THE ASSESSMENT OF THE AUTONOMIC RESPONSE TO ACUTE STRESS USING ELECTRODERMAL ACTIVITY

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Abstract

The response of autonomic nervous system to mental stress is currently studied as a key role factor in the pathophysiology of stress related diseases. Altered autonomic regulation can result in increased morbidity, potentially affecting (directly or indirectly) any of the organs. Cardiovascular system (CVS) is one of the most sensitive systems to the effect of autonomic outputs. The predictive value of the laboratory stress tests was proved in several studies with CVS pathology. In this study we aimed to assess the autonomic reactivity to different mental stressors (cognitive and emotional) in healthy subjects using electrodermal activity (EDA) as a sensitive psychophysiological marker of sympathetic activity. We found significantly increased EDA in response to all the mental tasks with decrease of the values during recovery periods. However, EDA did not return to the baseline values during recovery periods, potentially indicating the sympathetic arousal during complete stress profile protocol. We suggest EDA presents a well applicable marker of the sympathetic activation, offering a different information about central regulation processes regarding the sympathetic activity compared to cardiac autonomic indices.

Key words: mental stress, electrodermal activity, autonomic regulation, emotions, sympathetic reactivity

INTRODUCTION

Stress can be characterized as a state of disharmony or threatened homeostasis, evoking physiologically and behaviorally adaptive responses, which can produce a stress syndrome when the threat to homeostasis exceeds a threshold (1). The effect of mental stress depends on cognitive processing by cortical (mainly orbitofrontal and medial prefrontal) areas in cooperation with limbic system. Following modulation of the hypothalamus and brainstem activity results in modification of the autonomic, endocrine and somatomotoric nervous system (2). It is assumed that the hypothalamic-pituitary–adrenal axis and the sympathoadrenal system are the major components linking stress and stress-related diseases (3).

The autonomic nervous system (ANS) reactivity to stress can be studied using invasive or noninvasive methods. Invasive methods include recording of the peripheral neurons activity using microneurography, pharmacokinetic methods or measuring the plasma concentrations of the biomarkers of sympathoadrenal activation: catecholamines, their degradation products metanephrines (MNs) or chromogranin A (CgA). Besides the methodological difficulties and ethical aspects, there is a disadvantage of possible stress effect of the procedure itself. Therefore, noninvasive markers are preferred.

The noninvasive methods include both biochemical and physiological approach. From the biochemical methods it is possible to analyze substances excreted in saliva and urine. Salivary alpha-amylase is released by acinar cells innervated by both sympathetic and parasympathetic nerve fibers (4). It seems to be a valid and reliable stress marker, probably not closely related to other biological markers, thus providing a useful additional parameter of stress reaction (5). The amylase showed better sensitivity to psychological stress and

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shorter latency than salivary cortisol (6). The urine catecholamines controversial stability can potentially produce the preanalytical bias (7) in contrast to MNs, which don't require special conditions during processing (8). The concentration of CgA originating from secretory granules of endocrine cells and cosecreted with hormones (e.g. catecholamines)(9) can be assessed in saliva, serum or urine. The applicability of saliva CgA seems to be similar to plasma samples (10), urine values are less accurate (11).

The psychophysiological methods are based on the monitoring and analysis of basic physiological parameters such as the heart rate or blood pressure. For example, linear and nonlinear analyses of heart rate variability (HRV) were sensitive to detect a physiological shift of dynamic sympathovagal balance – decreased parasympathetic activity (vagal withdrawal) associated with sympathetic activation – in response to acute mental stress (12). Other studies revealed the significant effect of mental stress on blood pressure variability (13, 14, 15).

Electrodermal activity (EDA) is studied as a noninvasive sympathetic index in psychophysiological research. It was applied as a marker of autonomic arousal during mental stress with the aim to assess the effect of beta-blocking medications on cardiovascular reactivity (16). Thus, cholinergic mediation of EDA provides an advantage of applicability even under conditions of beta-blockade. It provides relatively direct presentation of sympathetic activity in contrast to most autonomic functions regulated by both ANS branches. The electrodermal responses are well distinguishable – it is possible to differentiate a reaction to even single presentation of stimulus (17) and to discriminate the effect of stress from response to cognitive load (18).

A potential disadvantage is the fact, that EDA presents information only about cholinergic part of sympathetic system. Further, it is influenced by multiple nervous processes (emotions, attention, activation…). Therefore, it can be difficult to control the experiment conditions and focus on the effect of a single variable. However, this can be as well an advantage in terms of applicability to study a wide range of psychophysiological states (17).

Based on these studies, we aimed to study electrodermal activity in response to different mental stressors in healthy subjects.

METHODS

We examined 20 students (males, age 22.9±0.1 years). Subjects were relatively healthy, non-smokers, without any medication or abuses. The effect of over/underweight was excluded by an anthropometric measurement using the InBody (Biospace, USA) device: body mass index 23.6±0.5 kg/m^2, body fat percentage 16.2±1.1%, waist-hips ratio 0.8±0.01. The study was approved by Ethics committee of Jessenius Faculty of Medicine in Martin.

The examinations were realized between 8.00 and 12.00 a.m. under standard conditions. The subjects were instructed to avoid consumption of meals and drinks containing any substances with potential effect on the ANS 24 hours prior to the examination and to avoid sleep deprivation. The stress profile protocol consisted of baseline period (first rest phase- RP 1) and three mental tasks: Stroop test, mental arithmetic (MA) and negative emotional stimulus (NES; represented by a video of dental caries treatment). The duration of each phase was 6 minutes followed by recovery phases (RP 2-4). Electrodermal activity was monitored using biofeedback device BioInfinity (Though Technology, Canada) during complete stress profile. The magnitude of EDA amplitude (μS) was evaluated in each phase.

The data were analyzed using SYSTAT (©2008, Cranes Software International Ltd, USA). Wilcoxon test was used for the assessment of differences because of nongaussian distribution of the data determined by Lilliefors test. Data are expressed as the mean±SEM.

RESULTS

The EDA was significantly increased in response to all mental tasks, indicating higher sympathetic activation during mental stress ($p<0.001$). A decrease of the values was observed during recovery periods ($p<0.001$); however, the EDA values were significantly higher in all periods (Stroop, MA, NES, RP 2-4) compared to baseline value ($p<0.001$) (Fig. 1), potentially reflecting persisting state of sympathetic arousal.

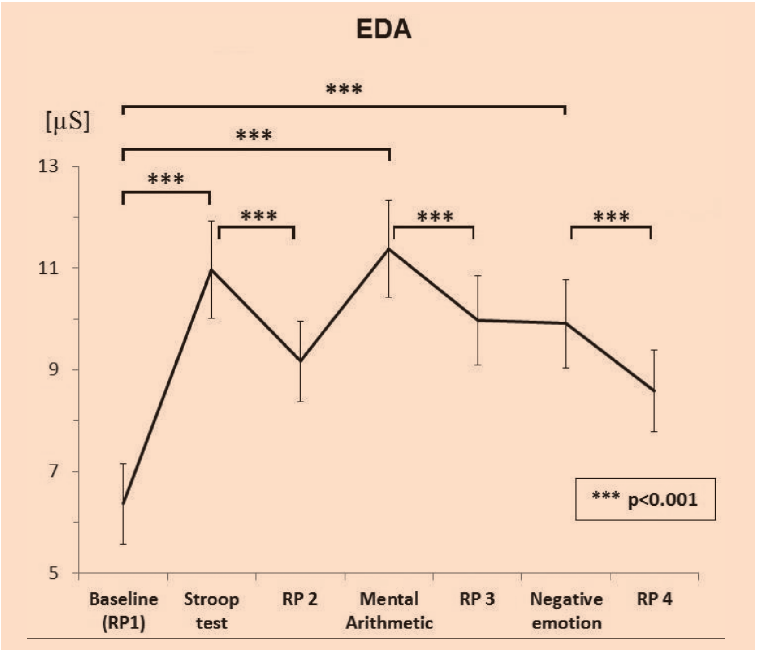

Fig. 1 Changes of electrodermal activity during stress profile (EDA- electrodermal activity, RP- recovery phase)

DISCUSSION

The major finding of this study was the higher EDA during complete stress protocol. It could indicate higher sympathetic activity in response to different kinds of stressors as well as in recovery phases. Compared to the previous results of HRV (12), we suggest that EDA can provide information about different kind of regulatory mechanism in response to stress, i.e. sympathetic cholinergic system innervating distinct peripheral organ (sweat glands in the skin) compared to the heart (expressed as 0V% parameter of HRV nonlinear analysis-symbolic dynamics expressing potential beta-adrenergic sympathetic activation). Therefore, one of the possible explanations can be effector-specificity of the stress response. Moreover, another mechanism could involve the neurophysiological characteristics of different neurotransmitters presented on the postganglional synapses (acetylcholine vs. norepinephrine).

The complex central EDA-regulation system consists of prefrontal cortical areas, amygdala and the anterior cingulate cortex (17). The integrative function of the ANS control system is situated in the hypothalamus. The sympathetic stimulation can be elicited by different structures of central nervous system (cortical areas, basal ganglia, hippocampus, thalamus, brainstem) with two main distinct groups: limbic-hypothalamic, which is emotionally and thermoregulatory driven and premotor-basal ganglia, occurring in preparation for motor movement (19).

EDA is widely used as a sensitive marker of emotion-related sympathetic activity (17, 19). It is related to the neurophysiological behavioral inhibition system, involved in passive avoidance and negative affections. Therefore, it should be a sensitive method to detect autonomic reactions elicited by discrete stimuli related to anxiety with no active coping mechanisms involved (17). In this study EDA values remained elevated within complete stress profile protocol. In the first and the second recovery phase (RP 2, RP 3) we can speculate about possible effect of the emotions related to the anticipation of the next task. The last rest period (RP 4) can be potentially affected by emotional ruminations (evoked by previously applied stimulus) which can result in delayed recovery (20).

As we can see, EDA is related to the activity of several cognitive and emotional centres modulating the autonomic response to mental stress. However, from evolutionary point of view, stress evoked sweating is assumed to offer an allostatic action convenient in typical fight or flight reaction. The evaporative sweating in the anticipation of an upcoming physical activity can play role in decreasing the body temperature as a compensation of reduced heat loss due to vasoconstricion (21). Perspiration of the palms allows better tactile differentiation (22) and protection against injury (23). The autonomic responses in the skin (sweating, piloerection, vasomotor changes) can serve also as a social signal in the interindividual interaction (24).

From this context, we could use mechanism, which was phylogenetically developed to improve the biophysical properties of the skin and express the social interaction during state of threat, for the study of underlying processes of autonomic reactivity to mental stress. The information provided by EDA is found to be interpretable even under a variety of suboptimal conditions (25), what can be an advantage in clinical application of this method. Its relevance in research of cardiovascular stress reactivity related morbidity is based on the presumption of significant role of autonomic dysregulation in the pathophysiology of such diseases.

CONCLUSIONS

The EDA is a sensitive marker used in psychophysiological research which can provide different information about autonomic, particularly sympathetic regulation compared to HRV determined mainly by vagal regulatory inputs. Importantly, the autonomic hypo/hyperreactivity to stress can result in increased risk of cardiovascular morbidity. Therefore, detailed study of autonomic (dys)regulations in response to stress is needed.

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