Release Ventilation (APRV)
- High Frequency Oscillation Ventilation (HFOV)
- Dual Modes
  - Pressure-regulated volume control (PRVC)
  - Minimum Mandatory Ventilation
- NAVA

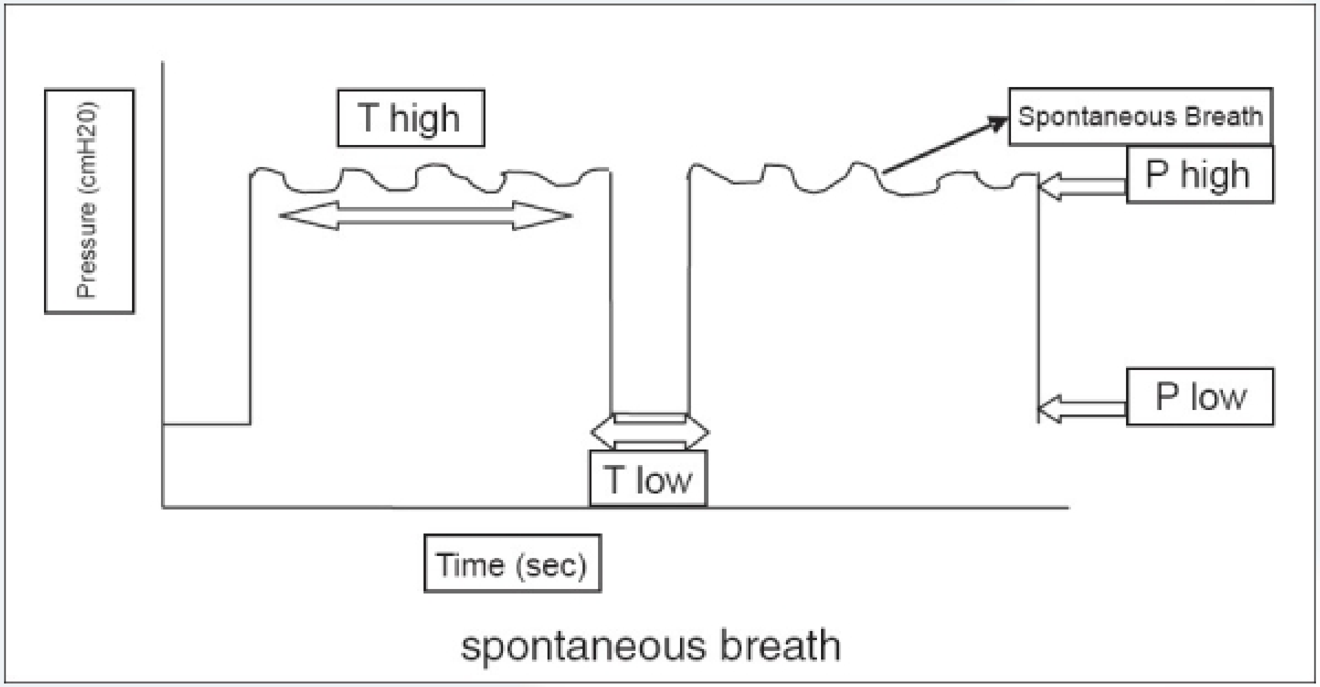
Airway Pressure Release Ventilation (APRV)

APRV

- A pressure controlled mode of ventilation
- First described in the 1980s
- An alternative to other pressure controlled modes when lung recruitment and maintenance is necessary
- Lung protective strategy
- “Inverse ratio” ventilation
- Allows spontaneous breathing
- An alternate to HFV

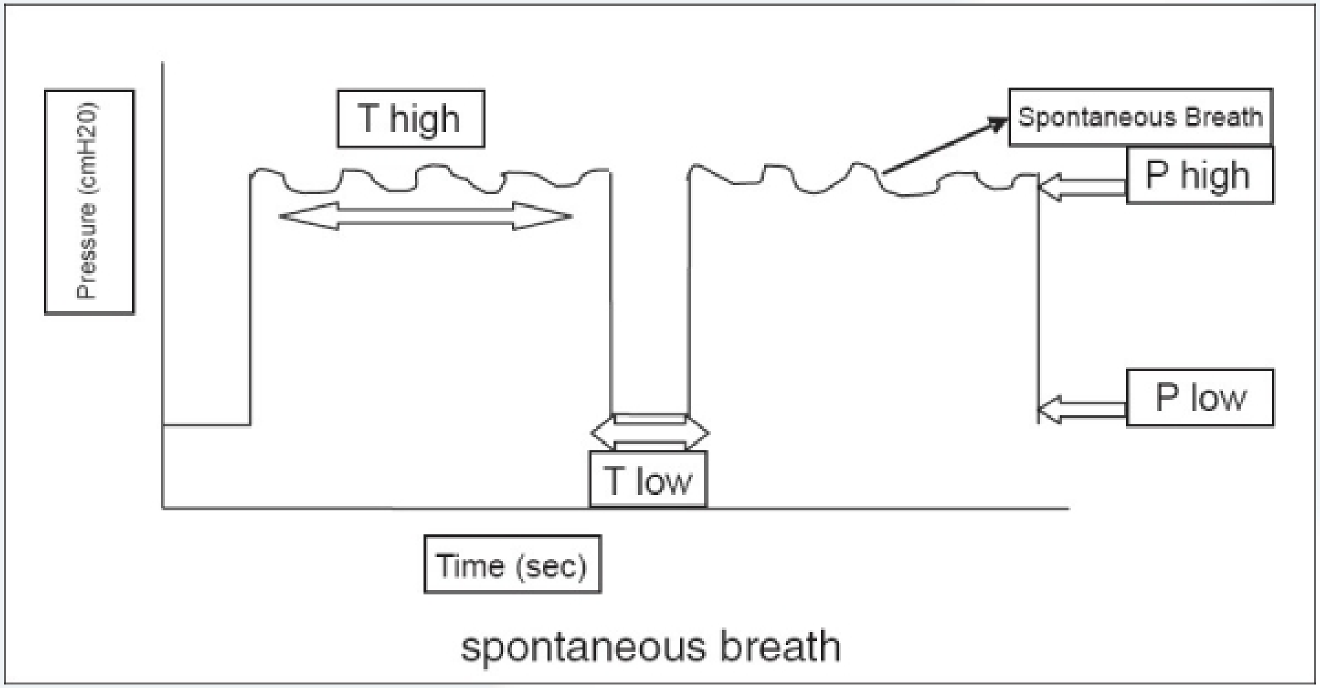
APRV

- Two levels of "CPAP" high and low
- Two "time frames" – time high and time low



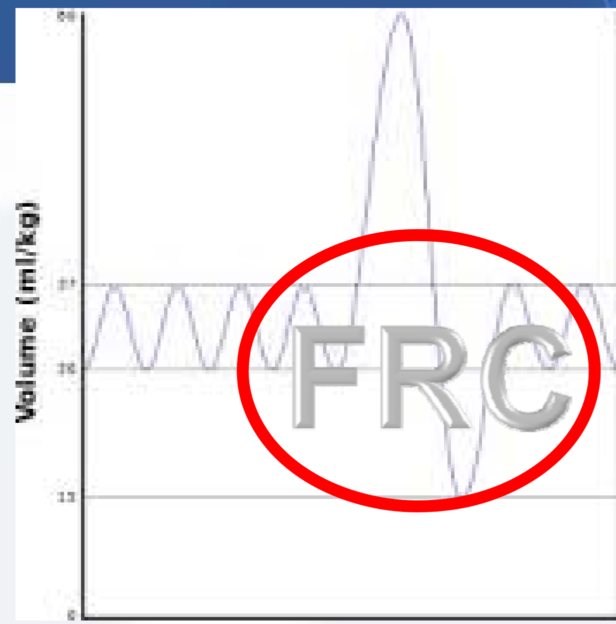
APRV

- Time high allows for recruitment
- Flow decrease to time low allows for CO2 release



http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2732103/

APRV Indications



- ALI/ARDS
- Atelectasis
- Pneumonia
- Pulmonary edema if hemodynamically stable
- Recommended in H1N1 strategies

http://www.canadiancriticalcare.org/h1n1_treatment.htm

APRV – Relative contraindications

- Underlying medical issue requiring high levels of sedation and paralysis
- Hemodynamically unstable
- Increases in intracranial pressure
- Air leak – untreated pneumothorax, bronchopleural fistula

http://www.ccjm.org/content/78/2/101.long

APRV – what's the proof

- Recent literature around H1N1
- Small study demonstrating decreased mortality
- No significant demonstrated benefit
- Usually used as "rescue" for difficult to oxygenate patients
- Concerns regarding PC ventilation – effect of compliance and resistance on volumes

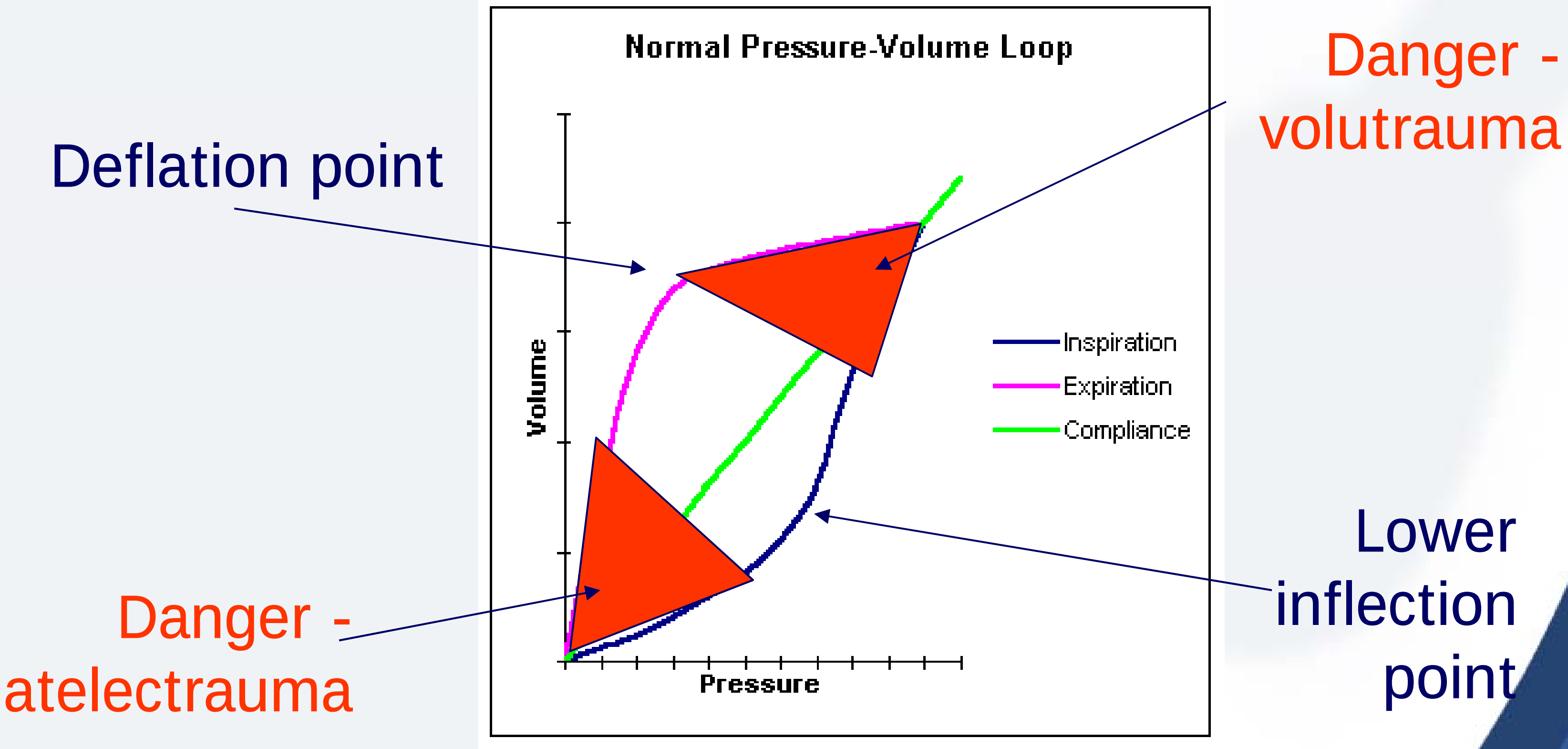
http://rc.rcjournal.com/content/57/2/282.short

High Frequency Ventilation

Theory and Practical Application

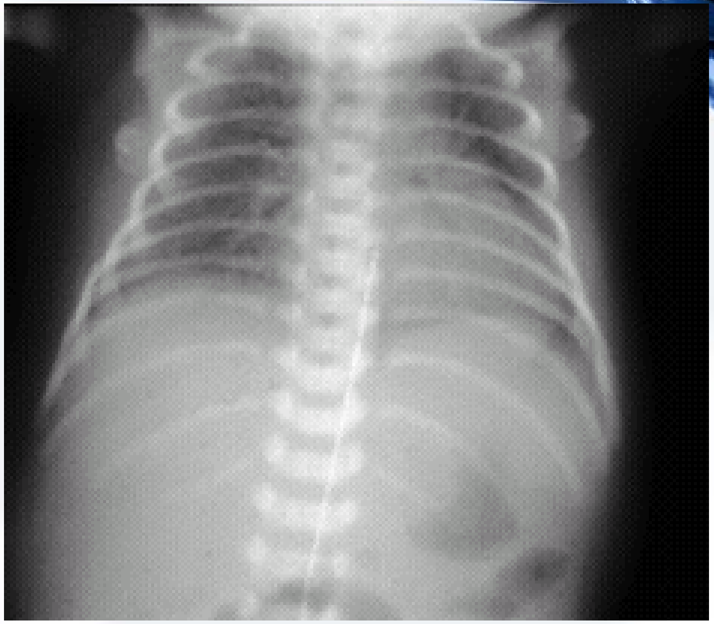
Theory of HFO

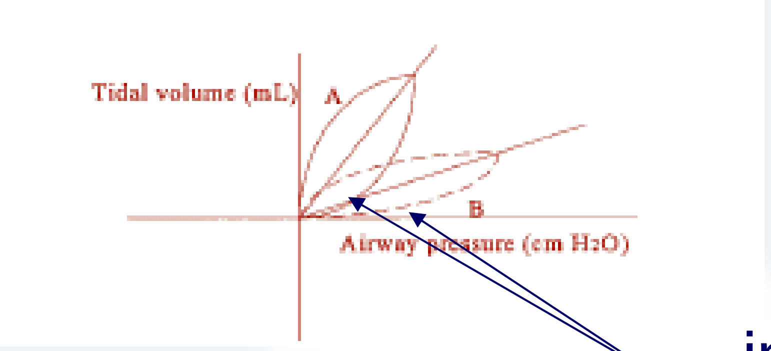
The “open lung concept”



Acute Lung Injury

- Surfactant deficiency RDS
- Surfactant inactivation ARDS
- Decreased compliance
- Prone to collapse





Lower inflection point

HFO - Open the lung and keep it open

Reduce the shearing forces of the inflation/deflation cycle of conventional ventilation

Reduce the production of inflammatory mediators – protect against biochemical injury

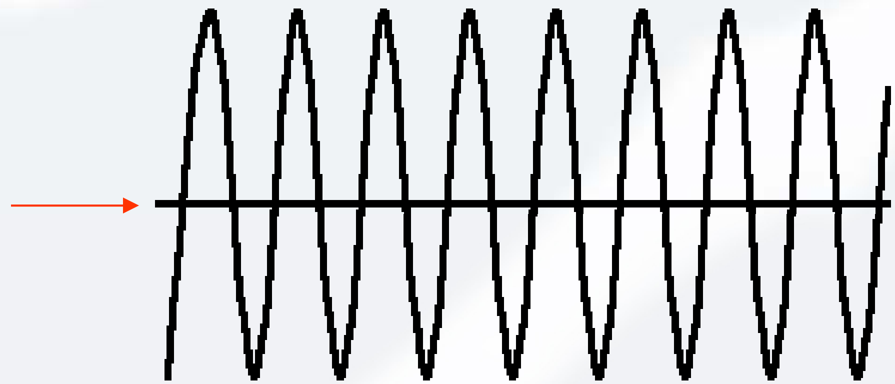
Oxygenation improves due to increased functional lung volume and surface area for gas exchange

Tidal volumes delivered are smaller than dead space and gas exchange is largely the product of molecular action and diffusion

Types of high frequency ventilation

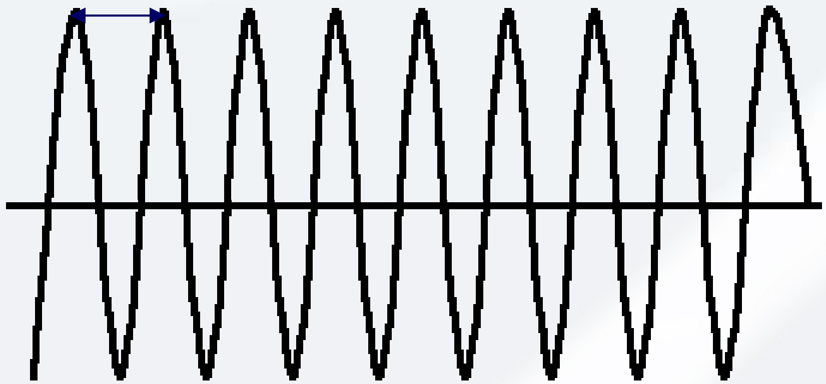
- Rapid Rate conventional ventilation
- Jet ventilation
- High frequency oscillatory ventilation
- High frequency flow interrupter
- High frequency percussive ventilation

Mean airway pressure



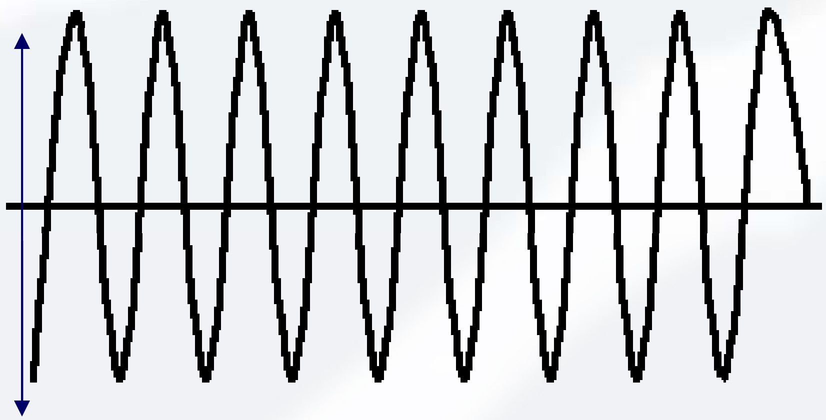
- MAP influences oxygenation
- Inadequate MAP can lead to atelectasis/collapse
- MAP is judged by lung inflation on CXR

Frequency (HZ) - bandwidth



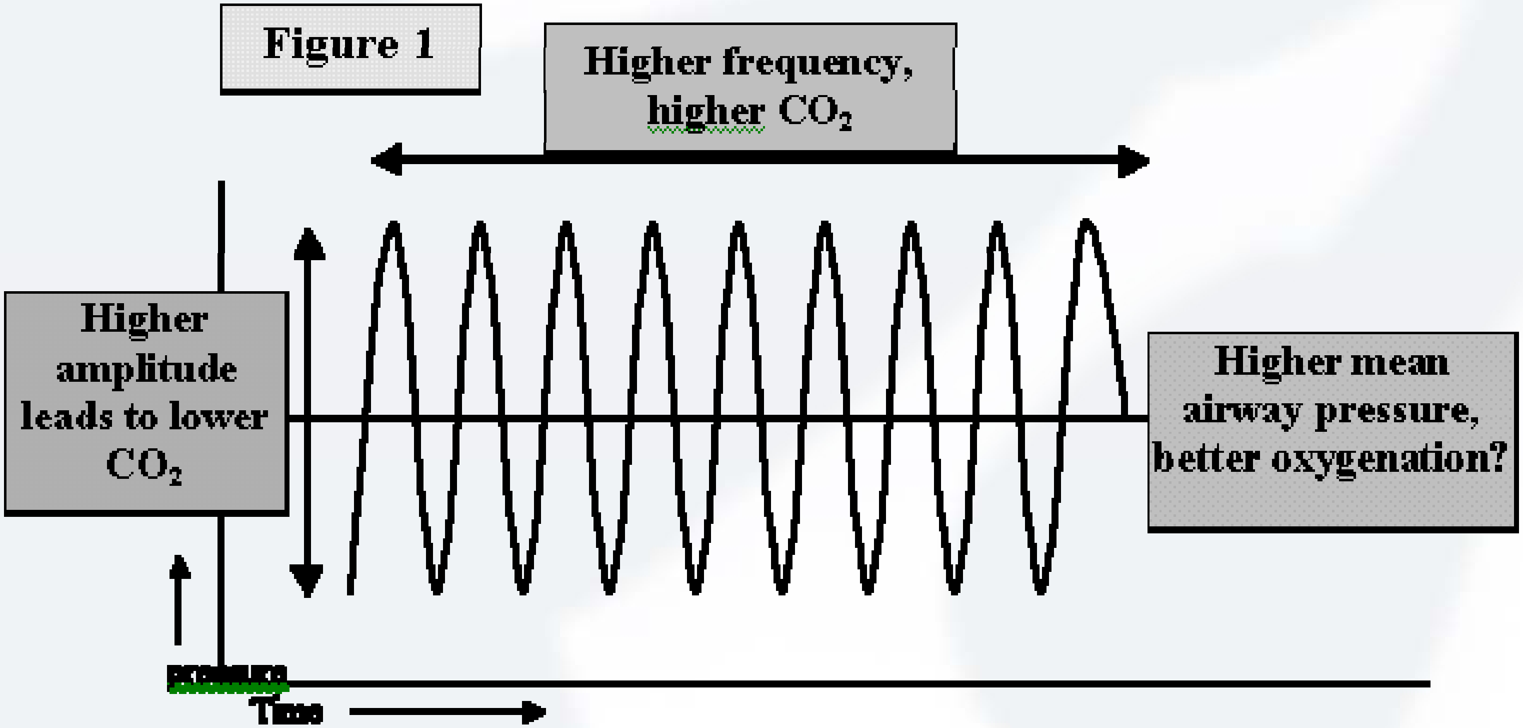
- Increasing HZ *increases* “rate” per minute
  - Increasing HZ, *decreases* “tidal volume” per breath
    - High HZ may increase incidence of air trapping because of decreased time for exhalation

Amplitude - the peak to trough gradient



- Increasing amplitude increases tidal volume – dependent on lung compliance
- Amplitude is judged by chest wiggle

Figure 1



Higher frequency, higher CO_2
Higher amplitude leads to lower CO_2
Higher mean airway pressure, better oxygenation?
pressure
Time

Pediatric and Adult strategies differ:

Pediatric / Neonatal

Hz – depends on size

Hz stable – change amplitude

Adult

Hz – 6ish (M vs F)

Amplitude stable – change Hz

HOB 30

Leak around ETT

Recruitment post suctioning

Adjuncts – NO, surfactant

Prone positioning

Other Parameters

Inspiratory Time – 33%

Bias Flow – fresh gas source, wash out of CO_2 in circuit, 15-20lpm adequate for neonates, need higher in pediatric and adult patients – up to 50+lpm

Practical Considerations

How does HFP influence monitoring the patient?

- The usual – HR, RR, BP – assessment specific to HFO
- CO2 monitoring – how???
- Blood Gasses
- CXR – how often
- Physical examination – assessment specific to HFO, auscultation of lungs, bowel and heart, palpation

Practical Considerations

HFO Safety issues?

- Circuit support
- Circuit alignment
- Two person suction, movement, etc
- In-line suction
- Manual ventilation equipment at bedside – PEEP compensated
- Position of ETT
- Alarms – on HFO and bedside monitor

Where is the evidence?

- Early meta-analysis included various HFV modes and strategies

First Intention
Oscillator
High lung
volume
strategy

Rescue
HFV/Jet/Flow
interrupter
Low lung volume
strategy

Where is the evidence?

2007 Cochrane:

Neonatal application of HFO- could not recommend for or against, increased IVH

2012 – Pediatric Application – beneficial effect on outcomes unclear. Question whether strategies have been optimized.

2013 Cochrane:

Adult application of HFO- promising yet unproven. Guarded use with patients susceptible to rapid changes in PaCO2 (head injured)

2013 – OSCILLATE study (adult) – not yet published

Dual Modes

- Combination of two or more conventional modes of ventilation
- PRVC / Autoflow / VC+
- MMV / Auto-Mode

Dual Modes

PRVC and MMV

Dual Modes

http://www.covidien.com

http://www.maquet-training.com

www.draeger.com

PRVC / VC+ / *Autoflow*

- Advantages of pressure and volume ventilation are combined into one mode
- Set VT and RR
- Ventilator measures patient's compliance and delivers the set VT at the lowest A/W pressure
- Utilizes PC advantages
  - Limiting pressure
  - Variable flow to respond to patient demand
- Utilizes VC advantages
  - Targeting VT and Minute Volume

Pressure-Regulated Volume Control (PRVC) Maquet Servo-I ventilator

- Servo-I video

PRVC/VC+/Autoflow Clinical Example

- ARDS patient with high A/W pressures on A/C (VC)
- **450 mls (6 mls/kg) x 28 b/min, 60% 02, PEEP 12 cmH20**
- **Pip 38 cmH20 and Pplat 34 cmH20**
- **ABG: pH 7.32, PaC02 49 mmHg, Pa02 65 mmHg, $HC0_3$ 25 mEq/L**
- ∆ PRVC or VC+
- Pip now 28 cmH20 with the same VT and RR

Mandatory Minute Volume (MMV)

- Combination of SPONTANEOUS breathing and VENTILATOR breaths
- Adjusts according to patient's own Minute Volume
- If patient breaths SPONTANEOUS > MV setting, all breaths are spontaneous
- If patient breaths < MV setting, ventilator delivers breaths to maintain minimum minute volume setting

Mandatory Minute Ventilation (MMV) Draeger XL ventilator

- MMV video

MMV - Clinical Example

- 2 hours post-op cardiovascular surgery
- Pt. on **SIMV 500 x 12 b/min, 50% and 5 PEEP**
- Pt. awakens and is asynchronous with the ventilator, coughing, aggitated
- Pt. Δ **CPAP/PS, PEEP 5 cmH20, PS 10 cmH20, 40% 02**
- With sedation for agitation and less stimulation, patient’s RR 5 b/min and VT 200 mls
- **ABG: pH 7.31, PaC02 52 mmHg, Pa02 75 mmHg, HC03 22 mEq/L**
- Δ MMV and patient can breath SPON when awake, but gets ventilated when sedated/apneic

Neurally Adjusted Ventilatory Assist

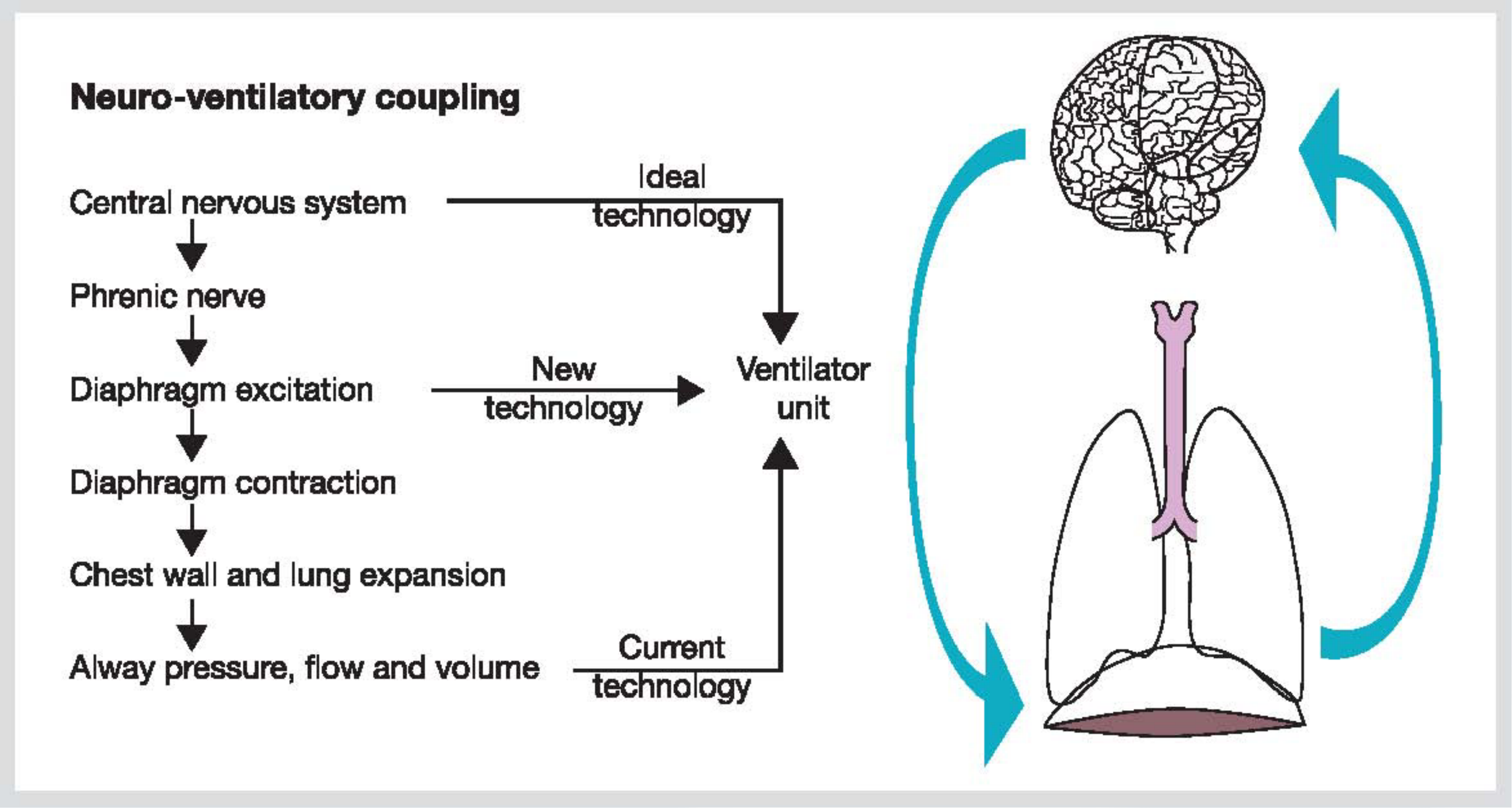
(NAVA)

NAVA

- Mode of ventilation available on the Servo I ventilator
- Uses electrical impulse from diaphragm to activate ventilator assist
- Level of assist is in proportion to the electrical activity of diaphragm
- Can be used as invasive or non invasive mode

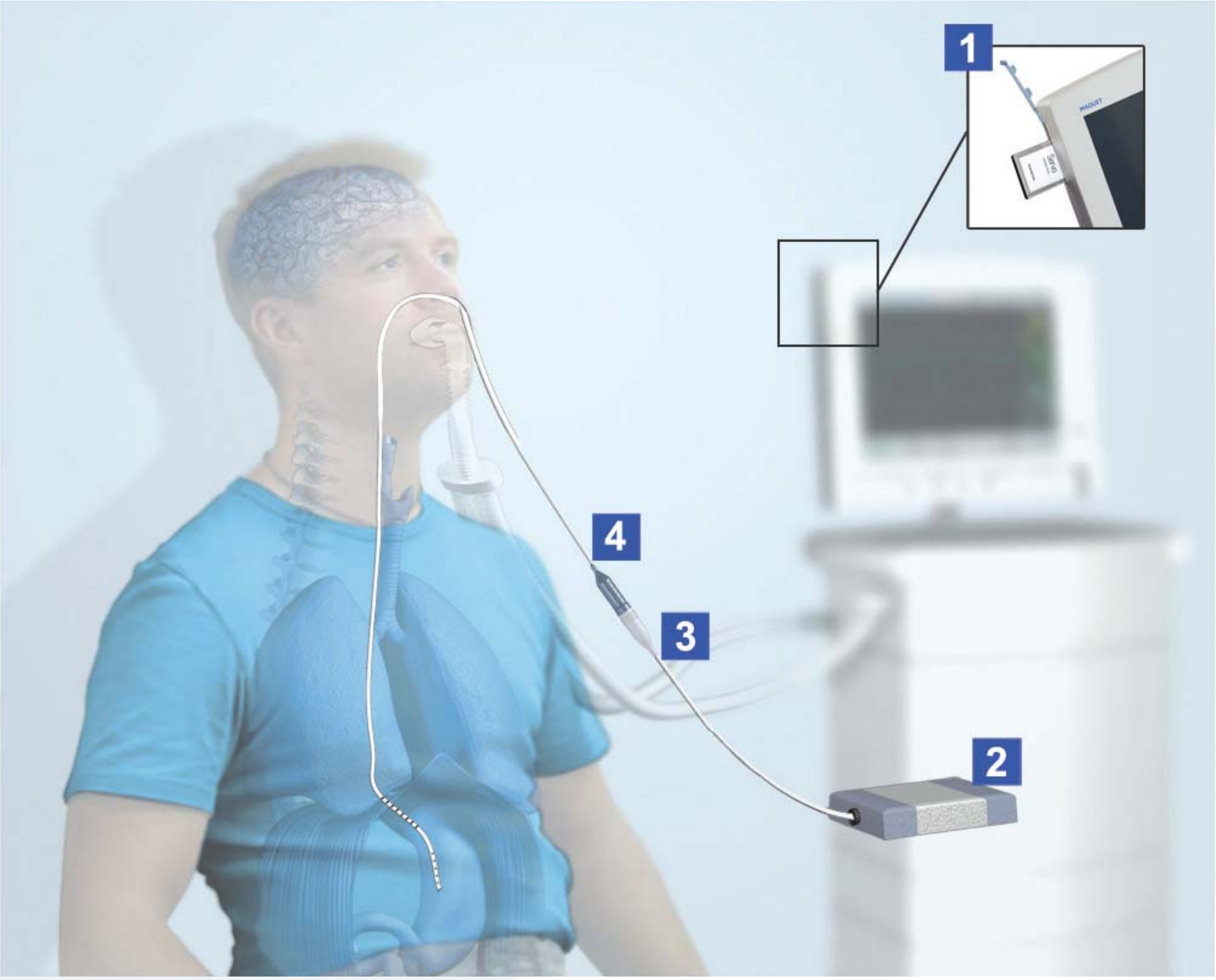
http://www.maquet.com/content/Documents/Brochures/SERVOI_BROCHU_MX-0353-1007_LoRes_EN_ALL.pdf

NAVA

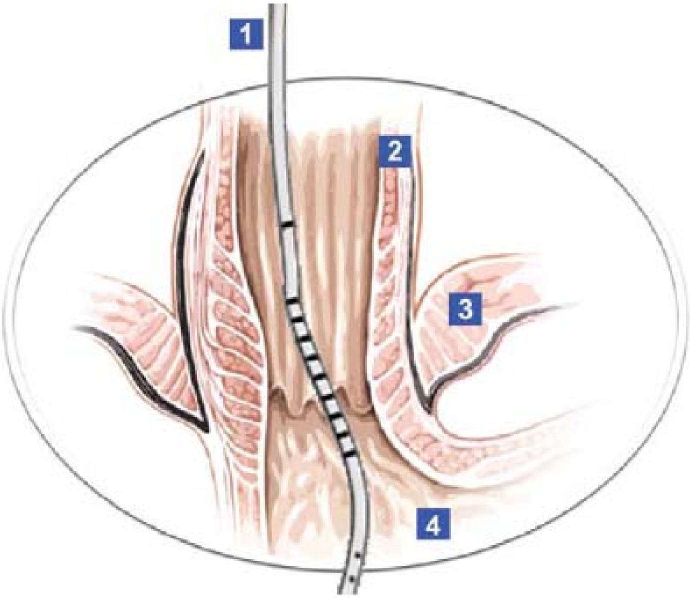


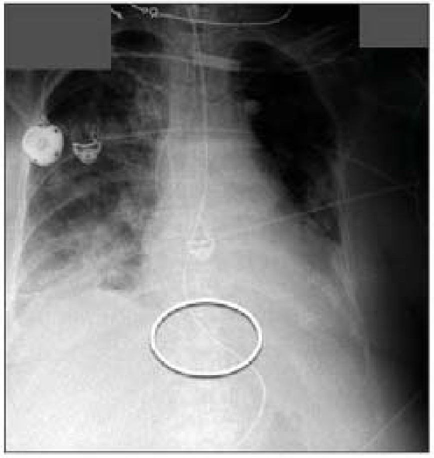
http://www.maquet.com/content/Documents/Brochures/SERVOI_BROCHU_MX-0353-1007_LoRes_EN_ALL.pdf

NAVA



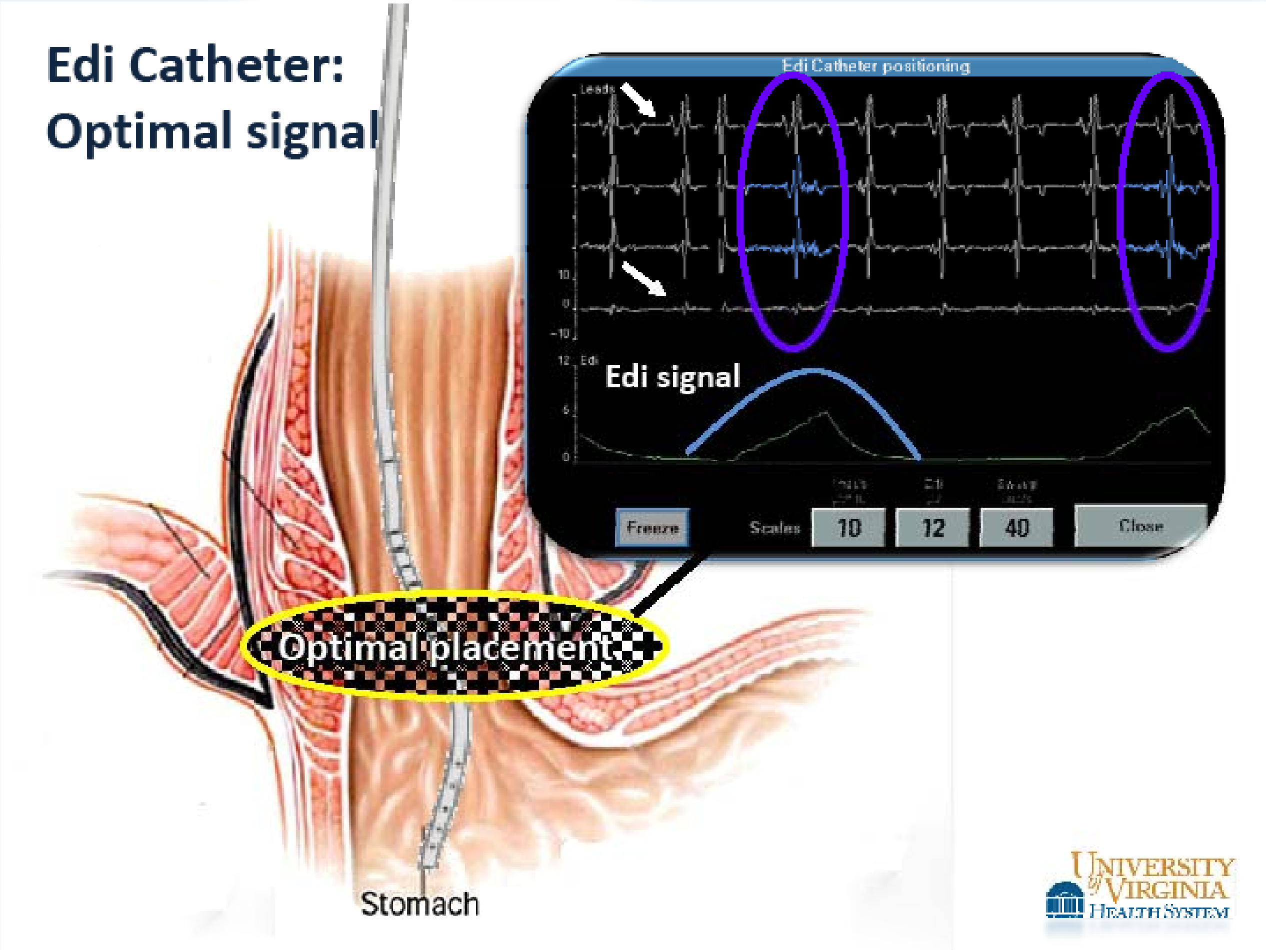
NAVA





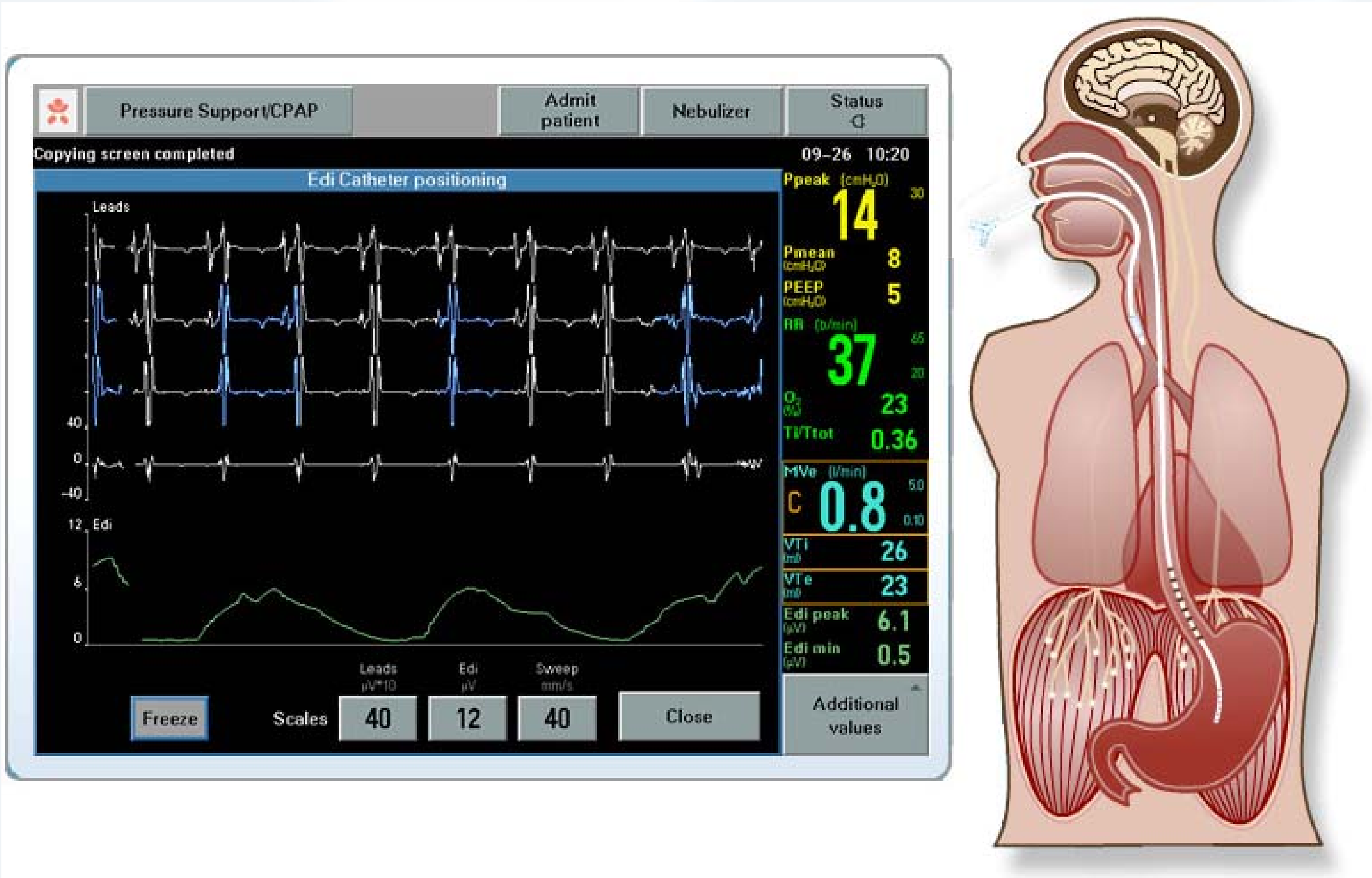
1. Edi Catheter
2. Esophageal wall
3. Diaphragm
4. Stomach

NAVA



UNIVERSITY of VIRGINIA HEALTH SYSTEM

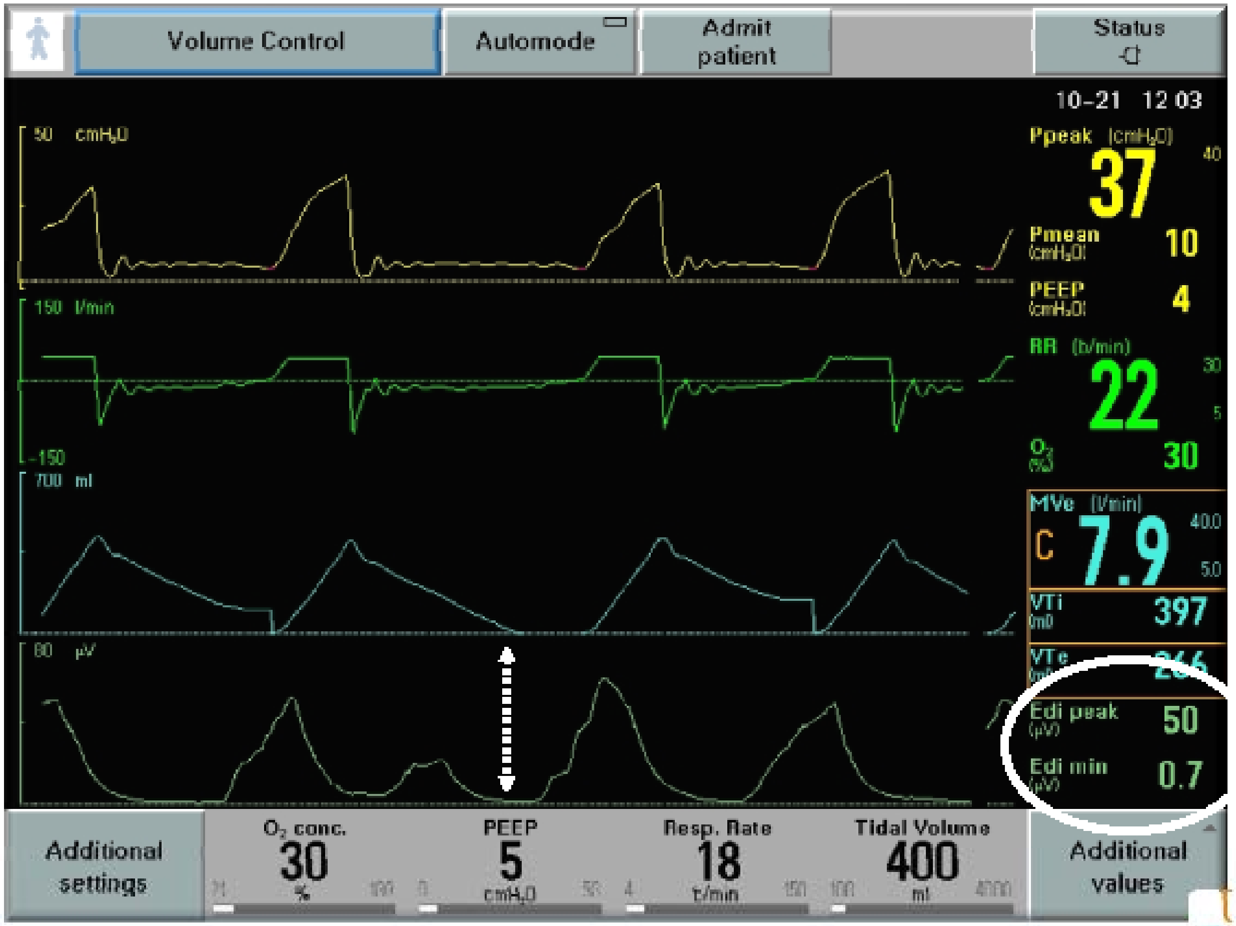
NAVA



Pressure Support/CPAP
Admit patient
Nebulizer
Status
Copying screen completed
09-26 10:20
Edi Catheter positioning
Leads
Edi
Ppeak
14
Pmean
8
PEEP
5
RR
37
23
Ti/Ttot
0.36
MVe
0.8
VTi
26
VTe
23
Edi peak
6.1
Edi min
0.5
Freeze
Scales
Leads
40
Edi
12
Sweep
40
Close
Additional values

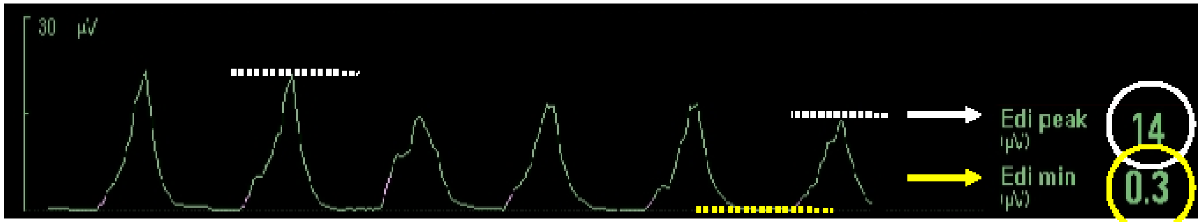
NAVA

New Physiologic Monitoring Tool



NAVA

Components of the Edi monitoring tool



- **Edi peak** – Displays the amount of impulse sent to generate tidal volume breath by breath.
  - directly proportional to workload
  - typically **≤ 10 µV** in healthy subjects

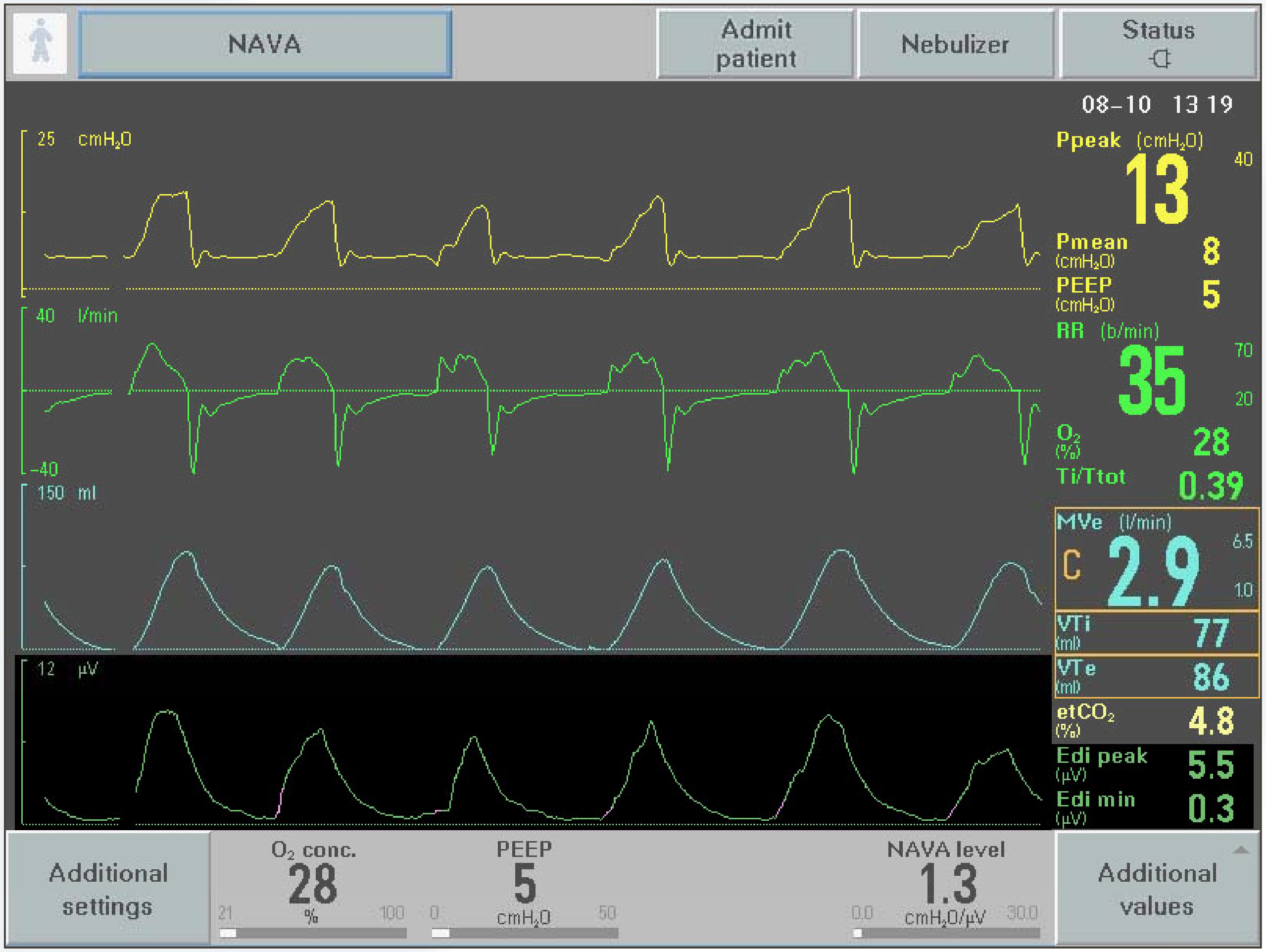
- **Edi min** – Measures the tonic activity of the diaphragm at rest. Physiologic reflection of derecruitment.

University of Virginia Health System

NAVA

Pressure Control
Automode
Admit patient
Nebulizer
Status
Operator–initiated breath
02–16 15 10
NAVA preview
NAVA press. estimate
NAVA level
2.0
0.0
cmH2O/μV
30.0
Close
20 cmH2O Pest
60 l/min
-60
400 ml
20 μV
Ppeak (cmH2O)
17
50
Pmean (cmH2O)
7
PEEP (cmH2O)
3
RR (b/min)
21
35
5
O2 (%)
38
MVe (l/min)
C
6.0
37.5
0.5
VTi (ml)
312
VTe (ml)
294
Edi peak (μV)
7.6
Edi min (μV)
0.1
Additional settings
O2 conc.
40
%
21
100
PEEP
3
cmH2O
0
50
Resp. Rate
14
b/min
4
150
PC above PEEP
14
cmH2O
0
120
Additional values

NAVA

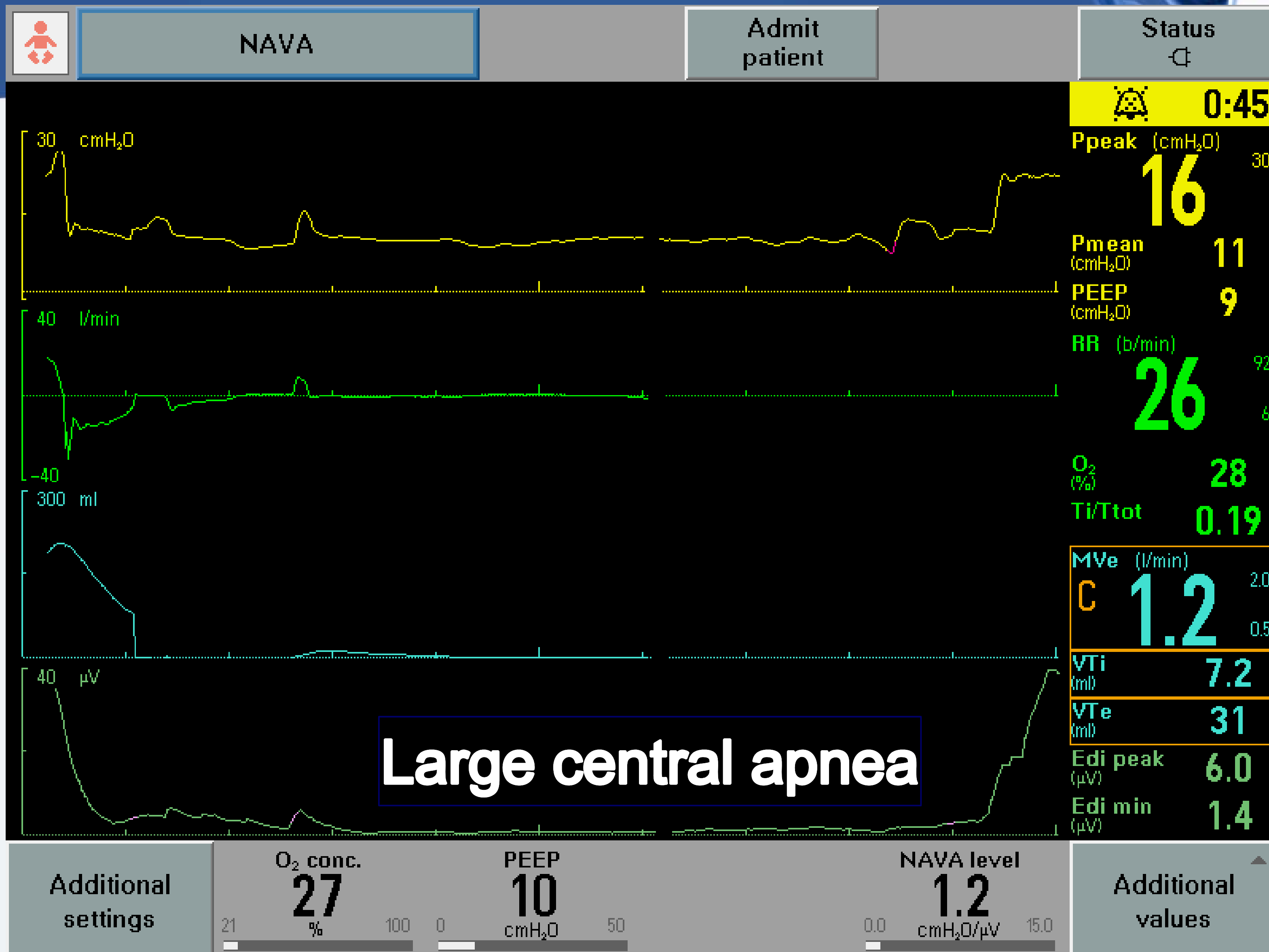


NAVA
Admit patient
Nebulizer
Status
08-10 13 19
25 cmH2O
40 l/min
-40
150 ml
12 µV
Ppeak (cmH2O)
13
40
Pmean (cmH2O)
8
PEEP (cmH2O)
5
RR (b/min)
35
70
20
O2 (%)
28
Ti/Ttot
0.39
MVe (l/min)
C
2.9
6.5
1.0
VTi (ml)
77
VTe (ml)
86
etCO2 (%)
4.8
Edi peak (µV)
5.5
Edi min (µV)
0.3
Additional settings
O2 conc.
28
%
21
100
PEEP
5
cmH2O
0
50
NAVA level
1.3
cmH2O/µV
0.0
30.0
Additional values

NAVA Patient examples #1

- 1 yr old
- History of cerebral palsy
- Admitted with query GI bleed
- PCO2 ↑150
- Intubated and ventilated
- SIMV PC/PS   PC10, PS 8
- PEEP 5
- RR 8
- FiO2 .25
- Normal ABGs on these settings.
- PS/CPAP ventilation revealed frequent apneas

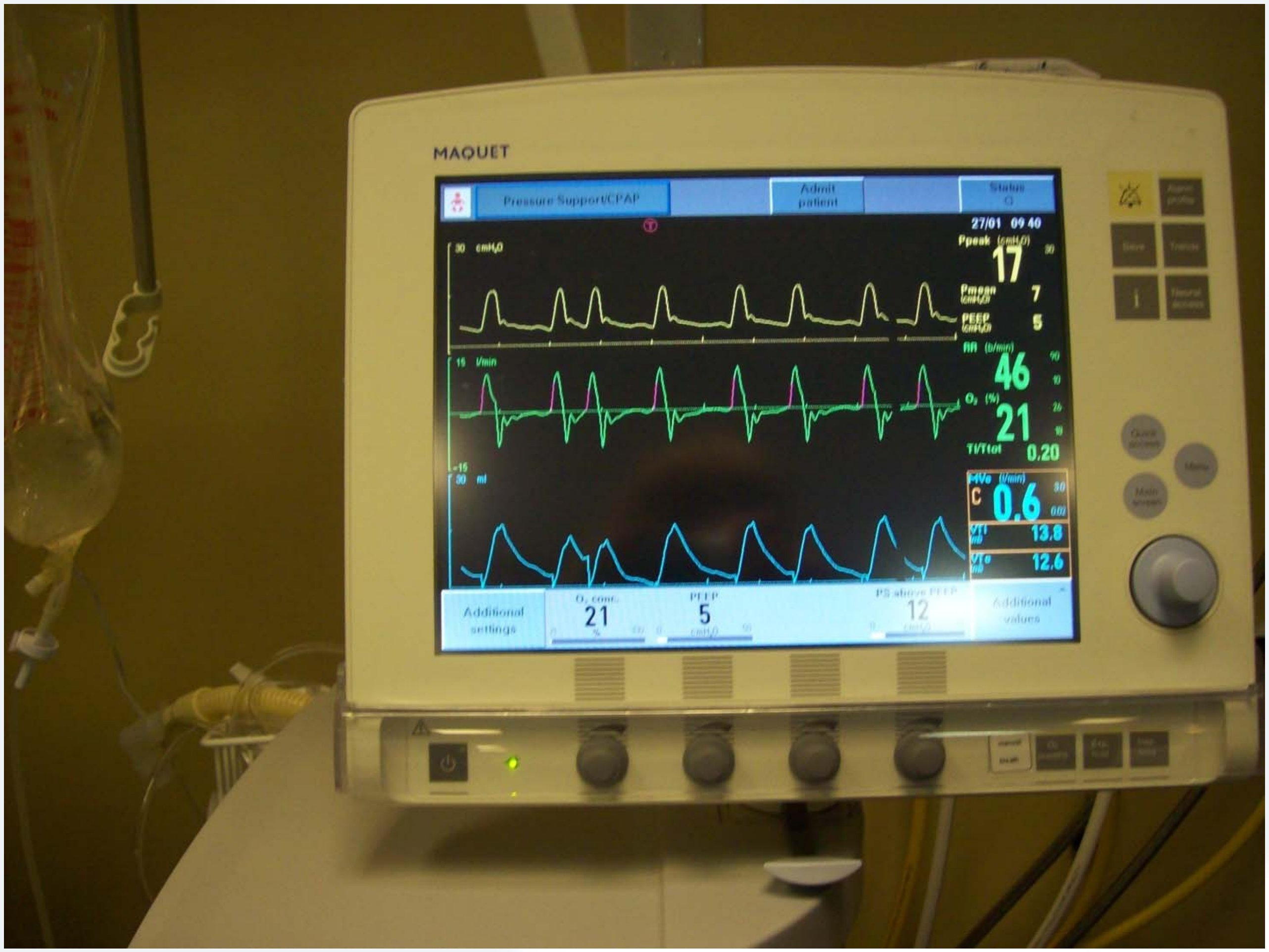
NAVA
Admit patient
Status
0:18
15 cmH2O
7 l/min
-7
50 ml
20 µV
Ppeak (cmH2O)
11
30
Pmean (cmH2O)
8
PEEP (cmH2O)
7
RR (b/min)
26
70
6
O2 (%)
27
Ti/Ttot
0.69
MVe (l/min)
C
0.9
3.0
0.10
VTi (ml)
15.0
VTe (ml)
3.8
Edi peak (µV)
24
Edi min (µV)
20
Additional settings
O2 conc.
27
21
%
100
PEEP
5
0
cmH2O
50
NAVA level
1.0
0.0
cmH2O/µV
15.0
Additional values


NAVA
Admit patient
Status
0:45
Ppeak (cmH2O) 16 30
Pmean (cmH2O) 11
PEEP (cmH2O) 9
RR (b/min) 26 92 6
O2 (%) 28
Ti/Ttot 0.19
MVe (l/min) C 1.2 2.0 0.5
VTi (ml) 7.2
VTe (ml) 31
Edi peak (µV) 6.0
Edi min (µV) 1.4
30 cmH2O
40 l/min
-40
300 ml
40 µV
Large central apnea
Additional settings
O2 conc. 27 % 21 100
PEEP 10 cmH2O 0 50
NAVA level 1.2 cmH2O/µV 0.0 15.0
Additional values

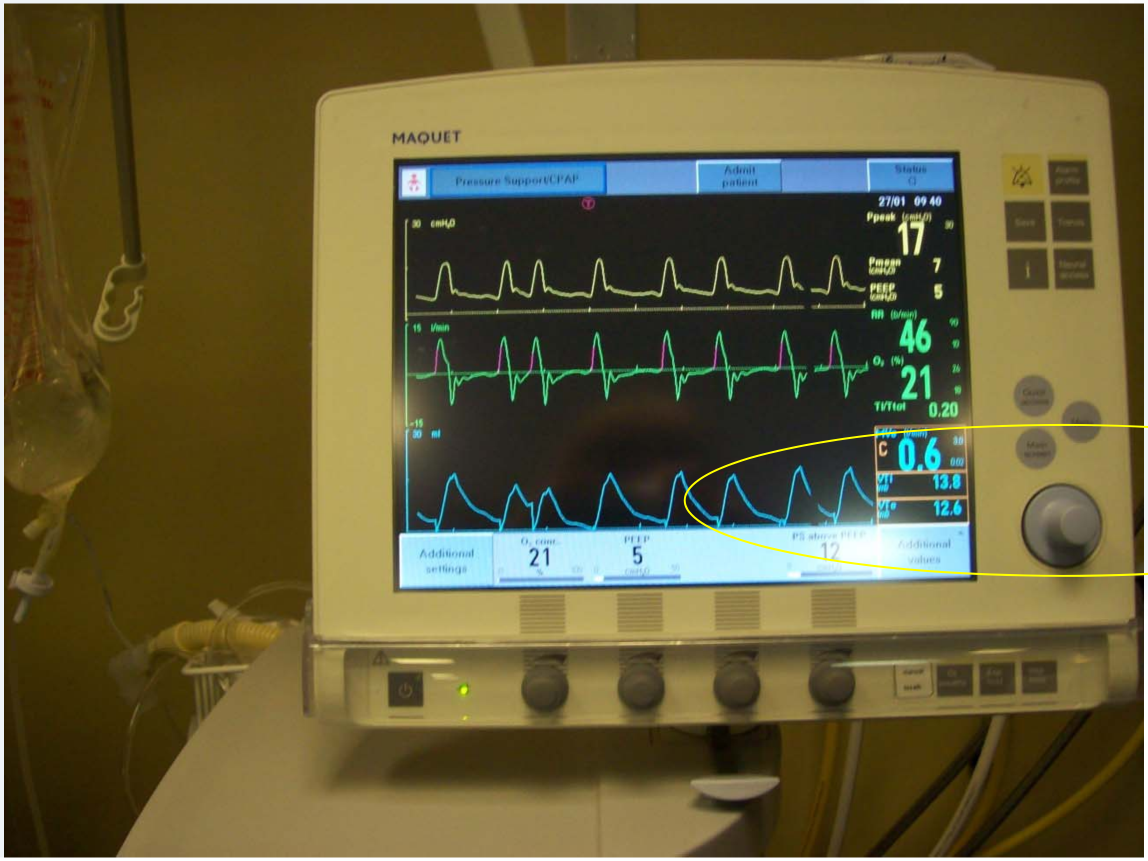
NAVA Patient examples #2

- Infant (6mo) PICU ventilated – very weak, poor muscle tone.
- Meds:
- On PSV – do not want this kid to fail extubation b/c difficult airway!
- Decided to start NAVA

Appears to have good efforts and synchrony with PSV



However, most of the breaths are auto-cycled by his leak. Baby actually had little spontaneous effort.



NAVA Patient example #3

- 4 year old with neuroblastoma
- Admitted with increasing SOB
- ARDS-like pattern – pneumo
- PRVC, sedated/nimbex X 1 week
- Day 8: chest tube out
- Day 9: no reaccumulation of air.

  Stop Nimbex

  Decrease Morphine

  Start on NAVA when possible

Day 1-2 on NAVA

24 hour trend showing initial poor efforts.

Day 3 on NAVA – total spontaneous breathing

Catheter position checked after moving patient back to bed

Catheter repositioned and rechecked

NAVA – what's the proof?

- Many studies – feasibility studies (neonates and children) or crossover design (between PSV or PC)
- Can be used in all age groups
- Appears to be more physiological in terms of respiratory parameters
- Detection of central apneas
- Improved patient synchrony and comfort
- Intact diaphragm essential
- May decrease PIP and FiO2

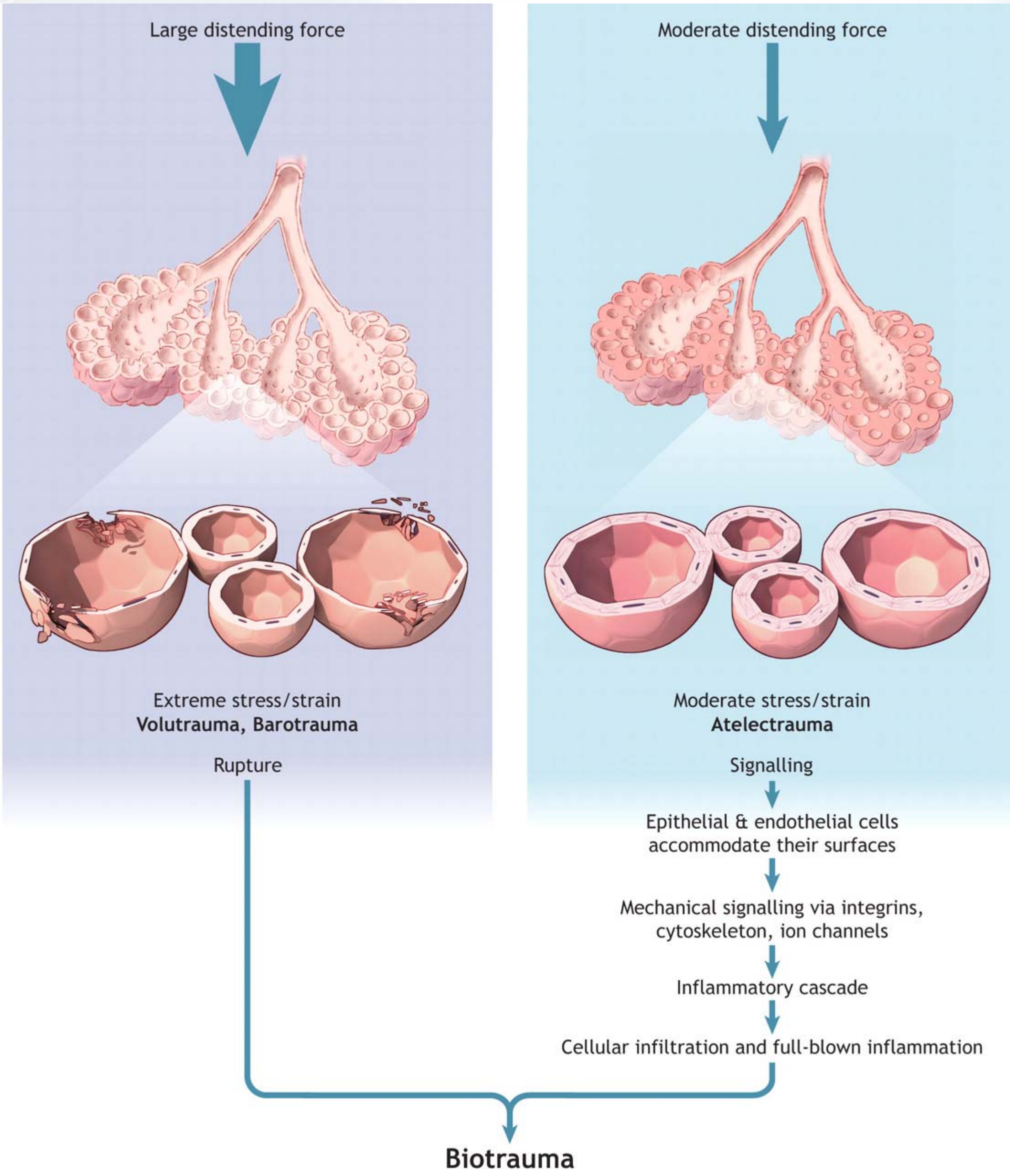
NAVA Drawbacks

- Only available on Servo I ventilator
- Costly
- Requires nasal/oral gastric catheter
- Investment in training time
- Surveillance when starting NAVA
- Need to recheck catheter every time patient is moved

THE ADVERSE EFFECTS OF POSITIVE PRESSURE VENTILATION

Barotrauma/Volutrauma/Biotrauma
Hemodynamic/Cardiovascular effects
Neuromuscular weakness
Infection; Ventilator-associated pneumonia

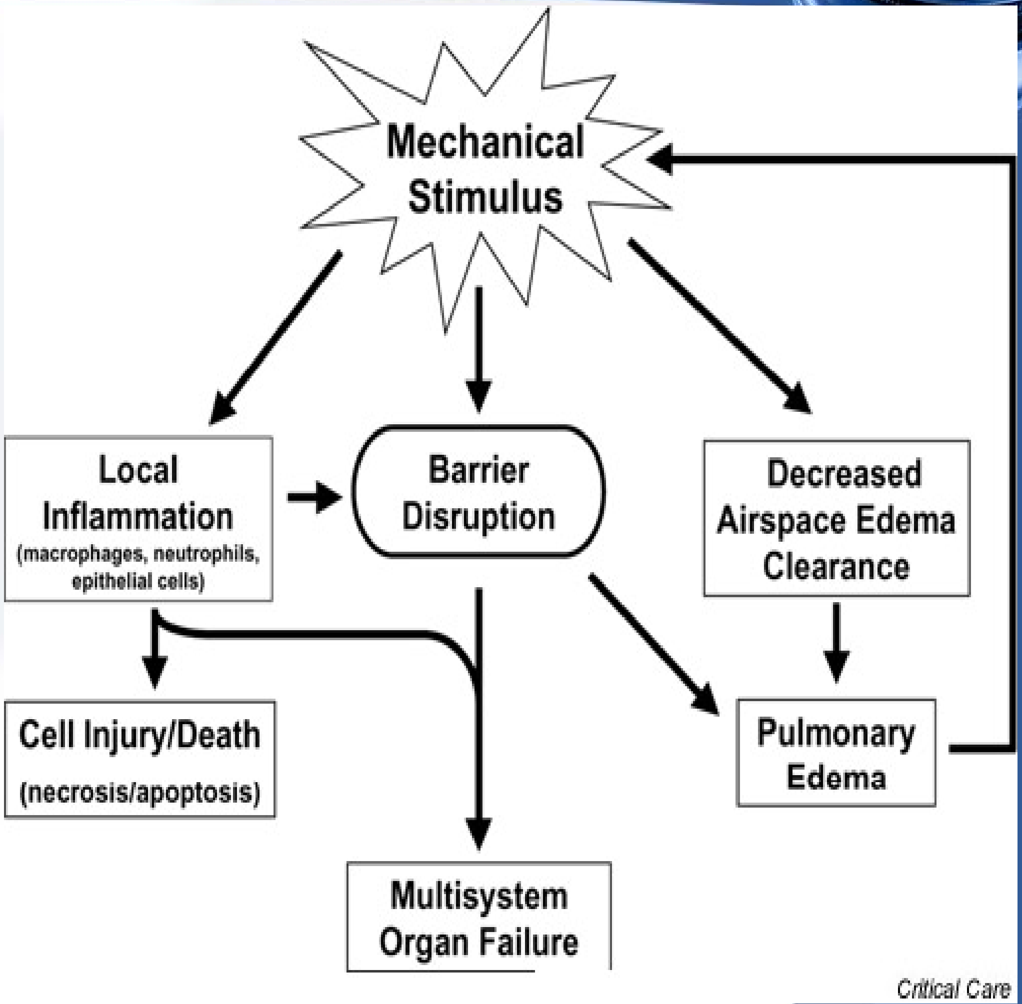
THE SCARIEST EFFECTS….



- BAROTRAUMA / VOLUTRAUMA
- DUE TO OVER DISTENSION OF LUNG TISSUE
- UNEVEN DISTRIBUTION OF VENTILATION
- *"SHEARING"* INJURY

Ventilator-induced lung injury (VILI)

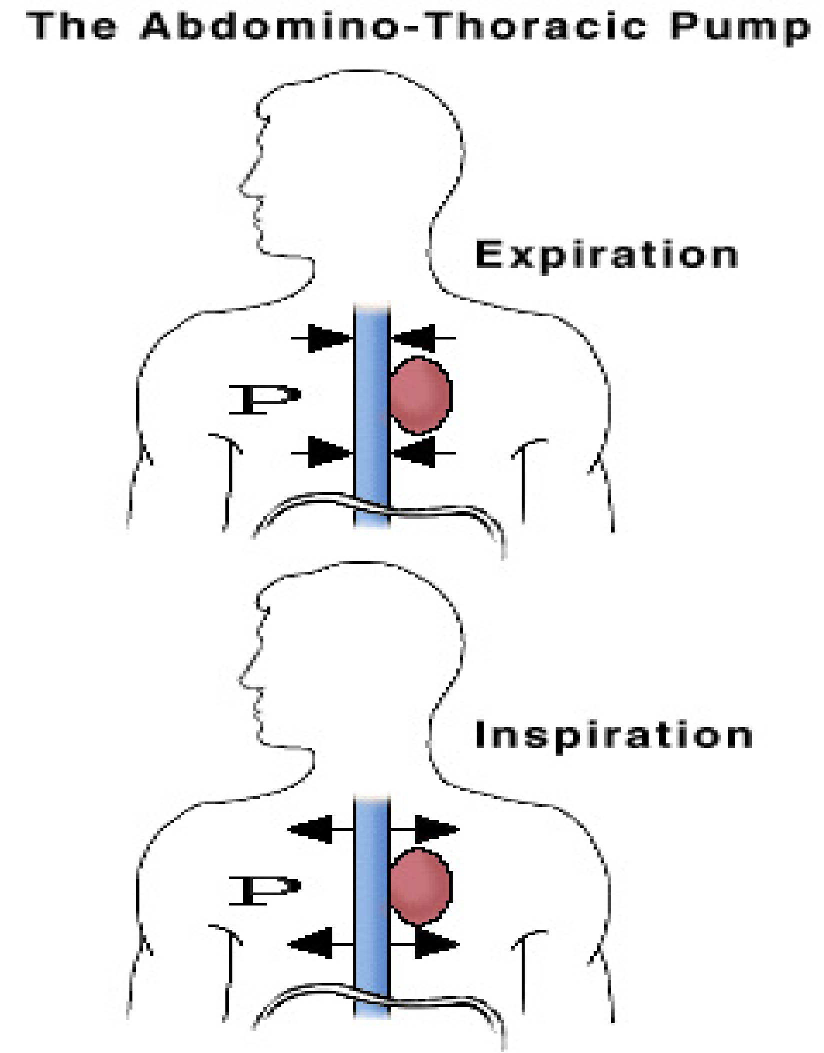
- Lower VTs (4-6 mls/kg) is the only strategy that has proven to improve survival in ARDS (ARDSnet.org,1999)
- ALVEOLI study determined no survival benefit with higher PEEPs vs lower PEEPs (Brower et al. *N Engl J Med*. 2004 Jul 22;351(4):327-36).



Frank and Matthay *Critical Care* 2003 **7**:233-241 doi:10.1186/cc1829

CARDIOVASCULAR EFFECTS

- ↑'ed INTRATHORACIC PRESSURE MAY ***↓VENOUS RETURN***
- LOSS OF ***"THORACIC PUMP"***
- ↓'ed ***RV PRELOAD , STROKE VOLUME, and CARDIAC OUTPUT***



How are our patients once they leave the ICU?

- Data collection at 1 year post-ICU for patients with sepsis, MODS and prolonged mechanical ventilation (Needham 2008)
- 49% returned to work
- 46% with ***neuromuscular dysfunction*** and mobility impairment
- Significant ↓ in QOL; attributed to
  - Loss of pulmonary function
  - Loss of muscle strength / fatigue
  - ↓ mobility

Early Mobilization of ventilated patients in the ICU

- Positive outcomes from early mobilization of ventilated patients (Schweickert et al. 2009, Li et al. 2012)
  - ↑ return to functional status (59% vs 35%, p=0.02)
  - ↓ ICU delirium (2 days vs 4 days, p=0.02)
  - ↑ ventilator-free days (21.1 vs 23.5, p=0.05)
  - One serious adverse event (Sp02 <80%) in 498 txs
  - d/c of tx due to destabilization of pt occurred in 19/498 txs; most common due to pt:vent asynchrony

Early Mobilization of Ventilated Patients in ICU

Early Mobilization of ventilated patients in the ICU

- Great example of interprofessional collaboration of the ICU team!
  - RNs, RTs and PTs

Needham (2008) JAMA

Ventilator Associated Pneumonia (VAP)

Ventilator-Associated Pneumonia

Centre for Disease Control (CDC) Definition:

Pneumonia is identified by using a combination of radiologic, clinical and laboratory criteria.

Ventilator associated pneumonia – onset of pneumonia in a patient who was intubated and ventilated at the time of, or within the 48 hours before, the onset.

NOTE: There is no minimum period of time that the ventilator must be in place in order for the pneumonia to be considered ventilator associated.

(CDC; *Device Associated Events,* 2012)

Ventilator-Associated Pneumonia

Impact of VAP

- Second most common healthcare acquired infection (HCAI)
  - 15% of all HCAI
  - 25% of all HCAI in ICU
- Approximate cost of VAP is as high as $40,000(US) per patient
- Accounts for 60% of deaths associated with HCAI
- Increases length of stay in ICU and in hospital (4 - 13 days, Kroeng & Turwit, Clin Micro Rev 2006).

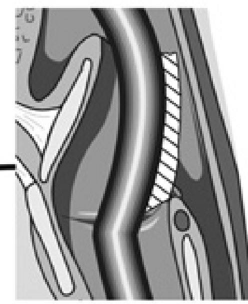
(CDC; ICD-9-CM, 2007)

Pathogenesis of Ventilator Associated Pneumonia

Endogenous Sources of Micro-organism

1) Impaired natural protection/clearance system allow increase colonization of nasopharynx

2) Colonized oropharynx & gastric fluid pool along tube in neonates



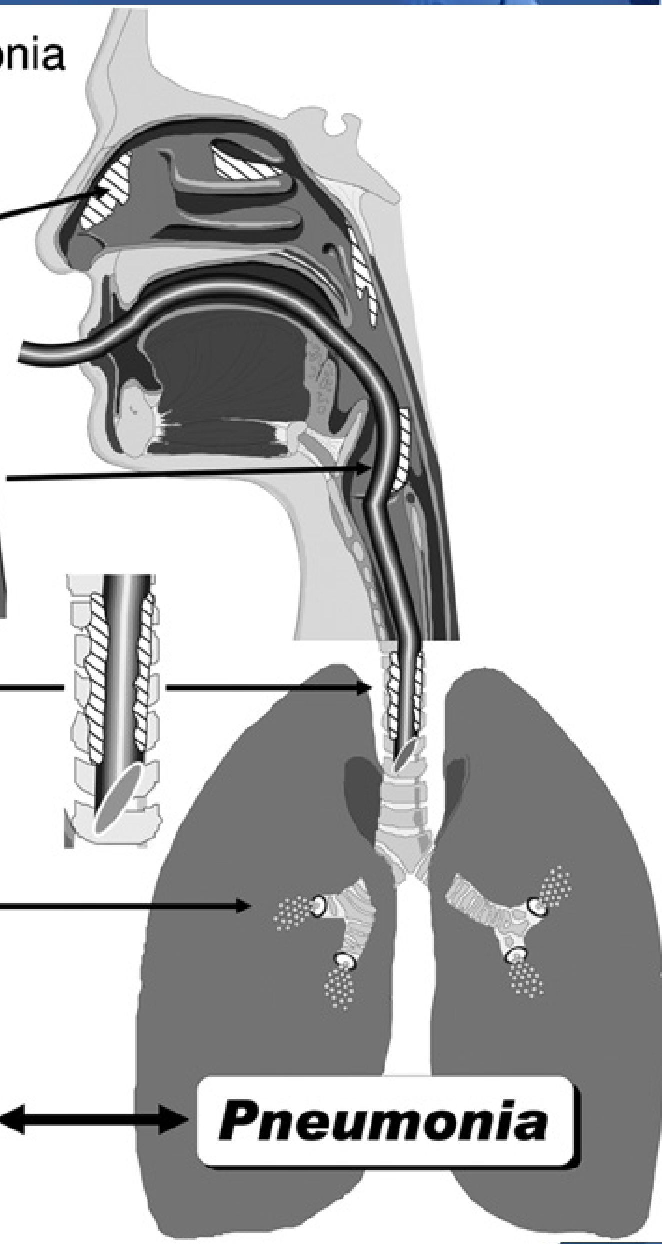
3) Colonized tracheal secretions

Mechanism for pneumonia

1) Aspiration of colonized fluids from any of the above sources into lungs can result in pneumonia

2) A hematogenous source seeding the lungs may rarely cause pneumonia

Blood ↔ **Pneumonia**



(Garland, Clin.Perinatol., 2010)

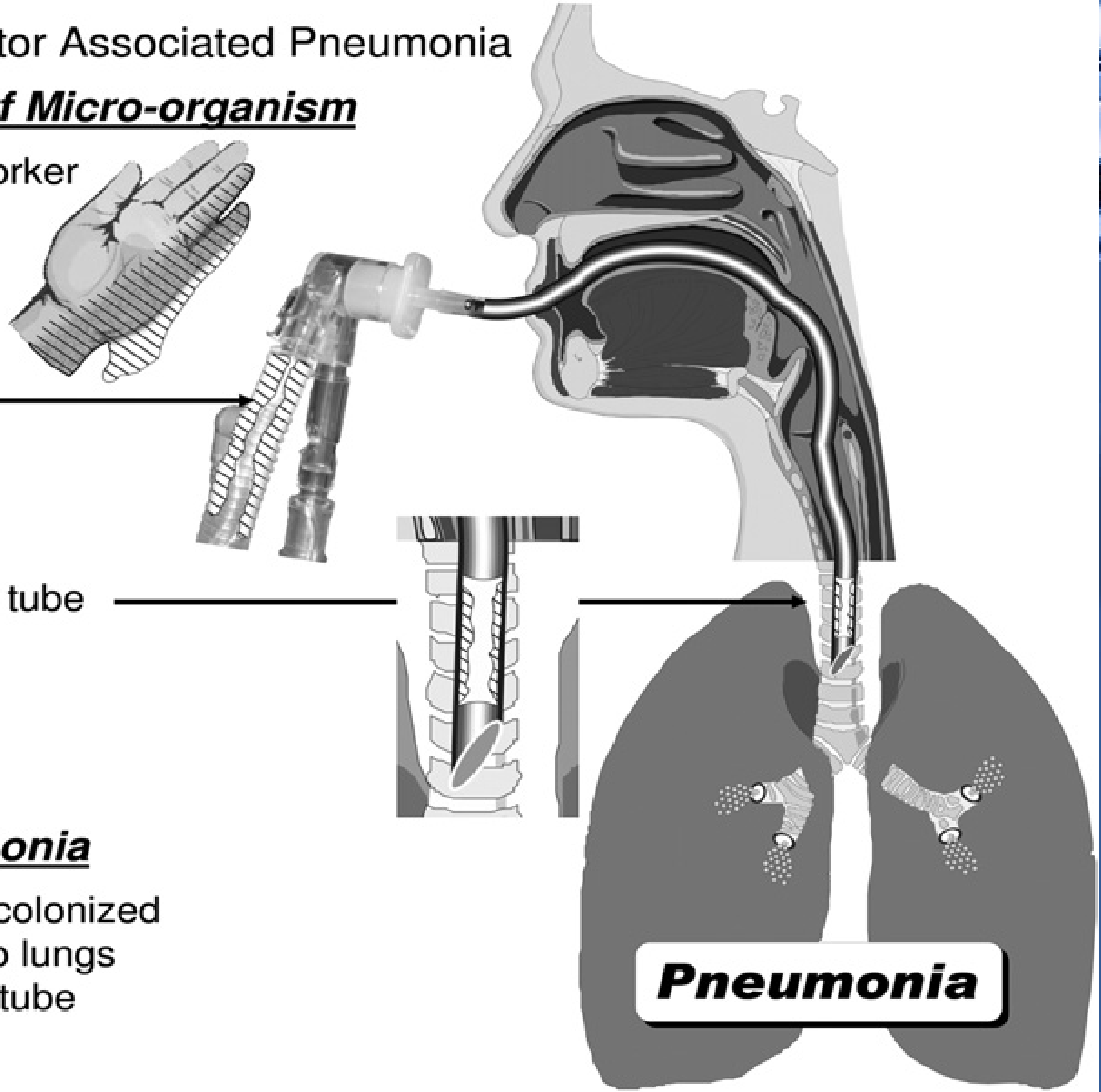
Pathogenesis of Ventilator Associated Pneumonia

Exogenous Sources of Micro-organism

1) Hands of healthcare worker

2) Ventilator circuit

3) Biofilm of endotracheal tube



Mechanism for pneumonia

Pneumonia occurs when colonized secretions are inhaled into lungs through the endotracheal tube

(Garland, Clin.Perinatol., 2010)

Prevention

Safer Healthcare Now! Campaign
How-to Guide: Prevention of VAP

safer healthcare now!

VAP Bundles

- Strategy to reduce VAP rates
- Group of practices
- Most data from adult literature extrapolated to pediatric and neonatal practice; small pool of pede/neo data
- Each unit can customize bundle to suit their needs
- Must be followed to assess whether practices are being followed and whether a difference is made in VAP rates.

Table 2
Interventions often included in bundles to prevent VAP

Adult Interventions to Prevent VAP Not Applicable to Neonates	Adult or Pediatric Interventions to Prevent VAP Applicable to Neonates	Adult Interventions to Reduce VAP Unknown Risk: Benefit in Neonates
Cuffed endotracheal tubes (II[a])	Caregiver education (IA)	Elevation of head of the bed (II)
Subglottic suctioning of secretions (II)	Hand hygiene (IA)	Oral care with antiseptic solution (II)
Silver-coated endotracheal tubes	Wearing gloves when in contact with secretions (IB)	Orotracheal vs nasotracheal intubation (IB)
Deep venous thrombosis prophylaxis	Minimize days of ventilation (IB) Prevent gastric distension Avoid unplanned extubation Change ventilator circuit only when visibly soiled or malfunctioning (IA) Disinfect respiratory equipment before storage (IA) Remove condensate from ventilator circuit frequently (IB) Avoid reintubation (II)	In-line (closed) suctioning Sedation vacation to assess extubation readiness Orogastric tube vs nasogastric tube

CDC categorization of evidence-based recommendations.

Recommendations categorized based on existing scientific evidence, theoretic rationale, applicability, and potential economic impact in adult patients.

Category IA: Strongly recommended for implementation and supported by well-designed experimental, clinical, or epidemiologic studies.

Category IB: Strongly recommended for implementation and supported by certain clinical or epidemiologic studies and by strong theoretic rationale.

Category II: Suggested for implementation and supported by suggestive clinical or epidemiologic studies or strong theoretic rationale.

[a] Category of recommendation for adult patients.

Prevention

Additional VAP Ventilator Bundle Components

- Elimination of routine instill during suction (CPIS, 2009)
- Oral suction before ETT suction and prior to movement of ETT; separate catheter or oral suction device (Curley, et al, 2006)
- Oral versus nasal ETT (Muscedere, 2008)
- Oral care in neonates – removing plaque from gums, intact mucus membranes, preventing xerostomia (CPIS, 2009)
  - sterile water
  - breast milk

Prevention

Additional VAP Ventilator Bundle Components

- Care of ventilator tubing
  - Dependent position for prevention of accidental instill (Curley et al, 2006)
  - Do not cross diaper line (AARC)
  - Empty condensate – double heated wire circuits, water traps, avoiding drafts in unit (Curley et al, 2006)

- Circuit change only when visibly soiled or malfunctioning (AARC, 2003)

- Positioning of infant (Hanly el al., 2008)

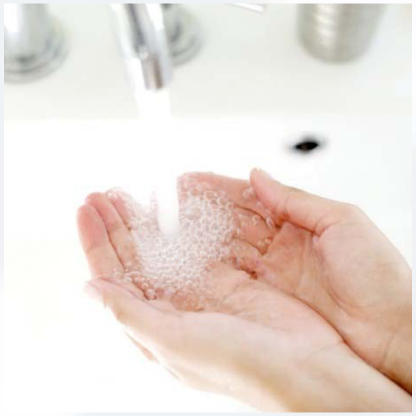
Prevention

Other considerations:

- Expiratory valve housing protection for HFO to prevent droplet spray (MacDonald et al, Ped.Crit.Care.Med., 2011)
- Disinfection of equipment (Sui et al, Resp.Care, 2012)
- Other sources: water supply, HVAC (Ryan et al, 2011), reservoirs (sinks, A/C)

Prevention

#1 –Hand Hygiene!



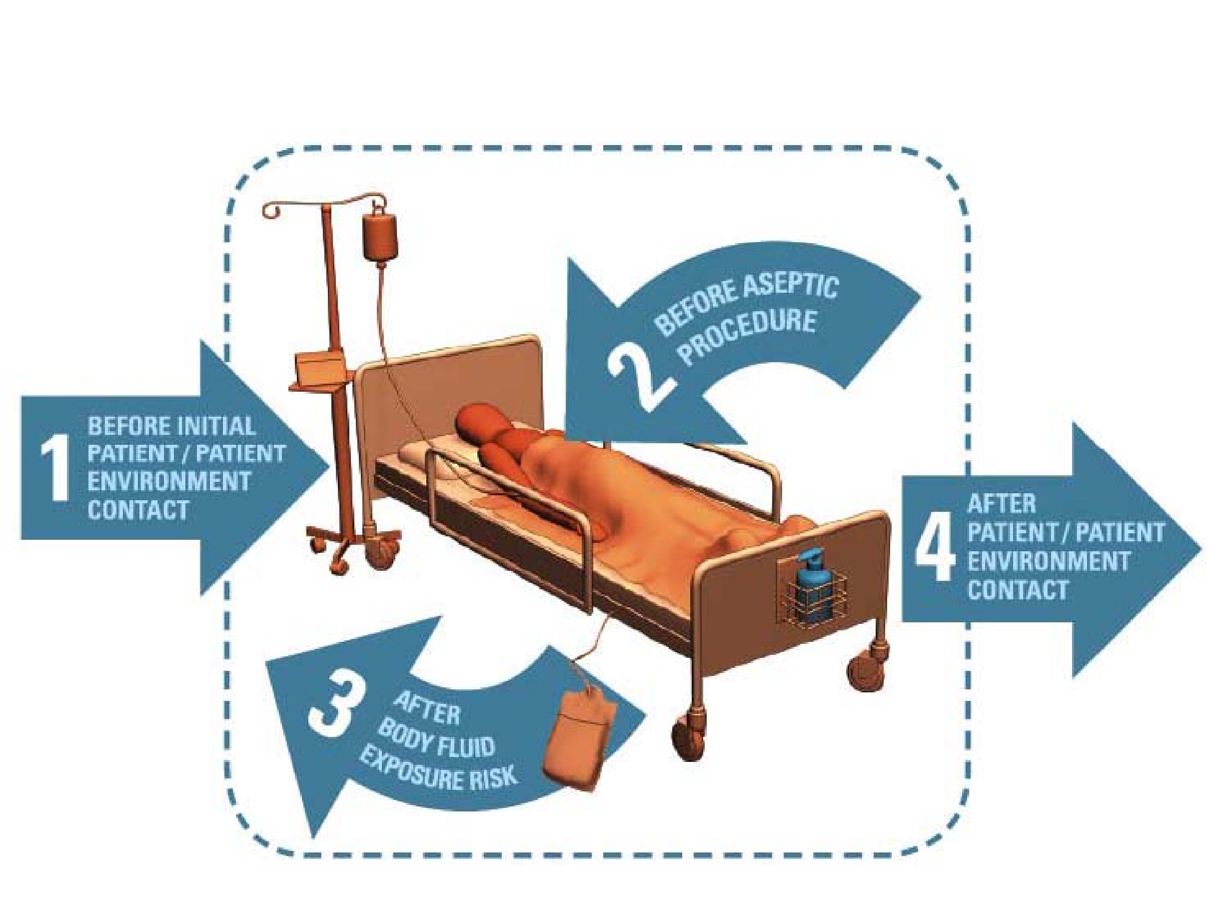
Hand hygiene compliance by healthcare workers as low as < 40%

An increase of 20% in compliance results in a 40% decrease in HCAI

(McGeer,, *Ontario Medical Review, 2008;)*

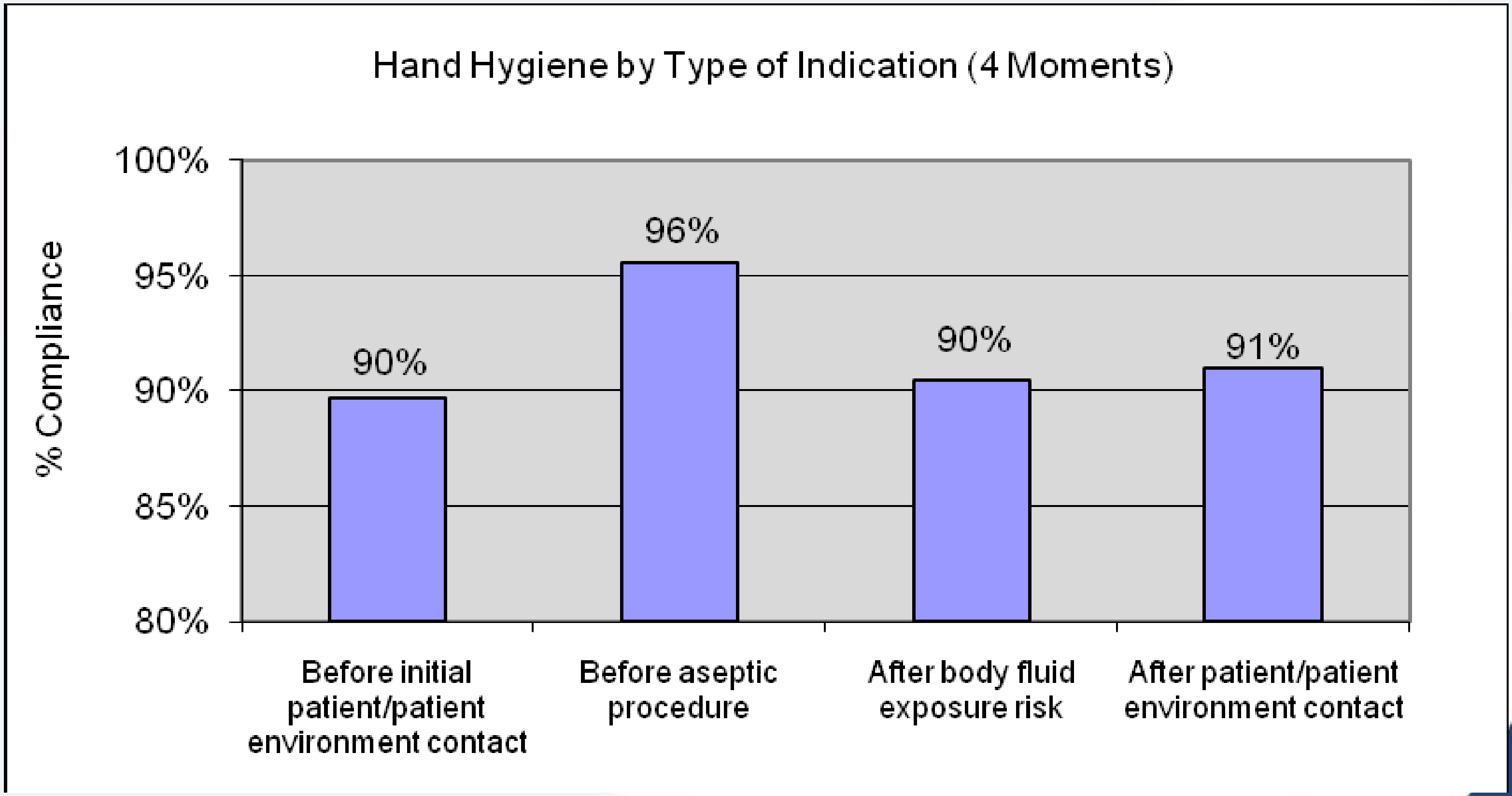
Prevention

IWK, June 2010: Educational components of Canada's Hand Hygiene program



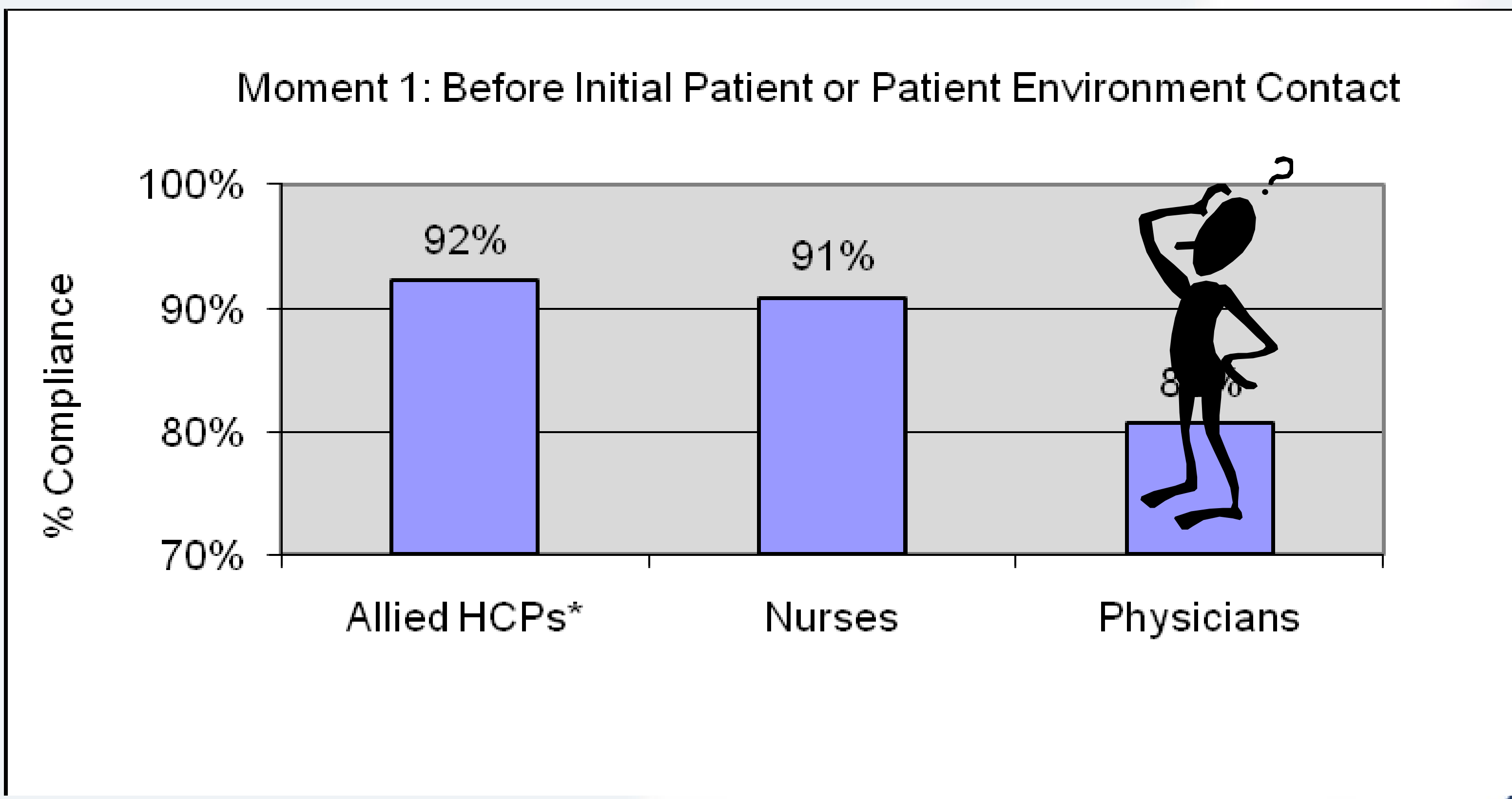
Prevention

Audit for compliance:



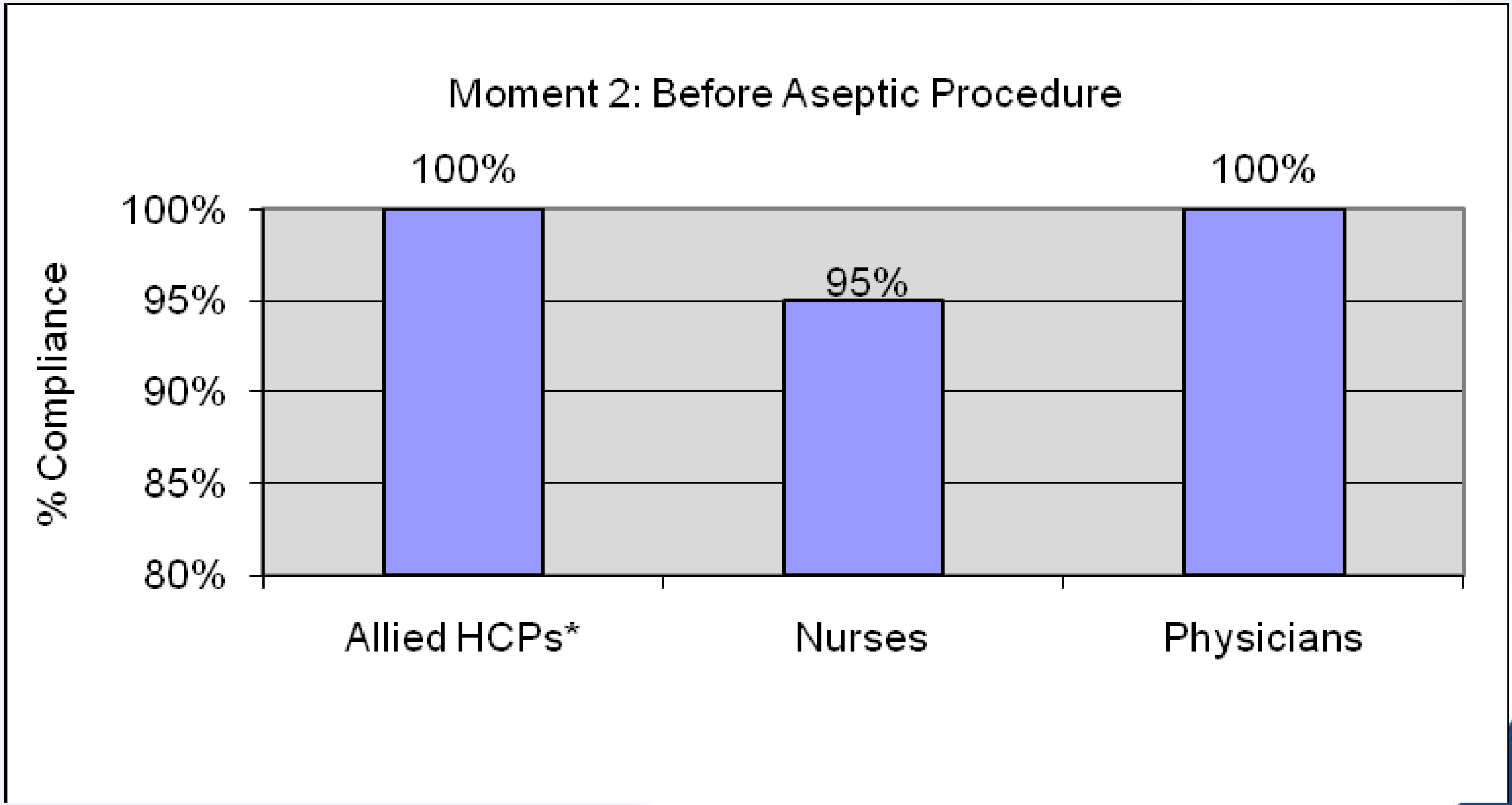
Prevention

Audit for compliance:



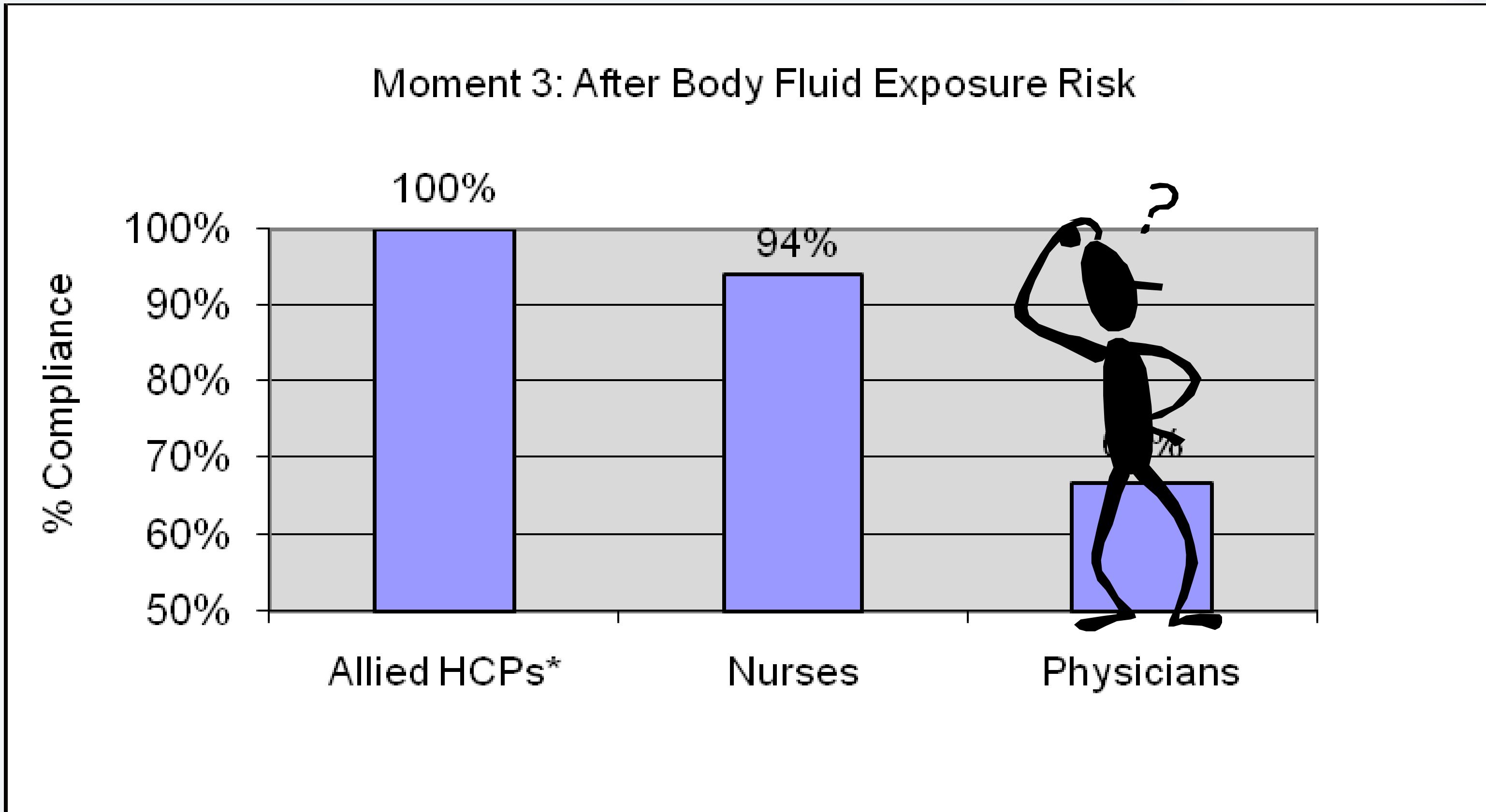
Prevention

Audit for compliance:



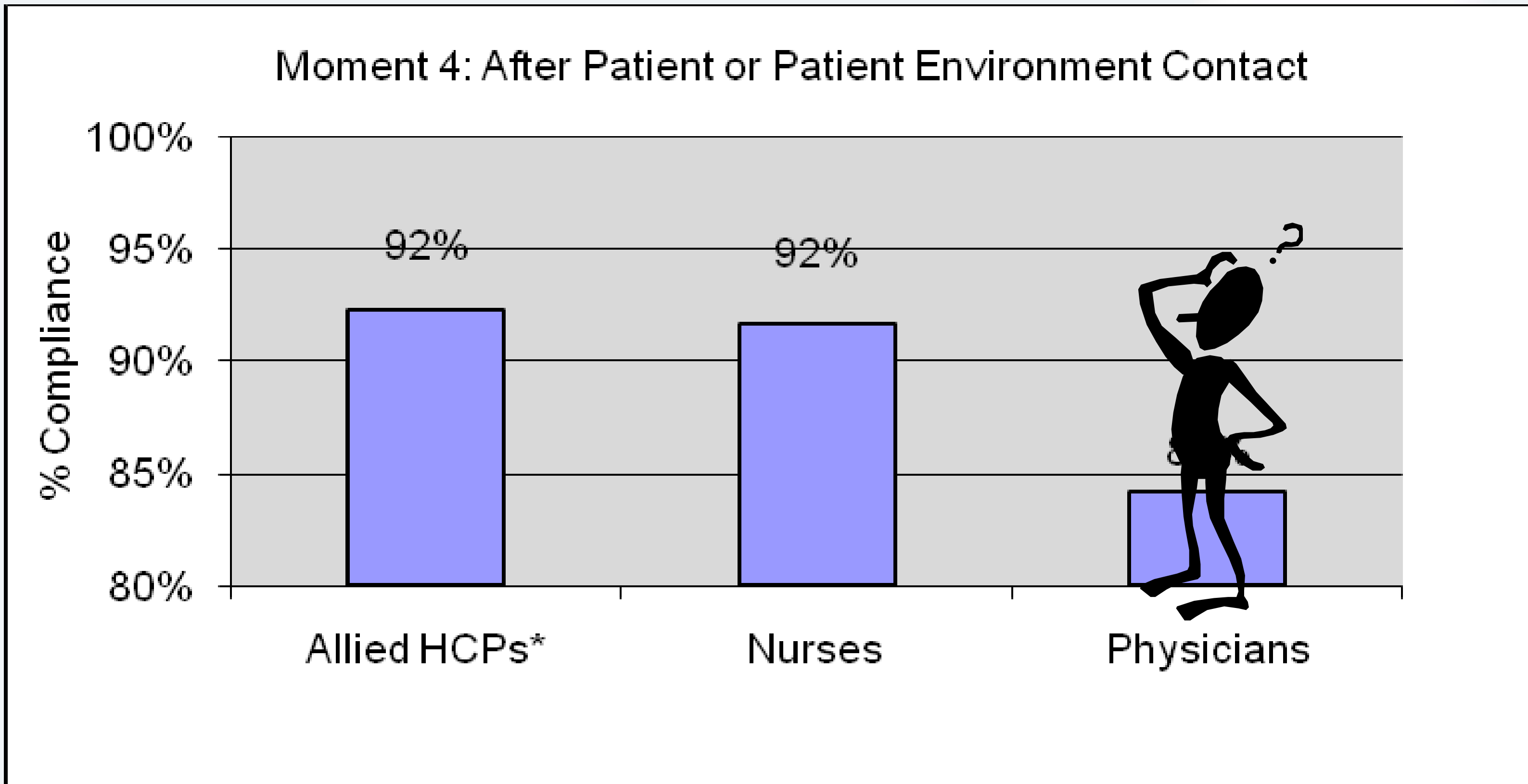
Prevention

Audit for compliance:



Prevention

Audit for compliance:



References

- Scanlan, C., Wilkins, R., and Stoller, J. (2009) *Egan's Fundamentals of Respiratory Care*, 9th ed. St. Louis: Mosby.
- Cairo, J. and Pilbeam, S. (2010). *Mosby's Respiratory Care Equipment,* 8th edition. St. Louis: Mosby.
- Oakes, Dana and Shortall, Sean (2009). Oakes Ventilator Management. Orono: Health Educators Publications

Questions? / Comments?