

Mini Review

Open Access

Autonomic and Hemodynamic Mechanisms in Chronically Low Blood Pressure

Stefan Duschek¹, Rainer Schandry² & Gustavo A. Reyes del Paso³

¹UMIT Tirol – University of Medical Sciences and Technology, Hall in Tirol, Austria

²University of Munich, Germany

³University of Jaén, Spain

Article Info

Article Notes

Received: May 18th, 2025

Accepted: June 10th, 2025

*Correspondence:

*Stefan Duschek, UMIT Tirol – University of Medical Sciences and Technology, Eduard-Wallnöfer-Zentrum 1, 6060 Hall in Tirol, Austria, Email. stefan.duschek@umit-tirol.at

© 2025 Duschek S. This article is distributed under the terms of the Creative Commons Attribution 4.0 International License.

Keywords

Hypotension
Blood Pressure
Autonomic Control
Cardiac Output
Baroreflex
Cerebral Blood Flow

Abstract

In addition to physical complaints like fatigue, reduced drive, dizziness, and cold limbs, chronic low blood pressure (hypotension) is associated with cognitive impairments and negative mood states. This review is concerned with autonomic and hemodynamic factors implicated in the origin of chronic hypotension and associated symptoms. There is strong evidence of reduced stroke volume and cardiac output, and prolonged pre-ejection period, at rest, during mental challenge and during sleep, indicating reduced myocardial contractility due to low beta-adrenergic drive in chronic hypotension. While studies investigating parasympathetic cardiac control and alpha-adrenergic vascular influences revealed inconsistent results, increased sensitivity of the cardiac baroreflex is well-supported in chronic hypotension. Moreover, cardiovascular stress reactivity is reduced. Low myocardial contractility constitutes the main factor in the manifestation of chronic hypotension; increased responsiveness of the baroreflex may play an additional role. Diminished cardiac output leads to insufficient blood supply of the organism and symptoms like low skin temperature and cold limbs; moreover, low beta-adrenergic activity is involved in hypotension-related mood impairments. Deficits in systemic hemodynamics are partly transferred to brain perfusion, expressed as lower cerebral blood flow at rest and impaired blood flow adjustment during cognitive activity in chronic hypotension. Blunted cardiovascular reactivity reflects reduced autonomic flexibility, limiting the adaptive resources of the organism.

Chronic hypotension

Chronic hypotension refers to an enduring state of inappropriately low blood pressure independent of further pathological conditions¹. It is frequently associated with complaints including fatigue, reduced drive, dizziness, headaches and cold limbs, which significantly reduce wellbeing and quality of life^{2,3}. The chronic form is distinguished from orthostatic hypotension (i.e., circulatory problems when assuming an upright position) and secondary (or symptomatic) hypotension, which occurs, for example, because of blood loss or medication use⁴. Although there is no generally recognized definition of chronic hypotension, in basic and clinical research, systolic baseline values of around 100 mmHg or lower are frequently used as an upper limit⁵⁻⁸.

In contrast to elevated blood pressure, which constitutes a major risk factor of cardiovascular diseases, chronic hypotension is commonly not regarded as a clinical condition requiring treatment¹. Moreover, in specific populations such as adolescents, especially females, or endurance athletes, low blood pressure values are frequent and, by definition, not clinically relevant^{9,10}. However, low

blood pressure confers increased risk in pregnancy, and longitudinal studies have shown associations with brain atrophy and cognitive decline in the elderly¹¹⁻¹³. Clinical research revealed a connection of hypotension with elevated risk of chronic kidney disease and worse outcomes in affected patients^{14,15}; and low blood pressure has been associated with increased occurrence of glaucoma^{16,17}. In clinical practice, further medical examination and consideration of antihypotensive treatment may only be required in individuals with extreme symptom distress or risk of sequela; and it should be ruled out that the symptoms relate to an underlying disorder instead of hypotension itself. While adrenergic agents may be a treatment option, information about their beneficial effects remains scarce^{18,19}. The same applies to alternative therapeutic strategies, such as physical training and increased liquid intake⁹.

Cognitive and affective symptoms

Various studies have compared individuals with chronic hypotension with those with blood pressure in the normotensive range in terms of their cognitive performance. Individuals with chronic hypotension performed poorer, for example, on tasks of selective, divided and sustained attention^{20,21}, working and long-term memory²⁰⁻²² and higher cognitive functions, i.e. executive control^{22,23}. Pharmacological blood pressure elevation in chronic hypotension led to cognitive performance enhancement, illustrating the causal role of low blood pressure in these deficits^{24,25}. Studies using electroencephalography demonstrated smaller event-related potential amplitudes, which constitute neural correlates of the attentional impairments seen in chronic hypotension^{26,27}. Moreover, the application of functional transcranial Doppler sonography in chronic hypotension showed blunted cerebral blood flow modulations during tasks requiring attentional and more complex cognitive processes^{8,23,28,29}. Positive correlations between the alterations in cerebral hemodynamics and performance measures implicate impaired regulation of brain perfusion in hypotension-related cognitive deficits.

While the bodily complaints of chronic hypotension have been confirmed using symptom checklists^{2,29}, further studies demonstrated associations of low blood pressure with reduced mental wellbeing³, negative mood states²¹, elevated prevalence of depression³¹ and increased pain sensitivity³². A more recent study showed negative correlations of affective impairments and depressive symptoms with myocardial contractility in chronic hypotension, suggesting that low beta-adrenergic activity is involved in the origin of the mood impairments³⁰.

Physiological mechanisms in the manifestation of chronic hypotension

Not much is known about the etiology of chronic

hypotension. Current research supports a contribution of genetic factors to interindividual differences in tonic blood pressure³³. While there are concerted research efforts into the genetic mechanisms relevant to the genesis of elevated blood pressure^{34,35}, such research has not yet been conducted for chronic hypotension. Insufficient liquid intake and low body weight are considered as risk factors of chronic hypotension, pointing toward a role of lifestyle factors⁹. However, relevant empirical studies are still lacking.

Growing evidence suggests that autonomic nervous system dysregulation is involved in mediating the occurrence of chronic hypotension. Alterations in parasympathetic and sympathetic cardiovascular control, as well as aberrant baroreflex function, have been considered and are discussed in the following.

A. Parasympathetic cardiac control

Chronic hypotension is associated with a lower heart rate (HR), suggestive of increased parasympathetic outflow to the sinus node^{7,22,29,36}. Parasympathetic cardiac influence is commonly quantified by means of heart rate variability (HRV) analysis³⁷. Time domain analysis of interbeat intervals recorded during sleep revealed a higher root mean square of successive differences (RMSSD) in chronic hypotension³⁸. In contrast, frequency domain analysis of nocturnal HRV did not show a difference in spectral power in the high frequency band (i.e., respiratory sinus arrhythmia) between hypotensive and normotensive samples⁶. Findings in awake individuals also remain equivocal. While a recent study demonstrated higher RMSSD in chronic hypotension at rest and during cognitive stress⁷, earlier studies revealed negative results^{22,36}. Though future research might help to clarify the sources of the discrepancies between the findings, an essential etiological role of increased parasympathetic cardiac tone in chronic hypotension seems unlikely.

B. Sympathetic cardiovascular control

Using impedance cardiography, numerous studies revealed reduced stroke volume (SV) and cardiac output (CO), as well as a longer pre-ejection period (PEP), in chronic hypotension at rest, during cognitive challenge and during sleep^{6,22,36,38}. Similar results were obtained in studies in which SV was derived from continuous blood pressure recordings by means of the Modelflow method^{7,39,40}.

As SV is positively related to myocardial contractility, and the ventricles are mainly innervated by the beta-adrenergic system, the SV reduction indicates diminished cardiac sympathetic drive in chronic hypotension⁴¹. In this regard, it must be noted that the magnitude of SV to some extent depends on HR, which at rest and during light physical activity is mainly under parasympathetic

control³⁷. Higher HR implies lower ventricular preload, which, mediated by the Frank–Starling mechanism, results in lower contractility and SV⁴¹. However, as individuals with chronic hypotension display reduced instead of increased HR^{7,22,36}, and as lower HR is associated with higher SV, a role of HR in the SV reduction is not plausible. Nevertheless, differences in ventricular preload could be relevant to the SV reduction seen in chronic hypotension, given the dependency of SV on venous blood return and overall volemia⁴¹. Hypovolemia, for example due to reduced hydration, is associated with lower blood return to the right atrium and ventricular filling, which in turn reduces myocardial contractility and SV⁷.

PEP is inversely related to myocardial contractility. Moreover, this parameter is virtually independent of HR⁴², such that its higher expression in chronic hypotension can be unambiguously interpreted as indicative of less beta-adrenergic inotropic influence. CO varies according to HR and SV⁴¹. Given that differences in HR at rest, during cognitive activity and during sleep cannot be ascribed to differences in sympathetic activity, the CO reduction in chronic hypotension cannot be attributed to specific alterations in one of the branches of the autonomic nervous system.

Most studies investigating total peripheral resistance (TPR) as an index of vascular tone did not reveal differences between individuals with chronic hypotension and controls^{6,22,36,39}. In a single study, chronic hypotension was associated with higher TPR at rest and during mental stress⁷; moreover, in contrast to controls, individuals with chronic hypotension showed a progressive rise in TPR during sleep, presumably as a response to decreasing CO³⁸. This effect may be mediated by compensatory activation of the renin–angiotensin system and the vasculature following CO decline⁷.

To conclude, the robust and replicated results regarding SV and PEP support a crucial etiological role of diminished myocardial contractility, mediated by reduced beta-sympathetic activity and lower venous return, in chronic hypotension. TPR is linked to alpha-adrenergic effects on vasomotor tone⁴¹; based on the small or absent group differences for TPR in the available studies, it has been argued that chronic hypotension is mainly caused by cardiac factors, whereas vascular factors play a subordinate role^{22,36,39}.

C. Baroreflex mechanisms

The baroreflex consists of a negative feedback loop in which activity changes in arterial baroreceptors, resulting from blood pressure fluctuations, precipitate compensatory changes in HR, myocardial contractility and vascular tone⁴³. The sensitivity of the reflex is most frequently quantified within its cardiac branch by analyzing the spontaneous

covariation of systolic blood pressure and heart cycle duration⁴⁴. Various studies showed that cardiac baroreflex sensitivity (BRS), expressed as change in heart cycle duration per unit blood pressure change, is increased in chronic hypotension^{7,22,36,45}.

It has been claimed that the baroreflex is involved not only in the buffering of transient changes in arterial pressure but also in the long-term setting of blood pressure, which is supported by reduced cardiac BRS in individuals with elevated blood pressure and an inverse relationship between cardiac BRS and blood pressure in the normotensive range^{46,47}. With respect to chronic hypotension, increased responsiveness of the reflex may lead to overcompensation of phasic blood pressure increases, and thus blood pressure stabilization at lower levels⁴⁵. The arterial baroreceptors respond to mechanical stretch of the vessel walls⁴³; it has been shown that chronic hypotension is associated with smaller frequency, slope and amplitude of short-term blood pressure oscillations, which implies reduced input to the baroreceptors⁷. Permanently low activity in baroreceptor afferents may cause reorganization of neural processing in the nucleus of the solitary tract and other cardiovascular control units in the brain stem. This may involve compensation for reduced baroreceptor load and the resetting of the operating point of the baroreflex, which in turn may contribute to the manifestation of chronic hypotension^{7,22,45}.

D. Interplay between regulatory mechanisms

In addition to increased cardiac BRS, a very recent study documented hypersensitivity of the vasomotor and myocardial branches of the baroreflex in chronic hypotension⁷. While the cardiac branch is the most important generator of HRV and thus parasympathetic chronotropic influence⁴³, the vasomotor and myocardial branches respond to blood pressure fluctuations through the alpha- and beta-adrenergic systems⁷. By definition, the baroreflex subsystems do not act independent of each other; moreover, they may contribute to low blood pressure in interactions with other mechanisms modulating sympathetic and parasympathetic outflow⁷. In addition to the autonomic nervous system, the renin–angiotensin system plays a role in acute and long-term blood pressure regulation by modulating blood volume, electrolyte balance and vascular resistance⁴¹. While the importance of this system in elevated blood pressure is beyond question⁴⁸, its contribution to chronic hypotension remains to be explored. Close interactions of the renin–angiotensin system with the baroreflex and adrenergic systems are well known⁴⁹, supporting the hypothesis of an interplay of multiple autonomic and endocrinological mechanisms in the manifestation of chronic hypotension.

Conclusions

Altogether, the state of research suggests that reduced sympathetically mediated cardiac contractility is crucially implicated in the origin of chronic hypotension. Low SV and HR cause substantially diminished CO; for example, Duschek et al.³⁹ reported a CO reduction of 25% at rest and of 33% during mental stress in individuals with chronic hypotension as compared to controls. Low CO may be associated with insufficient blood supply of the organism and typical symptoms like low skin temperature and cold limbs. Moreover, general sympathetic underarousal is linked to deficient energetic state and limited adaptive resources of the organism³⁶. As initially stated, low contractility was also associated with negative emotional state and depression symptoms in chronic hypotension³⁰. In line with this, a connection between decreased beta-receptor function and increased levels of anxiety, depressiveness and anger was reported in healthy adults⁵⁰.

In addition to reduced tonic contractility and HR, blunted reactivity of blood pressure, HR and myocardial contractility during acute stress was seen in chronic hypotension, suggestive of insufficient hemodynamic adjustment to situational requirements^{5,20,22,36,45}. Similarly, the decline in cardiac BRS, which is commonly observed during challenging conditions, is reduced in chronic hypotension^{22,43}. Baroreflex inhibition implies a reduced buffering effect of the reflex, which facilitates stress-induced blood pressure increases and enhancement of the metabolic supply of the organism; as such, the restricted cardiac BRS response to stress confirms the notion of impaired autonomic cardiovascular adaptivity.

It is important to note that alterations in peripheral hemodynamics are at least partly transferred to cerebral blood flow^{51,52}. Cerebral autoregulation is supposed to ensure virtual independence of brain perfusion from systemic blood pressure⁵³. Mediated by metabolic and vaso-myogenic factors, the constriction and dilation of cerebral resistance vessels buffers systemic blood pressure fluctuations and keeps cerebral blood flow relatively constant⁵³. However, autoregulation seems to be insufficient to fully compensate for chronic hypotension. Transcranial Doppler sonography recordings demonstrated lower resting blood flow in the medial cerebral arteries of both hemispheres in individuals with chronic hypotension than controls²⁸; moreover, a blunted rise in cerebral blood flow during mental activity^{8,23,28,29}, and increased dependence of cerebral blood flow modulations on fluctuations of systemic blood pressure⁵⁴, were reported in chronic hypotension.

To sum up, in addition to bodily symptoms, chronic hypotension is associated with impairments in a wide range of cognitive functions and an increased burden of affective problems, which may interfere with daily

activities and quality of life. Autonomic dysregulations and impaired cerebral hemodynamics are involved in the origin of these symptoms. The autonomic phenotype of chronic hypotension is mainly characterized by reduced beta-inotropic cardiac influences, increased responsiveness of the baroreflex and restricted autonomic adaptiveness to situational requirements.

Future Research Directions

Prospective research could aim to more precisely define the autonomic and hemodynamic dysregulations detected in chronic hypotension. The divergent findings pertaining to parasympathetic control may relate to differences in the applied HRV parameters, especially between indices derived in the time domain and those derived in the frequency domain^{7,22,36,38}. Future studies using multiple analytic methods may help to clarify this issue. More detailed knowledge about hypotension-related alterations in SV and CO could be gained by using thermodilution or cardiac magnetic resonance imaging; and experimental manipulations by pharmacological methods may help to further explore autonomic mechanisms^{55,56}. It would also be of interest to estimate the relative contributions of low beta-adrenergic inotropic drive and low venous return to the reduction of myocardial contractility seen in chronic hypotension. The isoproterenol sensitivity test (D_{25}), urine norepinephrine and bioelectric impedance analysis may be considered to assess beta-adrenergic activity, body liquid and volemia, respectively^{50,57,58,59}. In addition, the possible role of the renin-angiotensin system in chronic hypotension remains to be explored.

A substantial research gap remains concerning the involvement of genetic and lifestyle factors, and related physiological mechanisms, in the origin of low blood pressure. In a clinical context, the risk of possible sequela of chronic hypotension should be further investigated. This applies, for example, to the causal role of low blood pressure in chronic kidney disease, glaucoma, Alzheimer's disease and vascular dementia^{11,12,13,15,16,17}. Finally, there is no consensus regarding the necessity of antihypertensive treatment and optimal strategies for this purpose. Further research is clearly needed to establish a platform from which to develop clinical guidelines concerning the management of chronic hypotension³⁰.

Conflict of Interest Statement

The authors declare that they have no conflicts of interest.

References

1. De Buyzere M, Clement DL, Duprez D. Chronic low blood pressure: a review. *Cardiovasc Drugs Ther.* 1998;12(1):29–35.
2. Wessely S, Nickson J, Cox B. Symptoms of low blood pressure: a population study. *BMJ.* 1990;301(6748):362–5.

3. Rosengren A, Tibblin G, Wilhelmsen L. Low systolic blood pressure and self perceived wellbeing in middle aged men. *BMJ*. 1993;306(6872):243-6.
4. Freeman R, Wieling W, Axelrod FB, Benditt DG, Benarroch E, Biaggioni I, et al. Consensus statement on the definition of orthostatic hypotension, neurally mediated syncope and the postural tachycardia syndrome. *Clin Auton Res*. 2011;21(2):69-72.
5. Covassin N, de Zambotti M, Cellini N, Sarlo M, Stegagno L. Cardiovascular down regulation in essential hypotension: relationships with autonomic control and sleep. *Psychophysiology*. 2013;50(8):767-76.
6. De Zambotti M, Covassin N, Cellini N, Sarlo M, Stegagno L. Cardiac autonomic profile during rest and working memory load in essential hypotensive women. *Int J Psychophysiol*. 2012;85(2):200-5.
7. Reyes del Paso GA, Montoro CI, Davydov D, Duschek S. The cardiac, vasomotor and myocardial branches of the baroreflex in hypotension: indications of reduced venous return to the heart. *Clin Auton Res*. 2025;35(1):87-99.
8. Sarlo M, de Zambotti M, Gallicchio G, Devigili A, Stegagno L. Impaired cerebral and systemic hemodynamics under cognitive load in young hypotensives: a transcranial doppler study. *J Behav Med*. 2013;36(2):134-42.
9. Duschek S, Schandry R. Reduced brain perfusion and cognitive performance due to constitutional hypotension. *Clin Auton Res*. 2007;17(2):69-76.
10. Pemberton J. Does constitutional hypotension exist? *BMJ*. 1989;298(6674):660-2.
11. Heijer T, Skoog I, Oudkerk M, de Leeuw FE, de Groot JC, Hofman A, et al. Association between blood pressure levels over time and brain atrophy in the elderly. *Neurobiol Aging*. 2003;24(2):307-13.
12. Ruitenbergh A, Skoog I, Ott A, Aevarsson O, Wittman JC, Lernfelt B, et al. Blood pressure and risk of dementia: results from the Rotterdam study and the Gothenburg H-70 study. *Dement Geriatr Cogn Disord*. 2001;12(1):33-9.
13. Warland J, McCutcheon H. Is there an association between maternal hypertension and poor pregnancy outcome?: a review of contemporary literature. *Aust Coll Midwives Inc J*. 2002;15(4):22-6.
14. Kovesdy CP, Trivedi BK, Kalantar-Zadeh K, Anderson JE. Association of low blood pressure with increased mortality in patients with moderate to severe chronic kidney disease. *Nephrol Dial Transplant*. 2006;21(5):1257-62.
15. Villacorta Junior H, Villacorta AS. Hypotension and Renal Dysfunction: The Ghosts of Heart Failure. *Arq Bras Cardiol*. 2017;109(2):165-8.
16. Chung HJ, Hwang HB, Lee NY. The association between primary open-angle glaucoma and blood pressure: two aspects of hypertension and hypotension. *Biomed Res Int*. 2015;2015:827516.
17. Graham SL, Drance SM. Nocturnal hypotension: role in glaucoma progression. *Surv Ophthalmol*. 1999;43 Suppl 1:10-6.
18. Duschek S, Hadjamu M, Schandry R. Dissociation between cortical activation and cognitive performance in the pharmacological treatment of chronic hypotension. *Biol Psychol*. 2007;75(3):277-85.
19. Duschek S, Hadjamu M, Schandry R. Enhancement of cerebral blood flow and cognitive performance due to pharmacological blood pressure elevation in chronic hypotension. *Psychophysiology*. 2007;44(1):145-53.
20. Duschek S, Matthias E, Schandry R. Essential hypotension is accompanied by deficits in attention and working memory. *Behav Med*. 2005;30(4):149-58.
21. Costa M, Stegagno L, Schandry R, Bitti PER. Contingent negative variation and cognitive performance in hypotension. *Psychophysiology*. 1998;35(6):737-44.
22. Duschek S, Hoffmann A, Reyes del Paso GA, Ettinger U. Autonomic cardiovascular control and executive function in chronic hypotension. *Ann Behav Med*. 2017;51(3):442-53.
23. Duschek S, Hoffmann A, Bair A, Reyes del Paso GA, Montoro C. Cerebral blood flow modulations during proactive control in chronic hypotension. *Brain Cogn*. 2018;125:135-41.
24. Duschek S, Hadjamu M, Schandry R. Dissociation between cortical activation and cognitive performance in the pharmacological treatment of chronic hypotension. *Biol Psychol*. 2007;75(3):277-85.
25. Duschek S, Hadjamu M, Schandry R. Enhancement of cerebral blood flow and cognitive performance due to pharmacological blood pressure elevation in chronic hypotension. *Psychophysiology*. 2007;44(1):145-53.
26. Duschek S, Meinhardt J, Schandry R. Reduced cortical activity due to chronic low blood pressure: an EEG study. *Biol Psychol*. 2006;72(3):241-50.
27. Weisz N, Schandry R, Jacobs A, Miale J, Duschek S. Early contingent negative variation of the EEG and attentional flexibility are reduced in hypotension. *Int J Psychophysiol*. 2002;45(3):253-60.
28. Duschek S, Schandry R. Cognitive performance and cerebral blood flow in essential hypotension. *Psychophysiology*. 2004;41(6):905-13.
29. Duschek S, Hoffmann A, Montoro CI, Bair A, Reyes del Paso GA, Ettinger U. Cerebral blood flow modulations during antisaccade preparation in chronic hypotension. *Psychophysiology*. 2019;137:65-72.
30. Duschek S, Hoffmann A, Reyes del Paso GA. Affective impairment in chronic low blood pressure. *J Psychosom Res*. 2017;93:33-40.
31. Barrett-Connor E, Palinkas LA. Low blood pressure and depression in men: a population based study. *BMJ*. 1994;308:446-449.
32. Duschek S, Dietel A, Schandry R, Reyes del Paso GA. Increased sensitivity to heat pain in chronic low blood pressure. *Eur J Pain*. 2009;13(1):28-34.
33. van Rijn MJ, Schut AF, Aulchenko YS, Deinum J, Sayed-Tabatabaei FA, Yazdanpanah M, et al. Heritability of blood pressure traits and the genetic contribution to blood pressure variance explained by four blood-pressure-related genes. *J Hypertens*. 2007;25(3):565-570.
34. Agarwal A, Williams GH, Fisher ND. Genetics of human hypertension. *Trends Endocrinol Metab*. 2005;16(3):127-133.
35. Padmanabhan S, Newton-Cheh C, Dominiczak AF. Genetic basis of blood pressure and hypertension. *Trends Genet*. 2012;28(8):397-408.
36. Duschek S, Hoffmann A, Montoro C, Reyes del Paso GA. Autonomic cardiovascular dysregulation in chronically low blood pressure. *J Psychophysiol*. 2017;33(1):39-53.
37. Berntson GG, Quigley KS, Norman GJ, Lozano D. Cardiovascular psychophysiology. In: Cacioppo JT, Tassinary LG, Berntson GG, editors. *Handbook of psychophysiology*. Cambridge, UK: Cambridge University Press; 2016. p.183-215.
38. Covassin N, de Zambotti M, Cellini N, Sarlo M, Stegagno L. Nocturnal cardiovascular activity in essential hypotension: evidence of differential autonomic regulation. *Psychosom Med*. 2012;74(9):952-960.
39. Duschek S, Heiss H, Buechner B, Werner NS, Schandry R, Reyes del Paso GA. Hemodynamic determinants of chronic hypotension and their modification through vasopressor application. *J Physiol Sci*. 2009;59(2):105-112.
40. Wesseling KH, Jansen JR, Settels JJ, Schreuder JJ. Computation of aortic flow from pressure in humans using a nonlinear, three-element model. *J Appl Physiol*. 1993;74(5):2566-2573.

41. Hall JE, Hall ME. Guyton and Hall textbook of medical physiology. 14th ed. Philadelphia: Saunders Elsevier; 2020.
42. Hassan S, Turner P. Systolic time intervals: a review of the method in the non-invasive investigation of cardiac function in health, disease and clinical pharmacology. *Postgrad Med J*. 1983;59(693):423-434.
43. Duschek S, Werner NS, Reyes del Paso GA. The behavioral impact of baroreflex function: a review. *Psychophysiology*. 2013;50(12):1183-1193.
44. Parati G, di Rienzo M, Mancia G. How to measure baroreflex sensitivity: from the cardiovascular laboratory to daily life. *J Hypertens*. 2000;18(1):7-19.
45. Duschek S, Dietel A, Schandry R, Reyes del Paso GA. Increased baroreflex sensitivity and reduced cardiovascular reactivity in chronic low blood pressure. *Hypertens Res*. 2008;31:1873-1878.
46. Carthy ER. Autonomic dysfunction in essential hypertension: a systematic review. *Ann Med Surg*. 2013;3(1):2-7.
47. Hesse C, Charkoudian N, Liu Z, Joyner MJ, Eisenach JH. Baroreflex sensitivity inversely correlates with ambulatory blood pressure in healthy normotensive humans. *Hypertension*. 2007;50(1):41-46.
48. Fountain JH, Kaur J, Lappin SL. Physiology, Renin Angiotensin System. 2023 Mar 12. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-
49. Reid IA. Interactions between ANG II, sympathetic nervous system, and baroreceptor reflexes in regulation of blood pressure. *Am J Physiol*. 1992;262(6 Pt 1):E763-78.
50. Yu BH, Kang EH, Ziegler MG, Mills PJ, Dimsdale JE. Mood states, sympathetic activity, and in vivo beta-adrenergic receptor function in a normal population. *Depress Anxiety*. 2008;25(7):559-564.
51. Duschek S, Heiss H, Schmidt FH, Werner N, Schuepbach D. Interactions between systemic hemodynamics and cerebral blood flow during attentional processing. *Psychophysiology*. 2010;47(6):1159-1166.
52. Montoro C, Duschek S, Reyes del Paso GA. Variability in cerebral blood flow velocity at rest and during mental stress in healthy individuals: associations with cardiovascular parameters and cognitive performance. *Biol Psychol*. 2018;135:149-158.
53. Yang SH, Liu R. Cerebral autoregulation. In: Caplan LR, Biller J, Leary M, Lo E, Thomas A, Yenari M, Zhang J, editors. *Primer on cerebrovascular diseases*. New York, NY: Academic Press; 2017. p.57-60.
54. Duschek S, Schandry R. Deficient adjustment of cerebral blood flow to cognitive activity due to chronically low blood pressure. *Biol Psychol*. 2006;72(3):311-317.
55. Argueta EE, Paniagua D. Thermodilution Cardiac Output: A Concept Over 250 Years in the Making. *Cardiol Rev*. 2019;27(3):138-144.
56. Knobelsdorff-Brenkenhoff F, Pilz G, Schulz-Menger J. Representation of cardiovascular magnetic resonance in the AHA / ACC guidelines. *J Cardiovasc Magn Reson*. 2016;19(1):70.
57. Cleaveland CR, Rangno RE, Shand DG. A standardized isoproterenol sensitivity test. The effects of sinus arrhythmia, atropine, and propranolol. *Arch Int Med*. 1972;130:47-52.
58. Kennedy B, Ziegler MG. A more sensitive and specific radioenzymatic assay for catecholamines. *Life Sci*. 1990;47:2143-2153.
59. Kron J, Volkenandt J, Broszeit S, Leimbach T, Kron S. Can Bioimpedance Analysis Be Used to Estimate Absolute Blood Volume in Hemodialysis Patients? *Hemodialysis Int*. 2025; in press.