NEW RESPIRATORY THERAPY STRATIGIES IN THE NICU

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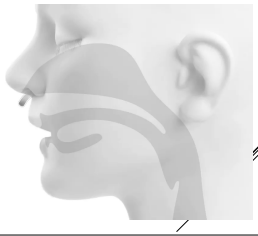
SMALL BORE
LARGE BORE

Work of Breathing Reduced Through Dead Space Ventilation

AT LEAST 30% OF INSPIRED TIDAL VOLUME IS DEAD SPACE VENTILATION

AT START OF INSPIRATION, ANATOMICAL DEAD SPACE IS FILLED WITH EXPIRATORY GAS, WHICH IS OXYGEN DEPLETED AND HIGH IN CO2

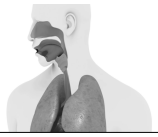
PRIMARY MECHANISM OF ACTION: DEAD SPACE FLUSH



VENTILATION EFFICIENCY – ALVEOLAR VENTILATION

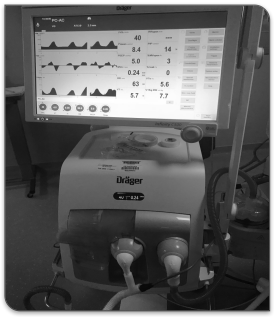
- **Minute Ventilation = Tidal Volume x Respiratory Rate**
- **Alveolar Ventilation = (Tidal Volume – Dead Space) x Respiratory Rate**
- **Alveolar ventilation improves with a reduction in dead space volume independent of tidal volume**
- **and respiratory rate**

Alveolar Ventilation
Volume of gas made available to the respiratory region of the lungs per minute



CONVENTINAL VENTILATION

- Most accepted form of ventilation
- Deliveries monitored press or volume
- Maintains gas exchange
- PRESSURE SIMV
- PC-AC VG



CONVENTIONAL VENTILATION

Definition: Ventilation is the exchange of gases, oxygen and carbon dioxide, between the alveoli and the pulmonary capillaries.

Modes of Ventilation:

SIMV – synchronized mandatory ventilation
PC-AC VG (pressure control-assist control, volume guarantee)

PRESSURE SIMV

RATE IS SET

PRESSURE IS CONTROLLED

VENTILATOR SYNCHRONIZES WITH PT

TIDAL VOLUME IS VARIABLE

PC-AC VG

- Pressure is variable with a set limit (P-max)
- Vt (tidal volume) is set
- Ventilator attempts to deliver target Vt by adjusting PIP up and down in an attempt to deliver set Vt (volume guarantee)
- Pt receives "full" ventilator breath with every spontaneous effort (assist control)

SURFACTANTS

Established Safe and Effective

In the early 1990's

Mixture of 90% lipids and 10%proteins

Main function to reduce surface tension

LIPIDS ROLE

PC

phosphatidylcholine

DPPC

Dipalmitoylphosphatidylcholine

GENERALLY ACCEPTED AS BEING RESPONSIBLE FOR NEAR ZERO SURFACE TENSIONS AT THE AIR/LIQUID INTERFACE

Bilayer lipid structures are attached to the monolayer and together they make up the surface film

These bilayer lipid structures form a LIPID RESERVOR

From which lipids and possibly proteins are inserted during inhalation

Tubular myelin

SURFACTANT SPECIFIC LATTICE LIKE LIPID STRUCTURE

PG

PHOSATIDYLGLYCEROL

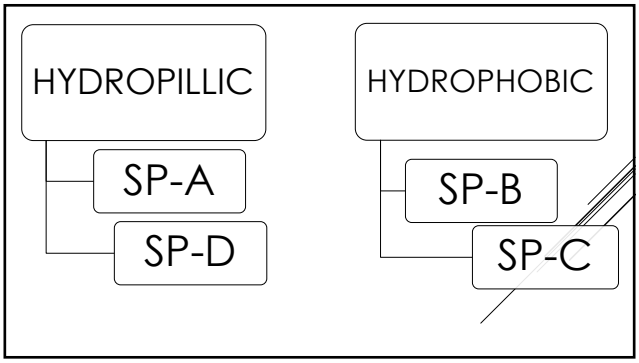
DPPC:PG

HAS INCREASED ABSORPTION ACTIVITY THAN PC ALONE

PROTEINS

FOUR SURFACTANT SPECEIFIC PROTEINS

- **Surfactant protein A (SP-A)**
- **SP-B**
- **SP-C**
- **SP-D**



SP-A

First surfactant protein detected

1) **Tubular myelin formation**
2) **Protection of the surface film against protein inhibition**
3) **Enhancement of SP-B's surface activity**
4) **Regulation of uptake and secretion of surfactant by type II cells**

SP-A's role in host defense

SP-B
Hydrophobic protein

- PROMOTION OF LIPID ABSORPTION AT THE AIR/LIQUID INTERFACE
- FORMATION OF TUBULAR MYELIN
- RESPREADING OF FILMS FROM THE COLAPSE PHASE
- REUPTAKE OF SURFACTANT BY TYPE ii CELLS
- STABILATION OF THE MONOLAYER FILMS

SP-C
THE ONLY TRUE SURFACTANT PROTEIN

IN CONTRAST TO SP-B,

ONLY A FEW ACTIVITIES HAVE BEEN DESCRIBED FOR SP-C

Most overlap SP-B'S activity

Animal

Vs

Artificial

WHEN TO TREAT WITH SURFACTANT THERAPY

Initial randomized trials

- Mortality
- Decreases incidence of air leak (pneumothorax, PIE)
- Lowers the risk of chronic lung disease or death

Early

Vs

Delayed Selective Surfactant Tx of RDS

INSURE

The Vermont Oxford Network Delivery Room Management Trial

Infants that received early administration had higher incidence of BPD or death than infants stabilized on CPAP alone