THE ROLE OF NEGATIVE EMOTIONS IN THE AETIOLOGY OF NON-IMPACT SPORTS INJURIES: INTERACTIONS OF THE STRESS RESPONSE AND MOTOR CONTROL SYSTEMS.

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Introduction

Athletes of all sports are subject to many injuries, which interfere with their training programs, progress and performance, and are a major cause for career-ending withdrawal from competitive sport. Although the nature and location of injuries are sport and gender specific, Iwamoto and colleagues' (2008) 14-year retrospective study, covering 2989 athletes, illustrated the prevalence of knee, low back and ankle injuries within the six sports with record of >100 injuries. Another epidemiological study (Caine et al, 2003) of female gymnast highlighted that incidence and severity of injuries are related to the competitive level and pre- competition and competition time. Further study data (Kerr & Minden, 1988) linked the high rate (83%) of pre-competition incidence and severity of injuries in elite gymnasts with stressful life events.

With the high status of competitive sport in modern society, many studies have been undertaken in the past few decades, attempting to identify and explain the principle causative factors involved in sports injuries – training styles and programs, equipment, training-recovery-competition balance, as well as other individual and lifestyle factors. For the last two decades the application of psychological approaches in sport has brought emotional, cognitive and personality aspects to the forefront of sports science research. The most prominent advances in this area are the Multi-dimensional Anxiety theory (Martens et al, 1990); Cusp catastrophe model (Hardy, 1996); Conscious Processing theory (Masters, 1992) and Processing Efficiency theory (Eyzenck & Calvo, 1992) which Eyzenck and colleagues further developed into the Attention Control Theory (2007). In the late eighties and early nineties Williams and Andersen adapted general stress theories to sports injury and developed the Stress and Injury model (Williams & Andersen, 1986; Andersen & Williams, 1988; Andersen & Williams, 1997). As the Stress-Injury model illustrates

well all the strengths and shortcomings of the psycho-social approach to sports injury, the next paragraph will discuss it in greater detail.

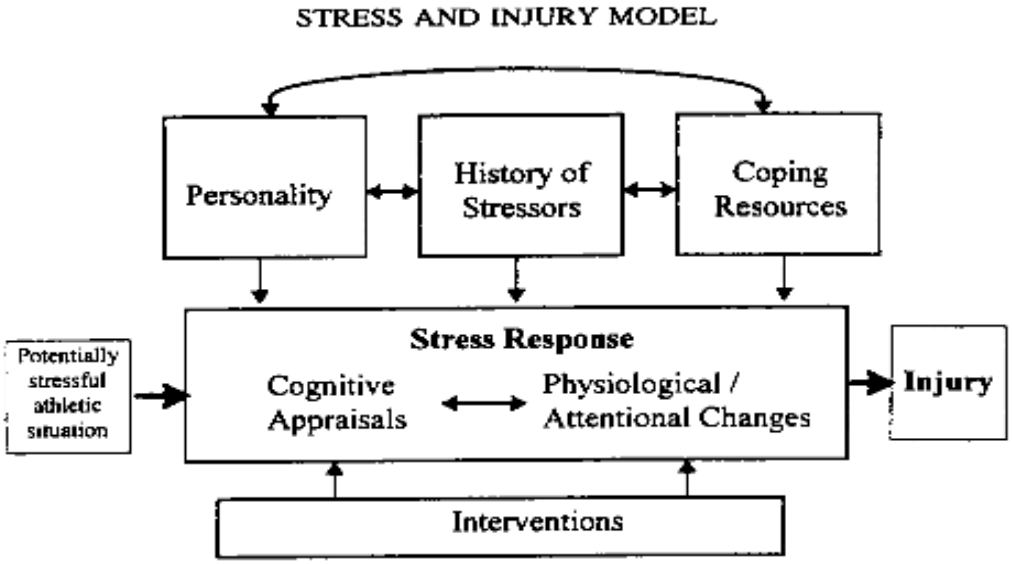

Figure 1. The revised version of the Stress-Injury model (Williams & Andersen, 1998)

The Stress-Injury model (figure 1. above) analyses in a comprehensive way the predisposing factors, associated in previous research with increased risk of injury. Williams and Andersen proposed that injury results directly from the athlete's stress response to a challenging situation (hard, demanding training; competition), which alters attention, muscle tension and visual perception. As the stress response severity is the direct mechanism of injury, Williams and Andersen concentrated their effort on identifying the internal (psychological) and external (negative life events, social support) factors involved. The evidence they presented, clearly demonstrates that both Major and Minor Negative Life Events, as well as previous injuries increased the risk of injury. The personality traits positively correlated with higher injury risk were external locus of control (negative perfectionism), trait anxiety, low affective states and negative mood states (defensive pessimism), as well as anger and aggression. Unsurprisingly positive mental states correlated negatively with risk of injury. As cognitive anxiety and perceived stress result from mismatch of demand and resources, social support, stress management and coping skills, as well as sensible lifestyle habits exerted a moderating effect on injury vulnerability. Hence, the interventions that Williams and Andersen found to reduce the occurrence of injuries were the ones that managed and reduced the stress response – psychological

interventions modifying cognitive appraisal of challenging events and situations, cognitive and biofeedback training and relaxation techniques.

The Stress-Injury model, similarly to other psycho-social theories, provides an insightful explanation of the complex interacting factors associated with stress as well as the cognitive/emotional mechanisms, which underlie attentional dysregulation and inadequate control of goal orientated behaviour (in this particular case – execution of complex movements) and predispose athletes to injuries. The weak point in all these theoretical models is the oversimplification of the exact neuro-physiological mechanisms, which link negative emotional states with motor control regulation of highly skilful, complex and physically challenging movements, as is the reality for athletes from beginner to highly advanced Olympic competitor level.

The follow-up paragraphs will attempt to briefly analyse from functionality aspect the neural substrates, which constitute the autonomic regulation and motor control systems. From whole body system's perspective, the most fundamental principle that underlies behaviour is the principle of dynamic integrity and stability (both structural and functional). The Autonomic nervous system (ANS) through the function of its two branches (Sympathetic and Parasympathetic) and neuro-endocrine and immunological regulation preserves the dynamic stability of the internal environment in the face of constant change (Chapman et al, 2008; Gold & Chrousos, 2002), as part of the general activity of the Central nervous system (CNS). The motor control system (MCS), as part of CNS, preserves the mechanical stability and integrity of the body (Panjabi, 1992) in the face of movement. In these terms the stress response can be viewed as behaviour, which disrupts the dynamic stability of the entire CNS and involves both the ANS and MCS. Viewed from this perspective, musculo-skeletal injuries are direct consequences CNS-MCS dysregulation, which manifests as altered, inadequate or inappropriate stabilisation strategy. To substantiate this hypothesis, this review will analyse the nature of the stress response and its connection with postural control and stability in anxiety, negative mood disorders and chronic pain conditions.

Emotion, behaviour and stress response

In the past few decades many different stress theories have evolved, approaching the complex interaction stimulus-psychological/physiological response from sometimes opposing angles. Some emphasize the importance of the characteristics of the environment (stimulus) and psychological and cognitive effects of life events. Others concentrate on the physiological aspects of the stress response and its neural and metabolic correlates, while there are also theories, which view the stress response as a type of evolutionary developed valence motivated behaviour. All these theories have many valid and common points, but when it comes to the specifics of meaning and use of common terminology like ‘stress’, ‘emotion’, ‘arousal’ and ‘balance/homeostasis’, the differences of their conceptual approaches become apparent, adding further confusion to this, already complex subject-area.

The most widely used, especially in bio-medical sciences, Response model of stress follows the psycho-physiological approach. It has its origins in Cannon’s (1929) ‘fight-or-flight’ response. Cannon defined stress as an aversive response to a threatening stimulus, which disturbs the homeostatic balance of the organism. Starting with Selye (1936), the continuous effort of many researchers has managed to locate, explain and inter-relate the specific neural structures and pathways, neurotransmitter systems, as well as endocrine and other specifics of the stress response. It is well accepted that the stress response alters the activity of the whole neuro-axis: from sensory and emotional/motivational and memory association cortices; through thalamus, hypothalamus and brainstem structures; to spinal cord and peripheral autonomic and somatic nerves (Wilson, 2003; Siegel & Sapru, 2006). The principal structures involved with emotional and motivational modulation and memory are the limbic structures of the Amygdala (central nucleus in particular, cAmg), Bed nucleus of the Stria Terminalis (BNST) and the Hippocampus (Chapman et al, 2008; Gold & Chrousos, 2002; Walker et al, 2009), which initiate and maintain fear and anxiety, while the ventral pre-frontal cortex (PFC) is associated with goal setting and motivation (Hansel & von Kanel, 2008). The general activating (arousal) physiological response is executed by Hypothalamic (mainly, but not exclusively Paraventricular Nucleus, PVN) and mid-brain and brainstem nuclear groups like Locus Coeruleus (LC), Raphe nuclear groups, Central and Para-aquaductal Grey and many parts of the Reticular Formation (RF), which activate the Sympathetic branch of the ANS (SNS) and the systemic secretion of adrenalin from the adrenal

medulla. With the activation of the pituitary by CRH from the PVN, but also noradrenalin from LC and serotonin from pontine Raphe nuclei, ACTH gets released, which triggers the secretion of cortical hormones from the adrenal cortex. Glucocorticoids in turn initiate a concerted alteration of metabolism of the entire organism, raising blood sugar and fat levels, suppressing immune function, as well as altering functions of many areas in the CNS (Chapman et al, 2008). Corticoids also serve as a terminator of the stress response through negative feedback to PVN, LC and the Raphe nuclear groups (Kvetnansky et al, 1993; Goczynska et al, 1995; Dinan, 1996).

As all other evolutionary reflexes, the stress response is very specific, effective and efficient in producing adequate life-saving behaviour when faced with major threat. In conditions of low-level but persistent threat (chronic stress), the stress response becomes maladaptive and it is widely accepted to be in the core of many chronic both affective (depression, anxiety and panic) and somatic (cardiovascular, metabolic, digestive) conditions. This assumption poses few conceptual problems. Either selective evolution is a faulty premise, or conditions of continuous low stress have never existed and are a modern day phenomenon. Quite the opposite is true - the insecurities of life (food and other resources) are the ones that define day-to-day existence for every organism on this planet. So the ability to withstand low-level persistent stress is what constitutes ‘survival of the fittest’ and the activation of the SNS and hypothalamo-pituitary-adrenal (HPA) axis reflects the mechanisms of physiological arousal only.

The physiological arousal executed by the altered activity of the HPA axis in connection with the activation of the SNS is part of the stress response and should not be equalled with the stress response itself. What defines the stress response in comparison to straightforward SNS-HPA physiological arousal is the elucidation of a specific behaviour of helplessness, resignation and withdrawal. This is well illustrated in experiments with animals where by manipulation of the context (meaning) of the threatening stimulation, opposite behaviours can be generated – either active behaviour (physiological arousal), or passive, withdrawn and resigned one (stress) (Seligman & Maier, 1967; Davies, 1989; Davies, 1997). The neural substrates in these two physiological states differ as well – the withdrawn behaviour is associated with different patterns of Amg/BNST and PFC activation of the peri-aquaductal gray

and the HPA-SNS system (Fanselow, 1995; Hansel & von Kanel, 2008). The passive, withdrawn behaviour represents the true nature of the stress response - a maladaptive state of dysfunction of central (cortical) and autonomic (sub-cortical) regulation – central-autonomic dysregulation.

Animal experimental models have provided many insights of the intimate physiological mechanisms of action and interaction of the different parts of the stress response system, but they have one major limitation – animals can not tell us how they feel. In the past twenty years a set of more than 600 emotionally calibrated pictures have been developed to test the emotional, motivational and physiological responses in humans (Lang et al, 1997). The two main factors in the classification of the pictures are the emotional valence (based on the dichotomous aspect of behaviour – attraction vs. aversion) and intensity of physiological and cognitive arousal. The pictures are classified as emotionally negative, positive or neutral in valence and low or high in arousal intensity. They can be used in combination with measures of emotional perception (self-rating), physiological activity (heart rate-HR, galvanic skin response-GSR, startle reflex-SR) as well as brain imaging techniques (PET and fMRI) to yield invaluable insights into the complex mechanisms of interaction between cognitive, psychological and physiological processes in both normal and pathological states (Lang et al, 1993).

A team from Florida published a comprehensive review (Lang et al, 1998) of the research accumulated using emotive picture viewing up to that date. The results from their analysis point clearly that there is a fundamental distinction between the two factors involved in emotional behaviour – emotional valence and physiological arousal. Both negative and positive emotion pictures (as measured by personal rating) were viewed for longer and produced higher arousal (measured by GSR) compared to neutral pictures, but positive pictures reduced Heart rate (HR) while negative pictures increased HR. Interestingly male subjects had a stronger response (both cognitive and physiological) to positive emotion pictures, compared to women, who responded stronger to negative ones. The results from brain activity (PET and fMRI) studies shed further light on the mechanisms of emotion processing. They confirmed that both positive and negative pictures produced arousal (GSR), but surprisingly neutral and pleasant pictures produced very similar patterns of brain activation in comparison to negative pictures.

The startle reflex (SR - blinking reflex in experimental human studies) is a defensive, evolutionary and subconscious reflex. SR is associated with evoked fear and is used to provoke and study the stress response. The results in healthy volunteers showed a marked alteration of the startle reflex (SR) in respect to the valence and arousal intensity of the pictures viewed. The positive emotion pictures reduced and delayed the SR in comparison to neutral and negative ones, while the negative pictures augmented and accelerated the reflex in comparison to neutral and positive ones. Studies of the interactions between emotional picture viewing and SR in anxiety and panic disorders (PD) have shown unexpected, contradictory at first glance, results. When viewing negative emotion pictures, PD participants (commonly associated with heightened sense of fear and stress response) showed similar increases in SNS activation and arousal (measured by GSR and self-report) compared to anxiety (simple phobic) and normal participants. At the same time they showed less increase in their heart rate (HR) and delayed, less augmented SR – which can be interpreted as lower stress response. After reanalysing the results, it became apparent that, although PD participants had lower increase rates of HR and SR, it was from much higher base level. These results indicated a very different cognitive, emotional and physiological stress response pattern, which was difficult to explain from the simple SNS-HPA activation mechanism of the conventionally understood stress response.

Another highly insightful review (Friedman & Thayer, 1998) observed the same abnormalities in the stress pathology of anxiety and PD, using completely different measure - heart rate variability (HRV). Their results linked PD with elevated HR, but lower HRV.

HRV is a measure, which reflects the relative contribution of the SNS and PSNS (vagal tone) of the ANS control over cardiovascular function. Spectral analysis of ECG reveals that internal oscillations exist within the ECG signal. Vagal and sympathetic efferents discharge at different rates: vagal - at ~0.25Hz are associated with the respiratory regulation of HR; sympathetic – at ~0.10 Hz reflect baroreceptor-mediated regulation (Malliani et al, 1991). The high frequency (HF) vagal regulation of HR is fast acting, able to achieve fine-tuned control over cardiovascular (CV) function. Sympathetic regulation, on the other hand, happens at a lower frequency (LF) and only produces generalised effect on CV function. HRV is the ratio between LF and HF, where low HRV combined with diminished HF power signifies reduced

vagal tone, which leads to poor HR control by the ANS and elevated HR. This is precisely the situation in PD in which the cardiac symptoms of tachycardia and palpitations are one of the predominant somatic symptoms.

In their review Friedman & Thayer (1998) pointed out that lower HRV and diminished vagal tone are also associated with stressful cognitive processes and worry, general anxiety disorder, major depression, cardiovascular disease, neurologic disorders, diabetes and foetal distress, all of which bear the hallmark of stress induced conditions. High HRV and vagal tone, in contrast, were associated with adaptive responsiveness, ability to maintain attention, mood control and increased capacity to aversive stimulation. In their view, reduced vagal tone, reflected in low HRV, indicated abnormal function of the ANS and was the underlying mechanism of central-autonomic dysregulation in all above-mentioned conditions.

Friedman & Thayer based their interpretations on the premise that the body is a self-regulating, open, non-linear, system with multiple parallel levels of control, where stability is maintained by continuous fluctuations of many interacting variables. In that respect homeostasis does not equal avoidance of perturbations, but totally the opposite – inability to change is a sign of system rigidity and un-adaptability, as is the case in many chronic disease states. In this light, the reduced startle reflex and HR increase in PD participants, in the above-mentioned emotive picture viewing studies, make complete sense. The diminished responsiveness and simultaneous high level of background arousal were due to the rigid state of the PD participants' system, which is well reflected in the dysfunctional restrictiveness of their general behaviour.

In summary, the above examples provide ample evidence that the stress response is not the same as simple activation of the SNS-HPA system. Furthermore, they point towards the fundamental function that the parasympathetic system (PSNS) plays as part of the ANS-CNS regulation. PSNS provides the axis of stability in the ever changing and adjusting organism, without inhibiting responsiveness and adaptability, and its robustness is what qualifies the 'fittest' to survive. PSNS dysfunction, as reflected in reduced vagal tone, affects all systems in the body and is what defines all states of true stress.

Emotion, posture and motor control

The Motor Control system (MCS), similar to the autonomic regulation system, is another typical example of an open, non-linear system, where movement is a product of simultaneous, stochastic in nature, control signals (van Galen & van Huygevoort, 2000). Movement is based on simple spinal (segmental and inter-segmental) reflexes upon which parallel, multiple levels of the CNS (sensory-motor cortex, Hypothalamus, Basal ganglia, cerebellum and many midbrain and brain stem nuclear groups) exert continuous and simultaneous control (Wilson, 2003; Siegel & Sapru, 2006). The MCS co-ordinates musculo-skeletal function for the dual purpose of maintaining mechanical stability and integrity and producing movement (Panjabi, 1992). The MCS employs both 'feed back' and 'feed forward' (anticipatory) processes, which are dependant on integrated sensory information and accurate self-image in relation to the environment. MCS effectiveness and efficiency is reflected in the strategies it employs to achieve stability without producing rigidity, which would compromise the expression of complex, purposeful movements.

In the past few decades many experimental studies have been carried out, consistently demonstrating that emotional factors alter motor control. Hillman and colleagues (2004) examined the effects of emotive picture viewing on postural control in healthy under-graduate students. Their results confirmed that both positive and negative emotion pictures increased arousal (GSR, self-report) and only negative pictures increased the Startle reflex. Interestingly, only negative emotion pictures were also associated with changes in posture (Centre of pressure) – female participants leant more backwards, while male ones leant more forward.

Another comprehensive study (Bolmont et al, 2002) established that the postural changes, associated with negative mood states (tension, depression, hostility, fatigue) and anxiety (self-report questionnaires) in healthy subjects, resulted from changes in sensory information processing and motor control. The negative mood states and anxiety impaired the ability of the participants to adequately utilise postural sensory information (visual, somato-sensory and vestibular) as well as increased the latency of their motor response (correcting posture after unbalancing perturbation).

The frequent occurrence of clinical co-morbidity of balance disturbances and anxiety (Eagger et al, 1992; Stein et al, 1994) has led to groundswell of research in that area. Different studies employed various experimental protocols to create anxiety and measure sensory and postural changes with consistently similar results – anxiety and alteration of posture control are equally linked in younger and older adults (Brown et al, 2006), children (Erez et al, 2004) as well as mice models (Lepicard et al, 2003). A culmination in the area of balance and anxiety research was reached by Balaban and Thayer's review published in 2001.

In their paper Balaban and Thayer refined and extended existing theories of CNS processing of afferent exteroceptive, interoceptive and proprioceptive information, as well as the central-autonomic and central-motor neural connections which control the two aspects of behaviour – metabolic and somatic. Central role in this circuitry is played by the Parabrachial nuclear (PBN) groups in the pons. As can be seen from the diagram below (Fig.2), the PBN's position allows it to receive and integrate sensory information from multiple sources. The close connections with the vestibular nuclei (VN) provide PBN with information from the inner ear organ, as well as visual information. Proprioceptive information from muscle spindles, tendon organs and other joint and skin receptors enters the PBN directly, or via the Thalamus-Amygdala link, which also relays emotionally interpreted information from the other senses. The Nucleus of the Solitary tract (NTrS) conveys vagal afferents' interoceptive and interior proprioceptive information. On the other hand, PBN is closely connected with circuits executing autonomic regulation (Hypothalamus, Rafae nuclear groups, LC and NTrS as well as ventro-medial Medulla) and motor control (VN, Reticular Formation, LC and RN group). Balaban and Thayer point out that the close reciprocal connections between the PBN and the cAmg, BNST and PFC are sufficient to explain the co-morbidity of balance disorders with anxiety and other negative mood and affect disorders

Another very interesting and, in their view, under-appreciated aspect of the PBN is its' ability to integrate information regarding gravitational forces, which affect not just the head (inner ear organ - VN), but also the musculo-skeletal system (various muscle, joint and skin receptors) and the vascular and visceral pressure (vagal afferents). PBN's central position explains its capacity to initiate such a wide-spread emotional, cognitive, autonomic and somatic response to disturbances of the sense

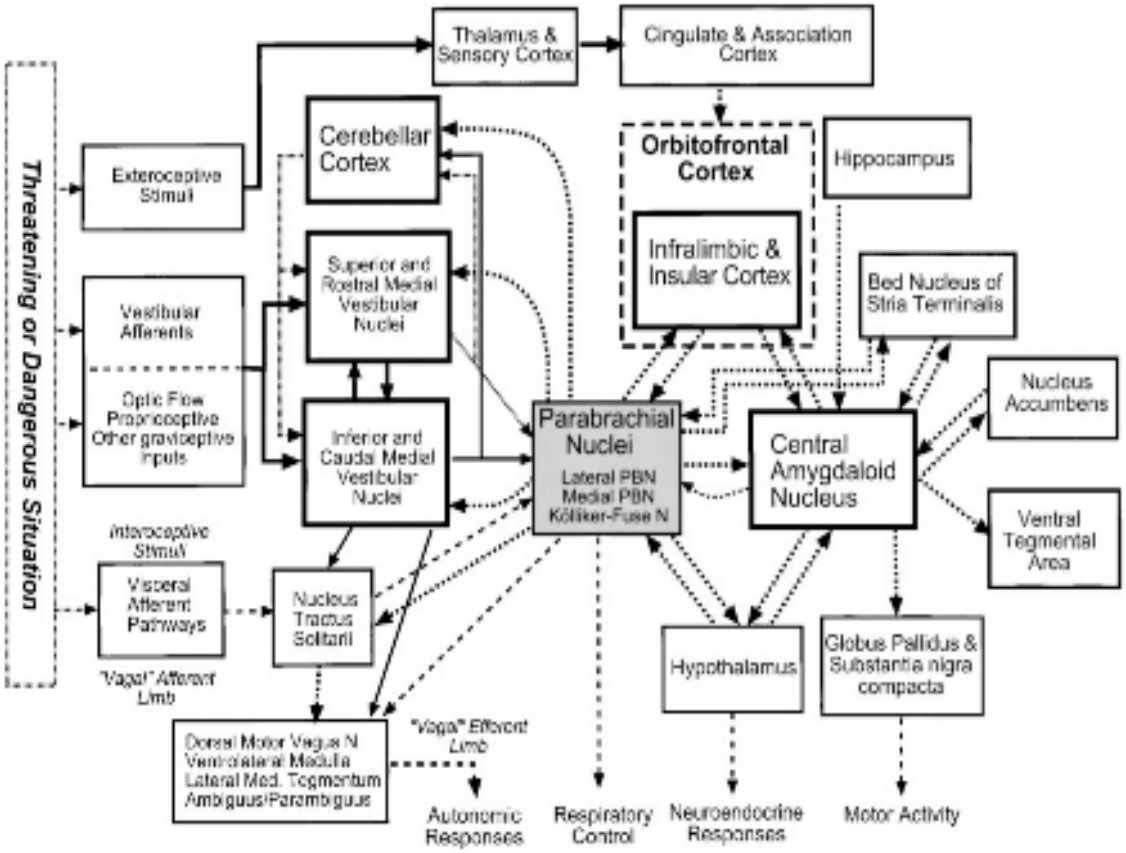

Figure 2. Principal connection of the Parabrachial Nuclear group (Balaban & Thayer, 2001).

of gravity. As out all the senses, both evolutionary and gestationally, the sense of gravity is likely to be one of the first to develop (this is true at least for life on this planet). The principle characteristic of gravity as a type of stimulation is its constancy and centripetal orientation of its force. The sense of gravity is fundamental to the sensory self-determination of the organism and plays a central stabilising and pivotal role in all functions of the CNS and especially in respect to the MCS. The PBN network links the sense of gravity, the affective and metabolic state of the body with the posture stabilising function of the MCS, so any disturbance in either of them simultaneously affects the whole system.

Evidence from chronic painful conditions (for a comprehensive review, see Hodges & Moseley, 2003) further confirms the above findings. Altered MCS strategy of stabilisation plays a crucial role in these conditions. Studies of chronic low back pain (LBP) have consistently found two main abnormalities of function of the trunk muscles: abnormal flexion-relaxation ratio of the back extensor (Erector Spinae) muscles (Watson et al, 1997; Geisser et al, 2005) and delayed activation of the deep

abdominal (Transverse - TrA) muscles (Hodges, 2001; Hodges & Richardson, 1996; Hodges & Richardson, 1999) with dysfunction of the deep spinal axial (Deep Multifidi - MF) muscles (Hides et al, 1996; Hides et al, 1993; MacDonald et al, 2009). This shift in stabilisation strategy from the deep axial musculature to the superficial trunk muscles in LBP sufferers, results in a rigid and inefficient posture control (Brumagne et al, 2008). Studies of the effects of psychological and mental stress on posture control (Marras et al, 2000; Davies et al, 2002) have reached similar conclusions – both psychological and mental stress increased the co-activation of the superficial trunk flexors and extensors, which significantly augmented spinal loading and the risk of spinal injury.

The part the deep axial musculature plays in mechanical stabilisation resembles closely the part the PSNS plays in metabolic stabilisation. The ANS and MCS are only two aspects of the function of the CNS, so it is not surprising that any dysfunction of either one of them will dysregulate the whole CNS and ultimately affect the other one. There is a striking functional similarity between the inadequate stabilization of autonomic function in PD, as well as other stress conditions (depression for example) and posture stabilization in chronic painful conditions - they all result in low emotional mood states and rigid, un-adaptable and severely restricted behavior.

Conclusions

The human body is a self-regulating open system, where homeostatic balance is achieved by continuous fluctuations of many inter-related variables around a central axis of stability. The multi-level, hierarchical parallel organisation of the CNS is a good illustration of this. The CNS through the function of the ANS (SNS and PSNS) and neuro-endocrine and immunological regulation maintains flexible homeostatic stability; while through the function of the MCS, it regulates mechanical movement and stability of the body. The autonomic stability of metabolism relies on robust and resilient PSNS (as expressed in high vagal tone), while the MCS achieves mechanical stability through employing the deep axial (MF and TrA, as well as other upper and lower girdle muscles) musculature for this purpose.

The stress response is a behaviourally maladaptive state, which manifests as altered function of the CNS and psycho-physiology – central-autonomic dysregulation. In the core of the stress response system is the emotional and motivational circuit of the cAmg, BNST and PFT cortex, which activates the physiological arousal systems of the SNS and HPA axis as part of the stress response. The same circuit plays also central role in connection with the PBN in integration of sensory (especially gravitational) information and regulation of balance and posture via the VN, RF and other mid-brain and brain stem nuclear groups. Alteration of the activity of PBN-Amg/BNST-PFC network by negative emotional and mood states in stress is the direct mechanism of dysregulation of the MCS.

MCS dysregulation manifests as rigid, static and unresponsive postural strategy. As a behaviour, this rigid strategy is totally ineffective, when fast, precise and highly skilful and complex movements are required. It is also inefficient as it is achieved by co-contraction of large antagonistic groups of muscles, which has the dual effect of consuming excess energy and overloading the passive structures of the musculo-skeletal system (ligaments, bones and other joint structures). This inflexible and inappropriate stabilisation impairs the fine, precise control of movements and increases the likelihood of acute injury occurring. It also increases the accumulative wear on both the muscles and the passive structures of the musculo-skeletal system and is in the root of most chronic, overuse injuries.

As modern sport is characterised by performing physically challenging movements in conditions of heightened psychological pressure, it is not surprising the high occurrence of both acute and overuse sports injuries. Apart from athletes, many millions engage in physical exercise on a regular basis in order to reduce stress. It would be interesting to investigate how the rates of sports injuries in non-athletes relate to the specifics of their stress - the potential impact this could have on health resources and effectiveness of implementation of exercise and health promotion programs is substantial.

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