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LETTER TO THE EDITOR

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COVID-19 manifesting as syncope

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Letter to the Editor

With interest we read the article by Canetta et al. about a study of 35 patients infected with SARS-CoV-2 who manifested initially with a syncope 1. It was speculated that SARS-CoV-2 led to internalisation of angiotensin-converting enzyme-2 (ACE2) receptors in the solitary tract nucleus (NTS) or other midbrain nuclei impairing baroreflexes and chemoreceptor responses and thus inhibiting reflectory tachycardia during hypocapnic hypoxemia 1 . We have the following comments and concerns.

The main shortcoming of the study is that causes of syncope other than orthostatic were not considered. Syncopes are multicausal and evaluation of the data should include determination of the exact cause of each syncope reported 2. Missing in this respect are the exclusion of cerebral causes by MRI, electroencephalography (EEG), and carotid ultrasound (CD) and by exclusion of causes due to affection of the peripheral nervous system (PNS) by nerve conduction studies, electromyography, or autonomic testing in all 35 patients included.

Another shortcoming is that the medication the 35 patients were taking prior to the syncope was not reported in detail. Knowing all drugs a patient with a syncope was regularly taking is crucial as drugs may

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have syncopes as a side effect 3.

Since the infection with SARS-CoV-2 may lead to hypercoagulability [4], it is conceivable that syncope may be due to thrombus formation intraventricularly or intra-arterially. Thus, it is crucial to know the echocardiographic and CD findings of all 35 included patients with a syncope.

Since the infection with SARS-CoV-2 can trigger the development of Guillain-Barre syndrome (GBS) [5] and since GBS may lead to autonomic disturbances, it is conceivable that syncope was due to polyradiculitis at least in some of the patients. This is why appropriate investigations (nerve conduction studies (NCSs), cerebro-spinal fluid (CSF) investigations) should have been carried out.

Furthermore, nine patients included had a history of traumatic brain injury (TBI) and another patient a history of subdural hematoma (SDH). We should know if the history in these patients was positive for seizures.

Missing is the family history, particularly if any of the first-degree relatives of the 35 included patients had syncopes, arrhythmias, cardiomyopathy, or epilepsy.

It is also unclear if there were recurrent syncopes and if different causes for the different syncopes were considered and appropriate differentials excluded.

The history of the 35 patients with a syncope at onset is insufficient. We should know in how many of the patients the syncope was associated with a secessus, postsyncopal myalgias, confusion, or with elevation of the creatine-kinase (CK). Though the latency between syncope and admission was registered it remains unclear how many of the patients had the syncope prior to admission and how many after admission. Knowing the exact time point of the syncope is crucial as not only the circumstances are different between pre- and post admission syncopes but also the medication and the disease course.

Blocking ACE2 receptors to prevent invasion of the virus into various cells is not conceivable given the statement that “loss of ACE2 receptors on the cell surface could precipitate cardiovascular, renal, or cerebral disease” 1 .

We also should know if any of the 35 patients had developed exsiccosis prior to the syncope.

Lastly, the NTS is not located in the midbrain but the rhombencephalon.

Overall, the interesting study by Canetta et al. has a number of shortcomings which should be addressed before drawing final conclusions. There is a need for more profound investigations of the syncopy group and exclusion of cerebral, arterial, and nerval causes of syncope before attributing a syncope to orthostasis.

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