Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

baseline reality that many of our patients (along with their friends and loved ones) lack the cognitive and relational skills to be fully capacitated for a range of medical decisions, when they are together in the hospital, they function as living, choosing bodies in ways that reassure primary teams about the path going forward. Having visitors in the room bolsters the patient side of care relationships in a manner to which we have become accustomed. Their absence leaves our internists and surgeons concerned that the patient's voice has lost an amplifier when those visitors are less present and only occasionally connected via remote technology.

The increase in requests for capacity assessment may also reflect heightened concerns about doing the right thing in these difficult times. We have seen what happened in Italy and fear the specter of rationing. So even at this stage with adequate resources for all of our inpatients, there is a desire to ensure the ethical rectitude of our own part in medical decisions and the practice that proceeds from them. There is more pressure to perform our work in a way that not only helps patients and fits with our own professional sense of duty but also holds up under outside scrutiny.

As psychiatrists, our C-L group has also been curious about other layers of meaning embedded in this change in our interdisciplinary experience. Metaphorically and dynamically, it may be that medical teams fear *they* lack some "capacity" in this time. They may worry more about making the wrong decisions about assessment and treatment when the emergence of a new disease and the evolving practice around it leaves standards unclear. Physicians themselves (ourselves?) may lack capacity to manage uncertainties inherent to caring for the sick and vulnerable when the close physical contacts doctors share with their patients to gather information and solidify the bonds of their working relationships have been limited by fear of coronavirus disease spread. We believe our service has been asked to contain more distress through a veiled communication of these concerns in the form of increased requests for evaluation of decision-making capacity.

I have decided that such requests – which on the surface may appear to be an improper use of our resources when the answer to that question about capacity is obvious – reflect a need for which we must exhibit the capacity to be present and helpful to patients and their care teams on multiple levels. C-L psychiatrists have become accustomed to being consulted in unclear ways about unclear things. Now, more than ever with the emergence of a pandemic threat, it is clear to me that the wisdom I attribute to our own Dr. Maryland Pao has never been more relevant – "There's no such thing as a bad consult."

Sincerely,
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Re-examining the Association Between COVID-19 and Psychosis

TO THE EDITOR: We read with great interest the recent report by Ferrando et al.,[1] which described 3 patients who presented to the emergency department with similar symptoms including agitation, disorganization, paranoid ideation, and auditory hallucinations. They were all tested for the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), albeit the method of testing was not specified, and found to be positive for the Coronavirus Disease 2019 (COVID-19). We have some thoughts on the observed association and hope this would generate greater discourse on the subject.

First, possible infective origins of mental illness were probably first hypothesized in 1845 by the French neurologist Jean Esquirol, and the theory was later refined by Swiss psychiatrist Eugen Bleuler in the 19th century.[2] Given the systemic

effects of SARS-CoV-2, which are well-reviewed in literature, it is certainly plausible that the coronavirus could, directly or indirectly, affect the brain and the central nervous system. However, in this case, we are mindful that the observed association in this report could also be fortuitous as this is a small sample of 3 individual cases, and in terms of epidemiological evidence, the onset of first-episode psychosis in the 30s is not uncommon. In a study of 555 individuals with first-episode psychosis, the median age of onset was found to be 27.3 years (interquartile range: 21.2, 36.5).[3]

Second, the authors appropriately mentioned the possibility of a stress-related trigger from the COVID-19 pandemic in psychiatrically vulnerable individuals. We recognise that all 3 of the patients presented had a prior psychiatric diagnosis: patient 1 and patient 2 both had a prior diagnosis of panic disorder while patient 3 had opioid use disorder, on methadone maintenance. Anxiety is the mother of psychopathology in predisposing, precipitating, and perpetuating symptoms. All three patients also appeared to respond to clonazepam, lorazepam, and low doses of quetiapine, which all exert anxiolytic effects.

In terms of their physical symptoms (or the lack thereof) and stable hemodynamics, there was no evidence of excessive immunologic response. Interestingly, Esquirol himself was also of the view that some mental illnesses may be caused by emotional disturbances rather than by organic brain damage.[2] If we accept the prevailing biopsychosocial model of mental illness, psychosis is likely to have multiple causes, including genetic, neurobiological, psychological, and environmental factors. Psychiatric manifestations may be the indirect ("reactive" stress) result of this pandemic rather than the direct pathophysiological effects of the virus.

Third, the classification of acute-onset psychotic disorders is rather inexact, and although the authors reported that the patients did not verbalise preoccupation with COVID-19, this may be difficult to elicit from the patients when they are in an acute agitated and psychotic state.

Fourth, clinically, the elevations in C-reactive protein (0.6–1.9 mg/dL) were relatively minor and could well be within normal variations. "Normal" C-reactive protein levels have not been rigorously studied or defined, and the levels are known to vary with population, age, and ethnicity.[4,5] To play devil's advocate, we know that struggling against restraints, physical agitation, or other common events could all result in an elevated C-reactive protein. Owing to the highly variable causality, a single C-reactive protein value at admission lacks clinical utility and is difficult to interpret in isolation.

Last but not least, it would be of value to follow up these patients and their symptoms longitudinally. Outcomes of patients with first-episode psychosis are known to be heterogeneous, ranging from complete recovery to recurrent and resistant psychotic symptoms. This may give us new insights beyond that of a "snapshot" picture or cross-sectional mental state examination. The SARS-CoV-2 is undoubtedly a novel virus, and there is much to be learnt from it—and it does surprise us sometimes.

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A Response to Nejad S et al.: Phenobarbital for Acute Alcohol Withdrawal Management in Surgical Trauma Patients—A Retrospective Comparison Study

TO THE EDITOR: While we appreciate the efforts of Nejad et al. to study the treatment of alcohol withdrawal in patients with trauma, we caution readers about the interpretation of their results. First, the readers are misinformed about the modern mortality of alcohol withdrawal, which should be close to nil with appropriate therapy.[1] Next, Nejad's comparator uses the wrong regimen of an inferior benzodiazepine. In their seminal work, Saitz et al.[2] demonstrated the superiority of symptom-triggered benzodiazepine therapy over the fixed interval dosing used here. In addition, benzodiazepines with long-acting metabolites such diazepam or chlordiazepoxide offer many theoretical and practical advantages for most patients.[2,3] Furthermore, the benzodiazepine-treated patients in this report were given additional medications (such as neuroleptics) which are not only inadequate to treat withdrawal but potentially harmful.[4]

There are clear difficulties with retrospective data analyses, the most important of which is a lack of a true control group. Other than the provided mean doses received in each group, there are no data concerning adherence to the regimen chosen for each patient. Furthermore, while the phenobarbital protocol includes tapering instructions over 7 days, no such information is provided for the benzodiazepine regimen. Likewise, the patients studied had alcohol use disorder and were not necessarily suffering from withdrawal at the initiation of therapy. Because it is well accepted that not all patients with alcohol use disorder progress to withdrawal,[5] it is unclear whether Nejad is providing preventive therapy or actual treatment.

Thus, even if this were a randomized controlled trial and we accepted the results, we would contend that phenobarbital is equivalent or superior to an inadequate regimen of a less than ideal benzodiazepine that is supplemented by potentially injurious medications. Although we applaud their efforts to further this discussion, the only conclusion is that a well-designed and implemented double-blind randomized controlled trial is necessary to answer the question at hand. Pending that answer, symptom-triggered benzodiazepine therapy has the strongest support in evidence-based medicine, and there is no reason that this therapy would not be appropriate in patients with trauma.

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