J. Phys. Ther. Sci.
24: 257–259, 2012

Autonomic Nerve Responses in a Psychological Stress Task and Subsequent Slow Breathing

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Abstract. [Purpose] To examine autonomic nerve responses during a psychological stress task and subsequent breathing. [Subjects and Methods] Slow and normal breaths were measured randomly at more than two days apart. The Uchida-Kraepelin test was executed for 15 minutes as a psychological stress task after a 5-minute rest sitting on a chair. Subsequently, slow breathing or normal breathing was carried out for 5 minutes. Salivary alpha-amylase and heart rate variability were analyzed as autonomic nerve responses. [Results] Salivary alpha-amylase increased with stress, and decreased with slow breathing. The HF and LF/HF ratio were synchronized with neither stress nor slow breathing. [Conclusion] The findings of our study suggest that salivary alpha-amylase is a more sensitive test of psychological stress than heart rate variability.
Key words: Autonomic nerve, Psychological stress, Slow breathing

(This article was submitted Sep. 22, 2011, and was accepted Nov. 2, 2011)

INTRODUCTION

The autonomic nervous system controls involuntary functions such as cardio respiratory function, digestion and absorption, sweating, metabolism, and reproduction. The autonomic nerve function is evaluated by heart rate, blood pressure, plasma norepinephrine, peripheral vascular resistance, and heart rate variability (HRV)[1–3)]. Power spectral analysis of HRV evaluates the distribution of the sinoatrial node in the dynamics of the autonomic nervous system. The power spectrum can be divided into parasympathetic activity and sympathetic nerve activity[1–3)]. The heart is controlled by both the sympathetic and parasympathetic nervous systems, with nerves modulating the effects of the two rival heart control systems. Heart rate increases with stimulation of the sympathetic nervous system and decreases with parasympathetic nervous system stimulation. Under stress, production of catecholamines, steroid and sex hormones increases. Adrenaline, noradrenaline, and dopamine of the catecholamine group are also increased by stress. Stress-induced catecholamines stimulate the body, and become a source of energy for a fight or flight reaction. These reactions are suppressed or reinforced by blood circulation disorders, and granulocytes are increased by hyper activity of sympathetic nerves.

The stress response of salivary alpha-amylase (sAA) has been suggested as an index of sympathetic nervous system activation[4–6)]. Stress-induced increases in amylase levels correlate with increases of amylase output[4)]. The autonomic nervous system regulates the process of salivation, and the concentration of alpha-amylase provides a reliable outcome measure of the sympathetic response[7)]. The salivary amylase level shows a greater increase and more rapid reaction than cortisol in response to psychological stressor[8)]. Activity of sAA is increased by acute psychosocial stress and the increase is correlated with increased norepinephrine[9)]. The purpose of this study was to investigate the autonomic responses during a psychological stress task and subsequent slow breathing as measured by sAA and HRV.

SUBJECTS AND METHODS

Fifteen healthy females aged 21–22 years (mean: 21.8 ± 0.7 yrs) participated in this study after providing their informed consent. Measures of sAA and HRV were taken under two conditions of slow and normal breathing after a stress task. The order of the conditions was chosen at random, and the two conditions were recorded two or more days apart. Subjects sat on a chair and remained at rest for 5 min. Then they took the Uchida-Kraepelin test for 15 min to induce psychological stress, and afterwards performed slow or normal breathing for 5 min. Slow breathing was performed while watching a stopwatch, with expiration adjusted to 6 seconds and inspiration adjusted to 4 seconds.

Normal breathing was spontaneous voluntary breathing. The salivary alpha-amylase level was measured after 4 min of rest before the start, at 12 min into the stress task, and at 4 min into the slow or normal breathing. Measurement of sAA was done using a monitor and disposable test strips (CM-2.1, Nipro Co., Ltd, Osaka, Japan). All saliva samples were collected by direct placement of a disposable test strip under the tongue. The strip was then transferred to a reagent

strip containing a chromogen with a saliva transcription device. Changes in color density were measured with an optical analyzer. To avoid the effects of food and drink, no measurements were performed immediately after a meal. The measurement environment was kept at a temperature of 23–26 degrees C, and 30–50% humidity. The electrocardiogram was recorded throughout the three-point guidance method for measuring HRV. The collected data were analyzed by computer, and heart rate, high-frequency components, low-frequency components, and the LF/HF ratio were obtained (MemCalc/Tarawa, GMS companies, Tokyo, Japan).

SPSS for Window, version 12.0 (SPSS Inc, Tokyo, Japan) was used for the statistical analysis. A P value below 0.05 was considered statistically significant. Statistical analysis consisted of two-way analysis of variance (ANOVA) and multiple comparisons. To explore the reliability, the intraclass correlation coefficient (ICC) was calculated using the first set of two conditions when at rest. The local ethics committee of International University of Health and Welfare approved all aspects of this study. Informed consent was obtained from all subjects.

RESULTS

The sAA values of the slow breathing study at rest, under stress, and slow breathing were 45 ± 21.3 KU/L, 83.2 ± 33.3 KU/L, and 40.7 ± 16.3KU/L, respectively. The sAA values of the normal breathing study at rest, under stress, and normal breathing were 51.7 ± 17.9 KU/L, 86.2 ± 23.0 KU/L, and 69.8 ± 33.2KU/L, respectively. In the slow breathing study, there was a significant difference between the values measured under stress and slow breathing.

Heart rate values of the slow breathing study at rest, under stress, slow breathing, and subsequent rest were 81.4 ± 9.6 beats/min, 88.7 ± 10.6 beats/min, 84.1 ± 10.3 beats/min, and 81.7 ± 12.0 beats/min, respectively. Heart rate values of the normal breathing study at rest, under stress, normal breathing, and subsequent rest were 81.6 ± 8.8 beats/min, 88.4 ± 8.2 beats/min, 81.1 ± 10.4 beats/min, and 81.4 ± 10.3 beats/minutes, respectively. No statistically significant differences were found.

HF values of the slow breathing study at rest, under stress, slow breathing, and subsequent rest were 363.5 ± 250.6msec^2, 273.0 ± 404.3 msec^2, 374.2 ± 332.3 msec^2, and 256.4 ± 321.1 msec^2, respectively. HF values of the normal breathing study at rest, under stress, normal breathing, and subsequent rest were 458.9 ± 372.5 msec^2, 220.4 ± 200.0 msec^2, 531.1 ± 510.9 msec^2, and 409.6 ± 361.9 msec^2, respectively. No statistically significant differences were found.

LF/HF ratio values of the slow breathing study at rest, under stress, slow breathing, and subsequent rest were 3.8 ± 2.5, 3.7 ± 2.6, 25.5 ± 25.9, and 9.0 ± 6.8, respectively. A statistically significant difference was found between slow breathing and the other three times. LF/HF ratio values of the normal breathing study at rest, under stress, normal breathing, and subsequent rest were 3.5 ± 2.4, 4.6 ± 3.3, 3.1 ± 2.4, and 3.4 ± 2.4, respectively. No statistically significant differences were found.

ICC of the sAA was 0.895. ICC of the HF was 0.676. ICC of the LF/HF ratio was 0.728. ICC of the heart rate was 0.746, and all P values were below 0.01.

DISCUSSION

All ICCs were high indicating the reproducibility of the measuring instruments was excellent.

Under stress, sympathetic nerve activity increases and parasympathetic nerve activity decreases[10]. The fact that sAA values increased while the subjects were taking the Uchida-Kraepelin test indicated that they were subjected to a psychological stress. However, the LF/HF ratio, an index of cardiac sympathetic nerve activity, at this time hardly increased. As there was an increase in heart rate at this time, we speculate that sympathetic nerve activity had increased. During slow breathing after experiencing stress, a significant stress reduction was shown, as indicated by the significant increase in the LF/HF ratio value. The lessening of stress was further confirmed by the heart rate returning to the resting heart rate value. Without slow breathing, elevation in the sAA value, indicative of increased stress, did not return to its resting value. This indicates that mental stress was alleviated by slow breathing, suggesting that the sAA value is an indicator that reflects mental stress. On the other hand, the LF/HF ratio value, which showed a response to slow breathing, did not respond at all in the absence of slow breathing.

Data obtained in this study corroborate the findings of other studies that show sAA levels are affected by psychological stress[8, 9, 11, 12]. There was virtually no increase in cardiac sympathetic nerve activity during the Uchida-Kraepelin test and we consider this indicates the low sensitivity of cardiac sympathetic nerve activity to these psychological problems. Despite the results of a psychological stress study involving 30 young males, which reported the association of psychological stress with sAA and the LF/HF ratio[13], we found no such association. Lung stretch reflex occurs due to slow breathing, and increases in sympathetic nerve activity[14, 15]. In synchrony with central inspiratory motor activity, an increase in the sympathetic nerve activity occurs[14]. Sympathetic nerve activity, in particular, increases when inspiration and expiration are differently regulated[15]. We believe this lack of synchrony is linked to the increase in sympathetic nerve activity at the time of slow breathing.

Muscle sympathetic nerve activity (MSNA) is increased by deep breathing[16]. Suppression of MSNA during an elevation of arterial pressure in healthy people is due to the aortic arch baroreflex[17]. Slow breathing is known to make the aortic baroreflex more sensitive, inducing inhibition of MSNA during elevation of arterial pressure in healthy subjects. In this study, however, there was no obvious explanation for the increase in sympathetic nerve activity, a topic which remains to be investigated in future research. Slow breathing increases the baroreflex sensitivity more than spontaneous breathing and subsequently decreases sympathetic nerve activity[18]. All in all, cardiac sympathetic nerve activity, rather than a measure of comfort or discomfort, seems to be a better indicator of how homeostasis of the body is maintained.

Autonomic nerve responses during a psychological stress

task and subsequent slow breathing were measured using sAA and HRV in this study. The measure of sAA increased with stress, and it decreased with slow breathing. The HF and LF/HF ratio were synchronized with neither stress nor slow breathing. The findings of this study suggest that sAA is a more sensitive index of stress than HRV analysis.

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