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Impact of stress on the psychological functions and the psychosomatic cardiovascular conditions in humans: assessment by the heart rate variability method

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Abstract. *According to the neurovisceral integration model, several neural structures are involved in adaptations to psychoemotional stress, i.e. medial prefrontal cortex (mPFC), insular cortex, amygdala, hypothalamus, medullar autonomic centers. These structures are organized hierarchically and the higher centers control and inhibit the lower ones. The mPFC is involved in the regulation of cognitive functions, emotion and social cognition, and cardiovascular functions. It constantly inhibits the amygdala and the sympathoexcitatory subcortical circuits responding to stress. The flexible brain system involved in adaptation may be evaluated through the heart rate variability (HRV). Individuals with greater ability for emotion regulation depending on the environment and the goals set have been shown to have greater levels of resting HRV. This parameter may reflect the level at which affective conditions dynamically influence the peripheral autonomic nervous system (ANS). The HRV fluctuations correlate with some somatic and psychological disorders. It is lower in a number of psychiatric conditions and is associated with the risk factors of the cardiovascular morbidity and mortality. Psychological processes, such as emotional and social cognition, as well as psychosomatic conditions affecting the nervous and the cardiovascular system under stress may be easily evaluated by the physiological parameter heart rate variability.*

Key words: heart rate variability, stress, neurovisceral integration model

Background

Stress is a state of threatened homeostasis. The adaptive response to stress includes physiological and behavioral reactions, thus restoring the organism's equilibrium. The key systems activated under stress include cortical and subcortical neural structures, the autonomic nervous system and the endocrine system. The autonomic centers in the brainstem are controlled by the endbrain cortex and by the hypothalamus. The hypothalamus affects most autonomic and endocrine functions, and the emotional behavior.

Neural structures involved in stress responses

In accordance with the neurovisceral integration model (Thayer, J.F., Lane, R.D., 2000; Thayer, J.F., Lane, R.D., 2009), adaptations to environmental dynamics are determined by physiological, emotional, behavioral, cognitive, social, and environmental factors. This allows the adaptation systems to flexibly react to changes. A system of structures functions in the brain, which integrates the signals from the external and the internal environment and regulates the cognitive, perceptual, motor and autonomic processes. It constantly monitors the information inflow for the existence of threatening or favorable stimuli for the organism's functioning. The amygdala and the medial prefrontal cortex (mPFC) are of particular importance for this system. The latter plays a major role for the representation of both internal and external context in the mind and for the use of this information to regulate behavior and physiological processes. The mPFC is characterized by a number of cognitive functions (Cerqueira JJ, Mailliet F, et al., 2007), e.g. the sense of the self (Kelley, W.M., Macrae, C.N., et al., 2002; Northoff, G., Heinzel, et al., 2006) and emotional appraisal (Urry, H.L., van Reekum, C.M., Johnstone, T., et al., 2006; Wager, T.D., Hughes, B., Davidson, M., et al., 2008). It shapes behavior in accordance with superior inner goals set. It integrates information from various stimuli and generates behavioral and physiological responses, such as fear responses (Delgado, M.R., Nearing, K.I., Ledoux, J.E., Phelps, E.A., 2008; Milad, M.R., Wright, C.I., Orr, S.P., et al., 2007; Schiller, D., Levy, I., Niv, Y., et al., 2008.), heart-rate changes related to social threat (Wager, T.D., Waugh, C.E., Lindquist, M., et al., 2009b) and other stress responses (Lane, R.D., Wager, T.D., 2009). Its functions are performed through connectivity with the brainstem (Saper, C.B., 2002; Wager, T.D., Waugh, C.E., et al., 2009b). The mPFC is involved in the regulation of cardiovascular functions (Resstel LBM, Corrêa FM, 2006; Tavares RF, Corrêa FM, Resstel LBM, 2009a) and is morphologically modified under stress, as indicated by volume loss and dendritic atrophy (Cerqueira JJ, Taipa R, Uylings HB, Almeida OF, Sousa N., 2006; Cerqueira JJ, Almeida OF, Sousa N., 2008). This leads to an autonomic imbalance in favor of the sympathetic system and to stress-related cardiovascular diseases (Hilz MJ, Devinsky O, Szczepanska H, Borod JC, Marthol H, Tutaj M, 2006).

The hypothalamus, in particular the n. posterolateralis (NPL) and the n. paraventricularis (NPV), are important integrative centers of viscerosensory information (Allen GV, Cechetto DF, 1992; Allen GV, Cechetto DF., 1993). NPV is functionally involved in cardiovascular reactivity under stress (Tavares RF, Pelosi GG, Corrêa FMA, 2009в), regulates sympathetic activity (Kannan H, Hayashida Y, Yamashita H., 1989) and secrets corticotropin releasing factor that activates the hypothalamic–pituitary–adrenal axis (Kageyama K, Suda T., 2009).

The amygdala has efferent connections with the autonomic nervous system (ANS), the endocrine and other regulatory systems. It is recognized as a rapid sensor of potential threats and an intermediator of adaptive "fear" responses (LeDoux, J., 1996). The "default" response to uncertainty, surprise, and threat is the sympathoexcitatory activation commonly known as the "fight or flight" response (Thayer, J.F., Lane, R.D., 2009. ; Herry, C., Bach, D.R., Esposito, F., et al., 2007). The mPFC may also inhibit the amygdala's activity and reduce stress responses and fear behavior (Amat, J., Paul, E., Watkins, L.R., Maier, S.F., 2008; Quirk, G.J., Beer, J.S., 2006). Thus sympathoexcitatory, cardioacceleratory subcortical circuits responding to stress are under tonic inhibitory control by the prefrontal cortex (Thayer, J.F., 2006). In accordance with the Hughlings Jackson principle of hierarchical integration, higher brain structures constantly inhibit the lower and evolutionally older ones, while the latter are activated when the higher centers are inhibited, i.e. by disinhibition (Jackson, J.H., 1884). The ANS is also organized hierarchically. The inhibitive processes are of utmost importance for the adaptation and their deficit may lead to emotional and psychological disturbances (Beauchaine, T.P., 2001).

The ability to regulate emotion is closely related to the ability to flexibly shape perceptual and affective brain processes in response to changing contexts. Emotions represent a distillation of an individual's perception of personally relevant environmental interactions, including also the ability to respond to them (Frijda, N.H., 1986). An adequate emotional response represents a selection of an optimal response and inhibition of less appropriate responses from a variety of behavioral patterns.

The perception, analysis and generation of responses to the intentions, dispositions and behaviors of others are known as social cognition (Green, M.F., Penn, D.L., Bentall, R., et al., 2008). The recognition of facial expressions and of the likely mental states of other people are an important characteristic of social cognition and go beyond the traditional neurocognitive assessments of working memory, psychomotor speed, and attention (Bora E, Eryavuz A, Kayahan B, Sungu G, Veznedaroglu B., 2006). The mPFC and ANS are important neural circuitry involved in social cognition (Amodio, D.M., Frith, C.D., 2006; Appelhans, B.M., Luecken, L.J., 2006).

Porges' polyvagal theory (Porges, S.W., 2007) suggests that ANS is crucial for the survival, reproduction and social engagement of the species. An important role is played by the bidirectional connection between brain and body (through the nucleus ambiguus) and body and brain (via the nucleus tractus solitarius). When an individual is threatened, vagal tone is inhibited; when the surrounding environment is deemed safe, the vagal tone is increased thus inhibiting the sinoatrial node and promoting social behavior. The polyvagal theory suggests that optimal social adaptation, which includes also the recognition of emotion in faces, is facilitated by a calm physiological state (Porges, S., 2003).

The polyvagal theory and neurovisceral integration model highlight the important role that is played by the ANS in emotion recognition and social cognition. Efficient cardiac control facilitates more flexible engagement with the environment and more efficient emotion regulation (Appelhans, B.M., Luecken, L.J., 2006).

The proper functioning of the PFC is important for the precise evaluation of the stimuli as either threatening or safe, for preserving the system's integrity and for health protection. When the PFC is inhibited, the amygdala is disinhibited and the sympathetic nervous system is activated. If this state is prolonged, it produces excessive exhaustion of the system components and is characterized by McEwen as "allostatic status" (McEwen, B.S., 1998).

HRV AS A BRAIN FUNCTION INDEX

Neurocardiology examines the interconnections between the brain centers and the cardiovascular system. It is widely recognized that regulation of cardiac function is dependent on medullary centers, namely the nucleus of the solitary tract (NTS) and the rostroventrolateral medulla (RVLM) (Saper CB, 2004). NTS receives afferents from baroreceptors and the visceral sensorial information derived from cranial nerves, while the RVLM is mainly composed by excitatory neurons that are responsible for the generation of the sympathetic response. Modern research shows that cardiac regulation is also dependent on supramedullary regions.

The insular cortex is a critical area in controlling the sympathetic and the parasympathetic tones. It is estimated that stroke victims with insular damage are associated with a higher risk of autonomic disbalance and cardiovascular sudden death (Tokgozoglu SL, Batur MK, Topcuoglu MA, Saribas O, Kes S, Oto A., 1999). The loss of the inhibition exerted by the insular cortex over inferior areas of cardiovascular centers results in rhythm instability of the heart (Oppenheimer S., 1993).

Based on their studies, Thayer and Brosschot (Thayer, J.F., Brosschot, J.F., 2005) and Thayer and Lane (Thayer, J.F., Lane, R.D., 2000; Thayer, J.F., Lane, R.D., 2009) have proposed that the flexible brain system involved in adaptation may be evaluated through heart rate variability (HRV). This is a method for evaluating the quick and slow heart rate changes specified as time and frequency parameters. It allows for distinguishing the effects of both parts of the ANS on the sinoatrial node and for estimating the parasympathetic effects on the heart.

Dynamical systems are characterized by multiple processes influencing each other. When processes mutually limit one another, the system as a whole tends to fluctuate within a range of states. A balance between the various processes is achieved and thus the system can respond flexibly to a number of stimuli (Thayer, J.F., Sternberg, E., 2006). In case of a disbalance, a particular process comes to dominate and the system becomes more rigid, unresponsive and limited to a specific type of reaction. When applied to the heart, this model explains why the healthy heart is characterized by a high HRV, whereas a diseased heart shows a lower one.

The basic idea of the Neurovisceral Integration Model is that HRV is not just a heart function index, but it also reflects the activity of the brain integrative system for the dynamic adaptation of organisms under stress. The correlation between HRV and PFC activity shows that this brain area is part of the descending "visceromotor" system that controls the autonomic nervous system and the neuroendocrine functions based on the relevant emotional context. Disturbances in the PFC activity results in disinhibition of the amygdala and the cardioacceleratory center, with increased HR and decreased HRV.

Connection between HRV and emotional and social cognition

Emotion regulation depends on the interaction between the mPFC and the amygdala and is linked to the HRV (Appelhans, B.M., Luecken, L.J., 2006; Thayer, J.F., Brosschot, J.F., 2005). Individuals with greater ability for emotion regulation depending on the environment and the goals set have been shown to have greater levels of resting HRV (Appelhans, B.M., Luecken, L.J., 2006; Thayer, J.F., Lane, R.D., 2009). During successful emotion regulation by way of reassessment or inhibition, the HRV appears to be increased (Butler, E.A., Wilhelm, F.H., Gross, J.J., 2006). This is accompanied by cerebral blood flow changes in structures associated with emotional regulation and inhibitory processes (Lane, R.D., Wager, T.D., 2009). Individuals with low resting HRV show delayed recovery of the parameters of the cardiovascular, endocrine, and immune systems upon psychoemotional stress as compared to those with higher levels of resting HRV (Weber, C.S., Thayer, J.F., Rudat, M., et al., 2010). Thus, individuals with higher resting levels of HRV appear more able to produce context appropriate responses and show quicker recovery after the stressor has ended. The HRV is therefore a parameter showing organisms' stress levels as well as their endurance, flexibility and adaptivity. HRV may as well be a parameter for the activity of the "PFC – brain stem – physiological reactions" axis and may reflect, in psychological terms, the level at which affective conditions dynamically influence the peripheral ANS. The inhibitory role of the PFC is also important for cognition (Thayer, J.F., Lane, R.D., 2000).

Emotional intelligence can be evaluated by means of an RMET (the Reading the Mind in the Eyes Test – (Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., Plumb, I., 2001). Poor performance in the RMET is associated with autism (Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., Plumb, I., 2001) and depression (Lee, L., Harkness, K.L., Sabbagh, M.A., Jacobson, J.A., 2005). The oxytocin enhances the HRV (Kemp, A.H., Quintana, D.S., Kuhnert, R.-L., et al., 2012a.; Norman, G.J., Cacioppo, J.T., Morris, J.S., et al., 2011) and the RMET performance with mentally healthy people (Domes, G., Heinrichs, M., Michel, A., Berger, C., Herpertz, S.C., 2007) and youth with autism spectrum disorders (Guastella AJ, Einfeld SL, Gray KM, et al., 2010).

HRV IN CASE OF PSYCHIATRIC DISORDERS

The HRV fluctuations correlate with some somatic and psychological disorders (Beauchaine, T.P., 2001; Friedman, B.H., 2007). HRV is decreased in case of a number of psychiatric conditions such as depression, anxiety disorder and alcoholism (Thayer, J.F., Lane, R.D., 2000; Friedman, B.H., 2007; Kemp, A.H., Quintana, D.S., et al., 2012b.; Kemp, A.H., Quintana, D.S., et al., 2010; Ingjaldsson, J.T., Laberg, J.C., Thayer, J.F., 2003). This is an indicator of inadequate autonomic regulation and poor social adaptation in case of such conditions (Baron-Cohen, S., Ring, H.A., et al., 2000; Demenescu, L.R., Kortekaas, R., Boer den, J.A., Aleman, A., 2010; Hasin, D.S., Stinson, F.S., Ogburn, E., Grant, B.F., 2007). It is therefore estimated that HRV is lower in children with autism (Van Hecke, A.V., Lebow, J., Bal, E.,et al., 2009).

The parasympathetic tone to the heart at rest is important and provides cardiac stability and improved reactivity (Levy, M.N., 1990; Verrier, R.L., 1987). The high frequencies of the HRV (HF) reflect the Respiratory sinus arrhythmia (RSA) which is parasympathetically mediated (Thayer, J.F., Lane, R.D., 2000). Anxiety disorders are associated with lower levels of RSA and HRV (Llera, S.J., Newman, M.G., 2010; Hofmann, S.G., Moscovitch, D.A.,et al., 2005), thus showing a limited reactivity of the parasympathetic nervous system. "Physiological rigidity" (Hoehn-Saric, R., McLeod, D.R., Funderburk, F., Kowalski, P., 2004) and decreased adaptivity to environmental demands (Hoehn-Saric, R., McLeod, D.R., 2000) are observed. The increased heart rate and the decreased HRV are also due to the chronically increased sympathetic tone in case of such conditions (Grippo AJ, Johnson AK, 2009; Grippo AJ, Moffitt JA, Johnson AK., 2002). This explains the relationship between worry, poor cardiovascular health (Kubzansky, L.D., Kawachi, I., Spiro 3rd, A., et al., 1997) and the overall physical morbidity and mortality (Lipsitz, L.A., Goldberger, A.L., 1992; Peng, C.K., Buldyrev, S.V.,et al., 1994). The cortisol levels correlate with the increased HR and the decreased HRV under stress (Looser RR, Metzenthin P, Helfricht S, et al., 2010).

HRV AS AN INDICATOR OF PSYCHOSOMATIC EFFECTS ON THE CARDIOVASCULAR SYSTEM

The decreased HRV is associated with the risk factors of the cardiovascular morbidity and mortality, including psychosocial stress (Thayer, J.F., Lane, R.D., 2007; Thayer, J.F., Yamamoto, S.S., Brosschot, J.F., 2010b; Thayer, J.F., Hansen, A.L., Johnsen, B.H., 2010a).

Furthermore, anxiety disorders are associated with a higher risk of cardiovascular diseases such as myocardial ischemia, sudden cardiac death, and myocardial infarction (Frasure-Smith, N., Lesperance, F., 2008., Martens, E.J., de Jonge, P., Na, B., Cohen, B.E., Lett, H., Whooley, M.A., 2010).

Diminished vagally mediated RSA has been shown to correlate with congestive heart failure, arterial hypertension (Curtis BM, O'Keefe JH Jr., 2002) and cardiovascular morbidity (Stys, A., Stys, T., 1998). It is a significant risk factor for all-cause mortality (Tsuji, H., Venditti Jr., F.J., Manders, et al., 1994).

Chronic activation of the sympathetic system and/or decreased activity of the parasympathetic nervous system are also characteristic of cardiovascular diseases (Fauchier L, Babuty D, Cosnay P, Fauchier JP., 1999; Joyner MJ, Charkoudian N, Wallin BG., 2010). The sympathetic nervous system contributes to endothelial dysfunction, hypertension and atherosclerosis, insulin resistance, and dyslipidemia; it induces left ventricular hypertrophy, increases the incidence of arrhythmia, and promotes renal dysfunction by stimulating sodium and fluid retention, glomerulosclerosis and activation of the renin–angiotensin–aldosterone system (RAAS) (Lambert GW, Straznicky NE, Lambert EA, Dixon JB, Schlaich MP., 2010). The sympathetic nervous system is persistently activated in patients with heart failure and leads to its progression (Kaye D, Esler M., 2005).

Centrally activated RAAS participates in the development of hypertension and heart failure [84]. Apart from the effects on the vascular tone and the renal ion transport, RAAS regulates the metabolism and the cardiac rhythm. It modulates membrane and sarcoplasmatic reticulum ion channels and has therefore a proarrhythmic effect (Iravanian S, Dudley SC., 2008). The altered hormonal milieu (hyperleptinemia, hyperinsulinemia,hy percortisolemia and hyperaldosteronism) acts back in the brain and maintains the activation of the sympathetic system (Mark AL, Correia MLG, Rahmouni K, Haynes WG., 2002; Landsberg L., 1986). Aldosterone and angiotensin II also regulate the sympathetic tone by their central action in the RVLM.

Conclusions

Recent data shows that stress-related pathology and depression are found to represent not only neuropsychological disorders, but also factors leading to cardiovascular diseases. The heart–brain interaction is known to be bidirectional, but it seems that regulation of the brain over heart and vessels is more prevalent as shown by the increased risk of cardiovascular diseases in case of neuropsychological disorders, such as anxiety disorders, depression, epilepsy or stroke.

The proper functioning of the brain structures responding in case of psychoemotional stress influence the mental health and the social adaptation of individuals, as well as the autonomic balance and the cardiovascular system, respectively. These psychosomatic conditions may be easily and non-invasively evaluated by the physiological parameter heart rate variability. Considering the significant psychoemotional stress characteristic of the contemporary social environment and the increasing cardiovascular morbidity and mortality, the analysis of the relationships between the psychological and the neuroendocrine factors and the heart health is of utmost importance and will be a subject of future studie

References

Thayer, J.F., Lane, R.D. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. J. Affect. Disord. 61, 201–216.

Thayer, J.F., Lane, R.D. (2009). Claude Bernard and the heart brain connection: further elaboration of a model of neurovisceral integration. Neurosci. Biobehav. Rev. 33, 81–88.

Cerqueira, J.J., Mailliet, F., Almeida, OFX, Jay, T.M., Sousa, N. (2007). Theprefrontal cortex as a key target of the maladaptive response to stress. J Neurosci 2007;27:2781–7.

Kelley, W.M., Macrae, C.N., Wyland, C.L., Caglar, S., Inati, S., Heatherton, T.F. (2002). Finding the self? An event-related fMRI study. J. Cogn. Neurosci. 14, 785–794.

Northoff, G.., Heinzel, A., de Greck, M., Bermpohl, F., Dobrowolny, H., Panksepp, J. (2006). Self-referential processing in our brain—a meta-analysis of imaging stud- ies on the self. Neuroimage 31, 440–457.

Urry, H.L., van Reekum, C.M., Johnstone, T., Kalin, N.H., Thurow, M.E., Schaefer, H.S., Jackson, C.A., Frye, C.J., Greischar, L.L.,

Alexander, A.L., Davidson, R.J. (2006). Amygdala and ventromedial prefrontal cortex are inversely coupled during regulation of negative affect and predict the diurnal pattern of cortisol secretion among older adults. J. Neurosci. 26, 4415–4425.

Wager, T.D., Hughes, B., Davidson, M., Lindquist, M.L., Ochsner, K.N. (2008). Prefrontal–subcortical pathways mediating successful emotion regulation. Neuron 59, 1037–1050.

Delgado, M.R., Nearing, K.I., Ledoux, J.E., Phelps, E.A., (2008).Neural circuitry underly- ing the regulation of conditioned fear and its relation to extinction. Neuron 59, 29–838.

Milad, M.R., Wright, C.I., Orr, S.P., Pitman, R.K., Quirk, G.J., Rauch, S.L., (2007). Recall of fear extinction in humans activates the ventromedial prefrontal cortex and hippocampus in concert. Biol. Psychiatry 62, 446–454.

Schiller, D., Levy, I., Niv, Y., LeDoux, J.E., Phelps, E.A., (2008). From fear to safety and back: reversal of fear in the human brain. J. Neurosci. 28, 11517–11525.

Wager, T.D., Waugh, C.E., Lindquist, M., Noll, D.C., Fredrickson, B.L., Taylor, S.F., (2009b). Brain mediators of cardiovascular responses to social threat. Part I: reciprocal dorsal and ventral subregions of the medial prefrontal cortex and heart-rate reactivity. Neuroimage 47, 821–835.

Lane, R.D., Wager, T.D., (2009). The new field of brain–body medicine: what have we learned and where are we headed? Neuroimage 47, 1135–1140.

Saper, C.B., (2002). The central autonomic nervous system: conscious visceral perception and autonomic pattern generation. Annu. Rev. Neurosci. 25, 433–469.

Resstel LBM, Corrêa FM. (2006). Involvement of the medial Prefrontal cortex in central cardiovascular modulation in the rat. Auton Neurosci;126–127:130–8, Tavares, R.F., Corrêa, F.M., Resstel, L.B.M. (2009a). Opposite role of infralimbic and prelimbic cortex in the tachycardiac response evoked by acute restraint stress in rats. J Neurosci Res (2009a).;87:2601–7.

Cerqueira, J.J., Taipa, R., Uylings, H.B., Almeida, O.F., Sousa, N. Specific configuration of dendritic degeneration in pyramidal neurons of the medial prefrontal cortex induced by differing corticosteroid regimens. Cereb Cortex. 2007 Sep;17(9):1998-2006. Epub 2006 Nov 2.

Cerqueira, J.J., Almeida, O.F., Sousa, N. (2008). The stressed prefrontal cortex. Left? Right! Brain Behav Immun. 2008 Jul;22(5):630-8. doi: 10.1016/j.bbi.2008.01.005. Epub 2008 Feb 20. Review.

Hilz, M.J., Devinsky, O., Szczepanska, H., Borod, J.C., Marthol, H., Tutaj, M. (2006). Right ventromedial prefrontal lesions result in paradoxical cardiovascular activation with emotional stimuli. Brain 2006;129:3343–55,

Allen, G.V., Cechetto, D.F. (1992). Functional and anatomical organization of cardiovascular pressor and depressor sites in the lateral hypothalamic area: I. Descending projections. J Comp Neurol. 1992 Jan 15;315(3):313-32.

Allen, G.V., Cechetto, D.F. (1993). Functional and anatomical organization of cardiovascular pressor and depressor sites in the lateral hypothalamic area. II. Ascending projections. J Comp Neurol. 1993 Apr 15;330(3):421-38.

Tavares, R.F., Pelosi, G.G., Corrêa, FMA. (2009в). The paraventricular nucleus of the hypothalamus is involved in cardiovascular responses to acute restraint stress in rats. Stress 2009в;12:178–85,

Kannan, H., Hayashida, Y., Yamashita, H. (1989). Increase in sympathetic outflow by paraventricular nucleus stimulation in awake rats. Am J Physiol 1989;256: R1325–30.

Kageyama, K., Suda, T. (2009). Regulatory mechanisms underlying corticotropin-releasing factor gene expression in the hypothalamus.

Endocr J 2009;56:335–44,

LeDoux, J., (1996). Emotional networks and motor control: a fearful view. Prog. Brain Res. 107, 437–446.

Herry, C., Bach, D.R., Esposito, F., Di Salle, F., Perrig, W.J., Scheffler, K., Luthi, A., Seifritz, E., (2007). Processing of temporal unpredictability in human and animal amygdala. J. Neurosci. 27, 5958–5966.

Amat, J., Paul, E., Watkins, L.R., Maier, S.F. (2008). Activation of the ventral medial prefrontal cortex during an uncontrollable stressor reproduces both the imme- diate and long-term protective effects of behavioral control. Neuroscience 154,1178–1186.

Quirk, G.J., Beer, J.S. (2006). Prefrontal involvement in the regulation of emotion: convergence of rat and human studies. Curr. Opin. Neurobiol. 16, 723–727.

Thayer, J.F., (2006). On the importance of inhibition: central and peripheral manifestations of nonlinear inhibitory processes in neural systems. Dose–Response: Publ. Int. Hormesis Soc. 4, 2–21.

Jackson, J.H., (1884). The Croonian Lectures on evolution and dissolution of the nervous system. Br. Med. J., 703–707.

Beauchaine, T.P. (2001). Vagal tone, development, and Gray‘s motivational theory: toward an integrated model of autonomic nervous system functioning in psychopathology. Development and Psychopathology 13, 183–214.

Frijda, N.H., (1986). The Emotions. Ed. de la Maison des Sciences de l'Homme.

Green, M.F., Penn, D.L., Bentall, R., Carpenter, W.T., Gaebel, W., Gur, R.C., Kring, A.M., Park, S., Silverstein, S.M., Heinssen, R., (2008).

Social cognition in schizophrenia: an NIMH workshop on definitions, assessment, and research opportunities. Schizophrenia Bulletin: Presented at the Schizophrenia Bulletin, pp. 1211–1220.

Bora, E., Eryavuz, A., Kayahan, B., Sungu, G., Veznedaroglu, B. (2006). Social functioning, theory of mind and neurocognition in

outpatients with schizophrenia; mental state decoding may be a better predictor of social functioning than mental state reasoning. Psychiatry Res. 2006 Dec 7;145(2-3):95-103. Epub 2006 Oct 30.

Amodio, D.M., Frith, C.D. (2006). Meeting of minds: the Medial frontal cortex and social cognition. Nature Reviews Neuroscience 7, 268–277.

Appelhans, B.M., Luecken, L.J. (2006). Heart rate variability as an index of regulated emotional responding. Rev. Gen. Psychol. 10, 229–240.

Porges, S.W. (2007). The polyvagal perspective. Biological Psychology 74, 116–143.

Porges, S. (2003). Social engagement and attachment: a phylogenetic perspective. Annals of the New York Academy of Sciences 1008, 31–47.

McEwen, B.S. (1998). Stress, adaptation, and disease. Allostasis and allostatic load. Ann. N. Y. Acad. Sci. 840, 33–44.

Saper, C.B. (2004). Autonomic nervous system. In: Paxinos G, editor. The rat nervous system. 3rd edn. San Diego: Elsevier Academic Press; 2004. p. 761–96.

Tokgozoglu, S.L., Batur, M.K., Topcuoglu, M.A., Saribas, O., Kes, S., Oto, A. (1999). Effects of stroke localization on cardiac autonomic balance and sudden death. Stroke 1999; 30: 1307.

Oppenheimer, S. (1993). The anatomy and physiology of cortical mechanisms of cardiac control. Stroke 1993;24:13–5.

Thayer, J.F., Brosschot, J.F. (2005). Psychosomatics and psychopathology: looking up and down from the brain. Psychoneuroendocrinology 30, 1050–1058.

Thayer, J.F., Sternberg, E. (2006). Beyond heart rate variability: vagal regulation of allostatic systems. Ann. N. Y. Acad. Sci. 1088, 361–372.

Butler, E.A., Wilhelm, F.H., Gross, J.J., (2006). Respiratory Sinus arrhythmia, emo- tion, and emotion regulation during social interaction. Psychophysiology 43, 612–622.

Weber, C.S., Thayer, J.F., Rudat, M., Wirtz, P.H., Zimmermann Viehoff, F., Thomas, A., Perschel, F.H., Arck, P.C., Deter, H.C., (2010). Low vagal tone is associated with impaired post stress recovery of cardiovascular, endocrine, and immune markers. Eur. J. Appl. Physiol. 109, 201–211.

Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., Plumb, I. (2001). The "Reading the Mind in the Eyes" test revised version: a study with normal adults, and adults with Asperger syndrome or high-functioning autism. Journal of Child Psychology and Psychiatry 42, 241–251.

Lee, L., Harkness, K.L., Sabbagh, M.A., Jacobson, J.A. (2005). Mental state decoding abilities in clinical depression. Journal of Affective Disorders 86, 247–258.

Kemp, A.H., Quintana, D.S., Kuhnert, R.-L., Griffiths, K., Hickie, I.B., Guastella, A.J. (2012a). Oxytocin increases heart rate variability in humans at rest: Implications for social approach-related motivation and capacity for social engagement. PLoS ONE 7 (8), e44014.

Norman, G.J., Cacioppo, J.T., Morris, J.S., Malarkey, W.B., Berntson, G.G., DeVries, A.C. (2011). Oxytocin increases autonomic cardiac control: moderation by loneliness. Biological Psychology 86, 174–180.

Domes, G.., Heinrichs, M., Michel, A., Berger, C., Herpertz, S.C. (2007). Oxytocin improves "mind-reading" in humans. Biological Psychiatry 61, 731–733.

Guastella, A.J., Einfeld, S.L., Gray, K.M., Rinehart, N.J., Tonge, B.J., Lambert, T.J., Hickie, I.B. (2010). Intranasal oxytocin improves emotion recognition for youth with autism spectrum disorders. Biol Psychiatry. 2010 Apr 1;67(7):692-4.

Friedman, B.H. (2007). An autonomic flexibility-neurovisceral integration model of anxiety and cardiac vagal tone. Biological Psychology 74, 185–199.

Kemp, A.H., Quintana, D.S., Felmingham, K.L., Matthews, S., Jelinek, H.F. (2012b). Depression, comorbid anxiety disorders, and heart rate variability in physically healthy, unmedicated patients: implications for cardiovascular risk. PLoS One 7 (2), e30777.

Kemp, A.H., Quintana, D.S., Gray, M.A., Felmingham, K.L., Brown, K., Gatt, J.M. (2010). Impact of depression and antidepressant treatment on heart rate variability: a review and meta-analysis. Biological Psychiatry 67, 1067–1074.

Ingjaldsson, J.T., Laberg, J.C., Thayer, J.F. (2003). Reduced heart rate variability in chronic alcohol abuse: relationship with negative mood, chronic thought suppression, and compulsive drinking. Biological Psychiatry 54, 1427–1436.

Baron-Cohen, S., Ring, H.A., Bullmore, E.T., Wheelwright, S., Ashwin, C., Williams, S.C.R. (2000). The amygdala theory of autism. Neuroscience and Biobehavioral Reviews 24, 355–364.

Demenescu, L.R., Kortekaas, R., Boer den, J.A., Aleman, A. (2010). Impaired attribution of emotion to facial expressions in anxiety and major depression. PLoS ONE 5, e15058.

Hasin, D.S., Stinson, F.S., Ogburn, E., Grant, B.F. (2007). Prevalence, correlates, disability, and comorbidity of DSM-IV alcohol abuse and dependence in the United States: results from the National Epidemiologic Survey on Alcohol and Related Conditions. Archives of General Psychiatry 64, 830–842.

Van Hecke, A.V., Lebow, J., Bal, E., Lamb, D., Harden, E., Kramer, A., Denver, J., Bazhenova, O., Porges, S.W., (2009).

Electroencephalogram and heart rate regulation to familiar and unfamiliar people in children with autism spectrum disorders. Child Development 80, 1118–1133.

Levy, M.N. (1990). Autonomic interactions in cardiac control. Annals of the New York Academy of Sciences 601, 209–221.

Verrier, R.L. (1987). Mechanisms of behaviorally induced arrhythmias. Circulation 76 (1 Pt 2), I48–I56.

Llera, S.J., Newman, M.G.. (2010). Effects of worry on physiological and subjective reactivity to emotional stimuli in generalized anxiety disorder and non-anxious control participants. Emotion 10 (5), 640.

Hofmann, S.G., Moscovitch, D.A., Litz, B.T., Kim, H.-J., Davis, L.L., Pizzagalli, D.A., (2005). The worried mind: autonomic and prefrontal activation during worrying. Emotion 5 (4), 464–475.

Hoehn-Saric, R., McLeod, D.R., Funderburk, F., Kowalski, P. (2004). Somatic symptoms and physiologic responses in generalized anxiety disorder and panic disorder: an ambulatory monitor study. Archives of General Psychiatry 61 (9), 913–921.

Hoehn-Saric, R., McLeod, D.R. (2000). Anxiety and arousal: Physiological changes and their perception. Journal of Affective Disorders 61 (3), 217–224.

Grippo, A.J., Johnson, A.K. (2009). Stress, depression and cardiovascular dysregulation: a review of neurobiological mechanisms and the integration of research from preclinical disease models. Stress 2009;12:1–21.

Grippo, A.J., Moffitt, J.A., Johnson, A.K. (2002). Cardiovascular alterations and autonomic imbalance in an experimental model of depression. Am J Physiol Regul Integr Comp Physiol 2002;282:R1333–41,

Kubzansky, L.D., Kawachi, I., Spiro 3rd, A., Weiss, S.T., Vokonas, P.S., Sparrow, D. (1997). Is worrying bad for your heart? A prospective study of worry and coronary heart disease in the Normative Aging Study. Circulation 95 (4), 818–824.

Lipsitz, L.A., Goldberger, A.L. (1992). Loss of 'complexity' and aging. JAMA 267 (13), 1806.

Peng, C.K., Buldyrev, S.V., Hausdorff, J.M., Havlin, S., Mietus, J.E., Simons, M., Goldberger, A.L. (1994). Non-equilibrium dynamics as an indispensable characteristic of a healthy biological system. Integrative Psychological and Behavioral Science 29 (3), 283–293.

Looser, RR., Metzenthin, P., Helfricht, S., Kudielka, B.M., Loerbroks, A., Thayer, J.F., Fischer, J.E. (2010). Cortisol is significantly correlated with cardiovascular responses during high levels of stress in critical care personnel. Psychosom Med. 2010 Apr;72(3):281-9.

Thayer, J.F., Lane, R.D., (2007). The role of vagal function in the risk for cardiovascular disease and mortality. Biol. Psychol. 74, 224–242.

Thayer, J.F., Yamamoto, S.S., Brosschot, J.F. (2010b). The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. Int. J. Cardiol. 141, 122–131.

Thayer, J.F., Hansen, A.L., Johnsen, B.H. (2010a). The non invasive assessment of autonomic influences on the heart using impedance cardiography and heart rate variability. In: Steptoe, A. (Ed.), Handbook of Behavioral Medicine: Methods and Applications. Springer, New York.

Frasure-Smith, N., Lesperance, F. (2008). Depression and anxiety as predictors of 2-year cardiac events in patients with stable coronary artery disease. Archives of General Psychiatry 65 (1), 62.

Martens, E.J., de Jonge, P., Na, B., Cohen, B.E., Lett, H., Whooley, M.A., (2010). Scared to death? Generalized anxiety disorder and cardiovascular events in patients with stable coronary heart disease: the heart and soul study. Archives of General Psychiatry 67 (7), 750–758.

Curtis, B.M., O'Keefe, J.H. Jr. (2002). Autonomic tone as a cardiovascular risk factor: the dangers of chronic fight or flight. Mayo Clin Proc. 2002 Jan;77(1):45-54.

Stys, A., Stys, T. (1998). Current clinical applications of heart Rate variability. Clinical Cardiology 21 (10), 719–724.

Tsuji, H., Venditti Jr., F.J., Manders, E.S., Evans, J.C., Larson, M.G., Feldman, C.L., Levy, D. (1994). Reduced heart rate variability and mortality risk in an elderly cohort. The Framingham Heart Study. Circulation 90 (2), 878–883

Fauchier, L., Babuty, D., Cosnay, P., Fauchier, J.P. (1999). Prognostic value of heart rate variability for sudden death and major arrhythmic events in patients with idiopathic dilated cardiomyopathy. J Am Coll Cardiol 1999;33:1203–7.

Joyner, M.J., Charkoudian, N., Wallin, B.G. (2010). Sympathetic nervous system and blood pressure in humans: individualized patterns of regulation and their implications. Hypertension 2010;56:10–6,

Lambert, G.W., Straznicky, N.E., Lambert, E.A., Dixon, J.B., Schlaich, M.P. (2010). Sympathetic nervous activation in obesity and the metabolic syndrome: causes, consequences and therapeutic implications. Pharmacol Ther 2010;126:159–72

Kaye, D., Esler, M. (2005). Sympathetic neuronal regulation of the heart in aging and heart failure. Cardiovasc Res 2005;66:256–64,

Francis, J., Sguang, Wei, Weiss, R.M., Felder, R.B. (2004). Brain angiotensin-converting enzyme activity and autonomic regulation in heart failure. Am J Physiol Heart Circ Physiol 2004;287:H2138–46.

Iravanian, S., Dudley, S.C. (2008). The renin–angiotensin–aldosterone system (RAAS) and Cardiac arrhythmias. Heart Rhythm 2008;5:S12 7.

Mark, A.L., Correia, M.L.G., Rahmouni, K., Haynes, W.G.. (2002). Selective leptin resistance: a new concept in leptin physiology with cardiovascular implications. J Hypertens 2002;20:1245–50.

Landsberg, L. (1986). Diet, obesity and hypertension: an hypothesis involving insulin, the sympathetic nervous system, and adaptive thermogenesis. Q J Med 1986;61: 1081–90.

Влияние на стреса върху психологичните функции и психосоматичните сърдечно-съдови явления при хора: оценка чрез вариабилността на сърдечната честота

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Резюме. *Според невровисцералния интегративен модел, определени нервни структури участват в реакциите на стрес: медиалната префронтална кора (мПФК), инсуларната кора, амигдалата, хипоталамусът и медуларните вегетативни центрове. Тези структури са организирани йерархично и по-високо разположените контролират и инхибират по-ниско разположените. мПФК регулира също когнитивните функции, емоционалните и социални когнитивни процеси и сърдечно-съдовите функции. Тя подтиска тонично амигдалата и подкоровите нервни мрежи, активиращи симпатикуса и медииращи стресовите реакции. Тези нервни структури, участващи в адаптацията, могат да бъдат оценени чрез метода вариабилност на сърдечната честота (ВСЧ). Индивиди с по-големи възможности за емоционална регулация в зависимост от околните условия и поставените цели, имат по-висока ВСЧ в покой. Този показател отразява степента, в която емоционалните състояния динамично повлияват периферната вегетати вна нервна система. ВСЧ корелира с някои соматични и психологични нарушения. Тя е по-ниска при някои психиатрични заболявания и е свързана с рисковите фактори на сърдечно-съдовата заболеваемост и смъртност. ВСЧ може да бъде индикатор за психологични процеси като емоционален и социален когнитивен процес и за психосоматичните влияния върху нервната и сърдечно-съдовата система*

Ключови думи: вариабилност на сърдечната честота, стрес, невровисцерален интегративен модел

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