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DETERMINATION OF AUTONOMIC CARDIOVASCULAR RESPONSE PATTERNS UNDER EMOTIONAL STRESS STATES

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Summary. Exaggerated cardiovascular responsiveness to emotional stress may lead to pathophysiological processes. The relative degree of integration or differentiation of cardiovascular responses studied with Heart Rate Variability, Variability of Systolic and Diastolic Arterial Blood Pressure, Baroreflex Sensitivity, the QT Interval Variability may offer an important link in the understanding of the etiology of cardiovascular-based emotional states disorders. Integrated effects of emotional load and mental load are studied as well, as they may cause a strong cardiovascular reactivity that is believed to be a risk factor for essential arterial hypertension or coronary artery diseases. Therefore, studying autonomic cardiovascular response patterns during emotional states, mental work load, job stress in laboratory and field conditions promotes for identifying subject and subject groups at risk for development of cardiovascular diseases.

Key words: cardiovascular diseases/etiology, physiopathology; stress, psychological (source: MeSH)

Biopsychological models of cardiovascular diseases assume that cardiac activation due to psychological stress may foster maladaptive consequences to the organism and is implicated in the etiology of various cardiovascular disorders including arterial hypertension, coronary artery disease and related increased susceptibility to life threatening cardiac arrhythmias.

Exaggerated cardiovascular responsiveness to emotional stress may lead to pathophysiological processes. Experience of negative moods with higher intensity is associated with blood pressure (BP), and heart rate (HR) elevations that may contribute to the risk of hypertension (Shapiro et al., 2001). Parental history of hypertension, and expression or suppression of negative emotions are associated with higher levels of ambulatory BP (Goldstein et al., 2000). The same researchers observe that frequent negative emotion experience during the day is associated
with higher levels of blood pressure responses during sleep (Shapiro et al., 1997). Cardiovascular control centers in the brain mediate the effects of emotion and other psychological processes on the circulation (Reise, Le Doux, 1987). Increased sympathetic nervous activation in response to emotional stress may serve an important role in the initiation and progression of coronary artery disease (Herd, 1983).

The relative degree of integration or differentiation of cardiovascular and metabolic processes may offer an important link in the understanding of the etiology of cardiovascular-based emotional states disorders (Langer et al., 1986). Characteristic pattern of flight or fight physiological and emotional reaction that do not result in physical activity is excess of free fatty acids which are converted to very low-density and low-density lipoproteins, the latter of which have been implicated in the pathogenesis of coronary artery disease (Schneiderman, 1983). Schneiderman, 1987 suggested the hypothesis that stress-induced increase in sympathetic nervous system activity and the release of plasma catecholamines are important links between emotional states and atherogenesis. According to this hypothesis, the sequelae include both an increase in arterial blood pressure and the release of catecholamines, which may compromise the endothelial lining of arterial vessels. This link is examined in respect to the association between distressed and pressured moods, and the cortisol and catecholamines secretion: distressed moods are related to norepinephrine excretion; pressured moods with cortisol, epinephrine and norepinephrine secretion (Shapiro et al., 2001).

The effective integration of psychophysiology and cardiology is based on two conceptions: the role of hemispheric specialization in the mediation of emotional arousal and the role of lateralized imbalance in sympathetic input to the heart in cardiac arrhythmogenesis. The proposed hypothesis is that individuals who manifest more lateralized frontal lobe activity during emotional arousal may concomitantly generate more lateralized sympathetic input to the heart and be at increased risk for cardiac arrhythmias (Lane, Schwartz, 1987). Neuroanatomical results confirm this thesis, and reveal that pleasant and unpleasant emotions have different neural substrates with greater activation of the brain, measured by positron emission tomography (Lane et al., 1997), and electroencephalography (Heller, 1993).

Integrated effects of the mental load and emotional load may cause a strong cardiovascular reactivity that is believed to be a risk factor for essential arterial hypertension or coronary artery disease. Therefore, studying autonomic cardiovascular response patterns during mental workload (effort), job stress, and emotional states in laboratory and field conditions promote for identifying subjects and subjects groups at risk for development of cardiovascular diseases (CVD).

**ANGER AND ANXIETY EFFECTS ON CARDIOVASCULAR RESPONSE PATTERNS**

Every kind of emotion induces a specific cardiovascular changes profile (Ekman et al., 1983; Mulder, 1973; Schwartz et al., 1981) that vary as a function of differentiated positive and negative emotions (Krantz et al., 1986; Sinha et al., 1992). Anger is negative
emotion that is regarded mostly associated with enhanced vascular tonus, and large
DBP responses. Anger and anxiety are one of the most strongest emotional stressors as
they yield enhanced cardiovascular reactivity that may be implicated in the etiology of
CVD (Shapiro D. et al., 1997; Shapiro D. et al., 2001). Alternative modes of anger
patterns: anger expression, and anger suppression are investigated

Psychophysiological research has demonstrated that both anger expression
and anger suppression are related to cardiovascular responses (Engebretson et al.,
1989). Coping with anger by conscious inhibition of its expression is associated with
increases in both systolic blood pressure (SBP), and diastolic blood pressure
(DBP) (Goldstein et al., 1988). Suppressed anger is found to be related to SBP but
not to DBP (Dimsdale et al., 1986).

The relationship between anger expression and reactivity to stressors may
provide insights into the mechanisms linking anger, blood pressure and incidence
of CVD. The degree to which arterial blood pressure can be predicted is enhanced
by considering both the level of cardiovascular responses to stress, and psycholo­
gical measures of the experience and expression of anger (Johnson, 1989). Explora­
tion of possible associations between anger expression and reactivity to a stres­
sor shows that anger expressed outward is associated with lower heart rate and
norepinephrine reactivity to the stressor (Mills et al., 1989). Negative relationships
are observed between: anger-out expression score and the mid-frequency compo­
nent (0.1 Hz) of SBP, and anger-in mode of expression and the heart rate peak
during mental tests, suggesting that individuals who are often in anger-provoking
situations should have heightened blood pressure (Laude et al., 1997).

Behavioral pattern with suppressed anger and anxiety promotes a hyperdyna­
mic cardiovascular state (increase in heart rate, SBP and DBP) and enhances the
hypertensive development process in subjects with positive family history of hyper­
tension (Perine et al., 1988; Matthews et al., 1996; Manuck et al., 1985; Johnson,
1989; Jorgensen et al., 1992; Goldstein et al., 2000).

Normotensive subjects at risk for future development of hypertension may show
heightened stress-related cardiovascular responses if they tend to inhibit the expression
of negative emotions. Hypertensive subjects show higher levels of state anxiety
(Blumenthal et al., 1993), and suppression of negative emotions (Roter, Ewart, 1992;
Cottington et al., 1986). Assessment of the combined influence of biological risk for
hypertension and patterns of emotional control upon cardiovascular responses to mental
stress reveal that: hypertensive risk interacts with anxiety state with the greatest SBP,
and heart rate responses, accompanied by cardiac baroreflex inhibition; anger
experience and expression do not interact with hypertensive risk but had a direct effect
upon cardiovascular responses to mental stress (Vogele, Steptoe, 1992).

In psychophysiological research, the conceptions of the integrated effects of job
stress/mental effort, parental history of hypertension, anger, and the experience of
anxiety and anger on the cardiovascular system are incomplete. An analytical approach
to the determination of the autonomic cardiovascular response patterns during job
stress/mental effort, and emotional states: experience and expression of anger, and
anxiety is needed. Determination of the autonomic cardiovascular response patterns will elucidate cardiovascular mechanisms controlling stress/mental effort, anxiety and anger. There is a lack of evidences in the literature whether integrated effects of the anger and anxiety, and the stress/mental effort, and anger yield cardiovascular activation, and whether activating cardiovascular effect of the anger adds to the activating effect of the anxiety, respectively whether activating cardiovascular effect of the anger adds to the activating effect of the anxiety. There is no specification whether Autonomic Nervous System (ANS) response patterns based on heart rate variability differentiate stress/mental effort, anxiety and anger, and whether the heart rate variability will be affected under the integrated effects of the anger and anxiety, and the stress/effort and anger. Following HRV components are regarded as indicators of the activity of the autonomic cardiovascular control (S. Danev 1989; R. Nikolova 1993; Malik et al., 1996):

- Temperature band (0.02-0.06 Hz) or low frequency band (LF). Peripheral vascular resistance exhibits intrinsic oscillations with a low frequency. These oscillations can be influenced by thermal skin stimulation and are thought to arise from the thermoregulatory peripheral blood flow adjustments. The fluctuations in peripheral vascular resistance are accompanied by fluctuations with the same frequency in blood pressure and heart rate and are mediated by the sympathetic nervous system.

- Traube-Hering-Mayer band (0.07-0.14 Hz) or mid frequency band (MF). The so-called 10-second rhythm in heart rate originates from self-oscillation in the vasomotor part of the baroreflex loop. These intrinsic oscillations result from the negative feedback in the baroreflex and are accompanied by synchronous fluctuations in blood pressure (Traube-Hering-Mayer waves). The frequency of the oscillations is determined by the time delay of the system. They are augmented when sympathetic tone is increased and are decreased with sympathetic or parasympathetic blockade.

- Respiratory sinus arrhythmia band (RSA) (0.15-0.40 Hz) or high frequency band (HF). Due to inspiratory inhibition of the vagal tone, the heart rate shows fluctuations with a frequency equal to the respiratory rate. The inspiratory inhibition is evoked primarily by control irradiation of impulses from the medullary respiratory to the cardiovascular center. Variations in HF band can mainly be attributed to RSA. RSA can be abolished by atropine or vagotomy and is parasympathetically mediated.

There is no specification as well about the autonomic cardiovascular response patterns, and blood pressure responses in subjects with parental history of hypertension who are characterized by following personality characteristics: high hostility, low anger out, high anxiety.

The comprehensive understanding of the autonomic cardiac control requires independent indices of activities of both the sympathetic and parasympathetic branches. To study autonomic cardiovascular response patterns, it is necessary to examine measures of heart rate variability. Non-invasive measurement of the heart rate variability reflects autonomic cardiovascular control. Changes of the parasympathetic and sympathetic mediation of the heart rate are postulated as important determinants in the etiology of the CVD (Malik, 1996; Stein et al., 1995; Huikuri et al., 1996; Van Ravenswaaij et al., 1993; Kleiger et al., 1992).
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