ACG Virtual Grand Rounds

Join us for upcoming Virtual Grand Rounds!

Week 5: Refractory GERD: New Options for Treatment 2020
Philip O. Katz, MD, MACG
April 23, 2020 at Noon EDT

Week 6: Celiac Disease: 10 Things Every Clinician Should Know
Amy S. Oxentenko, MD, FACG
April 30, 2020 at Noon EDT

Visit gi.org/ACGVGR to Register

ACG will send a link to a CME & ABIM MOC evaluation to all attendees on the live webinar.

ABIM Board Certified physicians need to complete their MOC activities by December 31, 2020 in order for the MOC points to count toward any MOC requirements that are due by the end of the year. No MOC credit may be awarded after March 1, 2021 for this activity.

ACG will submit MOC points on the first of each month. Please allow 3-5 business days for your MOC credit to appear on your ABIM account.

MOC QUESTION

If you plan to claim ABIM MOC Points for this activity, you will be asked to: Please list specific changes you will make in your practice as a result of the information you received from this activity.

Include specific strategies or changes that you plan to implement. THESE ANSWERS WILL BE REVIEWED.

Disclosures:

Moderators:
Mark B. Pochapin, MD, FACG
David A. Greenwald, MD, FACG

Speakers:
Brian P. Bosworth, MD, FACG
Vivek Murthy, MD, FCCP
Katherine Hochman, MD, FHM
Rita Agarwal, MD
Ira M. Jacobson, MD, FACG
Mark B. Pochapin, MD, FACG
David A. Greenwald, MD, FACG

According to ACCME guidance, because there are no current preventive or specific treatments for coronavirus infection, there are no relevant conflicts of interest for any speakers or moderators.
Our Panel of Experts

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NYU Langone Medical Center
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Pulmonologist

Katherine Hochman, MD, FHM
NYU Langone Medical Center
Internist/Hospitalist

Ritu Agarwal, MD
Mount Sinai
Hepatologist

Ira M. Jacobson, MD, FACG
NYU Langone Medical Center
Hepatologist

The Tsunami Has Arrived

It Could Have Been Much Worse

Projected Needs for Hospital and ICU Beds in New York State - March 2020

New York City COVID-19 Gearing Up for the Surge

Brian P. Bosworth, MD, FACG
AGG Governor, Manhattan
Chief of Medicine, NYU Langone Health Tisch Hospital
Professor of Medicine, NYU Grossman School of Medicine
Brian.Bosworth@nyulangone.org

American College of Gastroenterology
Mid-January, 2020: First case outside China (Thailand); family clusters reported. Airport screenings in U.S.

Late January, 2020: First cases in S. Korea, US. Reports of HCW infections in China. WHO decides against emergency declaration. Multiple cases in US, France, Canada.


January 31, 2020: CDC quarantines flight from China, declares public health emergency.


Early February, 2020: China with >20,000 cases.


Mid-February, 2020: Retrospective case finding: > total cases >60,000 in China. Cruise ship quarantines in place.

Late February, 2020: Marked increase in infections in S. Korea, Iran, Italy. Estimated case-fatality rate 2-3%. Additional countries affected. Shortage of testing capacity in US. Social distancing encouraged.


March 10, 2020: >10,000 in Italy. New Rochelle, NY "containment zone".


March 14, 2020: France closes most businesses. Travel restrictions in Germany, Spain. South Africa declares national state of disaster.


COVID-19 in NYC

US data: 629,264
New York State: 213,779
Respiratory Pathogenesis of SARS-CoV2 Infection

- The respiratory tract is a primary target of coronaviruses with type II pneumocytes as a focus
- ACE2 receptor, highly expressed on type II pneumocytes, site of viral entry
- Close proximity to rich capillary bed may enable rapid transmission
- Normal functions of type II pneumocytes are generation of surfactant and alveolar repair

Pathologic findings reported with SARS-CoV2 and MERS
- Diffuse alveolar damage
- Denuded bronchiolar epithelium
- Extensive hyaline membrane formation
- Type 2 pneumocyte hyperplasia
- Edematous alveolar septa with lymphocytic infiltrate
- ARDS

Risk factors for hypoxic respiratory failure (19%)
- Age >60 years
- Male gender
- +DM, malignancy, immunocompromised status
- Progression may be rapid, within 12-24 hours of onset
- Median time to developing ARDS 8-12 days
- Median time to mechanical ventilation 10.5-14.5 days
- Median time to VAP after intubation was 8 days

Support of Oxygenation/Ventilation in the Setting of SARS-CoV2 Infection

- Titrate SpO2 to a range between 92-96%
- Chu et al in a 2018 meta-analysis of 25 RCTs in 16,000 critically ill pts requiring supplemental O2, higher target SpO2 >96% associated with increased mortality without improvement in other outcomes
- In the DCCO2 trial ARDS pts randomized to conservative (88-92%) vs liberal (98%) O2 arms—stopped early due to deaths in the conservative arm
Support of Oxygenation/Ventilation in the Setting of SARS-CoV2 Infection

- NIPPV: Avoid BiPap as much as possible unless the patient’s underlying comorbidities (e.g. COPD) strongly favor its use
- Extrapolating from data on 10 critically ill MEIRS pts, 92% of patients intubated on NIPPV ultimately required endotracheal intubation with no difference in mortality or ICU LOS
- In a meta-analysis of 1384 patients in 8 RCTs comparing HFNC vs NC or NIPPV prior to consideration of mechanical ventilation, both were inferior to HFNC
  - OR for requiring intubation 0.68 for HFNC vs NIPPV (0.51–0.81, p=0.005)
  - OR for ICU mortality 0.76 for HFNC vs NIPPV (0.62–0.94, p=0.014)

The Role of High Flow Nasal Cannula

- HFNC: May reduce # of intubations, probably doesn’t reduce mortality or hospital LOS, probably doesn’t increase risk of transmitting SARS-CoV2 significantly
- Rochwerger et al in a meta-analysis of 9 RCTs (2003 pts) found in patients randomized to HFNC vs NC
  - RR for intubation was 0.86 (95% CI 0.74–0.99)
  - RR for mortality was 0.75 (95% CI 0.62–0.93)
- In an analysis of 128 hospital transmission in the 2003 Toronto SARS-CoV outbreak, HFNC was not noted to be associated with increased risk of transmission, however NIPPV and other activities were

The Role of Non-Invasive Positive Pressure Ventilation ("CPAP" or "BiPap")

- Significant concern regarding risk of aerosolization of SARS-CoV2 with NIPPV compared to other modalities
- Odds ratio for "super-spreading" nosocomial infection events in the 2003 SARS-CoV outbreak (case-control study of 86 SARS wards at 21 hospitals in China):
  - NIPPV, OR 11.4 (95% CI 1.92–70.8, p=0.007)
  - Minimum distance between beds < 1 meter, OR 4.08 (95% CI 1.68–9.75, p=0.006)
  - Performing CPR, OR 5.81 (95% CI 1.04–31.87, p=0.04)

Management Schematic for Respiratory Failure in COVID-19 Infection
Modalities of Oxygen Therapy

- **Nasal cannula:** 24-44% FiO₂
- **Venturi mask:** 35-50% FiO₂
- **Non-rebreather:** 100% FiO₂
- **High flow nasal cannula:** 30-100% FiO₂
- **Continuous positive airway pressure (CPAP):**
- **Bi-level positive airway pressure (BiPAP):**
- **Mechanical ventilation**

Nasal cannula:
- Use in adults and children over 6 months of age.
- Flow rates range from 2 to 6 liters per minute.
- Provides comfortable therapy.
- Good for patients with mild to moderate hypoxemia.
- Can be used as a bridge to more invasive therapies.

Venturi mask:
- Provides a fixed ratio of oxygen and air.
- Useful for patients requiring supplemental oxygen.
- Adjustable settings for different flow rates and oxygen concentrations.

Non-rebreather mask:
- Provides 100% oxygen flow to the patient.
- Commonly used in emergency situations.
- Can be uncomfortable for long-term use.

High flow nasal cannula:
- Delivers high flow oxygen directly to the nasal cavity.
- Can be used in patients with acute respiratory distress.
- Requires a high flow rate to maintain oxygen levels.

Continuous positive airway pressure (CPAP):
- Provides positive pressure above the baseline airway pressure.
- Helpful in treating respiratory failure and sleep apnea.
- May be used as a bridge to mechanical ventilation.

Bi-level positive airway pressure (BiPAP):
- Switches between two pressure levels.
- Used in patients with chronic obstructive pulmonary disease (COPD).
- Can be used to treat hypercapnia.

Mechanical ventilation:
- Requires mechanical assistance to maintain ventilation.
- Used in severe respiratory failure.
- Can be invasive or non-invasive.

All modalities require careful monitoring of oxygen saturation (SpO₂) and clinical response.

Additional notes:
- Use caution in patients with hypercapnia.
- Oxygen therapy should be adjusted based on clinical response and SpO₂ levels.
- Monitoring of carbon dioxide levels (CO₂) is crucial.
- Hypercapnia can indicate underlying respiratory failure.
- Monitor for signs of respiratory distress and hypoxemia.
- Adjust oxygen therapy as needed to maintain SpO₂ between 90% and 95%. 

**References and Further Reading:**
- American Academy of Sleep Medicine (AASM) guidelines for the management of sleep-related breathing disorders.
- National Heart, Lung, and Blood Institute (NHLBI) recommendations for the treatment of chronic obstructive pulmonary disease (COPD).
- European Respiratory Society (ERS) guidelines for the management of chronic obstructive pulmonary disease (COPD).

**Additional Considerations:**
- Use of non-invasive ventilation (NIV) in selected patients with acute respiratory failure.
- Role of high-flow nasal cannula therapy in acute respiratory failure.
- Importance of patient education and family involvement in oxygen therapy management.

**Evidence Levels:**
- Level 1: Strong evidence based on randomized controlled trials.
- Level 2: Moderate evidence based on meta-analyses or systematic reviews.
- Level 3: Limited evidence based on case series or expert opinion.

**Key Terms and Concepts:**
- FiO₂: Fraction of inspired oxygen.
- SpO₂: Oxygen saturation.
- COPD: Chronic obstructive pulmonary disease.
- Hypercapnia: Increased carbon dioxide levels.
- Hypoxemia: Decreased oxygen levels in the blood.
- SpO₂: Oxygen saturation.
Medical Therapies for COVID-19 Infection

There are no definite effective therapies for SARS-CoV2 infection, but several classes of medication are being investigated:

- Antiviral agents (remdesivir, lopinavir/ritonavir)
- Hydroxychloroquine/chloroquine
- Immunosuppressors
- Anti-IL-6 (sarilumab, tocilizumab)
- Anti-IL-1 (anakinra)
- Corticosteroids
- Anticoagulation
- Antibiotics (secondary bacterial infection)
- Convalescent plasma

Basic Approach to the Hypoxic SARS-CoV2 Patient

- On arrival, check a chest x-ray, EKG, ABG; avoid CT Chest if possible unless another dx is suspected
- Consider secondary bacterial pneumonia if worsening and start empiric antibiotics
- Maintain on minimum supplemental O2 to maintain SpO2 92-96%
- Escalate to HFNC if possible before using a non-rebreather (reasonable to touch base with Pulm/CC at this point); avoid CPAP/BiPAP unless there is a very specific indication
- If underlying COPD/Asthma, use metered dose inhalers rather than nebulizer therapy
- If in distress on oxygen therapy (lethargic/tachypneic), check an ABG and consider PE
- If on mechanical ventilation, consider daily ABG to aid in titration, though if tidal volume/rate are stable and the patient is overall improving, could also just wean FiO2/PEEP by 1%
Getting Started: Know How to Mine Your EMR

O2 requirements
Inflammatory Markers
COVID-directed therapy

Who gets hospitalized? Who gets intubated?

STRONGEST HOSPITALIZATION RISKS:
- Age
- >75 years (OR = 66.8)
- Admission O2 sat <88% (OR = 6.9)
- First Ferritin >3500 (OR = 6.9)
- First CRP > 200 (OR = 5.78)

STRONGEST CRITICAL ILLNESS RISKS:
- Admission O2 sat <88% (OR = 6.9)
- First Ferritin >3500 (OR = 6.9)
- First CRP > 200 (OR = 5.78)

A Snapshot of the NYC Experience

4,109 COVID (+) patients
1,999/4,109 (48.7%) hospitalized
2,110/4,109 (51.3%) – not hospitalized

650/1,999 (32.5%) critically ill (ICU/ventilation/death/hospitalized)

455/650 (70%) received mechanical ventilation
162/445 (36.4%) died/hospice
245/445 (55.1%) still ventilated
16/445 (3.6%) discharged
22/445 (4.9%) extubated, still hospitalized

Follow ACE2

- SARS-CoV2 gains entry via ACE2
- ACE2 present in:
  - Lung
  - Kidneys
  - Heart
  - Vascular epithelium
  - Brain
  - Gut/Liver

COVID-19 Course of Infection

Course of COVID-19 Infection


American College of Gastroenterology
COVID and the Kidneys

**PREVALENCE**
- 7.8% troponinemia
- 52% of ICU patients showed cardiac injury
- Mortality = 51% cardiac injury vs 4.5% without cardiac injury

**CLINICAL PEARLS**
- 2 patterns of cardiac injury:
  - Non-cardiac symptoms: troponin tracks with inflammatory markers in cytokine storm
  - Cardiac symptoms: palpitations/chest pain; viral myocarditis/arrhythmias, ST elevations, low EF
- ERG on admission (useful for OC3 interval)
- Mild troponin elevation – no cardiac imaging
- Standard heart failure, arrhythmia therapy
- No change in ACE/AHR management

COVID and the Heart

**PREVALENCE**
- 7.8% troponinemia
- 52% of ICU patients showed cardiac injury
- Mortality = 51% cardiac injury vs 4.5% without cardiac injury

**CLINICAL PEARLS**
- 2 patterns of cardiac injury:
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COVID and the Blood

**PRESENTATION**
- WBC 4.7
- RBC ≥ 1.5 83% of patients (lymphopenic)
- AIC ≥ 700 patients in ICU
- AIC < 700 lymphopenic ratio
- > 3.13 – 50% severe
- < 3.13 = 9% severe disease

**CLINICAL PEARLS**
- Patients are very hypercoagulable and should be anticoagulated
- Not classic DIC picture (high d-dimers and fibrinogen but mild thrombocytopenia)

COVID and the Nervous System

**PREVALENCE**
- 16.6% neurological symptoms
- CNS (dizziness, headache, 13%, AMS, –7%, stroke –6%)
- PND (state impairment: 6%, small impairment –5%)
- Skeletal muscle injury: muscle pain and CPK >300 – 10%
- Severe COVID patients are more likely to have neurologic symptoms (43.5% vs. 32.5%)

**CLINICAL PEARLS**
- Cord patients with neurologic symptoms often lack typical cough and fever
- Many patients come in with syncope as a presenting symptom (autonomic instability)
SUMMARY RECOMMENDATIONS

• The care of the COVID patient is largely supportive
• Silent hypoxia is real (patients don’t realize that they are hypoxic)
• Patients can decline very rapidly with no warning
• Get a health care proxy signed on admission/involving palliative care early
• Prone whenever possible
• Don’t forget about patient’s diet order (or your diet!)
• Patients are terrified and lonely
• Make sure to speak with families

Evaluation of Abnormal Liver Enzymes in COVID-19 Patients

Ritu Agarwal, MD
Assistant Professor of Medicine
Director, Liver Fellows’ Practice
Division of Liver Diseases
Icahn School of Medicine at Mount Sinai

Evaluation of Elevated Liver Enzymes in COVID-19

Mostly Hepatocellular Injury
- Mildly elevated AST, ALT, elevated LDH, ferritin
- Mildly elevated normal bilirubin
- Elevated AST ALT: common sources outside liver (e.g. myositis)
- Low serum albumin is a marker of COVID severity

Might be reflective of:
- Drug induced liver injury
- Cytoskelet Release
- Ischemia and congestion
- Myositis

Liver Pathology

- Limited reports
- Nonspecific
- Microvesicular steatosis
- Mixed mild lobular and portal activity

Background

• ACE-2 is a molecular target for SARS-CoV-2
• ACE-2 receptors occur on liver and biliary epithelial cells
• Elevated liver enzymes are fairly common in hospitalized COVID-19 patients: 14-53%
• Liver injury in mild COVID-19 patients is typically transient and resolves with supportive care
• Seldom have liver failure
• Liver injury is more common in severe than mild COVID-19 patients

Explanation of Liver Injury

Remains Unclear
- Indication of underlying pre-existing liver disease
- Direct Virus Induced Effect
- Immune Inflammatory Response to the Virus
- Could DILI be present as well
Evaluation of Elevated Liver Enzymes in COVID-19

**History**
- Alcohol: How many drinks per day and last drink? History of withdrawal?
- Medications
  - Home medications
  - Specific to COVID:
    - Acetaminophen use at home (Dose and Duration)
    - Antibiotics (Azithromycin, Augmentin, Cephalosporins)
    - Vitamins
- Speak to family and household members

Treatment-Drug Induced Liver Injury

<table>
<thead>
<tr>
<th>Medication</th>
<th>Function</th>
<th>Side Injury</th>
<th>Side Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydroxychloroquine</td>
<td>Arthritis Therapy</td>
<td>Arthritis Therapy</td>
<td>Arthritis Therapy</td>
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<tr>
<td>Azathioprine</td>
<td>Immune Modulation</td>
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<td>Methotrexate</td>
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<td>Prednisone</td>
<td>Immune Modulation</td>
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<tr>
<td>Penicillin</td>
<td>Antibacterial</td>
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</tbody>
</table>

COVID-19 in Patients with Chronic Liver Disease

- No evidence that patients with stable chronic HBV, HCV, PBC, PSC, NAFLD have increased susceptibility to SARS-CoV-2
- NAFLD/NASH patients have comorbidities (DM, HTN, Obesity) increased risk of severe COVID-19
- Continue treatment for hepatitis B or C
- Continue surveillance for HCC (cirrhosis and HBV) as close to intervals as possible.
- Arbitrary 2 month delay reasonable. Review reason for delay with patient.
- Emphasize immunization for Streptococcus pneumoniae and Influenza

COVID-19 Immunosuppression

- Effects of immunosuppression remain unclear
- Immunosuppression possibly protective from the reactive immune response
- Age and metabolic comorbidities (Obesity, HTN, DM) in post transplant patients might be associated with increased risk of severe COVID-19 disease
Patients with Chronic Liver Disease and COVID-19

Immunosuppression

- In immunosuppressed patients without COVID-19
  - Do not make anticipatory adjustments to immunosuppression

- In immunosuppressed liver patients with COVID-19
  - Consider minimizing high dose prednisone
  - Individual decision based on severity of COVID-19, graft history, timing post transplant
  - Consult transplant hepatology colleagues

- Initiate immunosuppression in patients with or without COVID-19 who have strong indications for treatment (autoimmune hepatitis, graft rejection)

Management Conclusion

- Limit acetaminophen < 2 grams daily
- Careful history from patient and family for alcohol and home medications
- COVID-19 liver injury is typically hepatocellular, transient and mild
- Mechanism of injury unclear
- Consider Drug Induced Liver Injury and extra hepatic sources of injury
- Continue immunosuppression when appropriate but consider dose reduction in patients with severe COVID-19

Investigational Agents for COVID-19

Ira M. Jacobson, MD, FACG
Professor of Medicine
Director of Hepatology

The Virus and its Receptor

Membrane glycoprotein
Spike protein
Envelope protein
Nucleocapsid protein
Receptor ACE2

SARS-Cov2

The Scientist, February 19, 2020
SARS-CoV-2 Life Cycle

1. Spike protein on the virus binds to ACE2, a cell surface protein, triggering uncoating in endosomes.
2. The virion releases the RNA genome, which is translated in the cytoplasm to produce polyproteins.
3. CYP450 enzymes cleave the polyproteins to produce individual proteins, including the replication-competent virus.

Potential Therapeutic Targets in COVID-19

- Inhibits RNA-dependent RNA polymerase activity among a diverse group of RNA viruses including filoviruses (e.g. Ebola, Sudan, Marburg), paramyxoviruses (e.g. RSV, Nipah, Hendra) and pathogenic coronaviruses.
- Nonhuman primates: therapeutic efficacy of remdesivir against Ebola virus but survival inferior to monoclonal antibodies in PALM study (NEJM 2019).
- Activity against SARS-CoV and/or MERS-CoV in mouse infection models, non-human primates, and human airway epithelial cell assays.
- Cell culture studies show potent SARS-CoV-2 suppression at micromolar level.

Background on Remdesivir

- 61 patients, 53 with analyzable data
- 30/53 (57%) on mechanical ventilation
- 4/53 (8%) receiving ECMO
- Median follow-up 18 days
  - 36/53 (68%) had improvement in oxygen-support class
  - 17/30 (57%) receiving mechanical ventilation were extubated
  - 25/53 (47%) were discharged
  - 7/53 (13%) deaths
  - 6/34 (18%) receiving invasive ventilation died vs 1/19 (5%)
Cumulative incidence of clinical improvement: Overd2
Cumulative incidence of clinical improvement: Baseline oxygen support
Cumulative incidence of clinical improvement: Age

High rate of improvement
Patients on ventilation had less improvement
Age > 70 a differentiator

Authors pointed out:
- 28 day mortality 22% in lopinavir/ritonavir trial: only 1/199 were on ventilation
- 66% mortality in 44/67 on mechanical ventilation in Wuhan, China
- Need randomized trials acknowledged: large studies in progress

Several approved
High rate
Based on

Grein J
Ferner RE

1. 3.
8. Cao et al, medRxiv 2020.03.22.20040758. (Declarations of interest)
Investigative Agents for COVID-19 (Not All-Inclusive)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Genera</th>
<th>Mechanism</th>
<th>Pharmacokinetics</th>
<th>Mode of Action</th>
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<tbody>
<tr>
<td>Remdesivir</td>
<td>Oral</td>
<td>Nucleoside 5'-triphosphate</td>
<td>Oral, parenteral</td>
<td>Nucleoside analog</td>
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<tr>
<td>Hydroxychloroquine</td>
<td>Oral</td>
<td>Antimalarial</td>
<td>Oral, parenteral</td>
<td>Inhibits viral replication</td>
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<td>Azithromycin</td>
<td>Oral</td>
<td>Macrolide antibiotic</td>
<td>Oral, parenteral</td>
<td>Inhibits bacterial translation</td>
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<td>Remdesivir Gilead Nucleotide RNAi</td>
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<td>‐888 Takeda Polyclonal mAb vs (generic)</td>
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**HCV**
- RNA virus (family Flaviviridae)
- Non-enveloped virus
- Parenteral transmission
- Chronically common
- Genotypes:
  - Clinical differentiation
  - End-stage disease takes years
- Bone marrow from monospecific to specifically targeted direct-acting antiviral agents (DAA)
- Triplet takes a long time to eradicate
- Virologic clearance was pivotal goal
- Resistance mattered until 2nd gen drugs
- No vaccine after 30 years

**SARS-CoV2**
- RNA virus (coronavirus)
- Zoonotic ("jump" from animals to human)
- Respiratory infection
- Chronically mild (?)
- Does chronicity exist (?)
- Genotypic variation: any clinical implications?
- End-stage disease take: long
- Controversy: whether nonspecific flu work on immunosuppressive or antibacterial or both?
- Tray endpoints achieved quickly
- Success will be measured by clinical outcomes
- Resistance may not matter: short course, nucleotide (?), viral suppression may be enough
- Vaccine desperately needed

**Preparation**
- Encourage your hospital or lab to scale up COVID-19 testing now
- Conserve PPE
- If possible, obtain N95s or equivalent
- Break down all procedures into elective, semi-elective, urgent

**Procedure Classification**

<table>
<thead>
<tr>
<th>Elective (Delay)</th>
<th>Semi-Elective (Perform)</th>
<th>Urgent (Perform)</th>
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| Screening or surveillance endoscopy
  - Flexible or surveillance EGD in a patient with asymptomatic upper GI disease
  - Evaluation of nonurgent symptoms (e.g., EGD for non-alarm symptoms, such as vague abdominal pain, nausea, GERD, or non-alarm endoscopic procedures)
  - EGD for pancreatic cyst or small submucosal lesion
  - All minitry procedures (e.g., hepatic arterial/venous manometry, CT-angiography) | Upper and Lower GI bleeding
  - Symptomatic pancreatobiliary disease
  - Evaluation of suspected malignancy
  - Upper/lower GI symptom that will not cause diagnostic management of suspected disease that the patient and physician believe cannot wait 3 months to evaluate. | Upper and Lower GI bleeding
  - Symptomatic pancreatobiliary disease
  - Evaluation of suspected malignancy |

**Waves of Deployment**

**Wave 1 ICU**
- Critical care trained physicians currently not practicing in ICUs and non-medical ICU intensivists (Pulmonologists trained in critical care, Anesthesiologists trained in critical care, Cardiologists who cover the CCR, Surgeons who cover the surgical ICU)
- Young physicians and volunteer senior fellows who receive additional training in ICU procedures and ventilator management.
- These physicians are board certified in Internal medicine and just a few years our from their ICU experience as residents.
- Internists and subspecialists (gastroenterologists) who will act as medical attendings to backfill the vacant hospitalist positions.
Two Tiers of the COVID Army

- Medical Attending
  - Acting as a hospitalist
  - Interns, resident, APPs
  - Responsible for overall medical management and clinical decisions
  - 12-15 patients
  - Internal medicine trained (often subspecialist)
- Supplemental medical attendings (Super Residents)
  - Part of a team of other supplemental medical attendings and housestaff
  - Overseen by a hospitalist
  - Average of 5 patients
  - Surgeons, Ophthalmologists, Dermatologists, OB/GYN

Other COVID-19 Tips:

- Uniform communication messaging wherever possible with staff and patients
- Plan for limited reopening of ASCs/Offices once the number of new cases begins to decline (May / June?)
  - Bring in the Semi-urgent patients first
- Self-monitoring for symptoms—test or retest for ANY symptoms
- Test all doctors, nurses and other staff who are planning on working with patients
- Recognizing stress/anxiety for patients, staff, and MDs
  - Exacerbation of underlying anxiety and mental illness—all ramped up by severe limitations on “usual” activities
  - Uncertainty how long this will last
  - It is a marathon without knowing where the finish line is

Visit ACG’s COVID-19 Resource Page
www.gi.org/COVID19

How to Receive CME and ABIM MOC Points

LIVE VIRTUAL GRAND ROUNDS WEBINAR
ACG will send a link to a CME & ABIM MOC evaluation to all attendees on the live webinar.

ABIM Board Certified physicians need to complete their MOC activities by December 31, 2020 in order for the MOC points to count toward any MOC requirements that are due by the end of the year. No MOC credit may be awarded after March 1, 2021 for this activity.

ACG will submit MOC points on the first of each month. Please allow 3-5 business days for your MOC credit to appear on your ABIM account.
If you plan to claim ABIM MOC Points for this activity, you will be asked to: Please list specific changes you will make in your practice as a result of the information you received from this activity.

Include specific strategies or changes that you plan to implement. THESE ANSWERS WILL BE REVIEWED.