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Review Article

Stress and Periodontium

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ABSTRACT:
Stress is compatible with good health, being necessary to cope with the challenges of everyday life. Chronic stress has a negative impact on the occurrence, development, and response to the treatment of periodontal disease via indirect actions on the periodontium. Stress disrupts the homeostasis of this network, which in turn alters the immune function. This article is an attempt to discuss interrelationship between stress and progression of periodontal disease.
Keywords: Oral health, stress, periodontitis.

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INTRODUCTION:
Periodontitis is a multifactorial disease. The dental plaque which harbors specific periodontal pathogens is its primary aetiological factor.[1] Since 1950 emotional factors have been identified in periodontal diseases.[2] Additionally some risks and susceptibilities have been connected with periodontitis, like systemic diseases, tobacco smoking, some genetic polymorphisms, socio-economic or educational status, and psychological stress.[3,4] Chronic stress is frequently thought to have a net negative effect on the success of the immune response, resulting in an imbalance between host and parasites, and ergo resulting in periodontal break-down.[5,6] For necrotizing periodontitis, stress is being exhibited as a secondary aetiological factor. Multifold clinical studies have probed the possible linkage between psychological stress and periodontitis and have recommended that stress may be taken part in the maturity of periodontal disease.[7,8] In a longitudinal study, Linden et al. submitted a linkage between occupational stress and the progression of periodontitis.[9] For years, it is being considered that the police profession has been ranked among the top five of most stressful occupations. Multiple studies have shown a positive linkage between stress and the police profession. Stress is also connected with certain masochistic habits like lip biting or cheek biting and habitual grinding of teeth. Systemic disorders like ulcerative colitis, overeating, and gastritis are also allied with stress.[10]

Multiple definitions of stress[1]
Stress originates from Latin word ‘stringere’ which means tight or strained.

Cannon in 1935 expressed stress as the consequence of homeostasis and showed the influence of the sympathetic system.

Hippocrates thought of health as a harmonious balance of the elements comprising the quality of life while disease represented disruption of harmony among those elements.

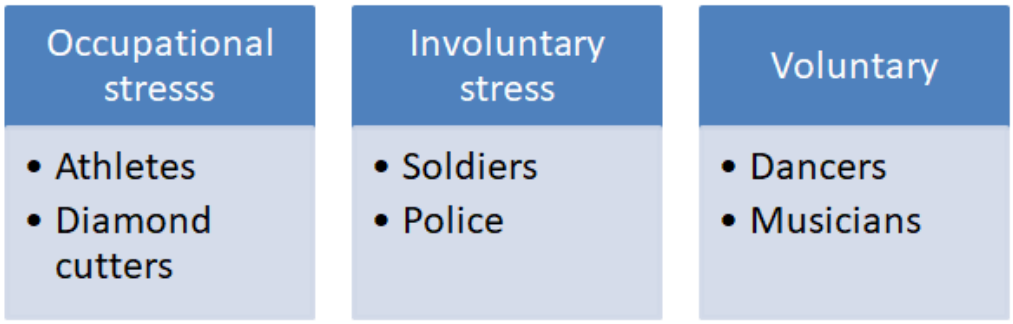
Sydenham suggested that pathological states represented diseases of adaptation – failure of the adaptive processes to restore well-being.

Hans Seyle(1992) Seyle defined stress as a response state of the organism to the forces acting simultaneously on the body which if excessive, that is straining the capacity of the adaptive process beyond their limits, lead to disease of exhaustion and death.

Coping is the response of the individual to stress (emotionally and physically).

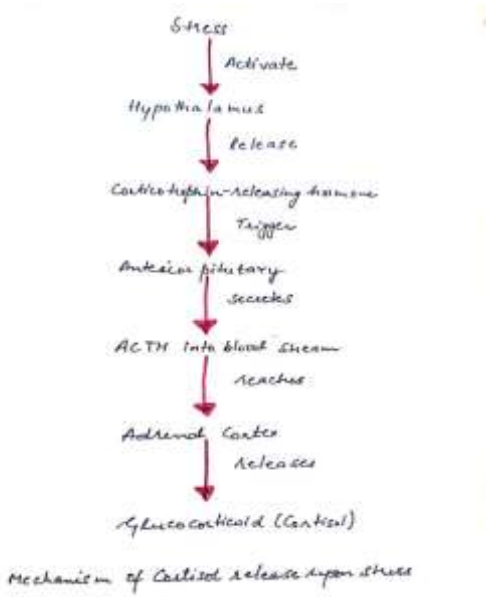
Stressors [11]

Types of stress [1]



Pathophysiology of stress and immune system[12]

In a review, Biondi displayed the impact of various psychosomatic conditions on the immune system. Stress can impair the deregulation of the immune system, mediated primarily through the hypothalamic-pituitary-adrenal and sympathetic–adrenal medullary axes. In response to a, an elegant sequence of events is initiated in response to stressful stimuli. Activation of the hypothalamic–pituitary–adrenal axis by results in the release of an increased concentration of corticotropin-releasing hormone from the hypothalamus. The pituitary gland is connected to the hypothalamus. Corticotropin-releasing hormone, in turn, acts on the anterior pituitary, thus leading to the release of adrenocorticotropic hormone (corticotropin). The adrenocorticotropic hormone then acts on the adrenal cortex leading to glucocorticoid hormones. The glucocorticoids then produce a myriad of effects suppressing the inflammatory response is one of them. It has been proved that pro-inflammatory cytokines, such as interleukin-1, can also activate the hypothalamic-pituitary-adrenal axis, leading to a feedback loop. Pro-inflammatory cytokines, such as interleukin-1 and tumor necrosis factor are two essential cytokines. The second major pathway to be activated is the sympathetic nervous system. Stress activates the nerve fibers of the autonomic nervous system. Thus the release of catecholamines can lead to hormonal secretion of norepinephrine and epinephrine from the adrenal medulla, thus modulating the immune response. Catecholamines, released during stress, contribute to the development of hyperglycemia. The sympathetic nervous system has a role in regulating immune cell activities.



Mechanism of Cortisol release upon stress

Stress and periodontal diseases

Gingivitis Stress diminishes saliva flow thus plaque formation is increased. Salivary pH and chemical composition are altered. Academic stress has also shown to be a risk factor for gingival inflammation with increased crevicular interleukin levels. In 1998 Axtelius showed the presence of cortisol in GCF. [13]

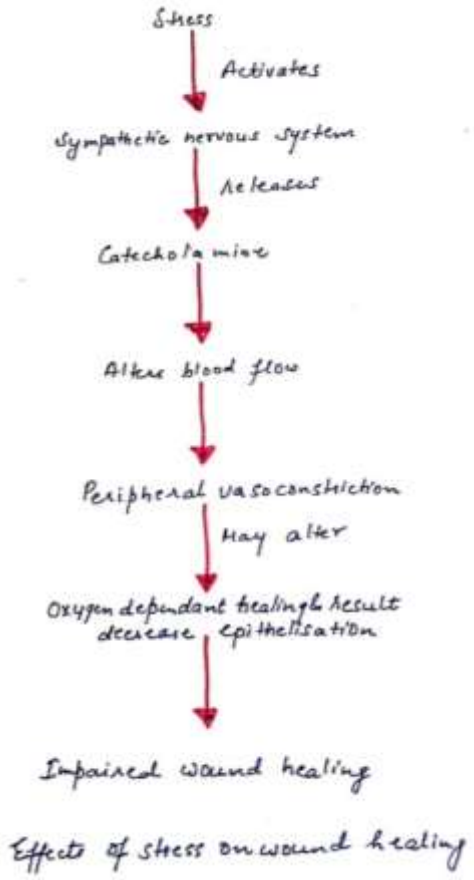
Acute necrotizing ulcerative gingivitis ANUG It is the most studied periodontal disorder concerning psychosocial factors. These factors will lead to increased bacterial overgrowth or weakening host resistance. Host resistance may be changed through ANS and endocrine glands thus it results in increasing levels of corticosteroid and catecholamine levels. Thus gingival microcirculation and salivary flow is reduced thus enhancing nutrition for Prevotella intermedia simultaneously also depress neutrophil and lymphocyte function. **Pindborg** showed a higher number of ANUG cases in military service.[11]

Aggressive periodontitis: Page et al. (1983) describes aggressive periodontitis as a particular disease and established the link existing between aggressive periodontitis and psycho-social factors and loss of appetite. **Kamma and Baehni (2003)** made a study to assess the clinical and microbiological status of patients with early-onset periodontitis who had acquired supportive periodontal care every 3–6 months for 5 years following active periodontal treatment. The results showed that supportive periodontal care was effective but was related to the progression of the disease with the following variables Porphyromonas gingivalis, Treponema denticola, total bacterial load, number of acute episodes, number of teeth lost, smoking, and stress. Thus there is a relation between psychosocial stress and periodontal treatment. [1]

Chronic periodontitis Linden et al.(1996) predicted the future attachment loss depending on the following criteria: age, a less satisfactory professional and socio-economical level, and a passive and dependent character. Psychosocial stress associated with financial problems and distress is risk indicator to develop periodontal disease.[1]

Effect on wound healing[12]

Stress releases highly active hormones like catecholamine, which causes altered blood flow, thus peripheral vasoconstriction takes place which may affect oxygen dependent healing mechanisms which impairs wound healing.



Effects of stress on wound healing

Risk factors of stress[14]

A risk factor is considered by longitudinal studies that relate a connection between factors and outcomes of diseases. On the other hand, risk indicators differ from risk factors as it is a connection between exposure event and outcome of disease but not demonstrated by longitudinal studies.

Periodontal disease is largely programmed by the influence of certain local and systemic risk factors that have an effect on the resistance of the host to pathogenic bacteria. Risk factors include diabetes, smoking a genetic variable called interleukin 1 polymorphism (IL1 genotype).

Diabetic patients have a much greater risk for progressive bone loss compared to nondiabetic patients.

Smokers are 4 times more likely to have periodontitis than nonsmokers.

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