

Body Mass Index May Influence Heart Rate Variability

Thalys Sampaio Rodrigues¹ and Levindo José Garcia Quarto²

University of Melbourne Department of Medicine Austin Health,¹ Heidelberg, Victoria - Austrália

Hospital Regional Norte,² Sobral, CE - Brazil

We read with interest the article by Bassi et al.,¹ titled “Effects of Coexistence Hypertension and Type II Diabetes on Heart Rate Variability and Cardiorespiratory Fitness”, published in the issue of July 2018. The authors investigated the influence of systemic hypertension on cardiac autonomic modulation in patients with type 2 diabetes mellitus (T2DM) and assessed the heart rate variability (HRV) on exercise capacity in these patients. They concluded that hypertension negatively affects cardiac autonomic function, with greater impairment in HRV, when compared to normotensive patients with T2DM.

Keywords

Hypertension/prevalence; Diabetes Mellitus, Type 2; Risk Factors; Cardiovascular Diseases; Autonomic Nervous Systems; Heart Rate.

Mailing Address: Thalys Sampaio Rodrigues •

145 Studley road. 3084, Heidelberg, Victoria – Australia

E-mail: thalys.sampaio Rodrigues@unimelb.edu.au

Manuscript received August 01, 2018, revised manuscript September 12, 2018, accepted September 12, 2018

Several aspects of this study require discussion. As previously reported, numerous factors may have impact on HRV indices, including sex, insulin resistance, body mass index (BMI), hyperlipidemia, hypertension, ischemic and non-ischemic cardiomyopathy, and smoking status.²⁻⁴ For instance, increased BMI can independently decrease HRV, particularly when central adiposity is present.⁵ Indeed, the hypertensive group had a higher BMI when compared to the normotensive group (28 ± 4.4 vs 31 ± 3.8 , $p = 0.031$). Given the lack of control for BMI between the two groups, the conclusions made by the authors should be regarded cautiously.

Finally, it is known that subclinical myocardial dysfunction is highly prevalent in diabetic patients and is independently associated with cardiac autonomic neuropathy.⁶ However, the authors only considered medical history consistent with ischemic heart disease for stratification/exclusion of patients for analysis. We believe that a more detailed cardiovascular assessment, including echocardiography to determine left ventricular mass and left ventricular diastolic dysfunction, would be important to better stratify the patients and strengthen their conclusions.

DOI: 10.5935/abc.20180201

References

1. Bassi D, Cabiddu R, Mendes RG, Tossini R, Arakilian VM, Caruso FC, et al. Effects of coexistence hypertension and type II diabetes on heart rate variability and cardiorespiratory fitness. *Arq Bras Cardiol.* 2018;11(1):64-72.
2. Benichou T, Pereira B, Mermillod M, Tauveron I, Pfabigan D, Magdasy S, et al. Heart rate variability in type 2 diabetes mellitus: A systematic review and meta-analysis. *PLoS One.* 2018;13(4):e0195166.
3. Liao D, Sloan RP, Cascio WE, Folsom AR, Liese AD, Evans GW. Multiple metabolic syndrome is associated with lower heart rate variability: the Atherosclerosis Risk in Communities Study. *Diabetes Care.* 1998;21(12):2116-22.
4. Vasconcelos DF, Junqueira Junior LF. Cardiac autonomic and ventricular mechanical functions in asymptomatic chronic chagasic cardiomyopathy. *Arq Bras Cardiol.* 2012;98(2):111-9.
5. Windham BG, Fumagalli S, Ble A, Sollers JJ, Thayer JF, Najjar SS, et al. The relationship between heart rate variability and adiposity differs for central and overall adiposity. *J Obes.* 2012;2012:149516.
6. Sacre JW, Franjic B, Jellis CL, Jenkins C, Coombes JS, Marwick TH, et al. Association of cardiac autonomic neuropathy with subclinical myocardial dysfunction in type 2 diabetes. *JACC: Cardiovasc Imaging.* 2010;3(12):1207-15.

Reply

Dear Editor,

We appreciate the authors' interest towards our article, “Effects of Coexistence Hypertension and Type II Diabetes on Heart Rate Variability and Cardiorespiratory Fitness”. We also appreciate the opportunity to respond to their comments. Their critique of our study mainly focused on 3 issues: 1) lack of methodological attention in relation to the participants' gender, BMI and insulin resistance; 2) lack of methodological details about the study population, regarding smoking habits

and hyperlipidemia; and 3) lack of consistent investigation on ischemic and non-ischemic cardiomyopathy. We appreciate the authors' concerns; however, we do not agree with many of their comments.

The first issue was clearly acknowledged in our paper. Since cardiac variability dynamics differ between genders, with higher parasympathetic activity and overall complexity for women, gender distribution must be considered when investigating heart rate dynamics.¹ However, in our study, gender

distribution is not significantly different for the investigated groups (Diabetes Mellitus (DM) and DM + Hypertension Systemic arterial hypertension (SAH), $p = 0.464$). In relation to insulin resistance, we agree with the authors' comments about differences possibly influencing HRV indices. However, we believe that the difference found between groups could be attributed to different weight and BMI. We kindly invite the authors to read a recent study from our group, demonstrating that obesity *per se* impairs aerobic-hemodynamic responses to exercise but that, however, metabolic syndrome (obesity, DM and hypertension) in young adults negatively impacts overall HRV, parasympathetic activity and HRV complexity, corroborating our findings.² In the present study, differences were observed between groups for BMI and weight, with patients in the DM group being overweight and patients in the DM + Hypertension group presenting grade 1 obesity; however, we would like to emphasize that, after age, sex and BMI adjustments, we concluded that these variables did not influence our results.

As for the second issue, current smokers were excluded from our study, as previous evidence showed that tobacco use represents an important cardiovascular risk and leads to HRV impairment.³ Even though it was not detailed in the exclusion criteria section, Table 1 clearly shows that none of the participants were current smokers. As for dyslipidemia, we agree with the authors that it may influence HRV; however, after adjustments for this variable, we concluded that dyslipidemia did not significantly influence our results ($p = 1.000$).

The last issue is related to the lack of consistent investigation on ischemic and non-ischemic cardiomyopathy in the present study. The authors have criticized that a simple clinical evaluation may not be sufficient to determine the presence

of ischemic conditions; however, clinical investigation may indicate the need for further exams, aimed at detecting ischemic and non-ischemic heart disease. In addition, the absence of effort-induced ischemic signs was evident during the cardiopulmonary test. Even though this was not clearly stated in the text, we emphasized that all participants underwent a thorough clinical evaluation, consisting of physical examination, resting electrocardiogram and maximal incremental exercise. We kindly invite the authors to read a relevant study about screening procedures for this kind of patients.⁴ Our patients presented no signs or symptoms of suspected ischemic disease, neither at rest, nor during effort. Thus, according to the most recent guidelines for investigation of ischemic patients with DM,⁵ no further examination was needed through echocardiography or other exams to investigate the presence of myocardial dysfunction.

Finally, it is well known that hypertension *per se* negatively affects HRV;⁶ however, no previous studies investigated linear and non-linear HRV indices in DM + SAH coexistence. Thus, we believe that our article provides a relevant contribution to the understanding of HRV alterations in pathological conditions.

In consideration of the fact that HRV is highly influenced by a number of variables, including demographic and anthropometric characteristics, the presence of obesity, associated comorbidities and cardiovascular risk factors, future, robust studies are needed to further investigate the influence of specific variables on linear and non-linear HRV indices, in order to confirm the preliminary findings of our study.

Daniela Bassi
Ramona Cabiddu
Audrey Borghi-Silva

References

1. Ryan SM, Goldberger AL, Pincus SM, Mietus J, Lipsitz LA. Gender- and age-related differences in heart rate dynamics: are women more complex than men? *J Am Coll Cardiol*. 1994;24(7):1700-7.
2. Carvalho LP, Di Thommazo-Luporini L, Mendes RG, Cabiddu R, Ricci PA, Basso-Vanelli RP, et al. Metabolic syndrome impact on cardiac autonomic modulation and exercise capacity in obese adults. *Auton Neurosci*. 2018 Sep;213:43-50.
3. Barutcu I, Esen AM, Kaya D, Turkmen M, Karakaya O, Melek M, et al. Cigarette smoking and heart rate variability: dynamic influence of parasympathetic and sympathetic maneuvers. *Ann Noninvasive Electrocardiol*. 2005;10(3):324-9.
4. Young LH, Wackers FJT, Chyun DA, Davey JA, Barrett EJ, Taillefer R, et al. Cardiac outcomes after screening for asymptomatic coronary artery disease in patients with type 2 diabetes. *JAMA*. 2009;301(15):1547-55.
5. Sociedade Brasileira de Diabetes. Diretrizes Sociedade Brasileira de Diabetes 2017-2018. São Paulo: Editora Clannad; 2017. 383p.
6. Lutfi MF, Sukkar MY. The effect of gender on heart rate variability in asthmatic and normal healthy adults. *Int J Health Sci (Qassim)*. 2011;5(2):146-54.

