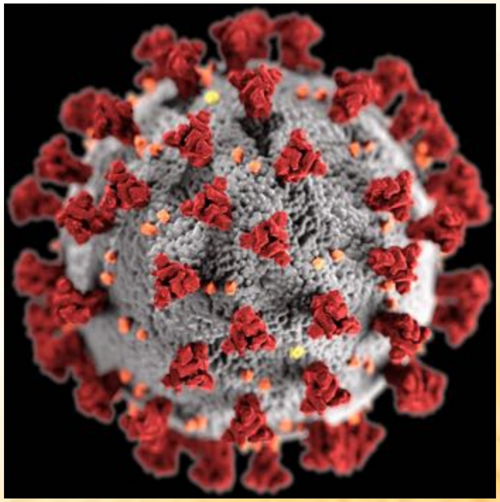
LEHIGH VALLEY HEALTH NETWORK

CHALLENGES AND STRATEGIES FOR VENTILATING PATIENTS WITH COVID-19



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Lehigh Valley Health Network
Allentown, Penna

Learning Objectives

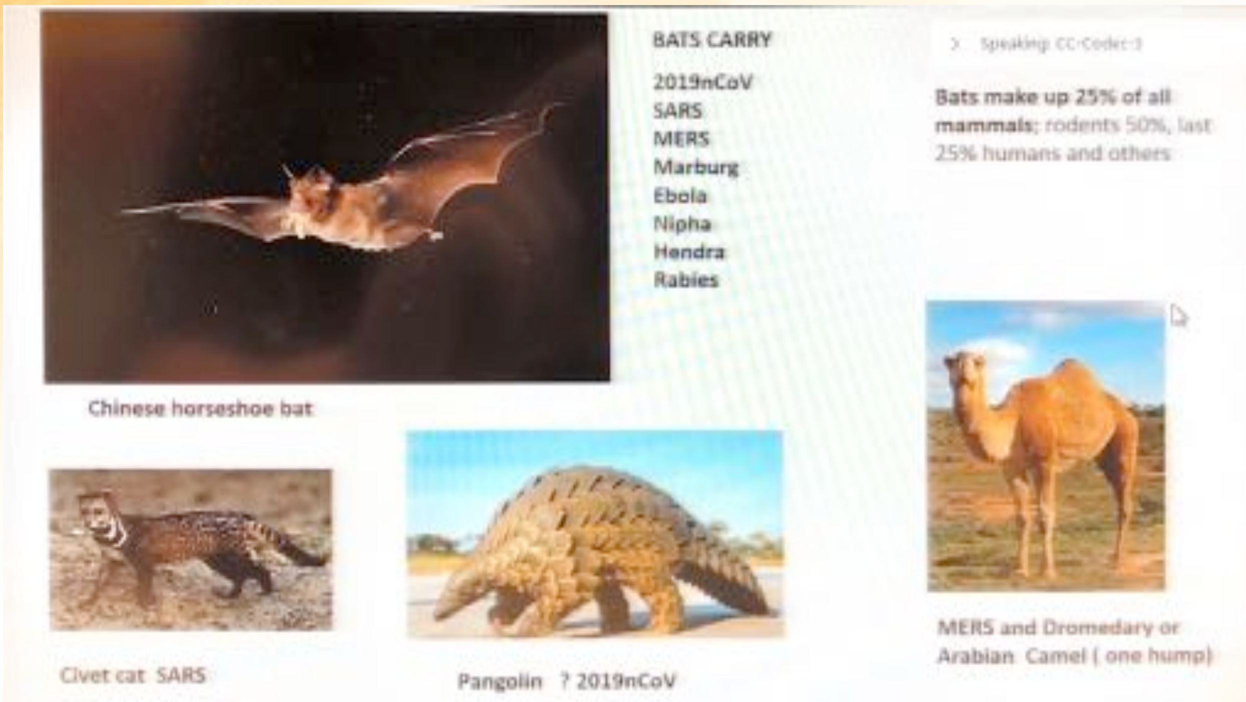
- Review the latest information on how the COVID virus affects the lungs
- Discuss known problems related to ventilating patients diagnosed with COVID-19
- Describe therapeutic interventions and ventilator strategies to improve outcomes of the COVID-19 patient population

CORONA VIRUS
SARS-COV-2
(COVID-19)

Moment of Silence

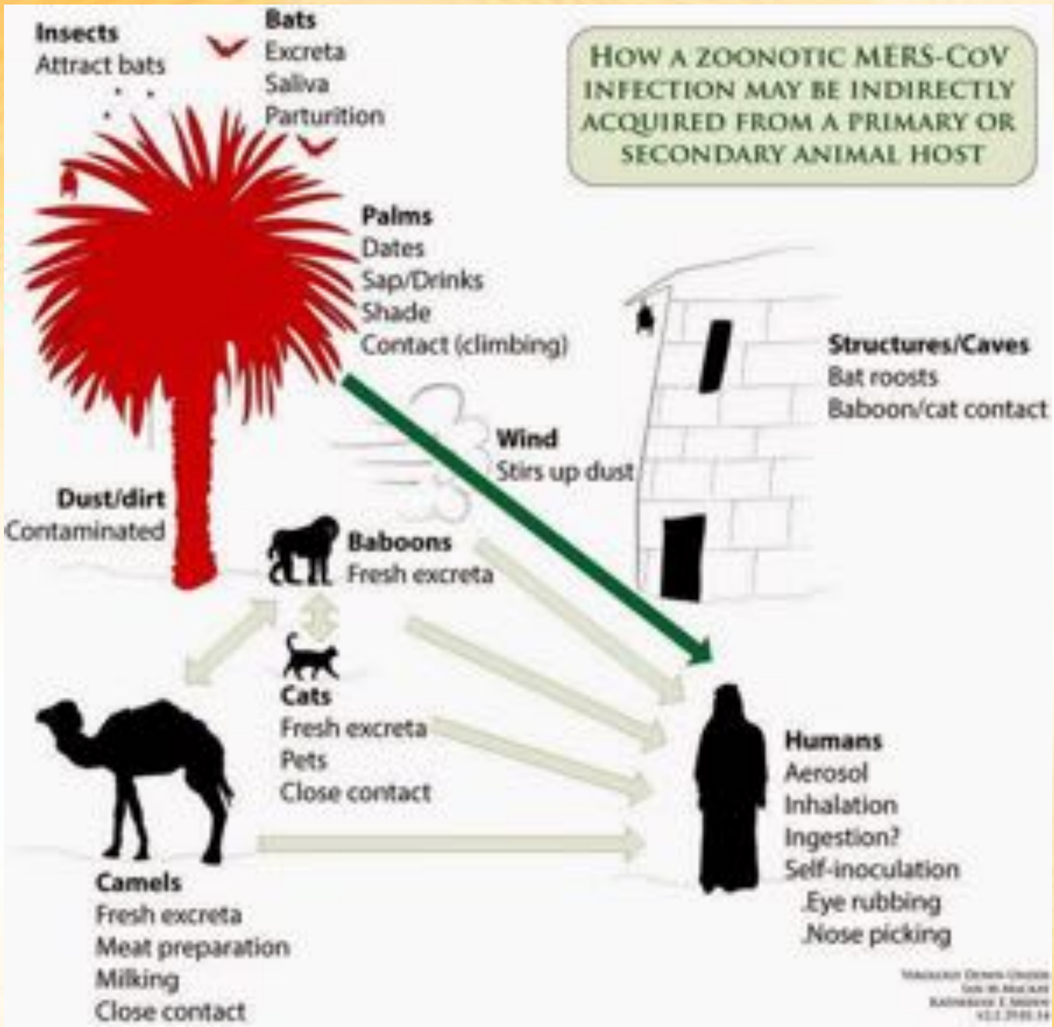
As of today, the AARC is aware of 13 respiratory therapists who have died of COVID-19 in the line of service. To honor their sacrifice, the Executive Committee of the AARC Board of Directors has authorized monies to be made available to the surviving families of licensed respiratory therapists who died while caring for patients with COVID-19.

How Did COVID-19 Happen?

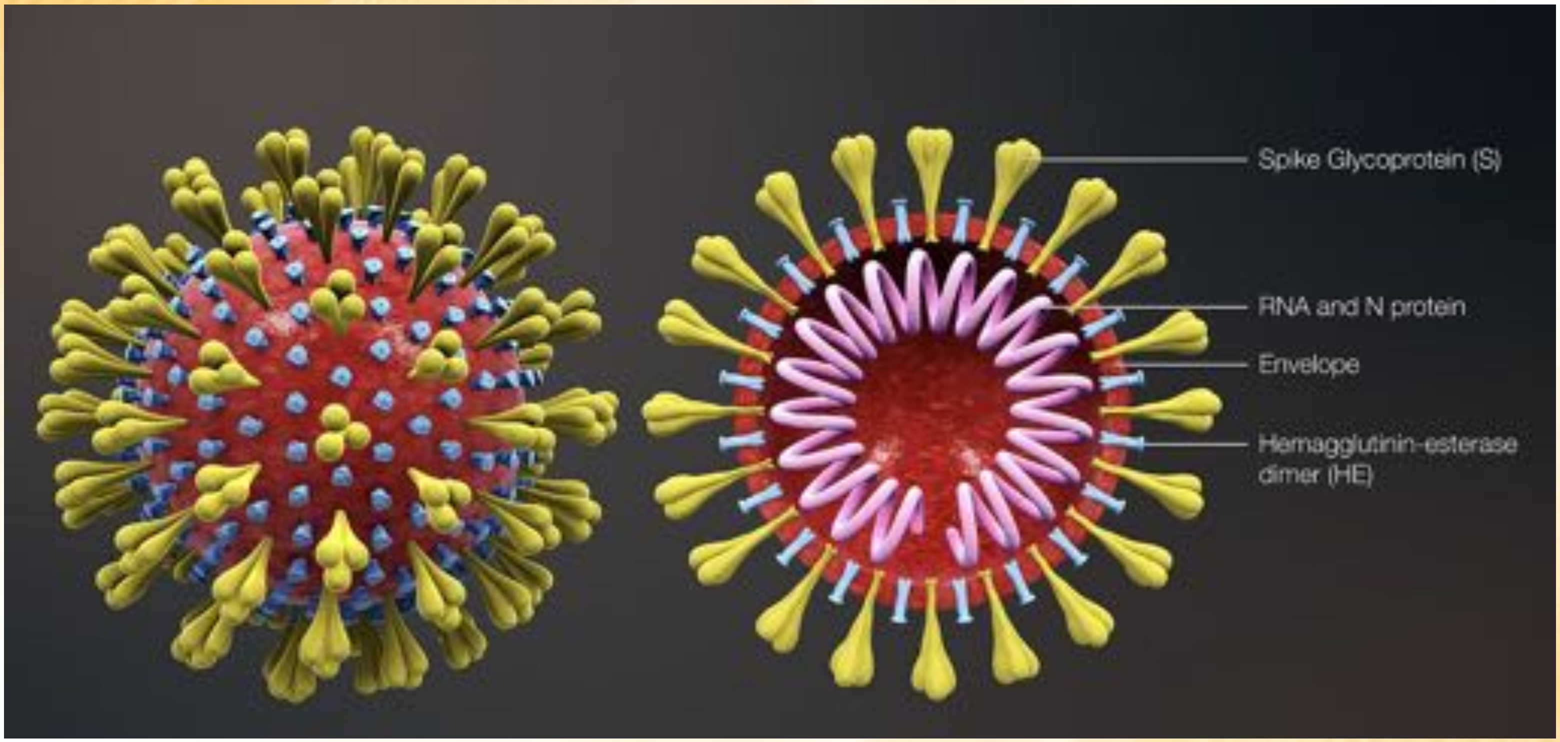


Chinese Wet Market?

Animal to Human Transmission

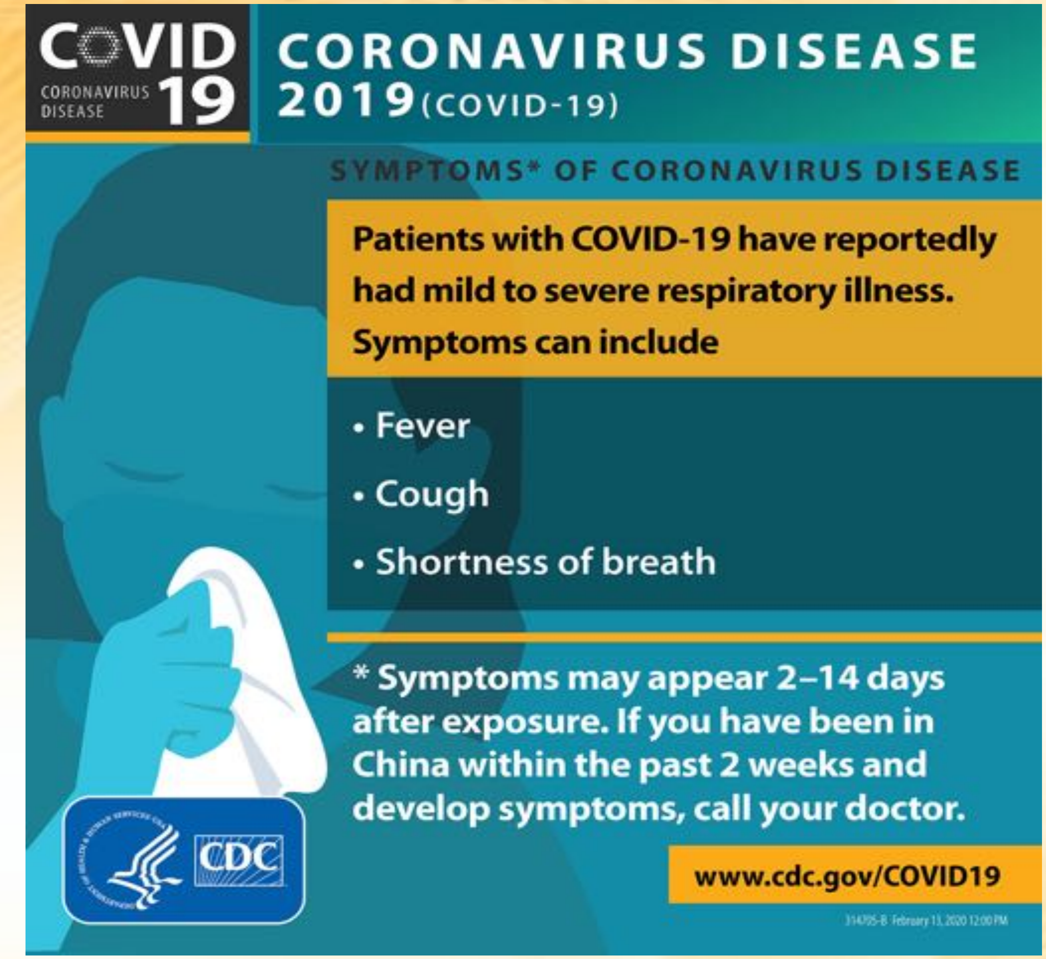


COVID-19 Virus Anatomy



Clinical Presentation of COVID-19

- Fever
- Dyspnea
- Dry spasmodic cough
- Secondary symptoms
    - Headache
    - Diarrhea
    - Blueish toe/fingers
    - Confusion
    - Stroke


COVID 19
CORONAVIRUS DISEASE
CORONAVIRUS DISEASE 2019 (COVID-19)
SYMPTOMS* OF CORONAVIRUS DISEASE
Patients with COVID-19 have reportedly had mild to severe respiratory illness. Symptoms can include
• Fever
• Cough
• Shortness of breath
* Symptoms may appear 2–14 days after exposure. If you have been in China within the past 2 weeks and develop symptoms, call your doctor.
CDC
www.cdc.gov/COVID19

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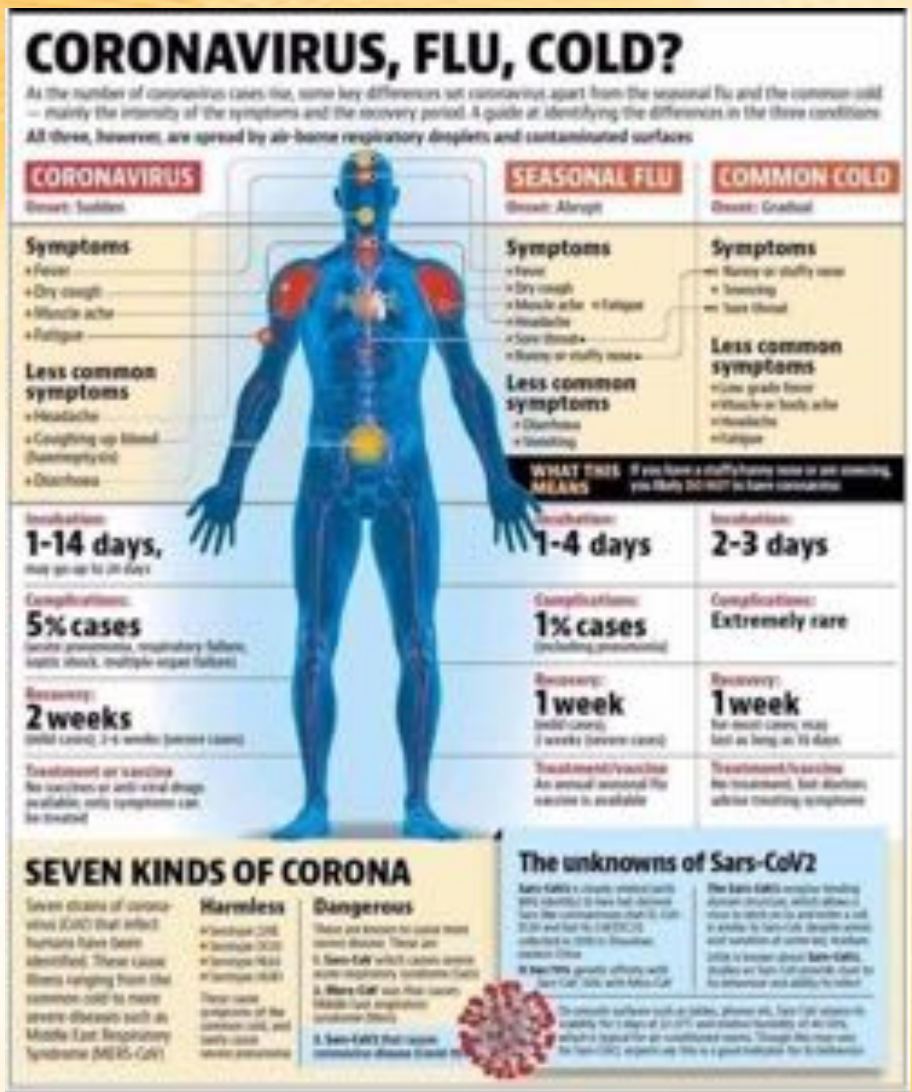
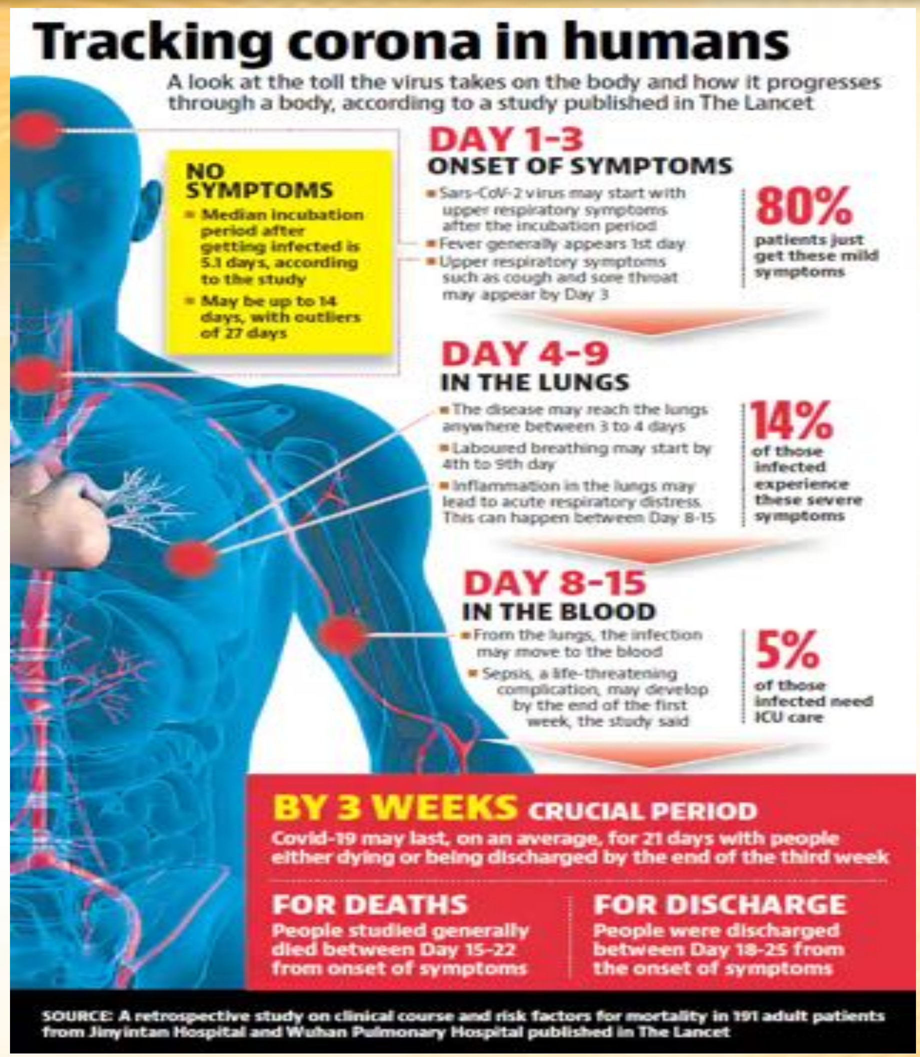
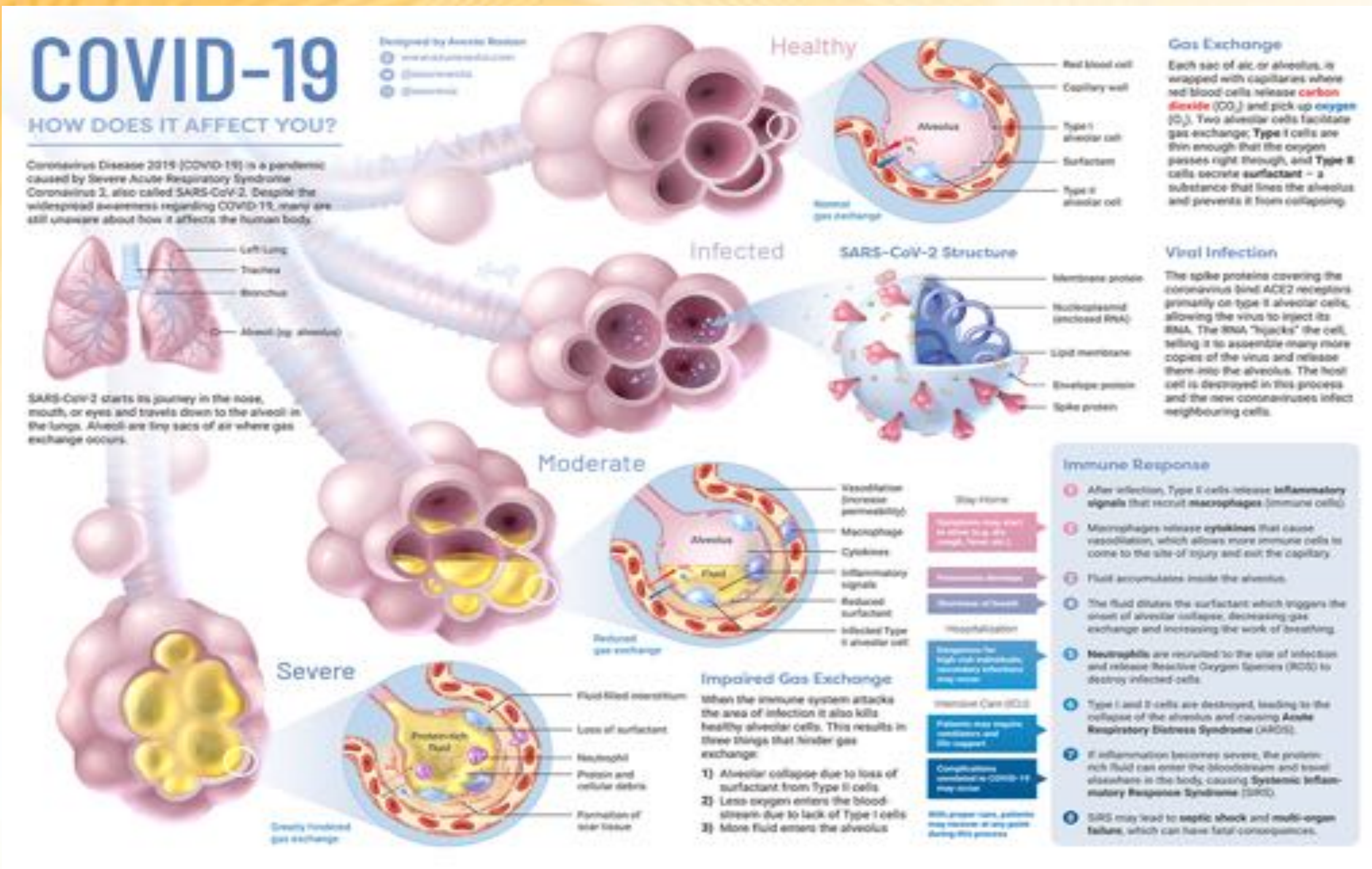
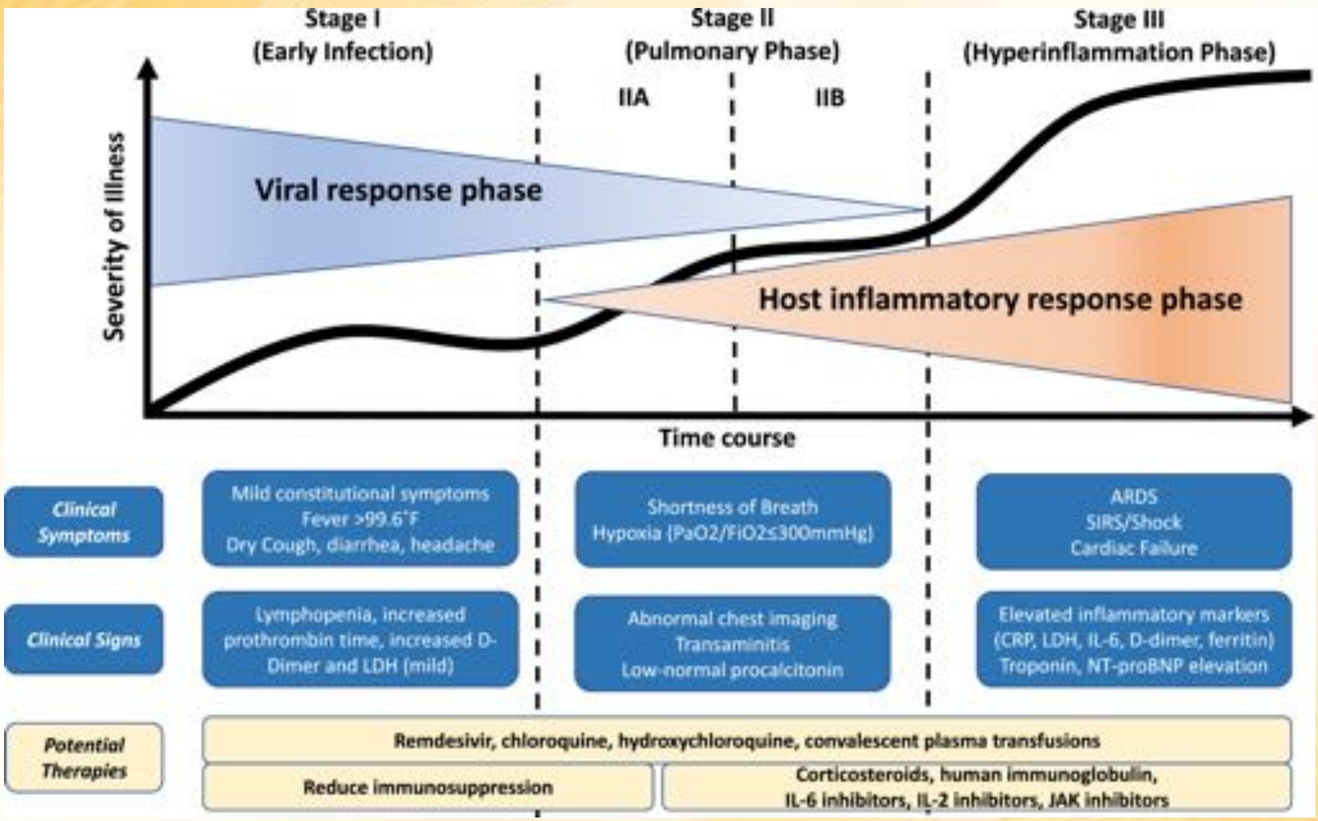



Tracking corona in humans
A look at the toll the virus takes on the body and how it progresses through a body, according to a study published in The Lancet
NO SYMPTOMS
Median incubation period after getting infected is 5.1 days, according to the study
May be up to 14 days, with outliers of 27 days
DAY 1-3
ONSET OF SYMPTOMS
Sars-CoV-2 virus may start with upper respiratory symptoms after the incubation period
Fever generally appears 1st day
Upper respiratory symptoms such as cough and sore throat may appear by Day 3
80%
patients just get these mild symptoms
DAY 4-9
IN THE LUNGS
The disease may reach the lungs anywhere between 3 to 4 days
Laboured breathing may start by 4th to 9th day
Inflammation in the lungs may lead to acute respiratory distress. This can happen between Day 8-15
14%
of those infected experience these severe symptoms
DAY 8-15
IN THE BLOOD
From the lungs, the infection may move to the blood
Sepsis, a life-threatening complication, may develop by the end of the first week, the study said
5%
of those infected need ICU care
BY 3 WEEKS CRUCIAL PERIOD
Covid-19 may last, on an average, for 21 days with people either dying or being discharged by the end of the third week
FOR DEATHS
People studied generally died between Day 15-22 from onset of symptoms
FOR DISCHARGE
People were discharged between Day 18-25 from the onset of symptoms
SOURCE: A retrospective study on clinical course and risk factors for mortality in 191 adult patients from Jinyintan Hospital and Wuhan Pulmonary Hospital published in The Lancet


COVID-19
HOW DOES IT AFFECT YOU?
Coronavirus Disease 2019 (COVID-19) is a pandemic caused by Severe Acute Respiratory Syndrome Coronavirus 2, also called SARS-CoV-2. Despite the widespread awareness regarding COVID-19, many are still unaware about how it affects the human body.
Left Lung
Trachea
Bronchus
SARS-CoV-2 starts its journey in the nose, mouth, or eyes and travels down to the alveoli in the lungs. Alveoli are tiny sacs of air where gas exchange occurs.
Healthy
Red blood cell
Capillary wall
Alveolus
Surfactant
Infected
SARS-CoV-2 Structure
Membrane protein
Lipid membrane
Envelope protein
Spike protein
Moderate
Alveolus
Fluid
Macrophage
Cytokines
Inflammatory signals
Reduced surfactant
Severe
Loss of surfactant
Neutrophil
Protein and cellular debris
Formation of scar tissue
Gas Exchange
Each sac of air, or alveolus, is wrapped with capillaries where red blood cells release carbon dioxide (CO_2) and pick up oxygen (O_2). Two alveolar cells facilitate gas exchange; Type I cells are thin enough that the oxygen passes right through, and Type II cells secrete surfactant – a substance that lines the alveolus and prevents it from collapsing.
Viral Infection
The spike proteins covering the coronavirus bind ACE2 receptors primarily on type II alveolar cells, allowing the virus to inject its RNA. The RNA "hijacks" the cell, telling it to assemble many more copies of the virus and release them into the alveolus. The host cell is destroyed in this process and the new coronaviruses infect neighbouring cells.
Immune Response
After infection, Type II cells release inflammatory signals that recruit macrophages (immune cells).
Macrophages release cytokines that cause vasodilation, which allows more immune cells to come to the site of injury and exit the capillary.
Fluid accumulates inside the alveolus.
The fluid dilutes the surfactant which triggers the onset of alveolar collapse, decreasing gas exchange and increasing the work of breathing.
Neutrophils are recruited to the site of infection and release Reactive Oxygen Species (ROS) to destroy infected cells.
Type I and II cells are destroyed, leading to the collapse of the alveolus and causing Acute Respiratory Distress Syndrome (ARDS).
If inflammation becomes severe, the protein-rich fluid can enter the bloodstream and travel elsewhere in the body, causing Systemic Inflammatory Response Syndrome (SIRS).
SIRS may lead to septic shock and multi-organ failure, which can have fatal consequences.
Impaired Gas Exchange
When the immune system attacks the area of infection it also kills healthy alveolar cells. This results in three things that hinder gas exchange:
1) Alveolar collapse due to loss of surfactant from Type II cells
2) Less oxygen enters the bloodstream due to lack of Type I cells
3) More fluid enters the alveolus

Immune Response: Cytokine Storm



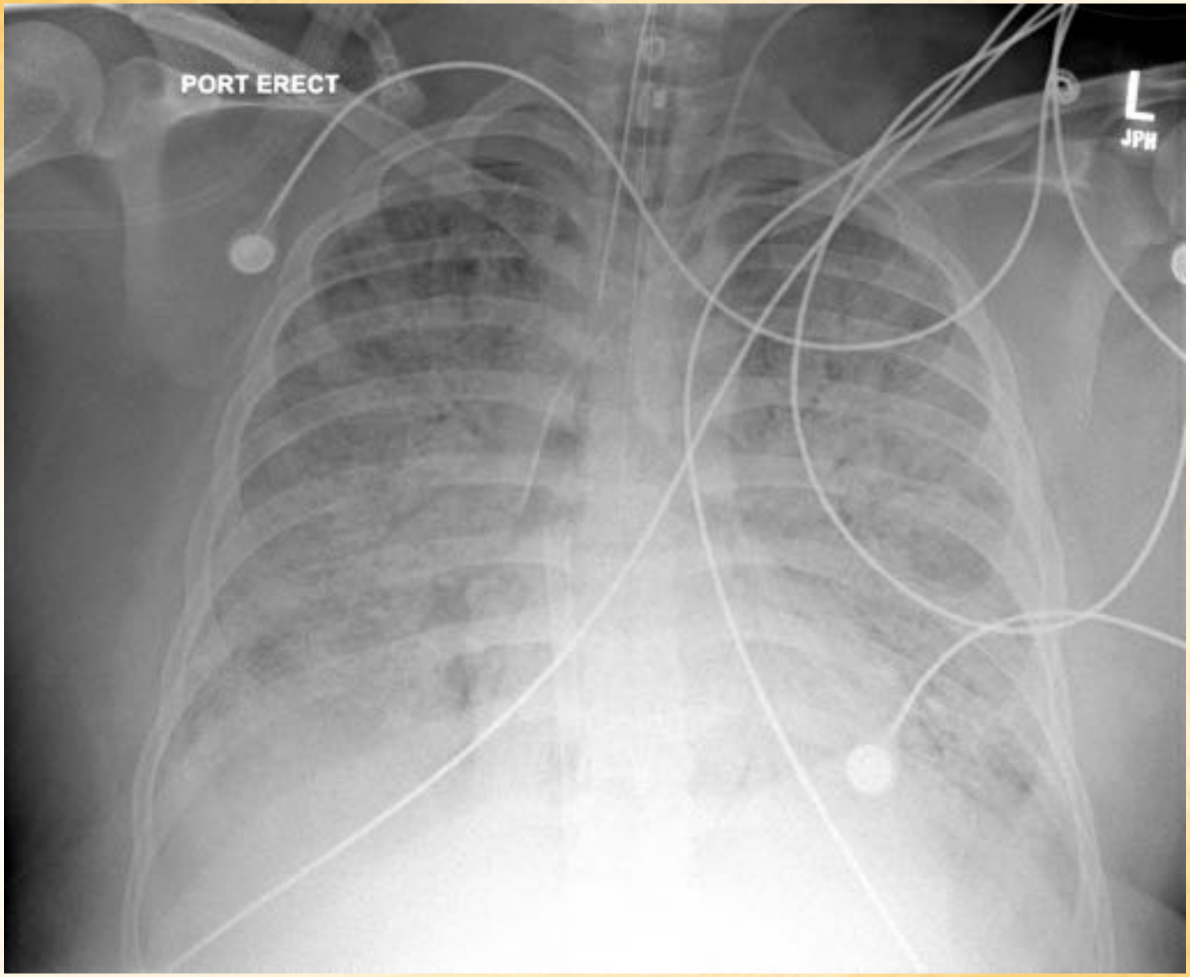
X-ray Findings

COVD-19
Patient
3-5 days after
developing
respiratory
symptoms

Profound ARDS

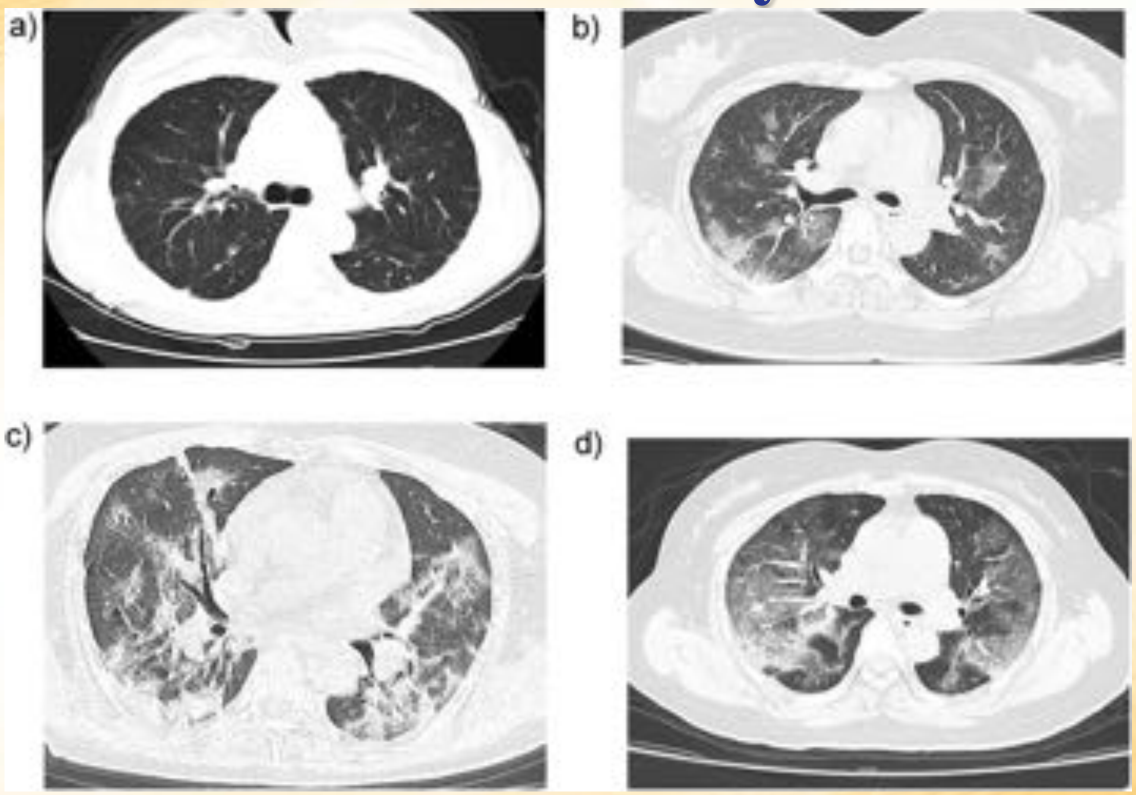
Barotrauma

End-stage
COVID



COVID-19 Patient
Cat-scan

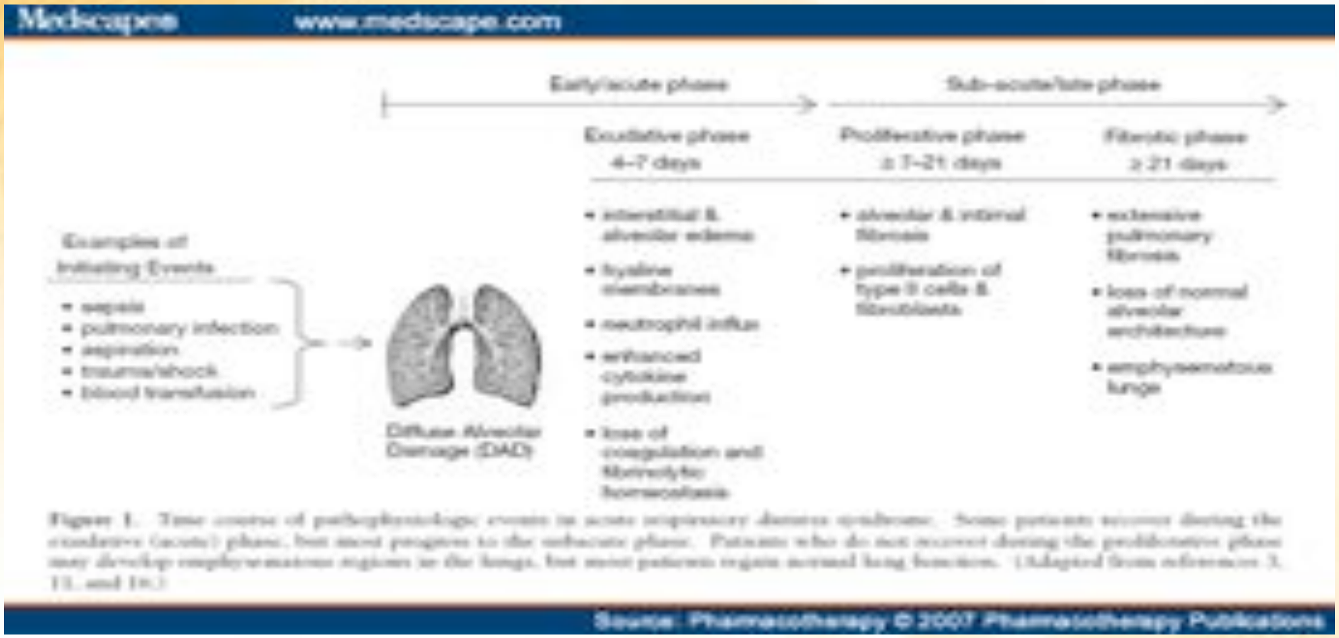
Progression of Disease
3-14 days



Berlin P/F Ratio Criteria

- **Mild**
  - P/F <300>200
  - Mortality 24%
- **Moderate**
  - P/F <200>100
  - Mortality 34%
- **Severe**
  - P/F <100
  - Mortality 44%
  - COVID-19 >50% China/Italy

Pathophysiology



http://www.medscape.com/viewarticle/558310_3

Pathophysiology

- The injured lung goes through 3 phases:

  Edema
  - Reparative

  Fibrotic

Edema Phase

- Occurs in the first week after onset of respiratory failure
- Inflammatory cells migrate into the lungs and release substances to cause **capillary leakage**
- Type I pneumocytes swell and detach from basement membrane
- Increased pulmonary vascular permeability
- Alveolar collapse

Homogeneous and Heterogeneous Alveolar Ventilation

Schiller et al. *Crit Care Med*. 2001;29:1049

Normal Lung

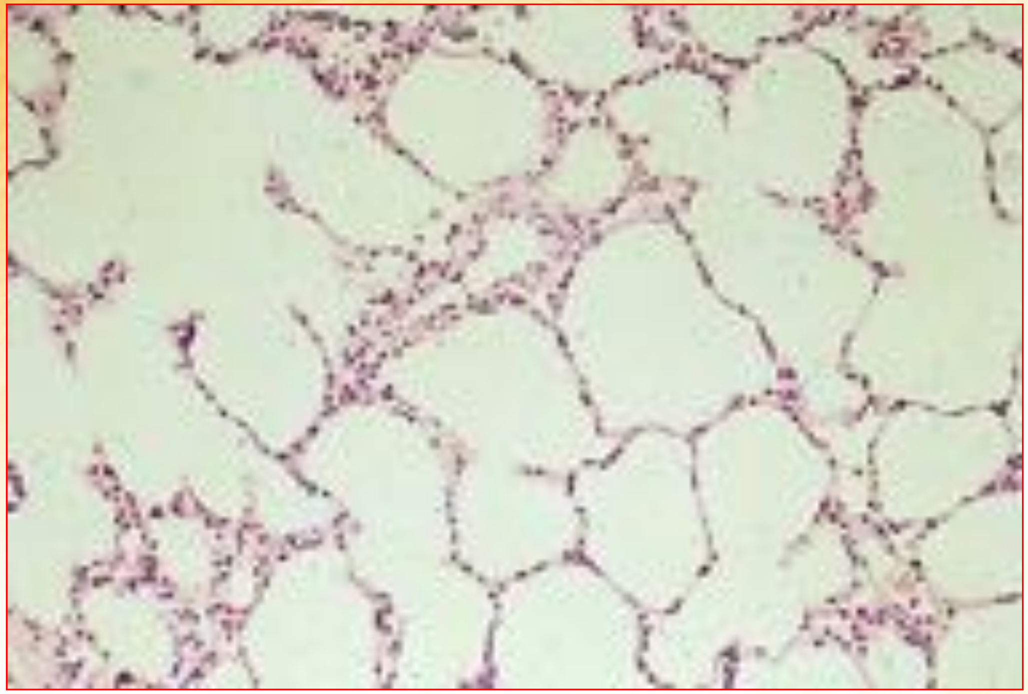
ARDS Lung

Uninjured Alveoli

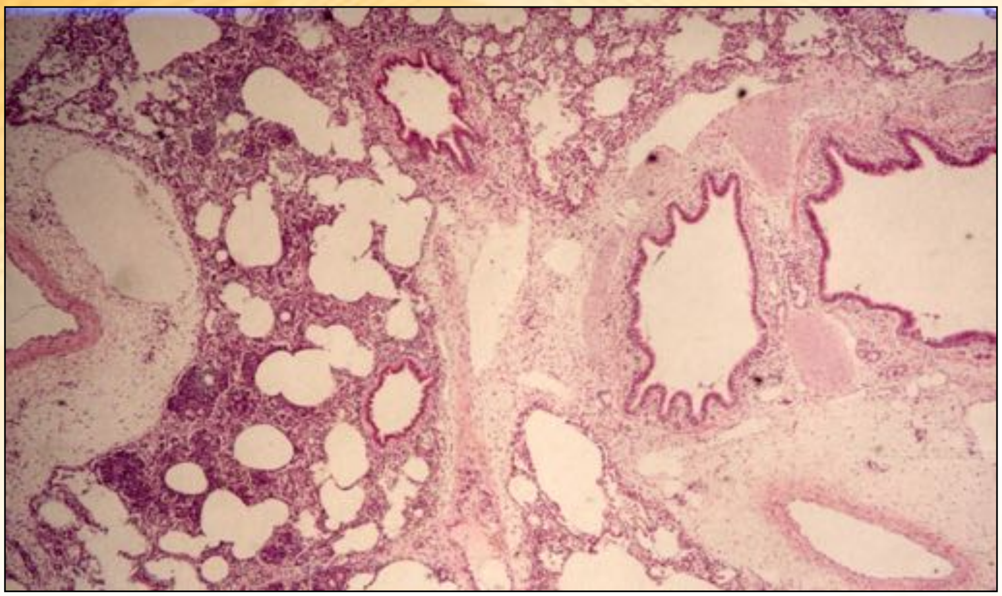
Permission granted from Gary Neiman

Injured Alveoli

Permission granted from Gary Neiman



Normal Lung



Consolidated Lung

Reparative Phase

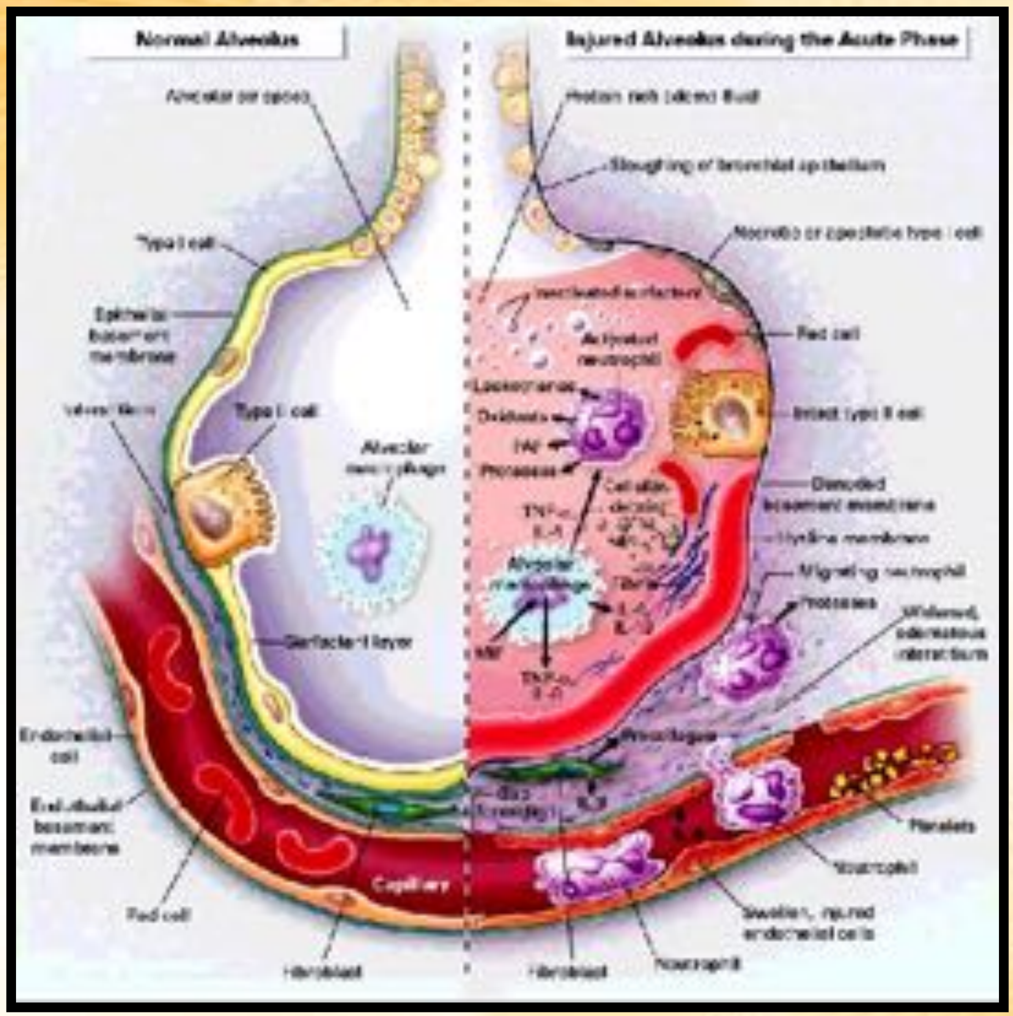
- May begin as early as 3rd day, but usually prominent in 2nd and 3rd week after onset
- Type II cells proliferate and reline membrane
- Fibroblast infiltration – migration through breaks in membrane forming granulation tissue
- Surfactant abnormalities occur – damage to Type II and alveolar flooding destabilize the surfactant layer- **Marked by poor gas diffusion**

Fibrotic Phase

- May begin as early as two weeks after injury
- Extensive remodeling by collagenous tissue
- Alveolar duct fibrosis
- Elastic collagen replaced by rigid collagen – resulting in stiff lung
- Extent of fibrosis correlates with mortality
- **VD/VT>60% Large amount of wasted ventilation good predictor of mortality**

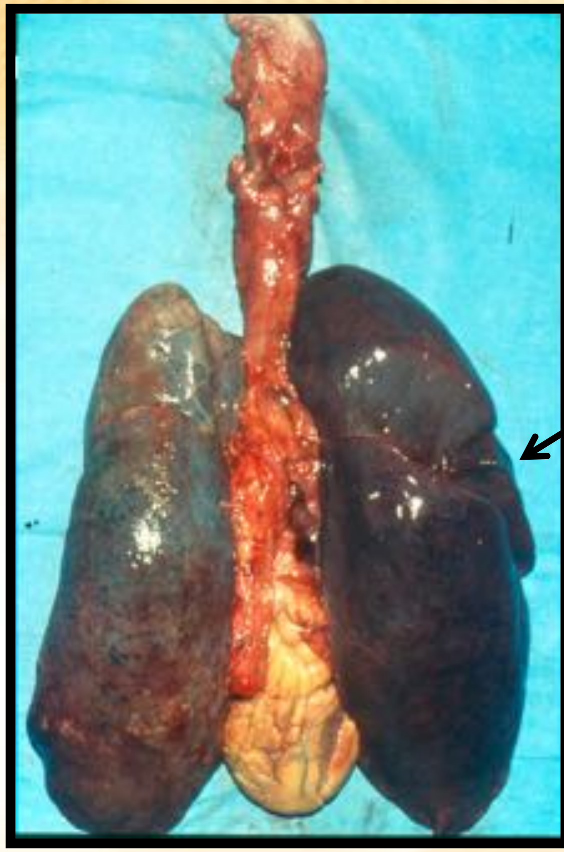
Structural Changes

- Damage to type I alveolar epithelial cells
- Increase edema influx
- Loss of surfactant
- Poor fluid clearance mechanism
- Development of a hyaline membrane
- Reduction in gas exchange
- Pulmonary Fibrosis development
- “Liver” lung appearance


Normal Alveolus
Injured Alveolus during the Acute Phase
Type I cell
Red cell
Denuded basement membrane
Hyaline membrane
Migrating neutrophil
Proteases
Platelets
Neutrophil
Capillary
Red cell
Fibroblast
Fibroblast
Neutrophil

Post-mortem Findings

- Dense infiltration with leukocytes and proteinaceous material
- Wet, heavy, congested lungs with collapsed alveoli
- Pneumonia revealed in up to 75 % of cases



Liver like appearance

Dependent hemorrhagic injury

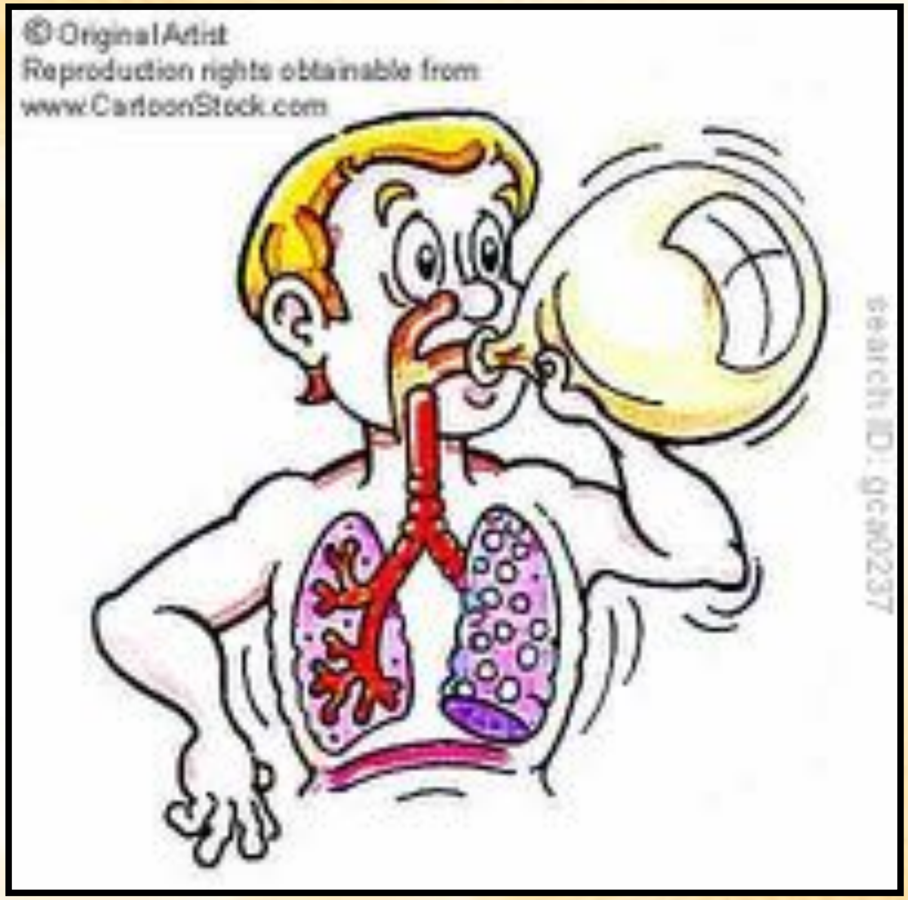
Wet Heavy Lung

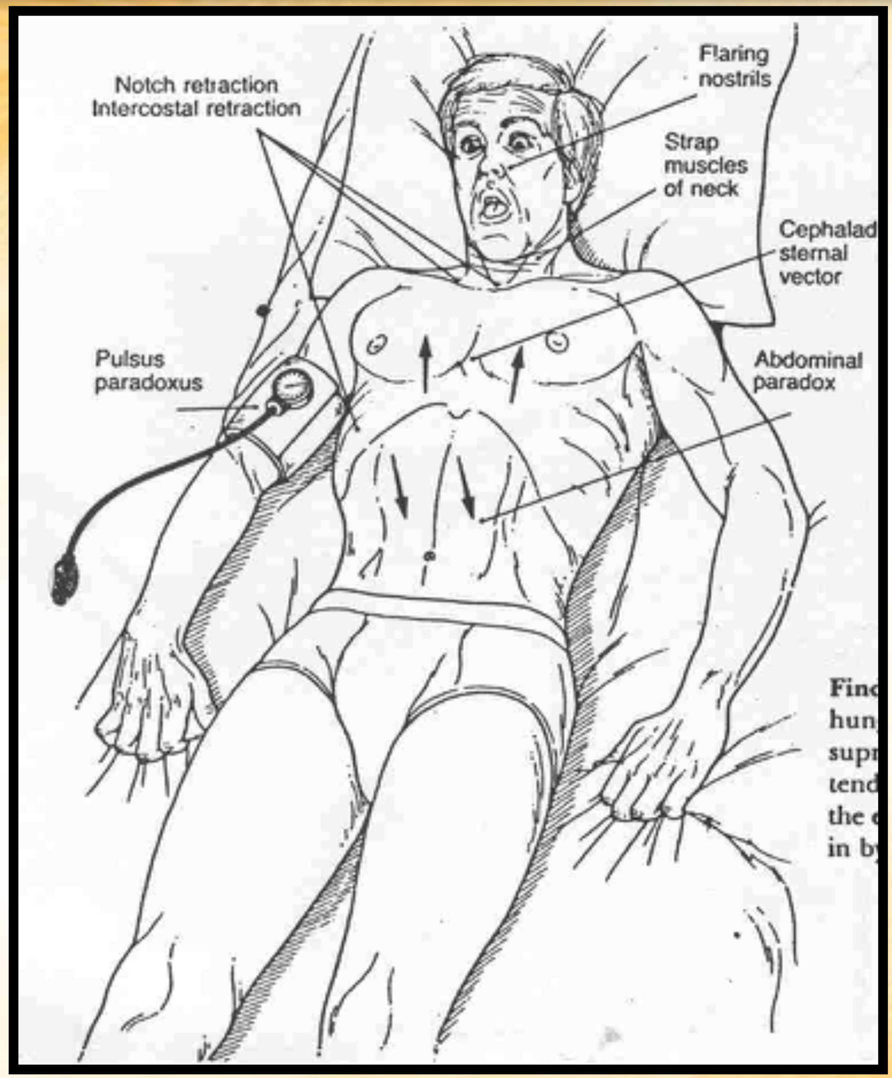
Pneumonia Induced ARDS

Clinical Manifestations

- Dyspnea
- Tachypnea (rapid, shallow breathing)
- Severe Hypoxemia – refractory to O2 therapy
- Intercostal and suprasternal retraction on inspiration
- CXR reveals diffuse bilateral infiltrates (not always a reliable study) **CAT SCAN Better!!**

WOB
Associated with
ARDS




Notch retraction
Intercostal retraction
Flaring nostrils
Strap muscles of neck
Cephalad sternal vector
Pulsus paradoxus
Abdominal paradox

COVID Two Different ARDS?

Luciano Gattinoni
Department of Anesthesiology and Intensive Care,
Medical University of Göttingen

COVID-19 pneumonia, Type L ("Happy hypoxemia")

At the beginning, COVID-19 pneumonia presents with the following characteristics:

- **Low elastance**: the nearly normal compliance indicates that the amount of gas in the lung is nearly normal CLT>30cm
- Low ventilation to perfusion (VA/Q) ratio: since the gas volume is nearly normal, hypoxemia may be best explained by the loss of regulation of perfusion and by loss of hypoxic vasoconstriction. Accordingly, at this stage, the pulmonary artery pressure, should be near normal.
- Low lung weight: Only ground-glass densities are present on CT scan, primarily located subpleurally and along the lung fissures. Consequently, lung weight is only moderately increased.
- Low lung recruitability: the amount of non-aerated tissue is very low, consequently the recruitability is low.

Acts like a diffusion deficient-poor DLCO

Treatment of Phenotype L

- Higher threshold for intubation
- Early prone
- High FIO2 administered >70%
- Lower PEEP

COVID-19 pneumonia, Type H

The Type H patient

• High elastance: The decrease of gas volume due to increased edema accounts for the increased lung elastance. CLT<30cm

• High right-to-left shunt: This is due to the fraction of cardiac output perfusing the non-aerated tissue which develops in the dependent lung regions due to the increased edema and superimposed pressure.

• High lung weight: Quantitative analysis of the CT scan shows a remarkable increase in lung weight (> 1.5 kg), on the order of magnitude of severe ARDS • High lung recruitability: The increased amount of non-aerated tissue is associated, as in severe ARDS, with increased recruitability .

The Type H pattern, 20 – 30% of patients in our series, **fully fits the severe ARDS criteria: hypoxemia P/F<100 torr**

Treatment of Phenotype H

- Early intubation
- High PEEP
- Paralytic
- Prone inhaled pulmonary Vasodilators
- ?ECMO

The research, which was published online in the *American Journal of Respiratory and Critical Care Medicine*, studies the electronic health records of 85 COVID-19 patients who died between Jan 9 and Feb 15, 2020 after treatment at two Wuhan hospitals.

Some of the clinical characteristics of the patient fatalities included:

•65.8 median age

•72.9 % were men

•Most common symptoms: fever, dyspnea, and fatigue

•Most common comorbidities: hypertension, diabetes, and coronary heart disease

•80%+ of patients had very low counts of eosinophils on admission

•Complications included: respiratory failure, shock, ARDS and cardiac arrhythmia

•Most patients received antibiotics, antivirals and glucocorticoids

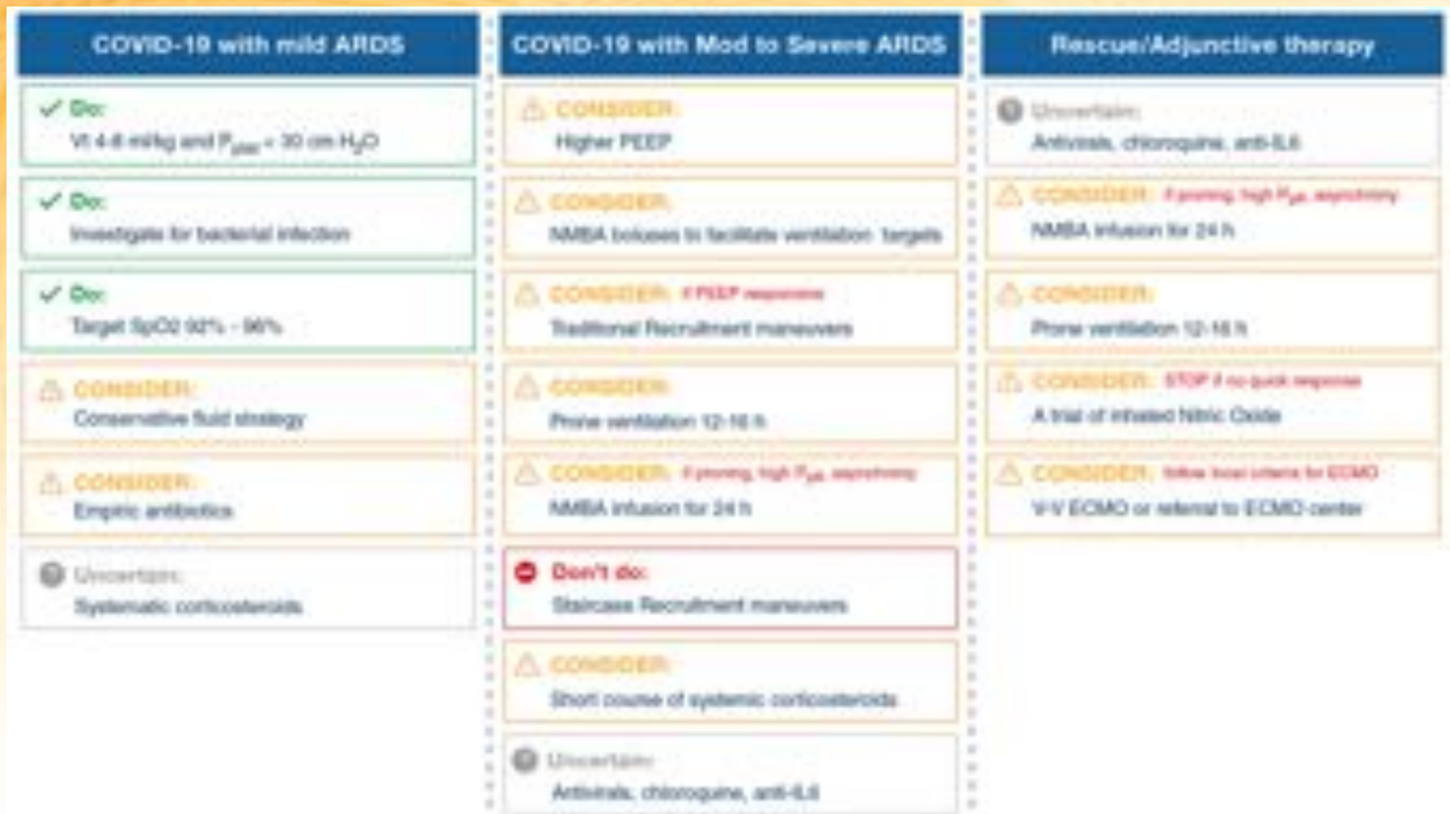
•Some were given intravenous immunoglobulin or interferon alpha-2b

•The majority of patients studied died from multiple organ failure

COVID-19 Fatalities Shared These Characteristics

Therapy	Implementation
High-flow nasal oxygen	Might prevent or delay the need for intubation
Tidal volume	Use 6 mL/kg per predicted bodyweight (can reduce to 4 mL/kg per predicted bodyweight)
Plateau airway pressure	Maintain at <30 cm H_2O if possible
Positive end-expiratory pressure	Consider moderate to high levels if needed
Recruitment manoeuvres	Little value
Neuromuscular blockade	For ventilator dyssynchrony, increased airway pressure, hypoxaemia
Prone positioning	For worsening hypoxaemia, PaO_2:FiO_2 <100–150 mm Hg
Inhaled NO	Use 5–20 ppm
Fluid management	Aim for negative fluid balance of 0·5–1·0 L per day
Renal replacement therapy	For oliguric renal failure, acid-base management, negative fluid balance
Antibiotics	For secondary bacterial infections
Glucocorticoids	Not recommended
Extracorporeal membrane oxygenation	Use EOLIA trial criteria[3]

Clinical Management



Recommendations from the ACCP, SCCM, AARC

Clinical management @ LVHN: The 4 Ps

- Paralytic
- Pulmonary vasodilators
- PEEP
- Prone

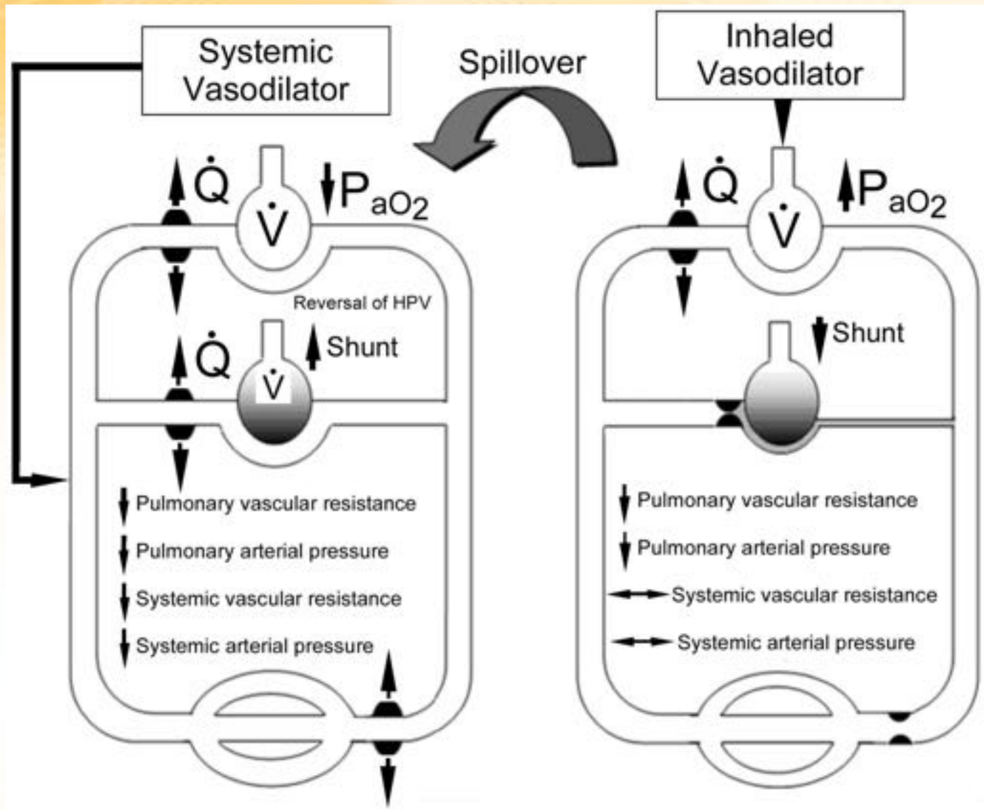
Paralytic Administration

- To minimize ventilator asynchrony
- To reduce oxygen consumption from fever severing
- Promote higher levels of PEEP and prone positioning


HYDROmorphone (DILAUDID) in NS infusion 50 mg/100 mL
midazolam (VERSED) in NS infusion 100 mG/100 mL (premix)
rocuronium (ZEMURON) 500 mg in NS 500 mL infusion
Modify
Discontinue

Inhaled Pulmonary Vasodilator

- Administered Veletri 20mcg/8ml or inhaled NO 20-40ppm
- Improve perfusion to patent alveoli
  - Improves oxygenation
- Reduces pulmonary resistance
  - Reduces pulmonary artery pressure


Systemic Vasodilator
Spillover
Inhaled Vasodilator
$\dot{Q}$
$\dot{V}$
P_{aO_2}
Reversal of HPV
Shunt
Pulmonary vascular resistance
Pulmonary arterial pressure
Systemic vascular resistance
Systemic arterial pressure

Desired Clinical Endpoints

-Increase SpO2 or P/F ratio by 20%.
-Reduction in Pulmonary Artery Pressure by 15%.
-Improvement of hemodynamics post intra-operative pump.

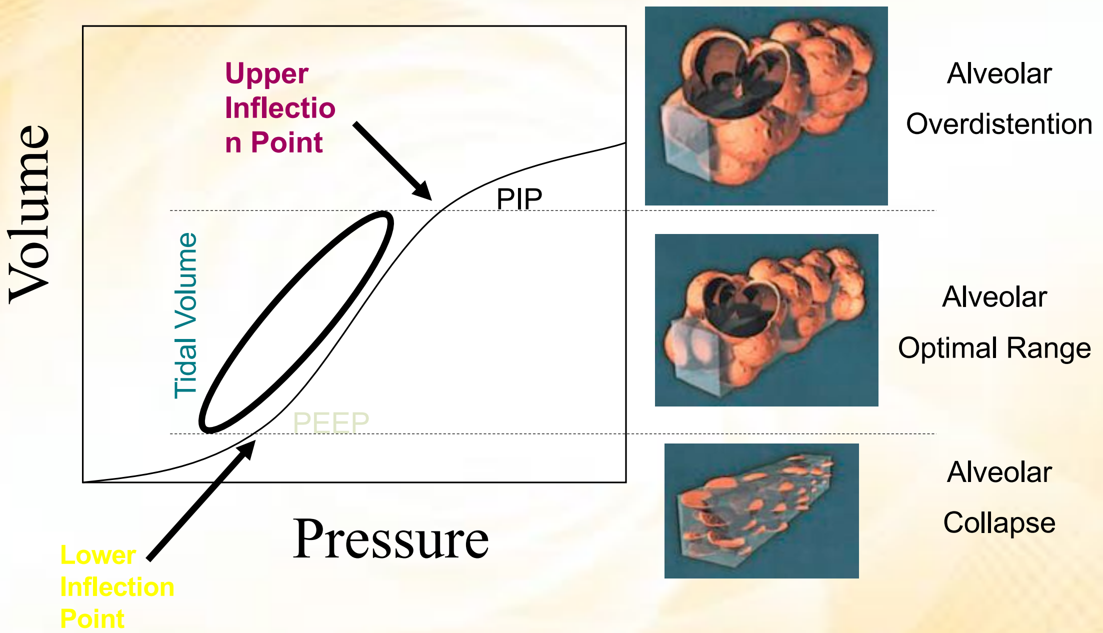
Optimizing PEEP

- Mean PEEP requirement was 14cm (8cm-28cm)
- Utilize P/V tool and/or transpulmonary monitoring to determine lower inflection point and transpE pressure

Setting PEEP Correctly

- Pressure/volume tool
- Decremental PEEP trial
  - Start at 30cm PEEP and reduce by 2cm until best compliance
- Incremental PEEP trial
  - Start at 5cm PEEP and increase by 2cm until best compliance
- Transpulmonary monitoring
- PEEP/FIO2 table
- Stress Index
  - Observe pressure/time curve
- Electrical Impedance Tomography
  - Chest wall imagery

PV Curve to determine PEEP



Improved Clt

Transpulmonary Monitoring: Clinical Rationale

- The measurement of esophageal pressure, used as a surrogate for pleural pressure, allows calculation of the pressure required to distend the lung and the chest wall. The distending force applied to the lung, called the transpulmonary pressure, is the pressure difference between the alveoli and the esophagus.
- This helps the clinical team to determine has much ventilator pressure (peak airway pressure) is actually going into the lung.
- Also helps to determine where to set the PEEP level.

Transpulmonary Monitoring

PIP=RAW/total CLT	41cm
PLT=Thoracic/pulmonary CLT	32cm
Transpl=pulmonary CLT	19cm

Transpl=what pressure the lung is receiving

Post esophageal balloon placement.

Peak airway pressure minus esophageal pressure equals: Transpulmonary pressure (pressure the lung is receiving) Goal<25cm

Note peak airway pressure is 42cm Esophageal pressure 21cm thus transpulmonary pressure (the lung receiving) only 19cm

PEEP set at 14cm
Based on PtransE
Between -2 to +2cm

Prone Positioning

- Improves Ventilation/perfusion matching in ARDS
- Helps reduce chest wall and abdominal impedance
- Facilitates secretion removal
- Prone 66% of all ventilated patients for>16 hrs. >3 days

Prone Positioning

- Improved oxygenation when turned to the prone position
- Prone positioning may promote alveolar recruitment in some patients and improves oxygenation in many patients
- 2001 study reported no survival benefit from the use of prone positioning in ARDS patients*
  - *Gattinoni N Engl J Med 2001;345(8):568-573
    - **2009 Meta-analysis reviews have demonstrated improved survival in those patients who's P/F ratio was <100 torr**

The NEW ENGLAND JOURNAL of MEDICINE

JUNE 6, 2013

Prone Positioning in Severe Acute Respiratory Distress Syndrome

Claude Guérin, M.D., Ph.D., Jean Reignier, M.D., Ph.D., Jean-Christophe Richard, M.D., Ph.D., Pascal Beuret, M.D., Arnaud Gacouin, M.D., Thierry Boulain, M.D., Emmanuelle Mercier, M.D., Michel Badet, M.D., Alain Mercat, M.D., Ph.D., Olivier Baudin, M.D., Marc Clavel, M.D., Delphine Chatellier, M.D., Samir Jaber, M.D., Ph.D., Sylvène Rosselli, M.D., Jordi Mancebo, M.D., Ph.D., Michel Sirodot, M.D., Gilles Hilbert, M.D., Ph.D., Christian Bengler, M.D., Jack Richecoeur, M.D., Marc Gainnier, M.D., Ph.D., Frédérique Bayle, M.D., Gael Bourdin, M.D., Véronique Leray, M.D., Raphaele Girard, M.D., Loredana Baboi, Ph.D., and Louis Ayzac, M.D. for the PROSEVA Study Group*

Results

- Significantly lower mortality in the prone group at day 28 compared to supine group (16% vs 32.8% respectively). This persisted at 90 days (23.6% vs 41%).
- 18 hrs. minimum of being in prone position

Proning team

- 2 RRTs
- 2 RNs
- 2 Physical Therapists

Anatomical Considerations

Occurrences in Pulmonary Compromised Patients

Supine Position:

- Weight of heart upon lungs
- Dorsal lung compression
- Abdominal contents press upward on diaphragm
- Limited lung compliance
- Ventilation/Perfusion - V/Q mismatch
- Facilitates hypoxemia

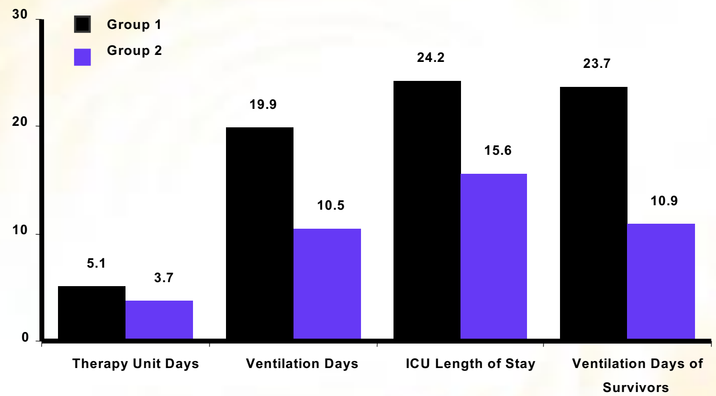
Prone Position

- Increases perfusion to apical and ventral lung units which are less inflamed and patent
- Oxygenation improves secondary improved V/Q matching
- Minimal 16 hrs. per prone session

Results

Group 1: 12 Hr of proning / 24 hr / 2 hr intervals

Group 2: 19:30 hrs proning / 24 hrs / :45 m supine vs. 3:15 hr prone



Sachin Sud et al. Effect of prone positioning during mechanical ventilation on mortality among patients with acute respiratory distress syndrome: a systematic review and meta-analysis. CMAJ July 8, 2014 vol. 186 no. 10.

Ventilatory Management During Prone Positioning

- **Pre-Prone: The Respiratory Therapist will:**

-Increase FIO2 to 100%

-Ensure proper transpulmonary reading/placement

-Ensure Endotracheal/tracheostomy tube is properly secured

-Ensure adequate ventilator circuit length

-Perform complete ventilator assessment

Use of Prone Prior to Intubation

- Any COVID pt. placed on oxygen> 4lpm
- Utilized in medical surgical patient population
- Utilized in all high flow oxygen pts.
- Recommended self-proning
- Reduced ICU intubation in >50%

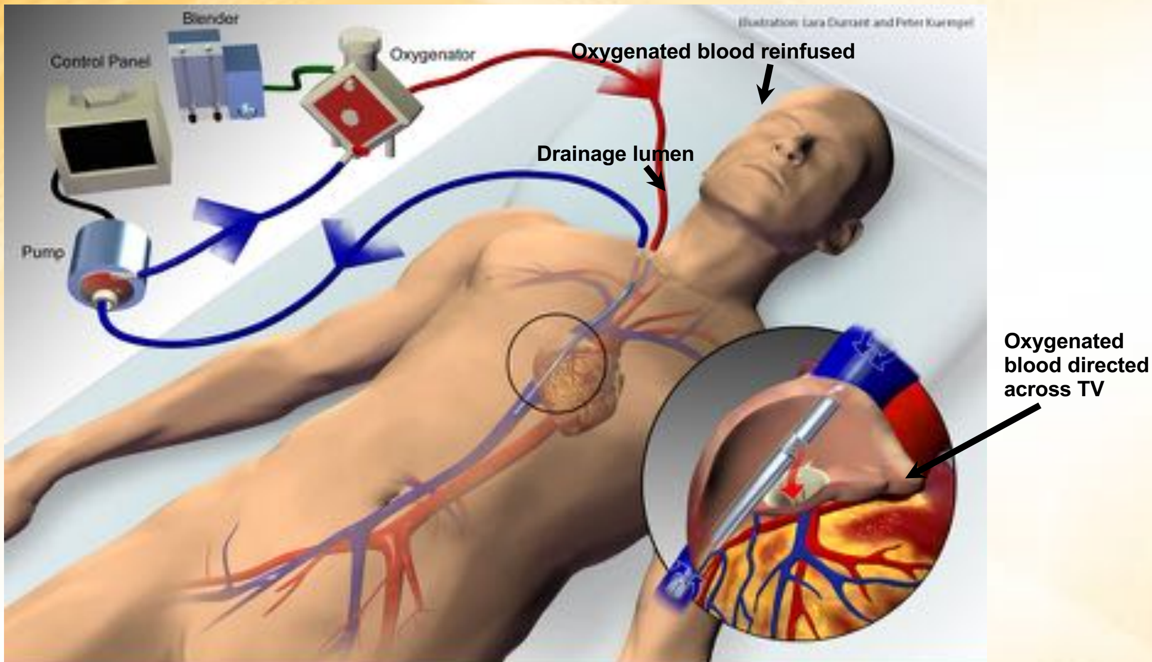
V-V
ECMO

"C'mon, c'mon—it's either one or the other."

Why Do V-V ECMO?

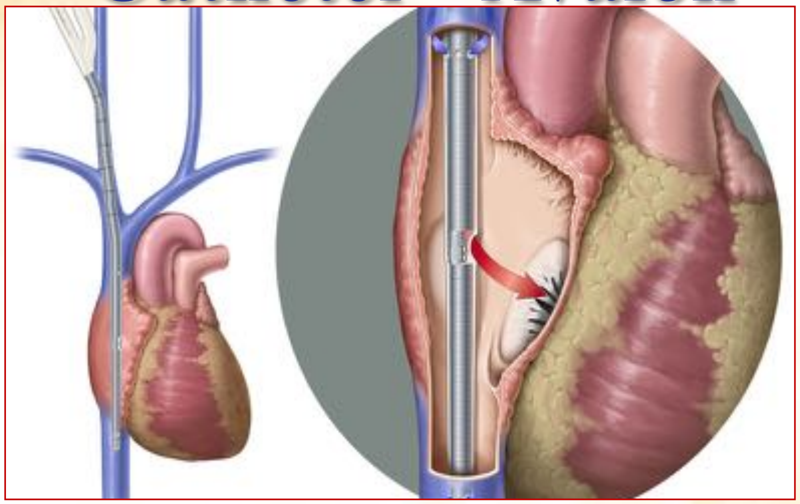
- Veno-venous ECMO takes over the ventilation and oxygenation properties of the lung
- It allows the ventilator to be set on "rest" lung settings while the lung recovers from the underlying illness
- The goal is to oxygenate/ventilate the patient with ECMO while *avoiding ventilator induced lung injury!*

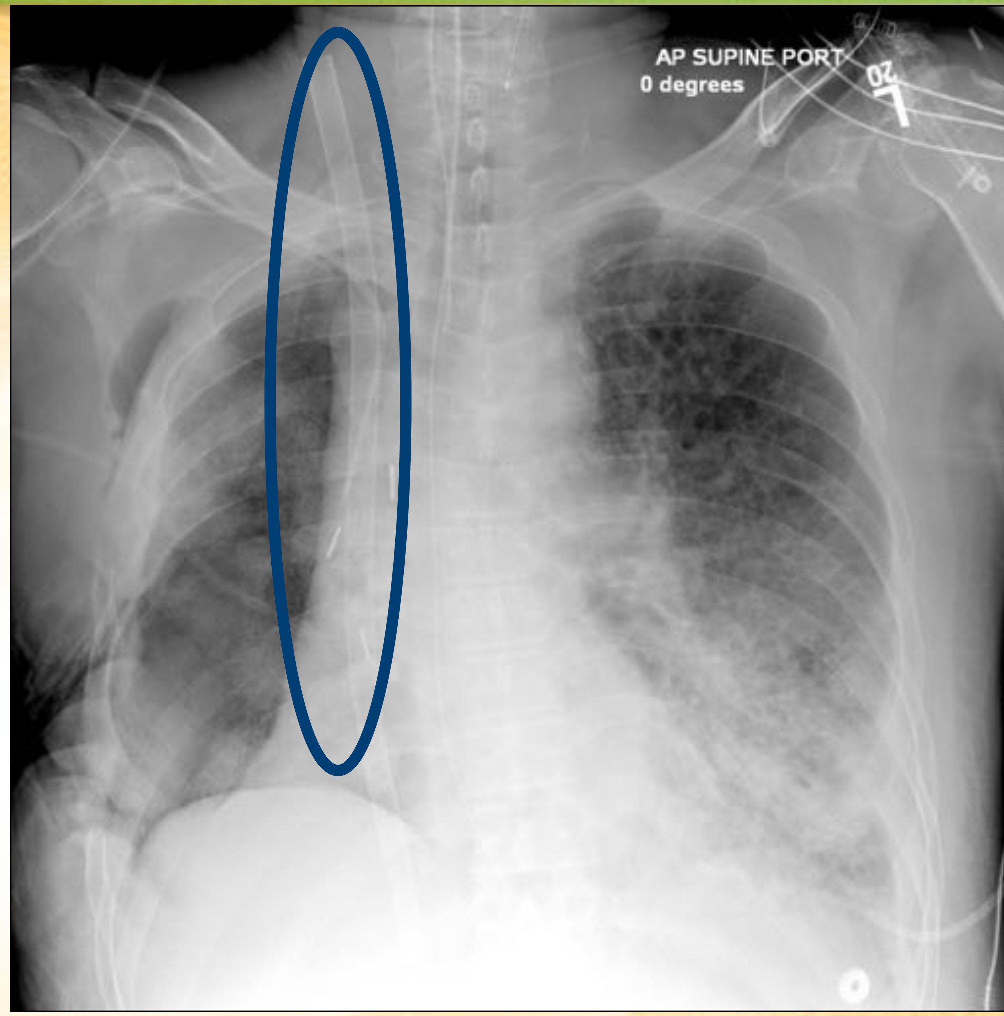
Dual Lumen Cannula for VV ECMO



Single-site approach to venovenous ECMO cannulation:
A dual-lumen cannula is inserted in the internal jugular vein (extending through the right atrium and into the inferior vena cava). Venous blood is withdrawn through one "drainage" lumen with ports in both the superior and inferior vena cava. Reinfusion of oxygenated blood occurs through the second lumen, with a port situated in the right atrium. Inset: The two ports of the "drainage" lumen are situated in the superior and inferior vena cavae, distant from the reinfusion port. The reinfusion port is positioned so that oxygenated blood is directed across the tricuspid valve and directly into the right ventricle. This arrangement significantly reduces recirculation of blood when the cannula is properly positioned.

Single Catheter, Double lumen Catheter - Avalon




AP SUPINE PORT
0 degrees

How We Identify the Potential ECMO Patient?

- P/F ratio
- Lung Compliance
- X-ray
- Ventilator settings
- Murray Score!!!

ns?

Points	0	1	2	3	4
P/F ratio (Fi02 of 1) in mmHg	>300	225-299	175-224	100-174	<100
CXR quadrants infiltrated	Normal	1	2	3	4
PEEP in cm H20	≤ 5	6-8	9-11	12-14	≥15
Compliance in ml/cm H20	≥80	60-79	40-59	20-39	≤19

Murray Score> 3.0=ARDS

Murry Score

Points	0	1	2	3	4
P/F ratio (Fi02 of 1) in mmHg	>300	225-299	175-224	100-174	<100
CXR quadrants infiltrated	Normal	1	2	3	4
PEEP in cm H20	≤ 5	6-8	9-11	12-14	≥15
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4 + 3 +3+ 4+=14/4=Murray Score 3.5=Candidate for ECMO

Mechanical Ventilation Goals During ECMO

- Maintain lung recruitment
- Minimize ventilator induced trauma
- Assist with oxygenation and ventilation if necessary during V-V cannulation
- Provide mucokinesis

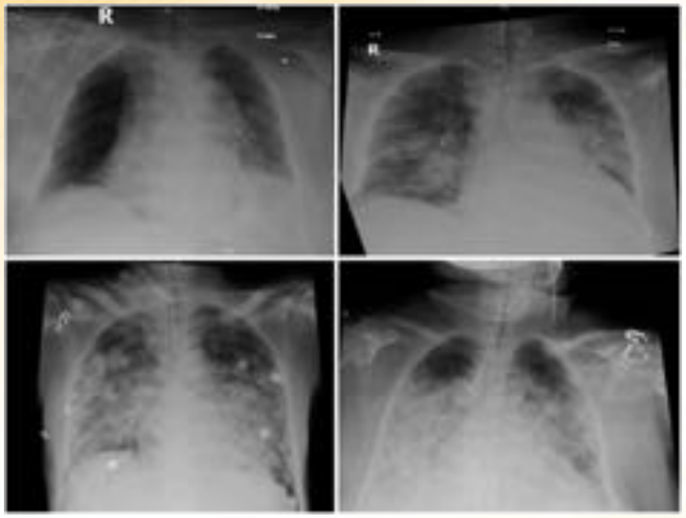
- Poor Survival rate in COVID-19 pts.?

Coronavirus survivor in U.S. receives double lung transplant

The patient, who is her 20s, was on a ventilator and heart-lung machine for almost two months before her operation last Friday at Northwestern Memorial Hospital.
The 10-hour procedure was challenging because the virus had left her lungs full of holes and almost fused to the chest wall, Dr. Ankit Bharat, who performed the operation, said Wednesday.

She was otherwise pretty healthy but her condition rapidly deteriorated after she was hospitalized in late April. Doctors waited six weeks for her body to clear the virus before considering a transplant.

X-Rays Pre/Post Transplant



L E H I G H V A L L E Y H E A L T H N E T W O R K

COVID-19 Ventilator Data @LVHN

Outcomes
July 10

Site	Total Ventilated Pt.	Liberation	Expired	Still ventilated
Muhl	46	27	18	1
CC	80	49	32	1
LVH-H	23	12	10	1
LVH-P	34	19	15	0
LVH-S	6	3	3	0
Total	179	99 (53.7)	77 (43.2)	3 (3.1%)

Ventilator Management@ LVHN

- First ventilated patient March 14 2020 at LVH-M
- Apex 46 ventilated COVID pts.
- Currently 3 ventilated pts.
- 60% of patients placed in placed in prone position for > 48 hrs.
- Mean starting PEEP:
  - March-June 14.5cm (6cm-28cm)
  - June-current 10.2 cm (5cm -20cm)
- Mean ventilator LOS of surviving pts. 27.8
- 100% received paralytics
- 80% received inhaled pulmonary vasodilators
- Early re-intubation (44%) rate especially in females
- BMI 36.6
- Males/Females 60%/40%
- All received Remdesivir, dexamethasone, and convalescent plasma once available

Rescue Therapies?

- High Frequency Percussive Ventilation
  - 3/20 survival 15%
- ECMO
  - 3/24 survival 12.5%

Future COVID Challenges

- Early intubation vs late intubation
- Utilize prone position early-oxygen therapy
- Extubation vs. early tracheostomy
- Balance between maximizing therapy and minimizing staff exposure
- Normalizing day to day operations
- Providing education for staff, students, fostering new technologies
- Is there a magic bullet??

Summary

- COVID ventilator management is very challenging and complex
- Proving lung protective ventilation continues to be mandatory
- The clinical management of the disease is evolving, as is the virus
- The ICU mortality rate remains high
- Is this the new norm?

Thank You For Everything You Have Done During This Pandemic!!

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