Study of sympathetic skin response in patients with COVID-19 infection

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Abstract
Objectives Many articles hypothesized the potential role of autonomic nervous system in the pathogenesis and outcome of COVID-19 infection. Several studies reported both central and peripheral nervous system involvement in COVID-19 as well. Up to our knowledge, there is no study evaluating whether this virus could invade the autonomic nervous system affecting its function adversely. Sympathetic skin response (SSR) has long been used as a method of evaluating the autonomic nervous system. Regarding the importance of the autonomic nervous system in hemostasis and wide consequences of COVID-19 infection, we designed this study to evaluate the autonomic nervous system function in patients recovered from COVID-19 compared with normal population who are not yet infected by this virus by the means of SSR.

Methods This case–control study included 70 patients surviving COVID-19 who met the inclusion and exclusion criteria that went under SSR. The data gathered were compared with those without the history of any symptoms attributable to COVID-19 during the pandemic.

Results There was a correlation between COVID-19 infection and abnormal SSR (p value < 0.0001) with the most effect on the latency prolongation of the action potential recorded from the median nerve at palms (effect size: right: 3.90, left: 3.69). Moreover, the greater severity of the disease correlated with more abnormality of parameters recorded by SSR technique.

Conclusions Abnormal SSR parameters could be a good indicator of autonomic nervous system involvement in patients with COVID-19 infection. It might be a predictor of disease severity, clinical outcomes and prognosis as well.

Keywords Autonomic nervous system · COVID-19 · Sympathetic skin response

Introduction
The pandemic viral illness COVID-19 is especially life-threatening in the elderly and in those with chronic medical conditions. The heightened risk in these patients may involve activation of the “extended autonomic system” (EAS). Traditionally, the autonomic nervous system has been viewed as consisting of the sympathetic nervous system, the parasympathetic nervous system, and the enteric nervous system. Over the past century, neuroendocrine and neuroimmune systems have expanded the meaning of “autonomic.” A hierarchical brain network—the “central autonomic network”—regulates these systems. Acute, coordinated alterations in homeostatic settings can be crucial for surviving stressors such as traumatic hemorrhage, asphyxiation, and sepsis [1]. However, intense or long-term EAS activation may cause harm. While required for appropriate responses in emergencies, EAS activation in the setting of chronically decreased homeostatic efficiencies (dyshomeostasis) may reduce thresholds for induction of destabilizing, lethal vicious cycles. Testable hypotheses derived from these concepts are that biomarkers of EAS activation correlate with clinical and pathophysiologic data and predict outcome in COVID-19 and that treatments targeting specific abnormalities identified in individual patients may be beneficial. Quantitative indices of EAS activation such as increases in plasma catecholamines, ACTH, AVP, and AII levels could provide...
valuable biomarkers by which to test whether EAS involvement explains the high mortality associated with aging and chronic disorders of regulation in COVID-19 [2]. To date, no reports have described levels of any of these compounds in COVID-19 patients. A recent correspondence noted associations of both elevated neutrophil/lymphocyte ratios and cortisol levels with decreased survival in COVID-19 patients [3]. There are several articles hypothesizing the potential role of autonomic nervous system in the pathogenesis and outcome of COVID-19 infection as well as both central and peripheral nervous system involvement by this virus [4–10]. The close association of the autonomic dysfunction with disease severity and significant pulmonary complications such as fibrosis was reported in patients with COVID-19 involvement [11]. Up to our knowledge, there are limited studies evaluating whether this virus could invade the autonomic nervous system affecting its function adversely. Sympathetic skin response (SSR) has long been used as a method of evaluating the autonomic nervous system [12]. Many studies showed the correlation between abnormal SSR and acute or chronic medical conditions such as electrical burn injury, osteoporosis as one of its late complications or in postmenopausal women with the higher frequency of vasomotor symptoms, urinary incontinence in partial spinal cord injury and so on [13–17]. Regarding the importance of the autonomic nervous system in maintaining the hemostasis and the very wide consequences of COVID-19 infection and the evidence of nervous system involvement in this disease, we designed this study to evaluate the autonomic nervous system function in patients recovered from COVID-19 compared with normal population who are not yet infected by this virus by the means of SSR.

Methods

Seventy patients surviving COVID-19 were recruited to this study and data gathered were compared with other 70 participants without the history of any symptoms attributable to COVID-19 during the pandemic (between July and October 2021). None of the participants had been received COVID-19 vaccination since that time. The Medical Ethics Committee of Shiraz University of Medical Sciences (SUMS) approved this study with the reference number “IR.SUMS.MED.REC.1400.099”. Inclusion criteria for the case group was positive COVID-19 PCR test 3 months before taking part in the study and for the control group negative history of a positive COVID-19 PCR test, or the symptoms of fatigue, body pain, chills and fever, any change in the hearing or smell senses. The range of the age for both groups was 18–70 years old. Exclusion criteria included positive history of all conditions resulting in peripheral or central neuropathy including diabetes mellitus (DM) before COVID-19 infection, consumption of medications interfering with sympathetic activity, any scar or skin lesion that interfere SSR recording at the palms and soles. We precisely described the purpose and the methodology of this research for each person to meet their interest in following the process. The written informed consent was signed by each participant. After recording the routine nerve conduction study (NCS), from different nerves, to rule out any peripheral neuropathy which interferes with our analysis, the SSR was recorded from four limbs of each included case. SSR was tested using a synergy multilinker EMG machine (Medelec Synergy electromyography instrument, Viasys Healthcare UK, Manor Way, Surrey, UK) in an ambient room with controlled temperature, lighting, and noise. The skin temperature was maintained above 31°C centigrade. The patients were in supine positions with closed eye albeit awake and alert. The active surface recording electrodes were attached to the center of the palms and soles and the reference electrodes on the dorsum of the ipsilateral hands and feet with a ground electrode on the distal part of the radial styloid and lateral malleolus, respectively. The median and tibial nerves were stimulated at the ipsilateral distal wrist crease and posterior to the medial malleolus, respectively. Each site was stimulated for ten times at an interval of 30 s to prevent habituation and the average was calculated. All of stimuli that failed to respond were deleted before averaging. The stimuli pulse duration was 0.2 ms with the intensity of 20–45 mA. Band pass filter set at 0.5–2000 Hz and sensitivity at 100–200 mV/division. The onset latency and peak to peak amplitude were measured. Normal ranges of the onset latency studies for upper and lower limbs were considered 1.39 ± 0.07 and 1.88 ± 0.11 s (S) as well as for amplitude of the action potentials recorded from upper and lower limbs, the ranges of 806 ± 322 and 640 ± 276 microvolt (µV) were considered normal, respectively [18].

In this study four grades of severity were determined for the COVID-19 symptoms as follows:

1. Just very mild symptoms that did not interfere with the activity of daily living.
2. Mild symptoms that resulted in complete bed rest but with good O2 saturation that excludes the need for hospital admission.
3. Moderate symptoms and signs that resulted in hospital admission.
4. Severe symptoms that resulted in ICU admission.

The statistical analysis was performed using SPSS 18. The number of participants (70 in each group) was determined using Med–Calc software with $\alpha = 0.05$, $\beta = 0.2$ and $r = 0.35$. The correlation between quantitative variables (SSR parameters) was tested using Pearson correlation coefficient or Spearman correlation. $p < 0.05$ was considered
statistically significant. We used Chi-square test for detection of the correlation between qualitative data such as sex. We categorized patients according to severity of their COVID-19 symptoms and used Kruskal–Wallis test as a second comparison for correlation of multiple levels of disease severity and abnormality of SSR parameters. Post hoc analysis was used for multiple comparison by the Mann–Whitney test as well.

Results

In this case-control study, a total of 140 participants with age between 22 and 70 years and mean age of 51.79 ± 10.29 years (51.9 ± 10.3 for case group as well as 51.6 ± 10.3 for control group) were included. Each group included 40 males and 30 females. There were no statistically significant differences between both case and control groups according to demographic variables such as age and sex (p value of 0.86 and 0.90, respectively).

The mean latency of the action potential recorded from median and tibial nerves of both right and left sides in the case group had statistically significant increase compared to the control group with p value < 0.0001 (median nerve mean latency (S) in case group: left: 2.40 ± 0.328, right: 2.40 ± 0.314 and in control group: left: 1.37 ± 0.237, right: 1.35 ± 0.228. Tibial nerve mean latency (S) in case group: left: 2.76 ± 0.312, right: 2.76 ± 0.305 and in control group: left: 1.79 ± 0.234, right: 1.81 ± 0.255) (Table 1 and Fig. 1).

Furthermore, the mean amplitude of the action potential recorded from bilateral palms and soles (median and tibial nerves) in the case group was significantly lower than related mean range in the control group with p value < 0.0001 (median nerve mean amplitude (µV) in case group: left: 234.12 ± 77.66, right: 237.06 ± 77.50 and in control group: left: 470.71 ± 70.34, right: 473.42 ± 66.85. Tibial nerve mean amplitude (µV) in case group: left: 216.86 ± 93.26, right: 214.31 ± 93.96 and in control group: left: 429.42 ± 62.75, right: 430.14 ± 58.25) (Table 1 and Fig. 1).

To show which parameter of the SSR had more correlation with COVID-19 disease, we calculated the effect size for each mean latency and amplitude of the action potential recorded from both hands and feet. The result showed the mean latency of the median nerve action potential had the most effect size (right: 3.90, left: 3.69) as compared to the latency of the action potentials recorded from tibial nerves bilaterally as well as mean amplitudes (mean median nerve latency > mean tibial nerve latency > mean median nerve amplitude > mean tibial nerve amplitude) (Table 1).

In the case group, 4.3% of participants have experienced very mild COVID-19 disease while 12.9, 52.9 and 30% of them had mild, moderate and severe disease during 2–3 months prior to this study, respectively. Among 21 patients with previous recent history of severe disease, seven cases had absent action potentials recorded by SSR technique. There was statistically significant difference among different levels of the severity of the disease and the mean latency as well as the mean amplitude of the action potential recorded from both palms and soles. The more severe disease the patient had experienced, the greater increase in latency or decrease in amplitude of the action potentials recorded from median and tibial nerves was detected (Table 2 and Fig. 2).

In summary, this study showed there was a correlation between COVID-19 infection and abnormal SSR parameters with the most effect on the latency prolongation of the median nerve recorded action potentials from the palms. Moreover, the greater severity of the disease correlated with more abnormality of parameters recorded by SSR technique.

Discussion

The main presentation of SARS-CoV-2 infection as the etiology of the recent coronavirus disease pandemic (COVID-19) is pulmonary system involvement [19]. However, studies showed it can be a neurotropic organism involving both central (CNS) and peripheral nervous system (PNS) as well. They reported headache and dizziness as its most common

| SSR parameters | Case group | Control group | p value | Effect size |
|----------------|------------|---------------|---------|-------------|
| L median N mean latency (S) | 2.40 ± 0.328 | 1.37 ± 0.237 | < 0.0001 | 3.69 |
| R median N mean latency (S) | 2.40 ± 0.314 | 1.35 ± 0.228 | < 0.0001 | 3.90 |
| L median N mean amp (µV) | 234.12 ± 77.66 | 470.71 ± 70.34 | < 0.0001 | 3.19 |
| R median N mean amp (µV) | 237.06 ± 77.50 | 473.42 ± 66.85 | < 0.0001 | 3.26 |
| L tibial N mean latency (S) | 2.76 ± 0.312 | 1.79 ± 0.234 | < 0.0001 | 3.55 |
| R tibial N mean latency (S) | 2.76 ± 0.305 | 1.81 ± 0.255 | < 0.0001 | 3.44 |
| L tibial N mean amp (µV) | 216.86 ± 93.26 | 429.42 ± 62.75 | < 0.0001 | 2.67 |
| R tibial N mean amp (µV) | 214.31 ± 93.36 | 430.14 ± 58.25 | < 0.0001 | 2.77 |

L left, R right, N nerve, Amp amplitude, µV microvolt
CNS involvement while taste and smell dysfunctions were the main PNS complications [20]. Multiple mechanisms to explain the pathogenesis of such above nervous system involvement were suggested like direct invasion to the CNS or PNS, hematogenous pathways, or the release of interleukin-6 resulting neurological and respiratory injuries as well as the expression of angiotensin converting enzyme 2 in the nervous system [21–23].

Moreover, recent studies showed the autonomic nervous system involvement in patients with COVID-19 infection during both acute and chronic phases. The pathogenesis may be due to the virus invasion, immune-mediated disruption of the autonomic fibers or interaction between their released neurotransmitters and inflammatory cytokines resulting in temporary or long-term orthostatic intolerance manifestations [24, 25]. The heart rate variability (HRV) was reported as the most valuable non-invasive test for evaluation of the function of the autonomic nervous system. Bail et al. showed it can be a good predictor for short and long-term clinical outcomes as well as respiratory sequelae of COVID-19 infection such as pulmonary fibrosis [11]. SSR is another simple non-invasive method to evaluate the unmyelinated axon involvement from autonomic nervous system by mediating sudomotor activity and changes in the voltage calculated.

![Fig. 1](image.png)

**Fig. 1** Correlation between mean values of the latency (S) and amplitude (µV) (from both sides of the hands and legs) in case and control groups for this sympathetic skin response (SSR) study. SSR: sympathetic skin response, Lt: left, Rt: right, A: amplitude, L: latency.
from the surfaces of the skin [12]. A number of studies have shown the importance of abnormal SSR in several medical conditions. For example, Roshanzamir et al. showed it could be a good predicting factor for the occurrence of the symptoms associated with autonomic dysfunction in patients after electrical burn injury. She concluded abnormal SSR could be an acceptable predictor for bone loss in such patients as well [26, 27]. In this study, we showed there was a statistically significant correlation between COVID-19 infection and abnormal SSR parameters \((p\text{ value} < 0.0001)\) with the most effect on the latency prolongation of action potentials recorded from median and tibial nerves at palms and soles with the preference of the first one (effect sizes: right palm: 3.90, left palm: 3.69, right sole: 3.44 left sole: 3.55). The result revealed the greater severity of the disease correlated with more abnormality of parameters recorded by SSR test as well. Pan et al. claimed HRV had significant association with the severity of the COVID-19 disease in such a way that the more disease severity, the greater autonomic dysfunction as well as abnormal HRV parameters recording by 24 h dynamic electrocardiography were detected. Therefore, it might be used in monitoring of courses of the illness and could be a good predictor for estimating the disease prognosis as well [24]. Based on our study findings, the amount of abnormal SSR parameters could be another good predictor of disease severity like HRV indicators in patients with COVID-19 disease. Yet, there were few studies evaluated SSR abnormality in patients developed this infection. Thus, this study would be one of the first researches considering this issue in the literature. Papadopoulou et al. conducted a case–control study to evaluate SSR to show if autonomic dysfunction might have association with long COVID-19 syndrome which was defined as ongoing symptoms usually for at least 3 months. The results showed there were

| SSR parameter            | Disease severity correlation | \(p\text{ value}\) |
|--------------------------|------------------------------|-------------------|
| Mean median nerve latency| Severe and very mild         | < 0.0001         |
|                          | Severe and mild              | < 0.0001         |
|                          | Severe and moderate          | < 0.0001         |
| Mean median nerve amplitude| Severe and very mild        | < 0.0001         |
|                          | Severe and mild              | < 0.0001         |
|                          | Severe and moderate          | 0.003            |
|                          | Moderate and very mild       | 0.047            |
| Mean tibial nerve latency| Severe and very mild         | < 0.0001         |
|                          | Severe and mild              | < 0.0001         |
|                          | Severe and moderate          | 0.031            |
|                          | Moderate and very mild       | 0.039            |
|                          | Moderate and mild            | 0.022            |
| Mean tibial nerve amplitude| Severe and very mild        | 0.025            |
|                          | Moderate and very mild       | 0.009            |
|                          | Moderate and mild            | 0.010            |

Fig. 2 Pairwise comparison test between different levels of severity and mean sympathetic skin response (SSR) parameters (latency and amplitude) recorded from median and tibial nerves (A severity and mean SSR amplitude of tibial nerve, B severity and mean SSR amplitude of median nerve, C severity and SSR latency of median nerve, D severity and SSR latency of tibial nerve)
significant longer latencies for cases with long COVID-19 in contrast to control group (healthy participants) (mean latencies in the upper limbs: 1.28 ± 0.24 s vs. 1.49 ± 0.19 s, \( p=0.010 \) in control group and patients, respectively, and in the lower limbs: 1.8 ± 0.31 s vs. 2.09 ± 0.34 s, \( p=0.014 \)). Amplitudes did not have difference between groups [28].

This research was somewhat similar to our study in the way that both showed the significant correlation between SSR abnormalities and autonomic dysfunction in such patients in contrast to control group. However, they had some differences. For example, our study showed there were significantly lower amplitudes of the action potentials recorded from both upper and lower limbs in the case group with different disease severity levels in contrast to the control group. We conducted a study with larger sample size as well. Another study by Ser et al. revealed neuropathic and autonomic complaints in more than one-third of patients with long COVID-19 and abnormal cutaneous silent period suppression index but normal SSR parameters [29]. One of the reasons for the difference between the results of this study and our research could be that the SSR evaluation in the mentioned study was performed on a limited number of patients who had neuropathic or autonomic symptoms after COVID-19 infection (14 out of 38 cases). Failure to investigate SSR parameters in a longer follow-up, case–control design, lack of standardized clinical evaluation for signs and symptoms of the autonomic nervous system dysfunction seemed to be the main limitations of this study. Moreover, we did not individualize cases with clinical features of autonomic dysfunction from those without these manifestations after COVID-19 infection and then, compared the abnormalities of the SSR parameters with each other as well as control group. Inability to control stressful condition well during nerve conduction studies in some cases which had negative effect on SSR parameters could be another shortcoming of this study. According to a study conducted by Emad et al., the height and limb length had significant effects on SSR latency [30]. Unfortunately, we did not evaluate these factors in this study. Whether the severity of the disease primarily caused the greater abnormality of the SSR parameters or the underlying autonomic dysfunction in our cases before developing COVID-19 infection predisposed them to experience more severe disease was still a controversial issue that needed more studies to know accurately.

Based on some studies, SSR abnormalities did not associate well with clinical features of autonomic nervous system dysfunction [12]. However, it could be a reliable indicator for conditions involving unmyelinated axons according to our interesting results in this study. It could be an alternative or adjunct method for HRV in evaluation of autonomic system in COVID-19 survivors so that it might be as a good predictor of clinical outcomes, disease severity and prognosis. Nevertheless, further evaluation in future is suggested.

### Conclusion

We showed there was a correlation between COVID-19 infection and abnormal SSR parameters with the most effect on the latency prolongation of the action potentials recorded from median and tibial nerves at the palms and soles with the preference of the median nerve. Moreover, the greater severity of the disease correlated with more abnormality of parameters recorded by SSR technique so that abnormal SSR might be a predictor of COVID-19 disease severity, clinical outcomes and prognosis like HRV indicators.

### Conflict of Interest

No potential conflict of interest was reported by the authors.

### References

1. Cannon WB (1914) The emergency function of the adrenal medulla in pain and the major emotions. Am J Physiol 33(2):356–372
2. Goldstein DS (2020) The extended autonomic system, dyshomeostasis, and COVID-19. Clin Auton Res 30(4):299–315
3. Tan T, Khoo B, Mills EG, Phylactou M, Patel B, Eng PC et al (2020) Association between high serum total cortisol concentrations and mortality from COVID-19. Lancet Diabetes Endocrinol 8(8):659–660
4. Del Rio R, Marcus NJ, Inestrosa NC (2020) Potential role of autonomic dysfunction in COVID-19 morbidity and mortality. Front Physiol 11:561749
5. Porzionato A, Emmi A, Barbon S, Boscolo-Berto R, Stecco C, Stocco E et al (2020) Sympathetic activation: a potential link between comorbidities and COVID-19. FEBS J 287(17):3681–3688
6. Bergmann CC, Lane TE, Stohlman SA (2006) Coronavirus infection of the central nervous system: host-virus stand-off. Nat Rev Microbiol 4(2):121–132
7. Iadecola C, Anrather J, Kamel H (2020) Effects of COVID-19 on the nervous system. Cell 183(1):16–27
8. Nampoothiri S, Saeude F, Ternier G, Fernandois D, Coelho C, Imerbon M et al (2020) The hypothalamus as a hub for SARS-CoV-2 brain infection and pathogenesis. bioRxiv. https://doi.org/10.1101/2020.06.08.139329
9. Politi LS, Salsano E, Grimaldi M (2020) Magnetic resonance imaging alteration of the brain in a patient with coronavirus disease 2019 (COVID-19) and anosmia. JAMA neurol 77(8):1028–1029
10. Song E, Zhang C, Israelov B, Lu P, Weizman O-E, Liu F et al (2020) Neu roinvasive potential of SARS-CoV-2 revealed in a human brain organoid model. bioRxiv. https://doi.org/10.1101/2020.06.25.169946
11. Bail T, Zhou D, Yuzhanjiang F, Wang D, Zhang D, Liu X et al (2022) Alteration of the autonomic nervous system is associated with pulmonary sequelae in patients with COVID-19 after six months of discharge. Front Physiol 12:805925. https://doi.org/10.3389/fphys.2021.805925
12. Shahani BT, Halperin J, Boulo P, Cohent J (1984) Sympathetic skin response—a method of assessing unmyelinated axon
13. Ashraf A, Mohammadi A, Roshanzamir S, Ayaz M, Tolide-ie H, Zafarghasempoor M (2012) Sympathetic skin response in electrical burn injury. Burns 38(2):232–235

14. Emad R, Zafarghasempour M, Roshanzamir R (2013) Sympathetic skin response in incomplete spinal cord injury with urinary incontinence. Ann Indian Acad Neurol 16(2):234–238

15. Roshanzamir R, Dabbaghmanesh M, Dabbaghmanesh A, Nejati S (2016) Autonomic dysfunction and osteoporosis after electrical burn. Burns 42(3):583–588

16. Ashraf A, Roshanzamir R, Bemana G, Mohammadi A, Jahani N, Naseri M (2015) Sympathetic skin response and vasomotor symptoms in postmenopausal osteoporotic women. Int J Community Based Nurs Midwifery 3(3):227–233

17. Satari S, Roshanzamir R (2016) A study of sympathetic skin response in burns with different mechanisms. J Biol 5(6):91–94

18. Amato A, Dumitru D (2002) Special nerve conduction techniques. In: Dumitro D, Amato AA, Zwarts JM (eds) Electrodiagnostic medicine. 2nd Philadelphia: Hanley & Belfus, Philadelphia, p P252

19. Bazrafshan H, Mohamadi Jahromi LS, Parvin R, Ashraf A (2022) A case of Guillain-Barre syndrome after the second dose of AstraZeneca COVID-19 vaccination. Turk J Phys Med Rehabil 68(2):295–299

20. Mohamadi Jahromi LS, Farour HR, Etminan H, Parvin R (2022) Ulnar mononeuropathy associated with COVID-19 infection, a case report. JRSR 9(2):93–96

21. Malekmohammad M, Hashemian S, Afsar BM, Jammati H (2020) Neurological manifestations of COVID-19: a case report. Tanaffos 19(2):160–164

22. Hutchins KL, Jansen JH, Comer AD, Scheer RV, Zahn GS, Capps AE et al (2020) COVID-19- associated bifacial weakness with paresthesia subtype of Guillain-Barre’ syndrome. AJNR Am J Neuroradiol 41(9):1707–1711

23. Khatoon F, Prasad K, Kumar V (2020) Neurological manifestations of COVID-19: available evidences and a new paradigm. J Neurovirol 26(5):619–630

24. Pan Y, Yu Z, Yuan Y, Han J, Wang Z, Chen H et al (2021) Alteration of autonomic nervous system is associated with severity and outcomes in patients with COVID-19. Front Physiol 12:630038. https://doi.org/10.3389/fphys.2021.630038

25. Dani M, Dirksen A, Taraborrelli P, Torocastro M, Panagopoulos D, Sutton R et al (2021) Autonomic dysfunction in ‘long COVID’: rationale, physiology and management strategies. Clin Med 21(1):63–67

26. Roshanzamir S, Dabbaghmanesh A, Ashraf A (2014) Predicting post-electrical injury autonomic dysfunction symptom occurrence by a simple test. Burns 40(4):624–629

27. Roshanzamir S, Keshavarz E (2020) Sympathetic skin response impairment: a good predictor of bone loss in electrical burn victims. Burns 46(2):394–399

28. Papadopoulou M, Bakola E, Papapostolou A, Stefanou M, Gaga M, Zouvelou V, et al (2022) Autonomic dysfunction in long-COVID syndrome: a neurophysiological and neurosonology study. J Neurol 10:1–2

29. Ser M, Calikusu F, Tanriverdi U, Abbaszade H, Hakyemez S, Balkan I (2022) Autonomic and neuropathic complaints of long-COVID objectified: an investigation from electrophysiological perspective. Neurol Sci. https://doi.org/10.1007/s10072-022-06350

30. Emad R, Roshanzamir S, Dabbaghmanesh A, Zafarghasempoor M, Eivazlou H (2016) Inclusion of height and limb length when interpreting sympathetic skin response. Iran J Med Sci 41(1):48–52

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