Control of breathing and ventilation

(Notes from :

“The Control of Breathing During Mechanical Ventilation “

”The Injurious Effects of Elevated or Nonelevated Respiratory Rate during Mechanical Ventilation”)

Normal control

T**ime constan**t - (best look at expiration - simpler)

T.C of emptying = **R x C**
(if increased compliance, the lung volume at beginning of expiration is larger, therefore longer to empty)

Normal compliance - 0.1 L/cm H20
Normal resistance = 5 cm/H20/L/sec

Normal time constant = 0.5 sec (i.e., volume and flow will be 1.3rd of peak - I.e., **66% empty**)
(at 1 sec = 1/9th)

Therefore it will reach FRC during normal expiration time. If R increases, it will exceed time to reach FRC and will lead to PEEPi - assynchrony.

NB. Driving pressure is tidal volume scaled to compliance

Respiratory Rate (RR)

RR changes minimally in response to : assist level, pCO2

In conscious volunteers -> **RR is an insensitive to assist nor pCO2**.

RR -> response minimal to CO2 between (23-45). **CO2 works mainly through inspiratory effort**.

Sleep- removing wakefulness stimulus to breath -> **RR depends on CO2**. Normal or **decreased CO2 by 3-4 mmHg from normal -> apnoea** = Apnoeic Threshold (AT)

During sleep - as CO2 decreases RR stable until -> AT =0

Between normal CO2 -> AT - ventilatory control only by change in effort (RR insensitive to change in CO2).

Beyond a level of **respiratory drive increase** which is **3-4 X higher than resting** -> **RR increases substantially**.

ARDSNet protocol allows RR during ventilation up to 35/min to keep pH >7.3 but a high RR can cause VILI

Higher centres

1) ***Sleep*** - control is mainly via chemoreceptors.

Increased pCO2 will increase respiratory **drive** not RR. At **3-4 X increase of drive above normal, then RR will increase.**

Hypocapnia - RR does not decrease until **pCO2 slightly below normal (3-4** mmHg) = **apneic threshold (AT) - therefore stops breathing.**

Therefore over ventilation continues until CO2 decreases below AT - recurrent apneas.

2) ***Awake*** - higher centres work here

-Respiratory center does **NOT cease below AT** - causing severe drop in pCO2.

-Behavioral response - breathing events not perceived unless different from what is expected - highly unpredictable response.

Desired breathing patterns -

Breathing pattern determined by RR and ventilation needed to satisfy metabolic demands

Vt is a **dependant** variable - it is **adjusted** to provide **ventilation at the prevailing RR.**

A **high RR does not necessarily denote distress or increased demand**.

RR normally **5-25**

To differentiate a ”normal” increased RR from distressed RR, **in distress, the RR decreases if ventilator assist is increased**, if not - not distressed related.

Normally there are large fluctuations of Vt - put a normal volunteer on a ventilator, give a fixed Vt, which equals the average Vt for that person - it is very uncomfortable! Comfort returns if you increase the Vt well above the average, but this leads to hypocapnia.

Control of breathing with different ventilatory modes

PEEPi - alveolar pressure at end expiration is **ABOVE** external PEEP.
To trigger, you must be below external PEEP.
Sometimes by **increasing external PEEP** - reduces the difference and **aids triggering.**

*1)**Volume cycled***

Vt and Ti are preset

*2) **Pressure cycled***

Ti is preset
Set pressure determines minute ventilation
NB. Patient’s RR does not decrease in response to hypocapnia.

These 2 modes tend to over ventilate. In alert patients, constant Vt is poorly tolerated unless ventilation is high - leading to hypocapnia. Therefore **tends to over assist leading to respiratory muscle dysfunction.**

Vt and Ti determine mean inspiratory flow

Peak flow - influence of respiratory drive and patient effort

If peak flow too low -> v. Uncomfortable (=**air hunger**)

If peak flow **too high** -> v. Short insufflation phase -> **"double triggering"** (neuro inspiratory time is longer than ventilators inspiratory time).

Ti -

Patient's Ti **varies** (**0.4 - 2.0 seconds)**, therefore difficult to match.
If Ti too short -> double trigger

Types of inappropriate triggering:

"Double triggering"
Neuro inspiratory time is longer than ventilators inspiratory time).

"Reverse trigger" -
A ventilator controlled triggered breath - due to increased lung volume (stretch receptors, etc) - can trigger a patient's inspiratory effort that would not have occurred otherwise.Cause eccentric diaphragmatic damage.

"reverse triggering" -> breath stacking -> increased Vt -> VILI

3) Pressure support ventilation

Set pressure determines minimum Vt
Unlike pressure control, **Ti is not fixed** and there is no backup rate. Therefore very different in control of breathing.

Inspiration ends when inspiratory flow - which peaks early - **decreases** to a **preset** level or a %

In **PSV**, minute **ventilation depends** on **assist** level, **mechanics** and **RR** - when ventilation -> a **drop in CO2** below normal -> only **inspiratory effort decreases** - **RR** does **not change**.

Rate of volume increase and inspiratory flow decline are **determined by the time constant**.

Long TC (ex. COPD)
a long TC -> long inhalation - therefore **inspiratory efforts may occur** ->
transient increase in flow above background flow decline.

For same reason, f**low rate declines slowly during expiration** -> **expiratory ineffective efforts (IEs)**. Therefore there are **several breath cycles during a single ventilator cycle.**

Solution to Its is to reduce pressure support so that respiratory **efforts** are **strønger** and **can overcome the PEEPi that results from a long TC**.
Change in pattern from slow, deep -> **rapid, shallow does not mean distress**, but **unmasks** the patient's **true RR** (NB. Not distress **unless** increase **use of accessory muscles** and/ or tachycardia, etc).

Short TC (ex. Stiff lung)

Patient receives Vt very quickly -> exhalation is also so fast, that there is no PEEPi, nor IE but pCO2 declines quickly -> central apnea -> persists until pCO2 rises above AT -> cycle continues.

PSV - marked **overventilation tends not** to develop.

Clinical consequences of asynchrony

60% of patients show **changes** (increases or decreases) of **diaphragmatic thickness** on U/S.

Reverse trigger - double cycling -> high delivered ventilation.
Diaphragmatic **contraction starts late** during insufflation -> **peak activity during exhalation** when **lung volume is decreasing and muscle lengthens = eccentric contraction**

Ineffective effort -> **fail to trigger** -> **most common form of assynchrony**.

The **patients RR is higher then the ventilator RR** -> therefore **most efforts during expiration (harmful** as inspiratory muscles activated while lung volume decreasing).

If PEEPi due to over assist -> ineffective efforts. Therefore **decrease assistance -> increased RR - this is nøt due to distress** but now each trigger is effective.

If **assistance is increased** -> decrease RR due to complex feedback reflexes.
Awake - RR will **not change if decrease in pCO2** but **could decrease respiratory drive** -> **ineffective efforts -> weaning failure**.

Insensitivity of RR to low CO2 resulting from a high assistance -> decrease effort -> **increase Ventilator Induced Diaphragmatic Dysfunction** (which is **2 X** more common the CIPPM).