Reverberations Of Stress On Periodontal Disease- A Review

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Abstract: Periodontal diseases are common chronic inflammatory diseases caused by pathogenic microorganisms which induce elevations of pro-inflammatory cytokines resulting in tissue destruction. Evolution of periodontal diseases is influenced by many local or systemic risk factors.Stress a term continually being re-defined in the scientific study of disease and illness,is nevertheless a confirmed and important factor in the etiology and maintenance of many inflammatory diseases, including periodontal disease. Stress is not what happens to us, it is our response to what happens and response is something we can choose. Stress results in delayed healing of the connective tissues and bone, apical migration of thejunctional epithelium and formation of periodontal pocket.Stress may negatively influence the outcome of periodontal treatment. In this review we will discuss the role of stress that might be playing in periodontal disease is considered. [Dr.Anita P NJIRM 2016; 7(4): 147-153]

Key words: Stress, Periodontal disease, immune system, behavorial changes

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Introduction: Periodontitis is a multifactorial disease.[1,2] Dental plaque which harbours specific periodontal pathogens is its primary aetiological factor. In addition several risks and susceptibilities have been associated with periodontitis, like systemic diseases, some genetic polymorphisms, socio-economic or educational status, tobacco smoking and psychological stress.[3,4]Chronic stress is commonlythought to have a net negative effect on the efficacy of the immune response, leading to an imbalance between host and parasites, and consequently resulting in periodontal break-down.[5] For necrotizing periodontitis, stress has been shown to represent a secondary aetiological factor.[6,7] Several clinical studies have investigated the possible relationship between psychological stress and periodontitis and have suggested that stress may play a role in the development of periodontal disease.[8– 10] In a longitudinal study, Linden e t al. suggested a relationship between occupational stress and the progression of periodontitis.[11]

In an exploratory case–control analysis of psychosocial factors and adult periodontitis, individuals with prevalent disease were found to report higher levels of social strain than individuals without prevalent disease. [12] So studying the effects of stress on periodontal disease will help to understand the proper course of both prevention and treatment.

Definitions: Definitions are important to understand stress because they are often misused. Stress originates from a latin word: 'stringere' which means 'tight', 'strained'. **Cannon(1935)** described stress as the result of the homeostasis and showed the influence of the sympathetic system.[13]

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. In the seventeenth century, Sydenham suggested that pathological states represented diseases of adaptation – failure of the adaptive processes to restore well being.[14]

Hans Seyle(1992) was largely responsible for giving the term stress its current saliency. 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 adaptive process beyond their limits, lead to disease of exhaustion and death.[15]

Stress is defined as a total transaction from demand to resolution in response to an environmental encounter that requires appraisal, coping and adaptation by the individual. Coping is the response of the individual to stress (emotionally and physically).[16]

Types of stress:

1. Occupational Stress: E.g. Athletes,boxers, Diamond cutters.
2. Involuntary Stress: E.g. Soldiers,recovery from General anesthesia.
3. Voluntary Stress: E.g. Dancers,musicians.

Stress is part of human condition, which is universally present, but to varying degree and with differenteffects on individuals.[16]

Selye(1976) defined forces that had the potential to challenge the adaptive capacity of the organism as 'stressors' and stated that stressors could be physical or mental (e.g. emotional). He recognized that stressors acting to produce changes in the body could be positive (e.g. exciting, pleasurable), leading to a response state he defined as 'eustress', or stressors could be negative, threatening homeostasis with pain, discomfort and physical pathology. He defined the negative response state as 'distress'.[17]

The following is the scale made by Holmes and Rahe in which all possible stressors received a value(Table-1).[18]

Table-1 Different stressors with their values

STRESSORS	VALUES
Death of spouse	100
Divorce	73
Death of close relative	63
Sickness	53
Marriage	50
Loss of work	47
Conjugalre conciliation	45
Retiring	45
Birth	39
To move in a new house	20
Holidays	13
Christmas	12

Stress coping strategies and its tools:

The stress reactions are all the biological, physiological or behavioural expressions that appear under certain conditions. The coping is the effort to try to reduce, control or tolerate the state of stress. It needs adjustment, adaptation and confrontation strategies

There are different coping strategies described here[19]:

1. Resigned coping: avoidance, escape, social withdrawal, resignation, self-pity,rumination.
2. Active coping: response control, situation control, positive self-instruction, minimization.
3. Distractive coping: distraction, search for self-affirmation, substitutegratification, need for social support.
4. Defensive coping: denial of guilt, self-blame, self-aggrandizement.
5. Coping with aggression and drug use: aggression, drug use.

These coping strategies may be used in generalized stressful situations. Individuals use coping measures as a response to stressors to reduce its intensity or toovercome stress altogether.

A successful coping is when the subject has the feeling to face the stress,he keeps the control of the given situation. An unsuccessful coping is when the subject is submerged by stressor agents and is in the reaction of stress. The reaction of stress is an imbalance between the demands perceived by the subject and the capability to face them. On the somatical level, hormonal and metabolical modifications appear. On thepsychological level, the patient has an exacerbation of vigilance and is in an emotional state with agitation.[19]

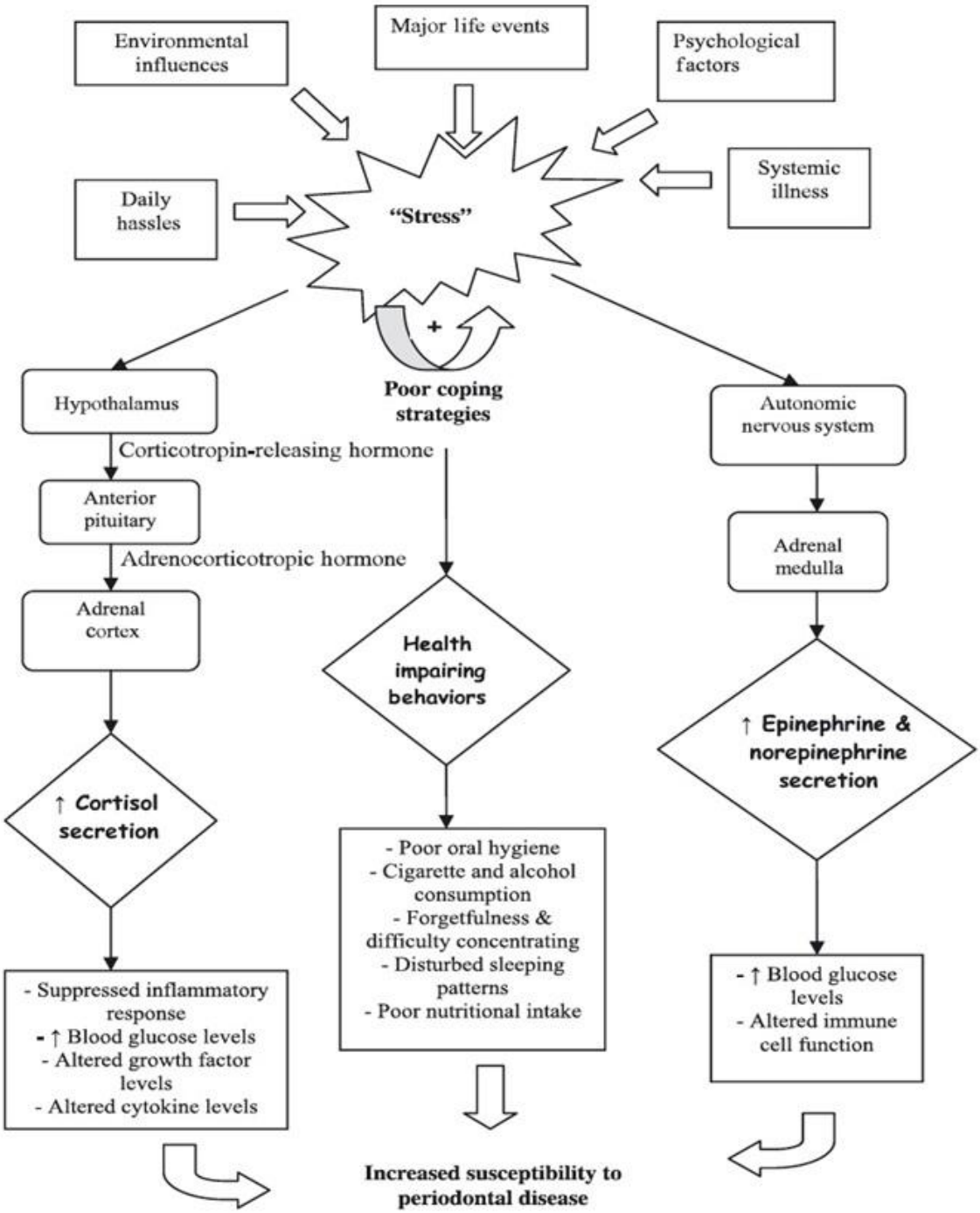
Tools for measurement of coping with stress:

1. Sign and symptom – related general physical examination: Blood pressure, skin response, pulse, skin temperature, various endocrine responses.
2. Various questionnaires- Stanford acute stress reaction questionnaire.
3. DSM-IV dissociation disorders.
4. Impact of event scale.
5. Post-traumatic stress disorder (PTSD) symptom scale.
6. Dissociative experience scale.
7. Life event scale by savoia.
8. Stress verarbatimfragebogan (SVF) scale.[20]

Pathophysiology of stress/ stress and immune system: In a review, Biondi[21] shows the impact of various psychosomatic conditions on the immune system.Stress can result in the deregulation of the immune system, mediated primarily through the hypothalamic– pituitary–adrenal and sympathetic–adrenal medullary axes [21] (Fig. 1).

In response to a variety of stressful stimuli, an elegant sequence of events is initiated. Activation of the

hypothalamic–pituitary–adrenal axis by stress results in the release of an increased concentration of corticotropin-releasing hormone from the hypothalamus. The pituitary gland is connected to the hypothalamus by the infundibulum, a stalk of tissue that contains nerve fibers and small blood vessels. Corticotropin-releasing hormone, in turn, acts on the anterior pituitary, resulting in the release of adrenocorticotropic hormone (corticotropin). The adrenocorticotropic hormone then acts on the adrenal cortex and causes the production and release of glucocorticoid hormones (predominantly cortisol) into the circulation. The glucocorticoids then produce a myriad of effects throughout the body, such as suppressing the inflammatory response, modifying cytokine profiles, elevating blood glucose levels, and altering levels of certain growth factors.[22,23]



Importantly, it has been established that pro-inflammatory cytokines, such as interleukin-1, can also activate the hypothalamic–pituitary–adrenal axis, leading to a feedback loop.[24] Immune function plays a critical role early in the wound healing cascade. Pro-inflammatory cytokines, such as interleukin-1 and tumor necrosis factor, are just two of the essential cytokines in this regard. It has been speculated that success in the later stages of healing is critically dependent on these early events.[25]After experimentally created blister wounds were inflicted in human subjects, it was found that women who reported higher stress levelsproduced lower cytokine levels than womenreporting lower levels of stress. In addition, the individuals exhibiting greater stress had higher salivary cortisol levels(Figure-1).[25]

The second major pathway to be activated is the sympathetic nervous system. A well-known example of this is the so-called flight or fight response to potentially harmful stimuli. Stress activates the nerve fibers of the autonomic nervous system, which innervate the tissues of the immune system. The adrenal medulla is actually a modified sympathetic ganglion. Its nerve bodies, instead of possessing axons, secrete their products directly into the bloodstream. The release of catecholamines results in the hormonal secretion of norepinephrine and epinephrine from the adrenal medulla, which results in a range of effects that may act to modulate immune responses. Catecholamines, released during stress, contribute to the development of hyperglycemia by directly stimulating glucose production and interfering with the tissue disposal of glucose.[22] In addition, the sympathetic nervous system has a role in regulating immune cell activities.[26]

From the above discussion it is apparent that the response of the human body to stressful stimuli is at once helpful and potentially therapeutic, even though a potentially harmful imbalance occurs when the stressful stimuli, or perceived stimuli, are prolonged.

Examples include chronic anxiety states and depression.[2]

Stress and behaviour changes: Ringsdorf and Cheraskin(1969), discovered that mental stress could influence life-style and dental hygiene habits. This influence was not only the decrease of the frequencyas well as thequality of the dental hygiene but also the increase of tobacco use and alcohol consumption, changes in food habits leading to a diminution of the general health.[28]This was also recently confirmed by Suchday et al.[29]

As the bacterial invasion is facilitated (poor oral hygiene) and the immune response becomes weaker it is logical to assume that periodontal disease will be enhanced by stress.

Stress and periodontal diseases: As stress influences the immune system and also changes the behaviour, it is extremely important to analyze if its influence is the same on the different forms of the periodontal disease.

Gingivitis: Stress diminishes saliva flow and increases dental plaque formation. Emotional stress modifies the saliva pH and its chemical composition like the IgA secretion.[30]A series of studies made by Deinzeret al. (1998-2001), examine the impact of academic stress by students at university during their examination period on periodontal health. Academic stress was shown to be a risk factor for gingival inflammation with increasing crevicular interleukin-1b levels and a diminution of the quality of the oral hygiene.[31–34] In a pilot study in 1998, Axtelius showed the presence of cortisol in gingival crevicular fluid.[35] A study in 2006 confirm the fact that the concentration of cortisol in the gingival crevicular fluid is higher by person showing depression signs.[36]

Necrotic periodontitis: Psychosocial factors are predisposing factors for the development of necrotic periodontitis. The first studies showing this influence were made by Pindborg (higher number of necrotic periodontitis during military service) and in 1963–1964 by Giddon (more necrotic periodontitis in college during examination period).[37, 38]Other studies like that of Cohen-Cole in 1983, have shown the influence of psychosocial factors.[39] The main risk factors for necrotic periodontitis and previous episode are: past episode of necrotic periodontitis, bad oral hygiene, bad sleep, unusual emotional stress, tobacco, alcohol, bad alimentation and recent illness. Many of those factors are often related to stress.

Aggressive periodontitis: Page et al. (1983) describes the aggressive periodontitis as a particular disease and established the link existing between aggressive periodontitis and psycho-social factors and loss of appetite.[40] In a case–control study in 1996, Monteira da Silva showed that people with aggressive periodontitis were more depressed and more socially isolated people than people with chronic periodontitis or a control group.[41]**Kamma and Baehni(2003)** made a study to evaluate the clinical and microbiological status of patients with early onset periodontitis who had received supportive periodontal care every 3–6 months for a period of 5 years following active periodontal treatment. The results showed that supportive periodontal care was effective, but some sites in some patients were still progressive. These variables were related to the progression of the disease: Porphyromonas gingivalis, Treponema denticola, total bacterial load, number of acute episodes, number of teeth lost, smoking and stress [42]. These studies show the relationship existing between aggressive periodontitis and psychosocial stress.

Chronic periodontitis: Linden et al.(1996) predicted the future attachment loss depending on the following criteria: age, socio-economical level, a less satisfactory professional life and a passive and dependant character.[43]Axtelius (1998) has suggested that patients with psychosocial strain and passive dependent traits did not respond as well as patients with less stressful psychosocial situation and with a rigid personality to periodontal treatment.[44] Psychosocial stress associated with financial problems and distress are risk indicators to develop a periodontal disease.[45]

Effect of stress on wound healing: Stress releases highly active hormones like catecholamine, which results in altered blood flow, peripheral vasoconstriction may affect oxygen dependant healing mechanism which impairs wound healing. (Figure-2)

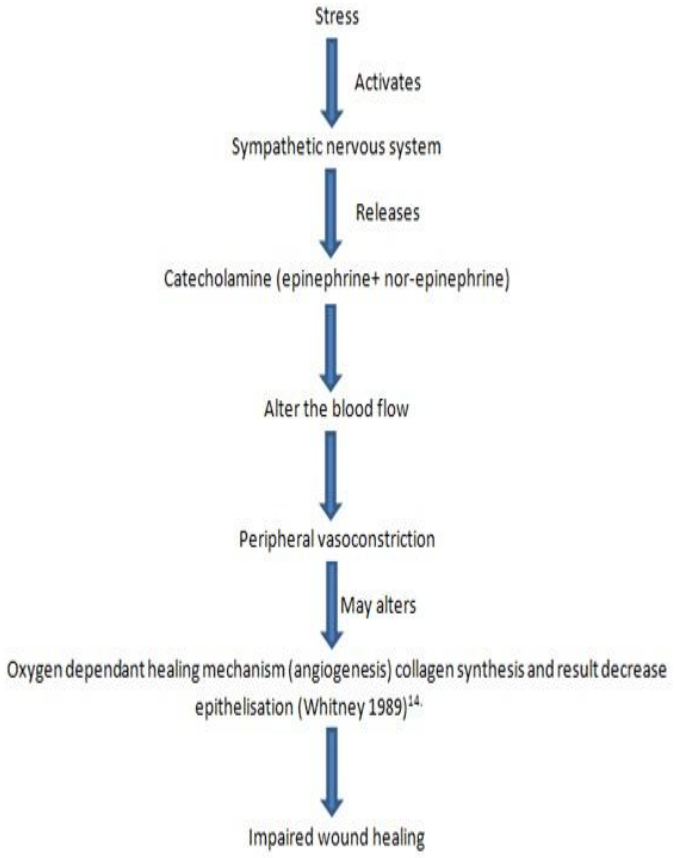


Figure -2[46] Effects Of Stress On Wound Healing

Treatment: Doctor needs to take a careful history to look for an underlying stress or psychological disorder that could be the source of patient's stress symptoms. Many times, a careful interview can be the best source of
information about the cause of patient's symptoms. Patient should be referred to a psychiatrist for help.

There are four basic approaches to dealing with stress:

_ Removal or alteration of the source of stress
_ Learning to change how you see the stressful event
_ Reducing the effect on your body that stress has
_ Learning alternative ways of coping

Breathing exercises and Guided imagery are simple ways of relieving stress and achieving wellbeing as a whole.[47]

Conclusion: Studies to date strongly suggest that stress, distress, and inadequate coping are important risk indicators for periodontal disease. Because of the better understanding of the stress dynamic and the progress made in the field of the psycho-neuro-immunology endocrinology and also because of the impact stress has on the behaviour, it is clear that stress influences periodontal disease. Stress also results in delayed healing of the connective tissues and bone in artificially induced gingival wounds but

does not affect the epithelium. In chronic stress osteoporosis of alveolar bone, apical migration of the junctional epithelium and formation of periodontal pocket occurs. These early beginnings will require extensive study to fully understand the molecular and cellular basis of the role of stress, and in turn these studies may lead to effective intervention strategies that minimize or negate stress as a contributing factor to periodontal diseases. The role of the dentist is to discuss lifestyle in a broader concept than just oral hygiene; they should be more psychologically oriented. It is very important to understand the patient's situation to help them to maintain a healthy periodontium. In addition all other risk factors for periodontal disease should be minimized, promoting good oral hygiene and smoking cessation.

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