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Response to 'Neuro-COVID due to the response against the virus' Letter

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To the Editors

I would like to thank Finsterer et al. [1] for their interest and feedback.

As stated in my article [2], neurological complications observed during or after the course of COVID-19 are consequences of direct mechanisms of SARS-CoV-2 penetration to the nervous system, or indirect mechanisms, as for example being triggered by the virus inflammatory response and immune dysfunction. Finsterer et al. propound a tertiary mechanism comprising neurotoxic or myotoxic side effects of anti-COVID drugs as well as drugs for comorbidities applied to COVID-19 patients. This finding may help to explain, at least in part, the differences in clinical manifestations and outcomes of SARS-CoV-2 infections.

In my opinion, this is a very valuable addition to our knowledge regarding COVID-19 neuropathogenesis and should be taken into consideration by physicians who prescribe medicine in the treatment of a SARS-CoV-2 infection. I am very grateful to Professor Finsterer for raising the issue of the iatrogenic effect of the drugs used during COVID-19 treatment, and I assure your readers that I will particularly highlight this subject in my future publications.

Finsterer et al. "do not agree that cerebral hypoxia is a mechanism underlying neuro-COVID." They argue that "most SARS-CoV-2 infected patients with pneumonia develop dyspnoea, which is usually adequately and quickly managed by the treating physicians."

I would like to point out that there are SARS-CoV-2 infected patients with extremely low blood-oxygen levels who do not complain of dyspnoea, and who are relatively well. This phenomenon, so called 'happy hypoxia' [3], can cause some patients with COVID-19 to delay their contact with physicians and thus the initiation of their treatment.

Moreover, I must point out that some studies have demonstrated the presence of stigmata of hypoxic brain damage. Microscopic examination of 18 brain specimens performed by Solomon et al. showed acute hypoxic injury in the cerebrum and cerebellum in all of the patients, with loss of neurons in the cerebral cortex, hippocampus, and cerebellar Purkinje cell layer. They did not find any encephalitis or other specific brain changes that could be shown to be related to the virus [4]. Neuropathological examination of four brains performed by Kantonen et al. showed various hypoxia-associated neuropathological features in COVID-19 patients, including severe ischaemic injury, abundant microhaemorrhages, and enlarged perivascular spaces, most pronounced in the white matter and deep grey matter [5]. Hypoxia was also postulated as a factor in charge of leukoencephalopathy with microhaemorrhages found in a patient with severe COVID-19 [6].

Therefore, based on these findings, I proposed hypoxia to be one of the possible mechanisms, although I did not extrapolate on this subject due to the constraints regarding word count for published articles.

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