PULMONARY MANIFESTATIONS OF COVID-19

MAY 27, 2020
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- President-Elect, American Academy of Emergency Medicine (AAEM)
- Professor of Emergency Medicine, Director of Research and Director of Diversity, Section of Emergency Medicine
- Louisiana State University Health Sciences, New Orleans, LA
BEYOND ARDS:
THE SEARCH FOR A MODEL OF COVID-19 LUNG INJURY AND ITS CLINICAL IMPLICATIONS

Cameron Kyle-Sidell, MD
Emergency Medicine and Critical Care Medicine
Maimonides Medical Center, New York, NY
ckylesidell@maimonidesmed.org
@cameronks
“There may be a possibility that we are dealing with a disease we have never seen... the constellation of symptoms seems to most mirror that of either decompression pulmonary sickness or high altitude sickness.”
THE COVID 19-ARDS DEBATE: IS COVID 19 ARDS?

Normal Alveoli

- Normal alveolus
- Surfactant layer
- Alveolar air space
- Type I cell
- Interstitium
- Epithelial basement membrane
- Type II cell
- Alveolar macrophages

Injured alveolus during the acute phase

- Skirving of bronchial epithelium
- Necrotic or apoptotic type I cell
- Inactivated surfactant
- Red blood cell
- Hyaline membrane
- Widened edematous interstitium
- Swollen, injured endothelial cell
- Platelets
- Neutrophils
- Migrating neutrophils
- Procollagen
- Gap formation
- IL-8
- TNF-α, IL-1β
- Leukotrienes
- PAF
- Oxidants
- Proteases
- Proinflammatory edema fluid

- Alveolar flooding
- Inflammatory exudates
- Widened interstitium
- Swollen endothelium

THE COVID-19-ARDS DEBATE: LUNG COMPLIANCE

The New England Journal of Medicine

VENTILATION WITH LOWER TIDAL VOLUMES AS COMPARED WITH TRADITIONAL TIDAL VOLUMES FOR ACUTE LUNG INJURY AND THE ACUTE RESPIRATORY DISTRESS SYNDROME

THE ACUTE RESPIRATORY DISTRESS SYNDROME NETWORK

ABSTRACT

Background

Traditional approaches to mechanical ventilation use tidal volumes of 6 ml per kilogram of body weight and an airway pressure measured after a 0.5-second pause at the end of inspiration (plateau pressure) of 50 cm of water or less, with ventilation with a lower tidal volume, which involved an initial tidal volume of 12 ml per kilogram of predicted body weight and an airway pressure of 50 cm of water or less, with ventilation with a lower tidal volume, which involved an initial tidal volume of 6 ml per kilogram of predicted body weight and a plateau pressure of 30 cm of water or less. The first group was treated with normal subjects at rest (range, 7 to 8 ml per kilogram), but they are frequently necessary to achieve normal values for the partial pressure of arterial carbon dioxide and pH. Since atelectasis and edema reduce arterial lung volumes in patients with acute lung injury and the acute respiratory distress syndrome,7,8 inspiratory airway pressures are often high, suggesting the presence of excessive distention, or "stretch," of the acerated lung. In animals, ventilation with the use of

Figure 2. Mean (+SE) Mortality Rate among 257 Patients with Acute Lung Injury and the Acute Respiratory Distress Syndrome Who Were Assigned to Receive Traditional Tidal Volumes and 260 Such Patients Who Were Assigned to Receive Lower Tidal Volumes, According to the Quartile of Static Compliance of the Respiratory System before Randomization.

Cstat: ≈ 25 – 45ml/cmH2O ≈ not so good

The interaction between the study group and the quartile of static compliance at base line was not significant (P = 0.49).
COVID-19 Does Not Lead to a “Typical” Acute Respiratory Distress Syndrome

Figure 1. (A) Distributions of the observations of the compliance values observed in our cohort of patients. (B) Distributions of the observations of the right-to-left shunt values observed in our cohort of patients.
- 20-30% of patients
- Stiff, heavy lungs
- Fit ARDS profile

- Over 50% of patients
- Thin, compliant lungs
- Don’t fit classic ARDS profile
THE BERLIN CRITERIA AND THE “ARDS VS NOT-ARDS” DEBATE

Within 1 week

Bilateral infiltrates

Not primarily cardiac

Hypoxemia (Low P/F)

ARDS
How cytopathic is COVID19?

How much direct alveolar damage?
Angiotensin-converting enzyme 2 protects from severe acute lung failure

Yumiko Imai1*, Keiji Kuba1*, Shuan Rao2, Yi Huan2, Feng Guo2, Bin Guan2, Peng Yang2, Renu Sarao1, Teiji Wada1, Howard Leong-Poi3, Michael A. Crackower4, Akiyoshi Fukamizu5, Chi-Chung Hui6, Lutz Hein7, Stefan Uhlig8, Arthur S. Slutsky9, Chenyue Jiang10 & Josef M. Penninger1

Figure 1 | Loss of ACE2 worsens acid aspiration-induced acute lung injury. a, Lung elastance after acid or saline treatment in wild type (WT) and Ace2 knockout (Ace2 KO) mice (n = 10 for acid-treated groups, n = 6 for saline-treated groups). P < 0.05 for the whole time course comparing acid-treated WT and Ace2 knockout mice.
b, Partial pressure of oxygen in arterial blood (p_{O2}) in acid-induced acute lung injury. c, Wet-to-dry weight ratios of lungs 3 h after acid injury. Single asterisk, P < 0.05; double asterisk, P < 0.01. d, Lung histopathology. Note the enhanced hyaline membrane formation, inflammatory cell infiltration and lung oedema in acid-treated Ace2 knockout mice (H&E staining, × 200). e, ACE and ACE2 protein expression in total lysates from control lungs and lungs 3 h after acid injury. Error bars indicate s.e.m.
MODEL OF COVID-19 LUNG INJURY: RELATIVE EXCESS OF ANGIOTENSIN II

SARS-CoV-2

Angiotensin II (Needs ACE to Form)

ACE2

Angiotensin 1-7

Counteract by ACEI / ARB

Pulmonary Endothelium

Relative Excess Angiotensin II

Relative Deficiency Angiotensin 1-7

Vasoconstriction
Pro-inflammatory
Pro-thrombotic

ACE2 Receptor Downregulation

ACE2 Receptor Unaffected

Imbalanced ACE > ACE2 Activity

[ Farid Jalali MD | May 2020 ]
ANG II mediated vasoconstriction, immunologically active endothelium
MODEL OF COVID-19 LUNG INJURY: VASOCONSTRICTION AND MICROTHROMBOSIS

Relative Excess
Angiotensin II

Vasoconstriction
Pro-inflammatory
Pro-thrombotic

Microthrombotic
Pulmonary Capillary
Acute Injury

Dorsal > Ventral
Peripheral > Central

Ischemic Injury
Fibrin Thrombi
Microvascular Occlusion

[ Farid Jalali MD | May 2020 ]
PULMONARY INTRAVASCULAR COAGULOPATHY

Extensive interstitial immunocyte activation with diffuse pulmonary bed extrinsic inflammation leading to microthrombotic immunopathology.
logic analysis of pulmonary vessels in patients with Covid-19 showed widespread thrombosis with microangiopathy. Alveolar capillary microthrombi were 9 times as prevalent in patients with Covid-19 as in patients with influenza (P<0.001). In lungs from patients with Covid-19, the amount of new vessel growth — predomi-
COVID-19 is hypoxic respiratory failure from acute respiratory distress syndrome (ARDS)\(^1\). To date, pulmonary endothelial cells (ECs) have been largely overlooked as a therapeutic target in COVID-19, yet emerging evidence suggests that these cells contribute to the initiation and propagation of ARDS by altering vessel barrier functions.

The proposed central role of ECs in COVID-19 disease escalation prompts the question whether vascular normalization strategies during the maladapted immune response could be useful. Indeed, a clinical trial...
Alveolar Epithelial Dominant ARDS

ARDS Heterogeneity

Endothelial Dominant ARDS

ANG II EXCESS LEADING TO CAPILLARY VASO-OCLUSION DISEASE

Microthrombotic immunopathology

Farid Jalali MD / May 2020
COVID-19 HYPOXEMIA:
SHUNTING TO AREAS OF IMPAIRED GAS EXCHANGE

www.thelancet.com/infection  Published online April 30, 2020

A bacterial infection than a viral infection. Overall, the combination of these imaging findings is novel for COVID-19 pneumonia.

Treatment for acute respiratory
BACKFLOW OF PULMONARY CIRCULATION BLOOD

No Evidence of PAH due to Flow Diversion via BPA

BPA: Bronchopulmonary Anastomosis
BPV: Bronchopulmonary Vein
BA: Bronchial Artery
BV: Bronchial Vein
2019-novel Coronavirus severe adult respiratory distress syndrome in two cases in Italy: An uncommon radiological presentation

International Journal of Infectious Diseases

Received 14 February 2020
Received in revised form 19 February 2020
Accepted 20 February 2020

Figure 6. a,b. (a) Baseline chest CT shows tubular size increase of segmental vessel with normally ventilated adjacent lung parenchyma, (b) where after 3 days there is a ground-glass opacities.
COVID-19 HYPOXEMIA: INTRAPULMONARY SHUNTING AND LOW V/Q MISMATCH

PRONING improves dorsal-predominant shunting
High Lung Volume ->
increased resistance of
intra-alveolar vessels

COVID-19
capillaries
already injured,
thrombotic,
vasoconstricted
LATE COVID-19 LUNG INJURY: DIFFUSE ALVEOLAR DAMAGE

Late Lung Injury is Characterized by Poor Lung Compliance
Progressive Interstitial Edema
Progressive Alveolar Edema and Damage
Progressive Bronchial Distortion

Cytokine storm

Multi-Organ Failure

Endothelial Protective Ventilation (EPV)?

Vent Induced Vascular Lung Injury (VIVLI)?

BPA: Bronchopulmonary Anastomosis
BPV: Bronchopulmonary Vein
BA: Bronchial Artery
BV: Bronchial Vein

Farid Jalali MD / May 2020
When to intubate?

VILI vs. P-SILI
DAMAGED PULMONARY ENDOTHELIUM AS THE “ENGINE” FOR MULTI-ORGAN FAILURE

Clinical Characteristics of Covid-19 in New York City

TO THE EDITOR: The world is in the midst of the coronavirus disease 2019 (Covid-19) pandemic,1,2 and ventilated. Clinical characteristics were more likely to be male, to have obesity, and to have elevated liver-function values and elevated CRP. Patients who were ventilated and had mechanical ventilation were more likely to need vasopressor support (95.4% vs. 1.5%) and to have other complications, including atrial arrhythmias (17.7% vs. 1.9%) and new renal replacement therapy (13.3% vs. 0.4%).

Among these 393 patients with Covid-19 who were hospitalized in two New York City hospitals, supplemental oxygen during the first 3 hours after presenting to the emergency department. Patients who received invasive mechanical ventilation were more likely to need vasopressor support (95.4% vs. 1.5%) and to have other complications, including atrial arrhythmias (17.7% vs. 1.9%) and new renal replacement therapy (13.3% vs. 0.4%).

Weill Cornell Medicine
Phase 1

Normal Pulmonary Microvasculature

Acute Pulmonary Microvascular Injury

Excess Endothelial Angiotensin II Activity

Endotheliitis
ARBS
ACE Inhibitors
Angiotensin 1-7
Statin
NAC
Vitamin C
XO Inhibition

Thrombosis
Antiplasmin
Thrombolitics
Anticoagulation
TXA2, Inhibition

Leukocyte Recruitment
Corticosteroids
IL-17 Inhibition
Omennin Na
Nicotinamide
Membrailast
Lornazepine

Vasoconstriction
ND (1st phase)
PGE2, Agonist (1st phase)
ET, Antagonist (1st phase)
TXA2, Inhibition (1st phase)

Endothelial
Angiostenin II
Male
Obesity
Older Age
Tobacco Use
Hyperlipidemia
Diabetes Mellitus
Hemocystinemia

Plausible Protective Factors

Phase 2

ACR: Receptor Downregulation

SARS-CoV-2 Induced Microvascular Injury

Increased Vascular Permeability

Severe Endothelial Dysfunction

Leukocyte-Platelet Recruitment

Progressive Vasoplastic Pro-thrombotic Pro-inflammatory Endothelial Milieu

Plausible Risk Factors

Microthrombotic Pulmonary Capillary Acute Injury

Dorsal > Ventral
Peripheral > Central

Ischemic Injury
Fibrin Thrombi
Microvascular Occlusion

Subacute Contemporary Pulmonary Microvascular Ischemia-Reperfusion (IR) Injury

Pulmonary Shunt
Low V/Q Mismatch

Ischemia-Reperfusion
Lung Injury Mediated By

NO Excess
Inducible NOS

ROS Production
NADPH Oxidase
Xanthine Oxidase

Leukocyte Adhesion
Macrophage (Early)
Neutrophil (Late)

Platolet Adhesion
Serotonin
Leukotriene
TXA2

Distal Vessel Dilation

Progressive Thrombotic Pulmonary Endothelial Injury

Poor Gas Diffusion

IR-induced Vasodilation

ACE2

Ischemia

ACE2 Sheding

Renal Recovery

Plausible Unique Factors in 2nd Phase

Worsening Reperfusion Injury
Inhaled NO

Worsening Pulmonary Shunt
Inhaled NO

PGII, Agonist
PDE, Inhibitors
ET, Antagonist
Calcium Channel Blockers
TXA2, Inhibition

Worsening Thrombosis
SSRIs

Improving Pulmonary Shunt
Prox-Decapine Cycles
(Re distributes Shunt Flow by Gravity)

@farid_jalali
# Vascular Normalization Strategies: Endothelial Stabilization in COVID-19

## Plausible Protective Factors

<table>
<thead>
<tr>
<th>Endotheliitis</th>
<th>Leukocyte Recruitment</th>
<th>Thrombosis</th>
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</thead>
<tbody>
<tr>
<td>ARBs</td>
<td>Corticosteroids</td>
<td>HRT / OCPs</td>
</tr>
<tr>
<td>ACE Inhibitors</td>
<td>IL-17 Inhibition</td>
<td>Surgery</td>
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<tr>
<td>Angiotensin 1-7</td>
<td>Cromolyn Na</td>
<td>Immobility</td>
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<tr>
<td>Statin</td>
<td>Nicotinamide</td>
<td>Malignancy</td>
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<td>NAC</td>
<td>Montelukast</td>
<td>Peripartum</td>
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<td>Vitamin C</td>
<td>Loratadine</td>
<td>Factor V Leiden</td>
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<td>XO Inhibition</td>
<td>Curcumin</td>
<td>Protein C/S Def</td>
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<td>ATIII Deficiency</td>
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<td>Sickle Cell Trait</td>
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<td>Prothrombin Mut</td>
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<td>Dysfibrinogenemia</td>
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<td>APS / PNH / PV</td>
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<td>Nephrotic Syndrome</td>
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## Risk Factors

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<thead>
<tr>
<th>Thrombosis</th>
<th>Endotheliitis</th>
<th>Plausible Risk Factors</th>
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<tbody>
<tr>
<td></td>
<td>Angiotensin II</td>
<td></td>
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<tr>
<td></td>
<td>Male</td>
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<td></td>
<td>Obesity</td>
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<td>Older Age</td>
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<td>Tobacco Use</td>
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<td>Hyperlipidemia</td>
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<td>Diabetes Mellitus</td>
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<td>Homocysteinemia</td>
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## Vasoconstriction

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<thead>
<tr>
<th>NO (1&lt;sup&gt;st&lt;/sup&gt; phase)</th>
<th>PGI&lt;sub&gt;2&lt;/sub&gt; Agonist (1&lt;sup&gt;st&lt;/sup&gt; phase)</th>
<th>PGI&lt;sub&gt;2&lt;/sub&gt; Inhibition (1&lt;sup&gt;st&lt;/sup&gt; phase)</th>
<th>TXA&lt;sub&gt;2&lt;/sub&gt; Inhibition (1&lt;sup&gt;st&lt;/sup&gt; phase)</th>
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<td>ETT&lt;sub&gt;A&lt;/sub&gt; Antagonist (1&lt;sup&gt;st&lt;/sup&gt; phase)</td>
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<td>TXA&lt;sub&gt;2&lt;/sub&gt; Inhibition (1&lt;sup&gt;st&lt;/sup&gt; phase)</td>
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</table>
COVID-19 VS DECOMPRESSION PULMONARY SICKNESS ??

COVID-19

Microthrombotic immunopathology

DCS

THANK YOU

PLEASE REACH OUT IF YOU HAVE ANY QUESTIONS

Cameron Kyle-Sidell, MD
Emergency Medicine and Critical Care Medicine
Maimonides Medical Center, New York, NY
ckylesidell@maimonidesmed.org
@cameronks
UPDATES FROM THE FRONT LINES: HYPERBARIC THERAPY

Keith W. Van Meter, MD, FACEP

- Chief, Section of Emergency Medicine, Professor of Clinical Medicine, Louisiana State University Health Sciences Center
- Clinical Professor of Surgery, Tulane School of Medicine
- New Orleans, LA
Soldiers hypoxic from “the blue death” were loaded in stake army trucks and were treated at 3 to 4 atmospheres of hyperbaric pressurization.

At depth they pined up, RR dropped, and they became comfortable.
Chapter 12

ACUTE RESPIRATORY DISTRESS SYNDROME IN PATIENTS AFTER BLUNT THORACIC TRAUMA: THE INFLUENCE OF HYPERBARIC OXYGEN THERAPY

Gennady G. Rogatsky, Edward G. Shifrin, and Avraham Mayevsky

I. INTRODUCTION

The rate of mortality from acute respiratory distress syndrome (ARDS) has reportedly reached as high as 50-75%.

The risk of ARDS development increases after severe blunt thoracic trauma (BIT) because of a higher likelihood for lung contusion and acute depression of cardiac function. Monitoring of oxygen transport in patients with ARDS has shown that oxygen delivery and consumption were significantly higher in the survivors compared to nonsurvivors. This suggests that maintenance of oxygen delivery at optimal levels can potentially enable the reversal of ARDS, in cases of severe BIT, these oxygen transport variables may be induced by early cardiopulmonary dysfunction, which requires inotropic support.

On the strength of these data, it is reasonable to conclude that the prevention and correction of oxygen deficiency are basic to intensive care during ARDS.

There are several reports in the literature on attempts to employ the most powerful of known anthosophic means, hyperbaric oxygenation (HBO₂), in treating ARDS. In spite of the favorable impression of the application of HBO₂ in patients with ARDS and the clearly positive results that were achieved when HBO₂ exposure was employed for the elimination of ARDS in various experimental models, however, investigations on this subject were not expanded upon.

The current study is a prospective analysis of the use of HBO₂ in clinical practice.

Neutrophil activation is complex, multiple pathways but two main categories:

- OUTSIDE-IN activation as with ischemia-reperfusion (HBO₂ inhibits this one)
- INSIDE OUT activation via G-proteins as with immune surveillance (NOT this one)

J. Biol. Chem. 283: 10822, '08
J. Biol. Chem. 286: 32854, '11
J. Biol. Chem. 287: 30346, '12
HBOT LESSENS THE VIRAL LOADS OF ENCAPSULATED VIRUS
HBOT AND THE POTENTIAL IMPAIRED OXYGEN CARRYING CAPACITY IN COVID-19

HBOT HAS THE POTENTIAL TO OVERCOME THE IMPAIRMENT OF HEMOGLOBIN OXYGEN CARRIAGE
HBOT AND MICROTHROMBOLYSIS OF MICROTHROMBOSIS IN MICROVASCULATURE OF ISCHEMIC ORGANS
HBOT LESSENS THE CYTOKINE STORM OF INFLAMMATION
Acknowledgment Letter

5/18/2020

Richard Clarke
National Baromedical Services
Nine Richland Medical Park, Suite 440
Columbia, SC 29203
UNITED STATES

Dear Richard Clarke:

The Center for Devices and Radiological Health (CDRH) of the Food and Drug Administration (FDA) has received your submission. This submission has been assigned the unique document control number below. All future correspondence regarding this submission should be identified prominently with the number assigned and should be submitted to the Document Control Center at the above letterhead address. Failure to do so may result in processing delays. If you believe the information identified below is incorrect, please notify the Program Operations Staff at (301) 796-5640.

Submission Number:  PEUA201147
Received:  5/18/2020
Applicant:  National Baromedical Services
Device: Hyperbaric Oxygen Chamber

We will notify you when the review of this document has been completed or if any additional information is required. If you are submitting new information about a submission for which we have already made a final decision, please note that your submission will not be re-opened. For information about CDRH review regulations and policies, please refer to http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/default.htm.

Sincerely yours,

Center for Devices and Radiological Health
THE INFLAMMATORY PULMONARY DAMAGE INDUCED BY COVID-19 IS CAPABLE OF PRODUCING PROFOUND HYPOXIA

Which HBOT Can Easily Overcome

At 1 Atmosphere of pressurization a FIO₂ of 100% Allows Only 2.3 vol % of Dissolved O₂ in Plasma

At 3 Atmospheres of Pressurization a SEFIO₂ of 300% Allows 6.6 vol % of Dissolved O₂ in Plasma

(easily demonstrated in the 1918 flu pandemic and most recently in the Covid-19 Pandemic by the Chinese and the Opelousas experience)
Hyperbaric oxygen therapy in preventing mechanical ventilation in COVID-19 patients: a retrospective case series

ARTICLE
The Outcomes of Hyperbaric Oxygen Therapy to severe and critically ill patients with COVID-19 pneumonia.

Ruiyong Chen 1*, Xiaoping Zhong 2*, Yanshao Tang 3, Yi Liang 4, Biju Li 5, Xiaomin Ti 2, Changbo Liao 1.

1. Division of Medical Research Department of Naval Medical Center, Naval Medical University, Shanghai China.
2. HBOT Department of General Hospital of the Yangtze River Shipping, Wuhan China.
3. The Third District of Airforce Special Service Hospital, Hangzhou China.
4. CT Department of General Hospital of the Yangtze River Shipping, Wuhan China.
5. CT Department of General Hospital of the Yangtze River Shipping, Wuhan China.

*Contributed equally
Correspondence to: Dr. Xiaoping Zhong, HBOT Department of General Hospital of the Yangtze River Shipping, Wuhan China. 
18771506628@189.cn
CURRENTLY NIH REGISTERED COVID-19/HBOT CONTROLLED HUMAN TRIALS

- Ochsner Medical Foundation, New Orleans, Louisiana, USA
- Karolinska University Hospital, Stockholm/USCD, San Diego, California, USA
- Direction Centrale du Service de Sante des Armees, Toulon, France
- Assaf-Harofeh Medical Center, Tel Aviv, Israel
HBOT VERSUS ECMO: INTERMITTENT NON INVASIVE ECMO

- US Facility Charge Average for 3 days of ECMO $350K (2020)
- US Facility Charge for 5 days of daily HBOT $10K (2020)
HOW CAN ONE GIVE PRESSURIZED OXYGEN FOR EQUIVALENCY OF 200-300% WITHOUT PRESSURE INJURY TO THE LUNGS?

**Safety - Pascals Law to the Rescue!**

During a HBOT treatment the whole body is pressurized as is the ventilator, so that the pressure differential provided to the patient is no different than that provided under normal circumstance.
UPDATES FROM THE FRONT LINES: ISRAEL EXPERIENCE WITH COVID-19

Ophir Lavon, MD
- Chairman, Israel Society of Toxicology
- Head, Clinical Pharmacology and Toxicology Unit, Carmel Medical Center
- Haifa, Israel
Israel:

Population ~9,000,000

- ~75% Jewish
- ~21% Muslim, Christian and Druze Arabs

West bank population
~2,800,000

Gaza population ~1,900,000

Size ~20,000 KM²
(the size of New Jersey State)
PUBLIC HEALTH SYSTEM + NATIONAL HEALTH INSURANCE

- 33 general hospitals
- ~16,000 beds (1.8 beds/1000 population)
- ~700 critical care beds
- ~1,400 available ventilators (Feb 2020)
- Community health services: 4 HMO’s

Israel Poison Information Center
Located at RAMBAM Health Care Campus, Haifa
Over 3,000 calls per month
Covid-19 Cumulative cases/deaths in Israel
Correct to May 25, 2020

Confirmed Cases Over Time
16,712
0.17% of Israel population

Early February: Flight ban from far east + Quarantine
Late February: First patients

January: Initial preparedness
Late January: Flight ban from China + Quarantine

Early March: Flight ban from Italy and other European countries + Quarantine
Mid March: Schools closed, No sport/culture events, Limited transportation
Late March: Closed sky + Movement restrictions
April: Full lockdown & Obligatory Masks
Late April: Dynamic lockdown
May: Gradual return to routine with restrictions

Deaths Over Time
279
deaths
1.67% of confirmed cases
Covid-19 Daily Incidence in Israel
Correct to May 25, 2020

Confirmed Cases Over Time
16,712 confirmed cases
Source: World Health Organization

Deaths Over Time
279 deaths
Source: World Health Organization

March 25, 2020
765 Daily Increase

April 8, 2020
13 Daily Increase
Active patients / recovered
Correct to May 25, 2020

RECOVERED
14,203

ACTIVE PATIENTS
2,230

Mild
2,157

Moderate
30

Severe
43

Hospitalized 121

Ventilated 34
Covid-19 Daily Incidence in the Palestinian territory
Correct to May 25, 2020

Palestinian territory:
Confirmed Cases Over Time
602 confirmed cases
Source: World Health Organization

Deaths Over Time
5 deaths
Source: World Health Organization
KEY POINTS:

- Early response: national containment and mitigation
- General Public & health system with orientation to emergency
- Compliant to regulation during emergency
- Public health system: professional, devoted, multicultural
- Addressing all ethnic and religious groups through their leaders
KEY POINTS:

• Assistance and collaboration of civilian and military systems

• Solidarity, especially with healthcare professionals
Q&A
ALL WEBINARS ARE RECORDED AND POSTED TO THE ACMT WEBSITE

www.acmt.net/covid19web

QUESTIONS?
WRITE TO: info@acmt.net
NEXT IN OUR COVID19 WEBINAR SERIES

TBA

Wednesday, June 3, 2020
3:00 PM EDT

www.acmt.net/covid19web