COVID-19 pneumonia
Time course, monitoring and treatment

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The main findings

Covid-19 Does Not Lead to a “Typical” Acute Respiratory Distress Syndrome
L. Gattinoni et al, AJRCCM 2020
VIRUS

Epithelial – endothelial receptor

Inflammation

Interstitial edema

Where?
Inflammation + focused stress

Peripheral densities
Time course of VILI development
In 40 hours

Lung Inhomogeneities and Time Course of Ventilator induced Mechanical Injuries
Cressoni et al,
Anesthesiology 2015
Lung vasoplegia
(undetermined mechanism)

Nitric oxide → cGMP → Phosphorylated myosin → Vasodilation ?

Angiotensin I → ACE2 → Angiotensin II
Gravity dependent VA/Q mismatch
Hypoxemia

Undetermined factors

Increased respiratory drive

VE – TV increase
Negative pressure ventilation:

\[ \Delta P_L = 0 \text{ cmH}_2\text{O} - (-\Delta P_{pl}) \]

Positive pressure ventilation:

\[ \Delta P_L = \Delta P_{aw} - (\Delta P_{pl}) \]

- \(-\) to 0 for Venous return
- + to ++++ for Positive pressure ventilation
- ++ to ++++ for Negative/positive pressure ventilation

Edema, Inflammation, Microdamage for Positive pressure ventilation

Hypoperfusion for Negative pressure ventilation
Positive pressure respiration and its application to the treatment of acute pulmonary edema
L. Barach,
Annals of Internal Medicine, 1938

Acute Respiratory Failure Following Pharmacologically Induced Hyperventilation: An Experimental Animal Study
D. Mascheroni et al,
ICM, 1988

Mechanical Ventilation to Minimize Progression of Lung Injury in Acute Respiratory Failure
L. Brochard, A. Slutsky, A. Pesenti
AJRCCM 2017
When does the patient become dyspneic?

- **Expected TV (ml)**
- **Received TV (ml)**

- **Normal compliance**

- **Dyspnea**

- **Decreased compliance**
COVID-19 pneumonia: different respiratory treatment for different phenotypes?
L. Gattinoni et al, ICM 2020 (in press)
**Possible interventions**

- **Pre-infection**: Community medicine…
- **Virus infection**: Antiviral drugs…
- **Inflammatory reaction**: Corticoids, anti IL-6…

**Disease stage**
Disease stage

Vasoplegia

Hypoxemia

Respiratory drive

Possible interventions

NO, almitrine, CO₂ …

FiO₂, CPAP, NIV, Prone position...

Opioids, sedatives…
Negative pressure

Assess and control

Progression of the disease

What can we do?

Viral biology:
Micro/macro thrombosis,

Anticoagulation?
Negative intrathoracic pressure

• **How to assess**
  - Esophageal pressure
  - Surface electromyography
  - Clinical signs

• **How to control**
  - CPAP?
  - NIV?
  - Mechanical ventilation
Non invasive support

**Pro**
- Increase oxygenation
- May decrease the Ppl swings

**Con**
- Increase PaCO₂
- May not decrease effort
- Right ventricular failure
- Acute Kidney Injury
How I set the ventilator

- Mode
- FiO2
- Tidal Volume
- Respiratory rate
- PEEP

Remember that it is a long lasting disease
1. Which patient am I treating?

**Type L**

**Type H**
1. **Which patient am I treating?**

**Type L**
- Low elastance
- Low VA/Q
- Low lung weight
- Low recruitability

**Type H**
- High elastance
- High shunt
- High lung weight
- High recruitability
**Mechanical ventilation**

- **Mode:** Volume Controlled Ventilation
- **FiO₂:** high as needed
- **Tidal Volume:** even > than 6 ml/kg
- **Respiratory rate:** try < 20 bpm
- **PEEP:** 8-10 cmH₂O
- **Prone position:** only as a rescue

**Type L**

- **Sedation and muscle relax**
Mechanical ventilation  
Type L

Sedation and muscle relax

In these high compliance patients (> 50 ml/cmH₂O) the plateau, driving pressure and mechanical power levels are well below the “classical” severe ARDS
Mechanical ventilation

• **Mode:** Volume Controlled Ventilation

• **FiO₂:** high as needed

• **Tidal Volume:** possibly 6 ml/kg

• **Respiratory rate:** to stay < 60 mmHg PaCO₂

• **PEEP:** possibly < 15 cmH₂O

• **Prone position:** daily

**Type H**

Sedation and muscle relax

**Why?**
In these low compliance patients (< 50 ml/cmH$_2$O) the plateau, driving pressure and mechanical power levels are the same of the “classical” severe ARDS
Weaning

COVID-19 pneumonia lasts long,
early weaning is problematic
Remember

1. More than 50% do not have a “classical” ARDS

2. It’s a long course disease

3. 10 cmH₂O of PEEP, sedation and especially patience is likely the best we can offer