

FUNCTIONAL SIGNIFICANCE OF AUTONOMIC CONTROL FOR GENESIS OF MORBIDITY WITH AUTONOMIC NERVOUS SYSTEM ORIGIN

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ABSTRACT

The purpose of the present study was to determine whether dysfunctional neural mechanisms might consider as aetiological factors for genesis of morbidity with Autonomic Nervous System (ANS) origin, specifically Cardiovascular Diseases (CVD). The autonomic cardiovascular control was examined with heart rate variability (HRV), heart rate, systolic and diastolic blood pressure (BP) in 122 individuals performing static and mental workload. Morbidity with neural autonomic genesis was studied. The significance of P_{RSA} , mean value of cardiointervals, P_{THM} , P_T , diastolic BP, occupation and age in pre-abnormal and abnormal control were revealed. Dependence of morbidity on P_T and P_{THM} in static workload, and dependence of morbidity on diastolic BP and P_{RSA} in mental workload were observed. Our study elucidated the functional effect and significance of parasympathetic, and sympathetic activity for genesis of morbidity with ANS origin.

KEY WORDS: Parasympathetic and sympathetic activity; Heart Rate Variability; Blood pressure; Mental and static workload; Morbidity

INTRODUCTION

Dysfunctional cardiovascular responses and the underlying neural mechanisms of regulation, result of exposure to mental and static workload, might induce pathophysiological states. Research studies show controversial results considering the effect of mental and static workload on the CNS and ANS and the relevant cardiovascular-regulated indices although is indicated the identical functional alteration in control mechanisms result of organism's

adaptive response. Studies on mental and static stress reveal increase in the ANS mediated responses such as heart rate, arterial blood pressure, cardiac output [1-4]. Mental stress decreases vascular resistance and increases blood flow as a consequence of the sympathetic withdrawal and β -adrenergic vasodilation whereas static exercise increases the sympathetic activity and vascular resistance [3; 4]. Mental and static workload is considered as contributing risk factor for genesis of CVD [2; 5]. Short-term stress-induced activation of the ANS and immune system generates adaptive response whereas prolonged activation might disturb the homeostasis. Prolonged exposure to cognitive and emotional factors elevates the efferent sympathetic cardiac activity, facilitates the process of endothelial dysfunction and mediates the progression of CVD [6]. Static and mental stress facilitate the process of myocardial ischemia which is preceded by vagal withdrawal [7]. Long-term stress activation, defined from McEwen, 2000 [6] as 'allostatic load' might cause metabolite and cardiovascular syndromes inherent for CVD, type 2 diabetes and obesity [8].

The purpose of the present study was to determine whether dysfunctional neural mechanisms might consider as aetiological factors for genesis of morbidity with Autonomic Nervous System (ANS) origin, specifically Cardiovascular Diseases (CVD).

MATERIALS AND METHODS

Two groups, each consisted of 61 male individuals, participated in the study: 1. Static workload (grain cleaning and packaging operators); 2. Mental workload (radio operators). Groups were divided into three sub-groups: referent, pre-abnormal and abnormal control according to referent Health Risk value (see Part I, Table 1). Operator's work activity was described in Part I of our study.

1. Heart Rate Variability. A computerized diagnostic system for the study of cardiovascular function was applied [9;10]. HRV data were determined from ten minutes of ECG recordings between 9 a.m. and 11 a.m. in supine position after a one-hour rest period.

Following indices were analyzed: 1.1. Time-domain HRV measure: 1.1.1. X (mean RR interval) (milliseconds), resp. mean heart rate (beats per minute); 1.2. Frequency-domain HRV measures. 1.2.1. Spectral power of RR intervals in the Temperature band (0.01-0.05 Hz) (P_T) (milliseconds²); 1.2.2. Spectral power of RR intervals in the Traube-Hering-Mayer band (0.06-0.14 Hz) (P_{THM}) (milliseconds²); 1.2.3. Spectral power of RR intervals in the Respiratory Sinus Arrhythmia band (RSA) (0.15-0.50 Hz) (P_{RSA}) (milliseconds²); 1.3. HRV-derived index: 1.3.1. Health Risk (HR) (%).

2. Arterial blood pressure. Systolic and diastolic blood pressure (BP) was measured by sphygmomanometer – “Riester”, No. 1360-107, Jungingen, Germany. Systolic and diastolic BP values were considered with the European guidelines for hypertension [11].

3. Morbidity. Morbidity was investigated [12; 13]. Diseases associated with dysfunction of the ANS and immune deficit: arterial hypertension, Ischaemic Heart Disease, neuroses, disturbance of ANS function, central neuro-othological syndrome, diabetes, colitis, viroses were registered at both groups. Depression, cancer disease and infractus myoardii were found in mental workload operators.

4. Data Analysis. Logistic regression analysis was used to define factors that affect pre-abnormal and abnormal control. A probability (P) for reference to pre-abnormal and abnormal control groups was defined as $P > 0.5$. Dependence of morbidity on workload – induced autonomic modes of control was determined with the χ^2 criterion. Logistic regression was used to determine the dependence of morbidity on cardiovascular indices. A p value < 0.05 was considered statistically significant.

RESULTS

Logistic regression analysis revealed variables that discriminate pre-abnormal and abnormal control from referent. The probability (P) an individual to refer to pre-abnormal and abnormal control is calculated with regressive equations (see Table 1). Significant effect for

generating pre-abnormal mode of control showed variables: occupation, P_{RSA} , diastolic BP, age. Significant effect for generating abnormal mode of control showed: occupation, X, P_T , P_{THM} , diastolic BP, age. The percentage for correct classification for the referent, pre-abnormal and abnormal autonomic modes of control were: 88.6%, 81.6% and 93.6%. χ^2 criterion showed significant dependence of morbidity on mental and static workload – induced autonomic modes of control ($p < 0.0001$). Distribution of healthy and diseased individuals was shown on Table 2. Logistic regression analysis differentiated dependence of morbidity on cardiovascular variables in mental and static workload (see Table 3). In static workload dependence of morbidity on P_T and P_{THM} was found. The percentage for correct classification in groups with healthy and diseased individuals is 89.1% and 46.7%. In mental workload dependence of morbidity on diastolic BP, and the P_{RSA} below the reference value ($P_{RSA} \leq 12 \text{ msec}^2$) was observed. The percentage for correct classification is 63.3% and 77.4%.

DISCUSSION

Factors with preventive and risk effect on pre-abnormal and abnormal control were determined. The results of our study revealed the preventive effect of parasympathetic function examined with P_{RSA} and risk effect of the occupation, diastolic BP and age on cardiovascular function in pre-abnormal control. The results of our study indicate also the preventive effect of sympathetic-to-parasympathetic equilibrium assessed with X and baroreflex examined with P_{THM} , and risk effect of occupation, sympathetically mediated P_T , diastolic BP and age on cardiovascular function in abnormal control. These results indicated the prognostic significance of HRV for determination of cardiovascular autonomic control.

Both groups registered diseases associated with dysfunction of the ANS and immune deficit. In static workload dependence of morbidity on the change in peripheral vascular resistance examined with P_T , and baroreflex assessed with P_{THM} was observed. In mental

workload dependence of morbidity on diastolic BP and parasympathetic function mediating P_{RSA} was found. Affected diseased individuals performing mental workload were not only with abnormal but also with pre-abnormal and referent mode of control. The later result showed the necessity of screening autonomic cardiovascular control with HRV and blood pressure even at asymptomatic referent mode of control. The functional significance of workload – induced alteration in autonomic modes of control and regulated cardiovascular variables that might mediate the genesis of morbidity with ANS origin is demonstrated. Our results are in agreement with the results of Kop et al., 2001 [5] who revealed the prognostic value of reduced parasympathetic activity mediating the P_{RSA} for CVD, aroused from exercise and mental stress-induced autonomic changes. Dysfunctional neural mechanism with a pattern of sympathetic activation, reduced parasympathetic activity and disturbed baroreceptor regulation affecting the peripheral vascular resistance, systolic and diastolic BP might consider as aetiological factors for genesis of morbidity with ANS origin, specifically CVD.

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Table 1. Dependence of pre-abnormal (A) and abnormal (B) control on cardiovascular indices, occupation and age.

$$A. \quad P = \frac{1}{1 + e^{-(-16.069 + 1.742 \cdot Occupation - 0.278 \cdot P_{RSA} + 0.176 \cdot diastolicBP + 0.77 \cdot Age)}}$$

$$B. \quad P = \frac{1}{1 + e^{-(-13.696 + 1.842 \cdot Occupation - 0.16 \cdot \bar{X} + 0.533 \cdot P_T - 0.59 P_{THM} + 0.202 \cdot Age)}}$$

Table 2. Distribution of healthy and diseased individuals in workload – induced autonomic control.

Control	Healthy: Mental/Static workload		Diseased: Mental/Static workload	
	Referent	20 (66.7%)	21 (45.7%)	3 (9.7%)
Pre-abnormal	10 (33.3%)	25 (54.3%)	9 (29%)	0 (0%)
Abnormal	0 (0%)	0 (0%)	19 (61.3%)	15 (100%)

Table 3. Dependence of morbidity (P_M) on cardiovascular indices in static (A) and mental (B) workload.

$$A. \quad P_M = \frac{1}{1 + e^{-(-8.896 + 1.337 \cdot P_T - 0.455 P_{THM})}}$$

$$B. \quad P = \frac{1}{1 + e^{-(-8.388 + 0.132 \cdot diastolicBP - 2.19 \cdot P_{RSA})}}$$

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