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Editorial

Heart and Brain: A Complex Relationship

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Editorial

Macey and colleagues have shown that heart rate (HR) responses are blunted and delayed in obstructive sleep apnoea (OSA) subjects. The blunted HR responses were observed during handgrip and cold test, which require integration of cold temperature, pain and proprioceptive input, and are representative for everyday activities. Such delay indicative of impaired autonomic processing in medulla and thalamic regions is consistent with the known structural brain changes in OSA [1]. Therefore, the brain impairment in OSA has an direct impact on cardiac function. However, how does heart affect brain in OSA? To respond to this question we need to summarize the most recent physiological research advances in this area.

For the first time long term brain microcirculation adaptation to decreased cardiac output has been reported in chronic left ventricle failure by Georgiadis et al [2]. Georgiadis et al. [2] found a significant relationship between the decline in left ventricle ejection fraction (LVEF) and the reduction in cerebrovascular reactivity. Ogoh et al. [3] demonstrated in an elegant study that blood flow velocity in the middle cerebral artery response to a rapid decline in systemic BP was highly related to unloading of the arterial baroreceptors, which suggests cardiac output involvement in the regulation of cerebral blood flow (CBF). One year later, our team reported positive correlations between changes in pial artery pulsation and LVEF, and between the systolic–diastolic cerebral blood volume fraction and LVEF in an animal model with stable blood pressure (BP) [4].

Introduction of wavelet transform analysis to cardiovascular research opened new area to investigate the cardiac contribution to cerebral perfusion [5,6]. Li et al. [7] reported a negative correlation between cerebral oxygenation and CBF velocity at respiratory and cardiac frequencies. Cui et al. [8] used wavelet coherence analysis to assess the relationship between spontaneous oscillations in changes in BP and the cerebral tissue oxyhemoglobin concentration (HbO_2); this analysis demonstrated a significant increase in wavelet coherence in elderly compared to young subjects at frequencies of 0.4–2.0 Hz, while no change in wavelet phase coherence was found at the same frequencies. Cui et al. [8] argued that increased sympathetic drive and diminished heart rate variability may results in increased cardiac contribution to spontaneous BP/HbO_2 oscillations in the elderly.

Very recent data coming from our lab suggest that cardiac contribution to the BP pial artery pulsation oscillations diminish during apnoea in healthy subjects [9]. In this study we used novel methodology that allows for non-invasive assessment of pial artery pulsation in humans [10]. We considered two potential explanations for our results. During apnea, the influence of the autonomic nervous system on the heart is characterized by co-activation of both branches of the autonomic nervous system and an increase in both sympathetic and parasympathetic outflow to the heart [11,12]. Increased parasympathetic outflow to the heart may result in decreased cardiac activity.

Alternatively, apnoea results in hypercapnia, hypoxia and augmented sympathetic activation, which in turn increases BP contribution to CBF [13,14]. Furthermore, hypercapnia dilates cerebral arteries [15-17] and can alter the pulse wave transmission characteristics of the cerebral vasculature by altering their Windkessel properties [17]. Progressive carbon dioxide retention results in the increase in intracranial pressure [18,19]. Increased intracranial pressure may, in turn, impair jugular outflow and does not allow for dampening the pulsation energy and actually exaggerates the pulsatile flow [20,21]. As a consequence of all factors mentioned above the relative cardiac contribution to BP cc-TQ oscillations may decrease. Interestingly, HR, together with carbon dioxide and BP, significantly modulate intracranial pressure [22].

Taken together, there is an increasing evidence that heart directly influences cerebral perfusion, brain microcirculation and intracranial pressure. Briefly discussed possibility of noninvasive assessment of pial artery function and intracranial pressure seems to be very well suited for OSA research. Recent advancement in physics, in particular the development of theory of chronotaxic systems [23,24], when applied to cardiovascular research, will most likely further increase our understanding about CBF control in human. The above described findings, coming mostly from young, healthy volunteers cannot be directly translated into OSA pathophysiology. Nevertheless, they may open new era in OSA research in the very near future.

References

1. Macey PM, Kumar R, Woo MA, Yan-Go FL, Harper RM. Heart rate responses to autonomic challenges in obstructive sleep apnea. PLoS One. 2013; 8: e76631.
2. Georgiadis D, Sievert M, Cencetti S, Uhlmann F, Krivokuca M, Zierz S, et al. Cerebrovascular reactivity is impaired in patients with cardiac failure. Eur Heart J. 2000; 21: 407-413.
3. Ogoh S, Tzeng YC, Lucas SJ, Galvin SD, Ainslie PN. Influence of baroreflex-mediated tachycardia on the regulation of dynamic cerebral perfusion during acute hypotension in humans. J Physiol. 2010; 588: 365-371.
4. Frydrychowski AF, Wszedybyl-Winklewska M, Bandurski T, Winklewski PJ. Flow-induced changes in pial artery compliance registered with a non-invasive method in rabbits. Microvasc Res. 2011; 82: 156-162.
5. Latka M, Turalska M, Glaubic-Latka M, Kolodziej W, Latka D, West BJ. Phase dynamics in cerebral autoregulation. Am J Physiol Heart Circ Physiol. 2005; 289: H2272-2279.
6. Stefanovska A. Coupled oscillators. Complex but not complicated cardiovascular and brain interactions. IEEE Eng Med Biol Mag. 2007; 26: 25-29.

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7. Li Z, Zhang M, Xin Q, Li J, Chen G, Liu F, et al. Correlation analysis between prefrontal oxygenation oscillations and cerebral artery hemodynamics in humans. Microvasc Res. 2011; 82: 304-310.

8. Cui R, Zhang M, Li Z, Xin Q, Lu L, Zhou W, et al. Wavelet coherence analysis of spontaneous oscillations in cerebral tissue oxyhemoglobin concentrations and arterial blood pressure in elderly subjects. Microvasc Res. 2014; 93: 14-20.

9. Winklewski PJ, Gruszecki M, Wolf J, Swierblewska E, Kunicka K, Wszedybyl-Winklewska M, et al. Wavelet transform analysis to assess oscillations in pial artery pulsation at the human cardiac frequency. Microvasc Res. 2015; 99: 86-91.

10. Frydrychowski AF, Guminski W, Rojewski M, Kaczmarek J, Juzwa W. Technical foundations for noninvasive assessment of changes in the width of the subarachnoid space with near-infrared transillumination-backscattering sounding (NIR-TBSS). IEEE Trans Biomed Eng. 2002; 49: 887-904.

11. Foster GE, Sheel AW. The human diving response, its function, and its control. Scand J Med Sci Sports. 2005; 15: 3-12.

12. Paton JF, Boscan P, Pickering AE, Nalivaiko E. The yin and yang of cardiac autonomic control: vago-sympathetic interactions revisited. Brain Res Brain Res Rev. 2005; 49: 555-565.

13. Battisti-Charbonney A, Fisher J, Duffin J. The cerebrovascular response to carbon dioxide in humans. J Physiol. 2011; 589: 3039-3048.

14. Mardimae A, Balaban DY, Machina MA, Battisti-Charbonney A, Han JS, Katznelson R, et al. The interaction of carbon dioxide and hypoxia in the control of cerebral blood flow. Pflugers Arch. 2012; 464: 345-351.

15. Vorstrup S, Henriksen L, Paulson OB. Effect of acetazolamide on cerebral blood flow and cerebral metabolic rate for oxygen. J Clin Invest. 1984; 74: 1634-1639.

16. Frydrychowski AF, Wszedybyl-Winklewska M, Guminski W, Lass P, Bandurski T, Winklewski PJ. Effects of acute hypercapnia on the amplitude of cerebrovascular pulsation in humans registered with a non-invasive method. Microvasc Res. 2012a; 83: 229-236.

17. Tzeng YC, MacRae BA, Ainslie PN, Chan GS. Fundamental relationships between blood pressure and cerebral blood flow in humans. J Appl Physiol. 2014; 117: 1037-1048.

18. Avezaat CJ, van Eijndhoven JH, Wyper DJ. Effects of hypercapnia and arterial hypotension and hypertension on cerebrospinal fluid pulse pressure and intracranial volume-pressure relationships. J Neurol Neurosurg Psychiatry. 1980; 43: 222-234.

19. Jennum P, Børgesen SE. Intracranial pressure and obstructive sleep apnea. Chest. 1989; 95: 279-283.

20. Bateman GA, Levi CR, Schofield P, Wang Y, Lovett EC. The venous manifestations of pulse wave encephalopathy: windkessel dysfunction in normal aging and senile dementia. Neuroradiology. 2008; 50: 491-497.

21. Frydrychowski AF, Winklewski PJ, Guminski W. Influence of acute jugular vein compression on the cerebral blood flow velocity, pial artery pulsation and width of subarachnoid space in humans. PLoS One. 2012b; 7: e48245.

22. Wszedybyl-Winklewska M, Wolf J, Swierblewska E, Kunicka K, Gruszecki M, Guminski W, et al. Pial artery and subarachnoid width response to apnea in normal humans. J Hypertens. [In press].

23. Suprunenko YF, Clemson PT, Stefanovska A. Chronotaxic systems: a new class of self-sustained nonautonomous oscillators. Phys Rev Lett. 2013; 111: 024101.

24. Suprunenko YF, Stefanovska, A. Generalized chronotaxic systems: Time-dependent oscillatory dynamics stable under continuous perturbation. Phys Rev E. 2014; 90: 032921.

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