Volume 1. Issue 1. 2018

ProClinS Cardiology

Review Article

Open Access

Sympathetic Overdrive is an Ignored, but a Popular Disease

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Received: June25, 2018; **Accepted:** August27, 2018; **Published:** August27, 2018

ABSTRACT

The sympathetic overdrive is considered as one of the common medical condition for many diseases. According to the principles of a disease, this is considered under disease. Some early warning signs include (high resting heart rate; high blood pressure; and obesity). There are some unspecified signs of the related medical conditions. The risk factors and the mechanisms are clear. Organ failures and morality is quite common. To identify the sympathetic overdrive as a disease and to treat the signs at the same time will prevent many common diseases. This article elucidates the hypothesis of the disease of sympathetic overdrive. It ought to be treated at the earliest instead of ignoring or misidentifying the disease which may drastically affect many people around the world.

KEYWORDS: Disease, Sympathetic Overdrive

INTRODUCTION

Sympathetic overdrive is considered as one of the mechanisms of primary hypertension [1-3]. Sympathetic overdrive is also described as one of the mechanisms of sudden death, chronic heart failure, atherosclerosis, diabetes and obesity [3-10]. This provides sufficient evidence to prove that Sympathetic overdrive is a disease. Thus, rather than treating it as a symptom it becomes necessary to consider it as an individual disease to avoid further Comorbidities [2, 8, 11]. To identify the DSO as a disease the review shall be discussed under key components of a disease. DSO must be practically considered for further improvement in prevention of its comorbidities.

Sign and symptoms

The original signs of the DSO include: high resting heart rate, high normal blood pressure and obesity/overweight. Although none of these signs have been confirmed as standard signs of a disease, they have shown by many clinical investigations to be risk factors of cardiovascular diseases, non-cardiovascular diseases, cancers [6, 9, 12]. Many large clinical investigations, such as Framingham studies [4, 6], French population survey and other European study [3] offer the evidences.

The first sign of DSO is high resting heart rate. The investigations of the correlation between high resting heart rate and diseases show that individuals with heart rate over 80 beats/min are in high risk of cardiovascular diseases [3, 8]. Studies of Chicago Detection Project [9] reported 10 times/min more of resting heart rate increased the rate of coronary heart disease and sudden death by 5～12%, non-cardiovascular death by 16% and cancer by 9%. Some other results showed the resting heart rate is a risk for diseases, such as T2DM, obesity, hypertension, sudden death, atherosclerosis and heart failure [4-8, 12, 13].

The reference normal resting heart rate is 60-70 beats/min, which associates with the lowest of the common diseases around the world [6, 8, 11]. The resting heart rate is an indicator of the activity of sympathetic system and could be considered as a clinical index to evaluate and manage the sympathetic system. It can be easily measured for routine

clinical practice [5-18]. So why not consider High resting heart rate to be the first sign of DSO. It will be very important in preventing and control of mortality rates. The second considerable sign is high blood pressure: It should be a considered at the earliest and treated immediately [2, 19, 20]. High blood pressure ranges between 120～139 / 80～89 mmHg. The high blood pressure accounts for arrhythmia and cerebrovascular events according to a 5 year survey reported [21]. A high school-based survey found that the blood pressure increased from normal (115.6/73.8mmHg) to abnormal (125/80.7mmHg) where in the cases of ECG abnormality gained the significance [22]. A report shows that elevated blood pressure is a leading global health risk. About 70% of DALYs and Death are caused due to the blood pressure over 140mmHg and about 30% of DALYs and Death are due to the blood pressure ranging between 110 to139 mmHg. There are some data from the same study of which DALYs and death are more in population who have the blood pressure ranges between 110~139mmHg . High Blood Pressure must be considered as a big problem and should be immediately treated and managed, as this is a huge public health problem frequently noticed. The high blood pressure and hypertension have been increasing in the past two decades [2].We in this review try to consider identifying high blood pressure as a sign of DSO. This is a step to deal with the problem and to reduce the increasing prevalence of the most common disease around the world. The newest guideline of blood pressure controlling is to put down the diagnosis standard to 130/80mmHg [23]. This is a big step, but not enough. It is, indeed, ignoring the relating problems. On the other hand, high prevalence of masked suboptimal blood pressure uncontrolled in patients (MUCH) is a risk factor [24]. The Sprint Study shows that intense controlling of the systolic blood pressure to 120mmHg in comparison with 140mmHg can gain more benefits [25-28]. It is important to learn about other remaining risks of high blood pressure. Some countries are going to change their guidelines [29, 30]. If only high blood pressure is considered as a sign of DSO in theory [19, 20, 31, 32], the new strategy and method of managements could be developed soon. Obesity and overweight is the 3rd sign of DSO. Obesity is considered to be an abnormal sign as it is connected with metabolic syndrome [14, 33]. Overweight and obesity is recently considered as a disease [34], but it seems not enough. Some results show that obesity, high waist/hip ratio could increase sympathetic activation and cardiovascular risks [14]. High BMI clues the parasympathetic system inhibited and then sympathetic overdrive induced [31]. Obesity is involved in hypertrophy and vasculature resistance [35]. Obesity is a key feature of the metabolic syndrome and causes the insulin resistance, sympathetic overdrive, oxidation, and inflammation [15]. Sympathetic overdrive is the crucial part between obesity and hypertension [32]. Leptin plays a role in activating central nervous system which increases the sympathetic activity in obesity hypertension patients [33, 36]. On account of overweight, there are some subclinical conditions, such as hypertrophy, vascular remolding, even before hypertension occurred [19, 31]. Therefore, overweight and obesity should be classified a sign of DSO and even agitate themselves. High resting heart rate, high blood pressure, overweight/obesity are the major and specific signs of DSO. More symptoms and signs may refer to tissue injuries/ damage or organ damages or related co-morbidities which are not discussed in this article due to the specificity varied from condition to condition. Until now, these major specific signs of DSO as well as DSO itself are not being accepted. In order to identify DSO and prevent the consequent diseases listed above, it is necessary to recognize these major signs first and then the DSO.

The risk factors

Each disease has some risk factors. There are many risk factors associated with the DSO. The unchangeable risk factors are age, gender [3,4,6,38,39] and hereditary medical conditions [32,40]. The changeable risk factors are smoke, drink, obesity, depression or anxiety, lifestyle, environment [1,7,15,16,18,38,41,46].

The mechanism(s)

Although Sympathetic drive is considered as a disease and mechanism too many related comorbidities, it is not really treated as a unique prior entity to a specific disease [11, 19]. The sympathetic overdrive is the basic and original mechanism of DSO [33, 37]. It could induce insulin resistance, adipokinin, inflammation, hyperlipidemia, renin-angiotensin system activated [15,17,18,34,38,47] to increase cardiovascular diseases and cancers [2,19,30,32,46].

The tissue and organs damage, morbidity and mortality

The tissue and organs, of which the disease of sympathetic overdrive might influence, are heart, artery system, kidneys, brain, and metabolic system. Since it is affecting so many tissue and organ damage, the morbidity and mortality rates are higher [3,5,6,47,48].

DISCUSSION AND CONCLUSION

To identify the disease of sympathetic overdrive (DSO) is meaningful, which may influence millions and millions of people throughout the world [14,23,33,41]. The higher prevalence of hypertension, diabetes, heart failure, atherosclerosis, sudden death and cancers might be going up in future, because of the earlier stage of these diseases ignored or mismatched [15,18]. To realize the DSO is an important step to aim and reduce the most common diseases and some kinds of cancers on the world. The newest guideline of hypertension controlling is still isolating it with other risk factors, of which shares the same mechanism. Things need to be remembered, anti-hypertension agents could even aggravate sympathetic overdrive and ensue more end-events and mortality in clinical community [49,50]. It is wise to control the signs together under the theme of the disease of sympathetic overdrive. There may be more questions about setting the conception and definition of the disease of sympathetic overdrive.

REFERENCES

1. Guido Grassi, Allyn Mark, Murry E Sleer. The sympathetic nervous system alterations in human hypertension. Circ Res. 2015; 116: 976-990.
2. Forouzanfar MH, Liu P, Roth GA, Ng M, Biryukov S, Marczak L. et al. Global Burden of Hypertension and Systolic Blood Pressure of at Least 110 to 115 mm Hg, 1990-2015.JAMA. 2017; 317: 165-182.
3. Benetos A, Rudnichi A, Thomas F, Safar M, Guize L. Influence of heart rate on mortality in a French population: role of age, gender, and blood pressure. Hypertension. 1999; 33: 44-52.
4. Gillum RF, Makuc DM, Feldman JJ. Pulse rate, coronary heart disease, and death: the NHANES I Epidemiologic Follow-up Study. Am Heart J. 1991; 121: 172-177.
5. Giannoglou GD, Chatzizisis YS, Zamboulis C, Parcharidis GE, Mikhailidis DP, Louridas GE. Elevated heart rate and atherosclerosis: an overview of the pathogenetic mechanisms. Int J Cardiol. 2008; 126: 302-312.
6. Ho JE, Larson MG, Ghorbani A, Cheng S, Coglianese EE, Vasan RS. et al. Long-term cardiovascular risks associated With an elevated heart rate: the Framingham Heart Study. J Am Heart Assoc. 2014; 8: 3.
7. Hong JW, Noh JH, Kim DJ. The Association of Resting Heart Rate with the Presence of Diabetes in Korean Adults: The 2010-2013 Korea National Health and Nutrition Examination Survey. PLoS One. 2016; 11: e0168527.
8. Parikh KS, Greiner MA, Suzuki T, DeVore AD, Blackshear C, Maher JF. et al. Resting Heart Rate and Long-term Outcomes Among the African American Population: Insights From the Jackson Heart Study. JAMA Cardiol. 2017; 2: 172-180.
9. Greenland P, Daviglus ML, Dyer AR, Liu K, Huang CF, Goldberger JJ. et al. Resting heart rate is a risk factor for cardiovascular and noncardiovascular mortality: the Chicago Heart Association Detection Project in Industry. Am J Epidemiol. 1999; 149: 853-862.
10. Grassi G. Sympathetic overdrive and cardiovascular risk in the metabolic syndrome. Hypertens Res. 2006 ; 29: 839-847.
11. Zhang D, Wang W, Li F. Association between resting heart rate and coronary artery disease, stroke, sudden death and noncardiovascular diseases: a meta-analysis. CMAJ. 2016; 188: E384-E392.
12. Stamler R, Stamler J, Riedlinger WF, Algera G, Roberts RH. Weight and blood pressure findings in hypertension screening of 1 million Americans. JAMA. 1978 ; 240: 1607-1610.
13. Tverdal A, Hjellvik V, Selmer R. Heart rate and mortality from cardiovascular causes: a 12 year follow-up study of 379,843 men and women aged 40-45 years. Eur Heart J. 2008; 29: 2772-2781.
14. Yadav RL, Yadav PK, Yadav LK, Agrawal K, Sah SK. et al. Association between obesity and heart rate variability indices: an intuition toward cardiac autonomic alteration - a risk of CVD. Diabetes MetabSyndrObes. 2017 ; 10: 57-64.
15. Conti FF, Brito Jde O, Bernardes N, Dias Dda S, Malfitano C. et al. Positive effect of combined exercise training in a model of metabolic syndrome and menopause: autonomic, inflammatory, and oxidative stress evaluations. Am J PhysiolRegulIntegr Comp Physiol. 2015; 309: 1532-1539.
16. InekeNederend, Nienke M. Schutte, Meike Bartels, Arend D. J. ten Harkel, Eco J. C. de Geus. Heritability of heart rate recovery and vagal rebound after exercise. Eur J Appl Physiol. 2016; 116: 2167-2176.

17. Moreira MC, Pinto IS, Mourão AA, Fajemiroye JO, Colombari E. et al. Does the sympathetic nervous system contribute to the pathophysiology of metabolic syndrome?. Front Physiol. 2015; 25: 234.

18. Williams KJ,Wu X. Imbalanced insulin action in chronic over nutrition:clinicalharm,molecular mechanisms ,and a way forward. Atherosclerosis. 2016; 247: 225-282.

19. Lambert EA, Lambert GW. Stress and its role in sympathetic nervous system activation in hypertension and the metabolic syndrome. CurrHypertens Rep. 2011; 13: 244-248.

20. Pal GK, Chandrasekaran A, Pal P, Nivedita N, Indumathy J. et al. Prehypertension status ,cardiometabolic risks,and decreased baroreflex sensitivity are linked to sympathovagal imbalance in salt-preferring individuals. Clin Exp Hypertens. 2015; 37: 609-615.

21. Iqbal P, Stevenson L. Cardiovascular outcomes in patients with normal and abnormal 24-hour ambulatory blood pressure monitoring. Int J Hypertens. 2010 ; 2011: 786912.

22. Dadlani GH, Wilkinson JD, Ludwig DA, Harmon WG, O'Brien R. et al. A high school-based voluntary cardiovascular risk screening program: issues of feasibility and correlates of electrocardiographic outcomes. PediatrCardiol. 2013; 34: 1612-1619.

23. Whelton PK, et al. ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC /NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults. Hypertension. 2018.

24. Nabil Naser, AlenDzubur, AzraDurak, Mehmed Kulic, Nura Naser. Blood Pressure Control in Hypertensive Patients, Cardiovascular Risk Profile and the Prevalence of Masked Uncontrolled Hypertension (MUCH). Med Arch. 2016; 70: 274-279.

25. The SPRINT Research Group. A Randomized Trial of Intensive versus Standard Blood-Pressure Control. N Engl J Med. 2015; 373: 2103–2116.

26. Williamson JD, Supiano MA, Applegate WB, Berlowitz DR, Campbell RC, Chertow GM. et al. Intensive vs Standard Blood Pressure Control and Cardiovascular Disease Outcomes in Adults Aged ≥75 Years: A Randomized Clinical Trial. JAMA. 2016; 315: 2673-82.

27. Xie X, Atkins E, Lv J, Bennett A, Neal B. et al. Effects of intensive blood pressure lowering on cardiovascular and renal outcomes: updated systematic review and meta-analysis. Lancet. 2016; 387: 435-443.

28. Ettehad D, Emdin CA, Kiran A, Anderson SG, Callender T. et al. Blood pressure lowering for prevention of cardiovascular disease and death: a systematic review and meta-analysis. Lancet. 2016; 387: 957-967.

29. Al Hamarneh YN, Houle SK, Padwal R, Tsuyuki RT. Yazid N. Hypertension Canada's 2016 Canadian Hypertension Education Program guidelines for pharmacists: An update.Can Pharm J (Ott). 2016; 149: 337–344.

30. Gabb GM, Mangoni AA, Anderson CS, Cowley D, Dowden JS. et al. Guideline for the diagnosis and management of hypertension in adults- 2016. Med J Aust. 2016; 205: 85-89.

31. Grassi G. Sympathetic overdrive and cardiovascular risk in the metabolic syndrome. Hypertens Res. 2006; 29: 839-847.

32. Pal GK, Adithan C, Dutta TK. et al. Association of hypertension status and cardiovascular risks with sympathovagal imbalance in first degree relatives of type 2 diabetics.J Diabetes Investig. 2014; 5: 449-455.

33. Rahmouni K. Obesity, sympathetic overdrive, and hypertension: the leptin connection. Hypertension. 2010; 55: 844-845.

34. NHLBI Obesity Education Initiative, National Heart, Lung, et al. The practical guide: identification, evaluation, and treatment of overweight and obesity in adults[M]. National Heart, Lung, and Blood Institute, 2002.

35. Pal GK, Chandrasekaran A, Hariharan AP, Dutta TK, Pal P. et al. Body mass index contributes to sympathovagal imbalance in prehypertensives. BMC Cardiovasc Disord. 2012; 12:54.

36. Haddock RE, Hill CE. Sympathetic overdrive in obesity involves purinergic hyperactivity in the resistance vasculature. J Physiol. 2011; 589: 3289-3307.

37. da Silva AA, do Carmo JM, Hall JE. Role of leptin and central nervous system melanocortins in obesity hypertension. CurrOpinNephrolHypertens. 2013; 22: 135-140.

38. Eyre EL, Duncan MJ, Birch SL et al. The influence of age and weight status on cardiac autonomic control in healthy children: a review. AutonNeurosci. 2014; 186: 8-21.

39. Appelman Y, van Rijn BB, Ten Haaf ME, Boersma E, Peters SA. Sex differences in cardiovascular risk factors and disease prevention. Atherosclerosis. 2015; 241: 211-218.

40. Pal GK, Adithan C, Ananthanarayanan PH, Pal P, Nanda N. et al. Effects of gender on sympathovagal imbalance, prehypertension status, and cardiovascular

risks in first-degree relatives of type 2 diabetics. Am J Hypertens. 2014; 27: 317-324.

41. Qi Z, Ding S. Obesity –associated sympathetic overactivity in children and adolescents: the role of catecholamin resistance in lipid metaboism. J Pediatr Endocrinol Metab. 2016; 29; 113-125.

42. Kivimäki M, Jokela M, Nyberg ST, Singh-Manoux A, Fransson EI. et al. Long working hours and risk of coronary heart disease and stroke: a systematic review and meta-analysis of published and unpublished data for 603,838 individuals. Lancet. 2015; 386: 1739-1746.

43. Pasquini M, Berardelli I, Biondi M. Ethiopathogenesis of depressive disorders.ClinPract Epidemiol Ment Health. 2014; 10: 166-171.

44. Farah D, Nunes J, Sartori M2, Dias DD, Sirvente R.et al. Exercise training prevents cardiovascular derangement indeced by fructose overload in developing rates. PLoS One. 2016; 8.

45. Zouhal H, Lemoine-Morel S, Mathieu ME, Casazza GA, Jabbour G. Catechotamines and obesity: effects of exercise and training .Sports Med. 2013; 43: 591-600.

46. Yadav RL, Yadav PK, Yadav LK, Agrawal K, Sah SK. et al. Association between obesity and heart rate variability indices: an intuition toward cardiac autonomic alteration – a risk of CVD. Diabetes MetabSyndrObes. 2017; 10: 57–64.

47. Bruno RM, Ghiadoni L, Seravalle G, Dell'oro R, Taddei S. et al. Sympathetic regulation of vascular function in health and disease. Front Physiol. 2012; 3: 284.

48. Smith MM, Buffington CA, Hamlin RL, Devor ST. Relationship between muscle sympathetic nerve activity and aortic wave reflection characteristics in aerobic- and resistance-trained subjects. Eur J Appl Physiol. 2015; 115: 2609-2619.

49. UE Beckenbauer, G Planz. Clonidine-diuretic combination in patients with essential hypertension. Modification of sympathetic activity and blood pressure as well as the reaction following sudden withrawal. Fortschr Med. 1984; 102: 802-806.

50. Brown MJ, Toal CB. Formulation of short-acting nifedipine tablets influences the heart rate and sympathetic nervous system response in hypertensive patients. Br J Clin Phamracol. 2008; 65: 646-652.