

# Treating COVID-19 in patients with ME/CFS & severe FM

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# Useful medical information when treating COVID-19 in patients with underlying myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS) and severe Fibromyalgia (FM)

If your patient has been diagnosed with ME/CFS/severe FM, you should assume they have a serious, chronic, multisystem illness that may negatively impact their prognosis. The intention of this booklet is to provide basic advice and medical/scientific information about ME/CFS/severe FM that can inform medical decisions.





ME/CFS is characterized by severe fatigue and easy fatigability, both physically and cognitively. Symptoms can worsen after physical or cognitive exertion, orthostatic stress, environmental and sensory stress.

**When subjected to these stressors, patients may experience more exhaustion, cognitive impairment, sleep disruption, pain, headaches, autonomic dysregulation, dizziness and flu-like symptoms.** Be aware of common comorbid conditions: small fiber neuropathies, hypermobility, autoimmune thyroid disease, euthyroid sick syndrome, Sicca Syndrome, pain amplification disorders (to include FM), postural orthostatic tachycardia syndrome (POTS), primary sleep disorders, multiple chemical sensitivities, small intestine bacterial overgrowth (SIBO), GI dysmotility, Celiac disease, etc.

# **QUICK TIPS**

## **for managing patients with ME/CFS/severe FM who become acutely ill:**

**Presume the patient is orthostatic and treat as if in “shock” (abnormal perfusion and circulatory failure)**

- \* Increase and maintain intravascular volume with IV saline (unless CHF or obvious fluid overload is present). Monitor orthostatic vital signs. Monitor and replace electrolytes. Consider alternating normal saline and lactated Ringer’s. Avoid hypotension and hypovolemia.
- \* Provide some oxygen even if not severely hypoxemic.
- \* Allow patients to lie down, or sit with feet elevated, limit activity and rest as needed. Avoid prolonged standing, or even sitting with feet on the floor for long periods of time. Provide a wheelchair for energy conservation.

**Reduce sensory stimuli (sensory stress) as much as possible**

- \* Reduce: bright light, loud music, obnoxious sounds, and scents/smells.
- \* Maintain adequate pain control.
- \* Limit nighttime sleep disruptions. Treat sleep, if needed and appropriate. Serial nights of sleep disruption may worsen all aspects of the illness.

## **Assume cognitive impairment (cognitive slowing) is present**

- \* Keep conversation simple. Avoid compound questions.
- \* Be patient and allow longer periods for patient responses to questions.
- \* Write down instructions.
- \* Allow family and caregivers to assist with communication.

## **Use medications thoughtfully and skillfully with close monitoring**

- \* Patients with ME/CFS may be unusually sensitive to medication effects and more likely to have allergic reactions.
- \* Start low (10-25% of usual) with medication doses. Allow the patient to report past intolerances and experiences.
- \* Be very cautious about abruptly stopping benzodiazepines and opioids, as the withdrawal can be amplified and dramatically provoke rebound symptoms.
- \* Consider "stress doses" of hydrocortisone (5-10 mg bid) as though the patient has adrenal insufficiency. Patients may have a centrally blunted "stress response" that signals pulsatile release of cortisol.



The current evidence-based **core clinical diagnostic criteria** for ME/CFS (symptoms must be moderate to severe and present 50% of the time for at least 6 months) are: **impaired ability to function in association with fatigue, post-exertional illness worsening, disordered sleep, cognitive impairment and/or orthostatic intolerance.** Most patients also experience **pain and immune** manifestations.

**Based on current evidence the underlying pathology of ME/CFS involves energy metabolism, the nervous system, and the immune system.**

- \* **Abnormal cellular metabolism.** Metabolomics have found deficits in pathways that generate energy from simple sugars, fatty acids, and amino acids. Increased lactate levels in cerebrospinal fluid may indicate impaired oxidative phosphorylation, with a consequent shift to anaerobic metabolism. Cardiopulmonary exercise testing (CPET) testing suggests a low anaerobic threshold. Invasively monitored CPET demonstrates impaired/reduced pre-load and reduced oxygen extraction from either perfusion abnormalities or inability of cells to utilize delivered oxygen.

- \* **Neuroinflammation and central sensitization.** Amplification of or increased sensitivity to sensory stimuli. Spinal fluid studies have shown elevated WBC and protein. MRI, functional MRI and PET studies have revealed hypoperfusion, elevated lactate, and widespread activation of glial cells. EEG studies show abnormal brain waves and connectivity of various brain regions.
- \* **Cognitive impairment.** High quality evidence supports cognitive slowing, deficits in attention, memory and reaction time. It is unclear how much this relates to a neuroinflammatory process versus impaired brain perfusion.
- \* **Impaired/dysregulated HPA-axis and ANS stress response system.** Orthostatic intolerance is common. Neuroendocrine studies demonstrate abnormalities of the hypothalamic-pituitary-adrenal axis, of growth hormone secretion, and central signaling of cortisol release. ANS studies have found strong evidence of disordered autonomic nervous system activity, impaired baroreflex function, exaggerated venous pooling, diminished red cell mass, and reduced plasma volume.
- \* **Abnormal immune function.** Evidence supports impaired natural killer cell function, increased numbers of activated CD8+ cytotoxic T cells, presence of various autoantibodies, particularly to targets in the CNS and ANS. Increased production of various proinflammatory cytokines correlate with the illness severity. Mast cell activation is present in some patients.
- \* **Risk of viral reactivation** (for example varicella zoster, Cytomegalovirus, Epstein–Barr virus) may be present.

*This information was prepared on 03/20/2020 by Lucinda Bateman, MD, of the Bateman Horne Center. It is intended to raise awareness about known aspects of ME/CFS. The TIPS are from Dr. Bateman, an expert in the management of ME/CFS and FM. There are currently no Treatment Guidelines for ME/CFS. The "underlying pathology of ME/CFS" in the previous section was written drawing heavily from the reference below. Every case should be carefully assessed and treated using the best judgement of the managing physician, and for each presenting medical problem, taking current scientific progress and guidance into account.*

**RESOURCE:**

*Inflammation correlates with symptoms in chronic fatigue syndrome: Anthony L. Komaroff. Proc Natl Acad Sci USA. 2017 Aug 22; 114(34): 8914-8916. Published online 2017 Aug 15. doi: 10.1073/pnas.1712475114 PMID: PMC5576849*

*Pam Harrison. Patients on Steroids With COVID-19 Might Need Rescue Steroids - Medscape - Apr 03, 2020*

**For more information about ME/CFS  
visit the U.S. ME/CFS Clinician Coalition's  
website at [mecfscliniciancoalition.org](http://mecfscliniciancoalition.org).**

*Whittemore Peterson Institute is proud to be a source of information about ME/CFS and FM. Our comments are based on professional advice, published experience, and expert opinion, but do not represent individual therapeutic recommendations or prescriptions. For specific information and advice, consult your physician.*



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