CURRENT STUDIES OF UKRAINIAN RESEARCHERS OF STRESS IMPACT ON CHEST ORGANS: LITERATURE REVIEW

AKTUALNE PRACE BADACZY UKRAIŃSKICH NA TEMAT WPŁYWU STRESU NA NARZĄDY KLATKI PIERSIOWEJ: PRZEGLĄD LITERATURY

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ABSTRACT

Introduction: Stress reactions allow the body to adapt to the various environmental factors using the universal complex of neurohumoral reactions. Excessive stress reduces adaptability of the body and can initiate the onset and development of various pathologies. In recent years, Ukrainian scientists have completed a series of studies on the analysis of various aspects of the stress effect on the body.

Aim: The paper was aimed at the analysis of the national medical publications, devoted to the study of the impact of stress on the chest organs.

Material and Methods: The bibliosemantic method has been used during the study. Findings of the contemporary scientific studies devoted to the effect of stress on the chest organs have been analyzed.

Results: The analysis shows that despite a long history of stress-related investigations, the study of the problem of stress remains relevant to date. Scientific researches elicit the new aspects of the impact of stress on the body, particularly on the chest organs.

Conclusion: Recent investigations of Ukrainian scientists assist in better understanding of the essence of the stress-related morphofunctional changes that occur in the lungs, heart and esophagus. The findings will be helpful in the search for ways to prevent and treat stress-induced pathological processes, since the problem of stress remains to be solved to date.

KEY WORDS: esophagus, heart, literature review, lungs, stress

Wiad Lek 2017, 71, 6 cz. l, 1114-1117

INTRODUCTION

The stress phenomenon, discovered by Hans Selve, belongs to a number of the fundamental manifestations of life, because it allows organisms to adapt to the various environmental factors due to the universal complex of neurohumoral reactions. The term of "stress" has transformed from medicine into the adjoining fields (biology, psychology, sociology), and became widely used in everyday communication due to the increase of stress in humans caused by urbanization, increasing pace of life and more apparent gap between the human biological nature and conditions of its social existence [1, 2, 3]. Basically, the cortex- hypothalamus-pituitary-adrenal cortex system is involved into the physiological adaptation to a variety of stressors, including the emotional ones [4]. Excessive stress reduces the adaptability of the body and can become the basis for the emergence of various diseases (mental, endocrine, cardiovascular, etc.). Therefore, the researchers consider stress as a pathogenetic basis for diseases and find ways to correction of its impact [5-10].

In recent years, Ukrainian scientists have completed a series of studies, investigating the various aspects of stress impact on the body.

THE AIM

The paper was aimed at the analysis of national medical publications devoted to the study of stress impact on the chest organs.

MATERIALS AND METHODS

The bibliosemantic method has been used during the study. Findings of the contemporary scientific studies devoted to the effect of stress on the chest organs have been analyzed.

RESULTS

Investigations made on the Department of Operative Surgery and Topographic Anatomy at the Higher State Educational Establishment of Ukraine "Ukrainian Medical Stomatological Academy" show that both the acute and chronic stress leads to the development of prominent morphofunctional changes in rats' lungs. Macroscopic analysis shows the development of such apparent hemorrhagic phenomena as accumulation of mucous hemorrhagic content in the bronchi lumina, the occurrence of numerous hemorrhages under the visceral pleura, into the lung tissue

and bronchi mucosa has been noted after the exposure to the acute immobilization stress. Microscopically, the acute immobilization stress causes a significant dilatation of the alveoli (by 63,8%, p<0,01 in the left lung and 70,6%, p<0,01 in the right lung), local destruction and thinning of the interalveolar septa (by 42,3%, p<0,01, on the left and 40,4%, p<0,01 on the right), thickening and lesions of the epithelial layer of mucous membrane of intrapulmonic bronchi (in the smaller bronchi: by 85,9% (p<0,01) in the left lung and 87,4% (p<0,01) in the right lung; in the medium sized bronchi: by 65,7% (p<0,01) on the left and 65,9% (p<0,01) on the right), impaired hemomicrocirculation with the development of blood stasis and occurrence of numerous hemorrhages from the dilated hemomicrovessels into connective tissue and alveoli lumina. Macroscopically, chronic immobilization stress leads to the development of the apparent emphysema (lungs are enlarged and inflated, edges of which cover the anterior mediastinum, not collapsed after removal from the chest) of the lungs of laboratory rats. At the same time lumina of intrapulmonic bronchi are densely filled with clear mucoid exudate. Microscopically, chronic immobilization stress causes significant enlargement of the alveoli (by 61,9%, p<0,01, in the left lung and 68,1%, p<0,01, in the right one), deformation and thinning of the alveolar septa (by 53,19, p<0,01 on the left and 51,75%, p < 0.01, on the right), thickening and destruction of the epithelial layer of the intrapulmonic bronchi mucosa (in the smaller bronchi: by 42% (p<0,01) in the left lung and 43,7% (p<0,01) in the right lung; in the medium sized bronchi: by 38,5% (p<0,01) on the left and 36,9% (p<0,01) on the right), impaired hemomicrocirculation with the phenomena of the red blood cells diapedesis into interstitial connective tissue and alveoli lumina. After exposure to the chronic immobilization stress, in contrast to the acute one, the phenomena of fibrosis have been detected in the lungs; origination of atelectasis and inflammatory infiltration in the lung tissue and walls of intrapulmonic bronchi has been found. The authors hypothesize, that such lesions can promote the origination and development of multiple pathologies of the respiratory organs [11-17]. Findings of experimental study, made by another author, show that on Day 7 of the exposure to chronic immobilization stress the apparent morphological changes in lungs were noted and their progression occurred on Day 14 and 21 of the experiment [18]. The findings provided with the opinion that postoperative stress reaction, caused by the restrained locomotor activity, should be considered as one of the factors that contribute to the onset and development of postoperative noncardiogenic pulmonary edema [19].

The role of stress as one of the pathogenetic factors for the onset and development of bronchopulmonary pathology has been also admitted by the other national scientists [20, 21].

In this way, the intensification of free radical oxidation of lipids concomitant with the total antioxidant blood activity disorder is one of the mechanisms of the progression of chronic obstructive pulmonary disease [22]. In addition, stress affects the barrier function of the lung. This leads to the development of the "stress lungs" syndrome, disruption of the oxygen delivery to the tissues of the body and hypoxia [23]. The bronchopulmonary pathology, in turn, is accompanied by the oxidative stress [24, 25]. Notably, nonnosocomial pneumonia in the acute period is accompanied by the excessive generation of reactive oxygen species, including the superoxide anion radical and products of nitric oxide metabolism concomitant with low control of antioxidant system [26].

Decompensated acidosis is detected in the majority of children with the acute stress non-nosocomial pneumonia, whereas alkalemia is found in the greater number of patients suffering from the chronic stress non-nosocomial pneumonia. In most cases the acute and chronic stressrelated lowering of the osmolar concentration of blood plasma is detected in sick children. At the time of hospital admission the temperature reaction of the body is higher than 39,0 °C in one-third of children with stress nonnosocomial pneumonia. Higher probability of occurrence of life-threatening conditions caused by the inflammatory pulmonary process is detected in children with stress [27].

It has been also found that oxidative stress has inductive effect on the intensity of apoptosis in small children with pneumonia; however, the processes of neutrophils' necrosis along with the weakening of the physiological death of cells become more active with the progressing severity of the disease and intensification of the oxygen free radicals' generation [28].

Experimental studies show a significant strengthening of the processes of lipid peroxidation and phagocytic activity of leukocytes concomitant with the substantial depression of the activity of enzymes antioxidant protection in the blood and lungs, especially at the late period (Day 6 and 16) of the experiment, observed at all stages of the development of pneumonia in combination with immobilization stress [29].

Likewise, stresses affect the heart, and, therefore, are considered as the leading risk factors for cardiovascular disease [30, 31, 32]. Microscopically, the acute immobilization stress is accompanied by significant subendocardial hemorrhages, plethora of vessels of cardiac microcirculatory flow with blood stasis events and edema of myocardial intersticium [33].

Prolonged immobilization in combination with hypoand hyperthermic shock, caused by the psychoemotional stress load, leads to the accumulation of irreversible changes in the cardiac hystiocytes of experimental animals that is proved by the increase in number of cardiac hystiocytes identified at the late stages of apoptosis and necrosis [34]. Stress effect of big doses of catecholamines leads to the development of the acute adrenal myocardiodystrophy associated with the suppression of cellular immunity level [35]. Oxidative stress causes the disturbance of the free radical processes and system of antioxidant protection with accumulation of the primary and secondary products of lipid peroxidation and decrease in content of nitric oxide in rats' atriums, especially in males [36].

Mechanisms of inflammation and oxidative stress, occurred due to interrelation between the reninangiotensin, sympathoadrenal and immune systems, can be crucial in pathogenesis of the cardiovascular lesion, damage and remodeling of the heart and vascular wall and development of arterial hypertension [37].

The impact of the acute emotional-and-pain stress and stress, complicated with hypercholesterolemia, on the state of the left ventricular endocardial endothelium has been investigated during the experiments on white rats. It has been shown that stress reaction caused damage to this structure, which is accompanied by desquamation of individual endotheliocytes. The degree of damage depends on the duration of the stress factor impact with maximum manifestation at the post-stress period. Elevation of the total cholesterol in the blood of animals which were on the atherogenic diet also affects the structure of the endocardial endothelium. Alimentary hypercholesterolemia potentiates the destructive effect of the stress and contributes to the increase in number of desquamated cells. The detachment of endothelial cell layers is often observed in the combined action of both pathogenic factors [38].

Experimental studies on rats have shown that in simulation of chronic stress the use of aspen bark extract during the period of immobilization has a cardioprotective effect, preventing the development of dystrophy and foci of heart muscle necrosis, reducing the violation of the state of microcirculatory flow, ensuring the endothelium-protective effect [39]. In experimental model of the acute emotional stress the effect of piracetam on the activity of the total lactate dehydrogenase, LDG1 cardiac fraction, proteinase in rats' blood serum and myocardial tissues has also been studied. The increase in the activity of the basic marker enzymes indicates about the development of stressogenic destructive and degenerative changes in the membrane subcellular structures of cardiac hystiocytes. Restriction of stress-induced fermentemia and proteolysis under the influence of piracetam is a manifestation of its membraneand cytoprotective action with regard to cardiac hystiocytes. In emotional stress preservation of the structural integrity of the cardiac hystiocytes can be regarded as a manifestation of antistress cardioprotective effect of piracetam [40].

Recent studies show that in the embryogenesis and postnatal development of the heart, as well as in the functioning of the mature myocardium both in normal conditions and during the adaptation to stress, as well as in the process of aging, characterized by the hypertrophy and remodeling of the cardiac muscle, the Wnt/ β -catenin signaling pathway is involved. Its control over the processes of cardiogenesis and adaptation is of complex and multistage nature, and beta-catenin is a promising candidate for the role of a target for the development of the novel approaches to the therapy of pathologies of the mature myocardium [41].

Among the chest organs stress has the apparent adverse impact on the esophagus, too, thus contributing to the development of ulcerogenesis [42].

Generally, the oxidative stress plays a leading role in the genesis of multiple organ failure since ultrastructural alterations in the heart, liver, kidneys, lungs and brain are caused by the excessive activation of peroxide oxidation of lipids [43].

CONCLUSION

Notwithstanding the long history of its study the problem of stress remains relevant to date. Numerous scientific studies are covering the novel aspects of the stress impact on the body, particularly on the chest organs. Similar investigations carried out by the Ukrainian researchers in recent years allowed better understanding of the essence of the stress-related morphofunctional changes that occur in the lungs, heart and esophagus. The findings will assist in the search for ways to prevention and treatment of pathological processes caused by stress reactions; however, the problem of stress still remains to be solved to date.

The paper has been written within the research scientific study, carried out at the Department of Clinical Anatomy and Operative Surgery of the Higher State Educational Establishment of Ukraine "Ukrainian Medical Stomatological Academy", entitled "Determination of mechanisms of morphogenesis of organs, tissues and vascular-nerve structures of the body in normal condition, during experiment and under the influence of external factors. Morphoexperimental grounding of the efficacy of new surgical suture material used in clinical practice". State registration number: 0113U00124.

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Received: 25.07.2017 Accepted: 20.11.2017