VIEWPOINT

Can controlled vestibular stimulation reduce stress?

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Abstract

Stress responsiveness is primarily regulated by two neuroendocrine axes: the hypothalamic-pituitary-adrenocortical (HPA) and sympathetic adrenomedullary (SAM) systems. A thorough review of literature revealed that vestibular stimulation inhibits both HPA axis and SAM axis and decreases cortisol level and heart rate and blood pressure within normal limits and brings to stress- less condition. Researchers testified the presence of inferior vestibular-hypothalamic connections. Vestibular stimulation directly inhibits the HPA axis and decreases cortisol levels. Vestibular stimulation can also inhibit HPA axis by increasing GABA release. Vestibular stimulation activates hippocampal formation and hippocampus inhibits HPA axis. Controlled vestibular stimulation decreases heart rate and blood pressure within normal limits. Vestibular stimulation decreases salivary alfa amylase levels slightly by inhibiting SAM axis. From above observations we conclude that controlled vestibular stimulation can reduce stress.

It is the need of time to identify the importance of vestibular stimulation and to start translational research for the well being and peak performance of human being and also for patient care and treatment.

Keywords: Hypothalamo-pitutary-adrenal axis, Sympathetic-adrenomedullary axis, hippocampal formation.

Introduction

"Stress is life and life is stress".

The stress system is essential for individual and species survival. Normal stress system function is crucial for maintenance of mental and physical health. The human body reacts to stress by activating a complex repertoire of behavioral and physiological responses. Selye stated that all states of stress are not noxious. Eustress is the one mild brief and controllable states challenged homeostasis, perceived as pleasant positive stimuli to emotional, intellectual growth and development. Distress is one which is severe and uncontrollable physical and psychological challenge. The vestibular system is the sensory system that responds to the position of the head in relation to vestibular motion, specifically, gravity and accelerated or decelerated motion. The vestibular mechanism and the cerebellar and proprioceptors in the muscles, tendons and joints serve are regulate posture, equilibrium, muscle tone and the orientation of the head and body in space. The close neuro-anatomical relationship of the vestibular system with other regulators of sensory-motor functions is well documented.[1,2,3,4] Research has shown many benefits from vestibular stimulation including decreased self stimulation, decreased hypersensitivity, increased postural security, increased concentration and attentiveness, increased balance, increased body awareness, calming effects, reduction of abnormal muscle tone at slow speeds and increased alertness at high speeds. Korner and associates found that newborns tend not only to stop crying when provided with vestibular input, but also become visually alert. [5,6]. Research has proved vestibular stimulation as an effective non verbal intervention method for the facilitation of spontaneous language.[7] Vestibular stimulation allows many individuals to work far more effectively, often for long periods of time. Hammam E *et al* (2012) proved that low frequency galvanic vestibular stimulation modulates skin sympathetic nerve activity.[8] Sixty-seven studies employed some form of vestibular stimulation as the independent variable. Fourteen of these studies met criteria consistent with traditionally accepted standards of empirical inquiry in the behavioral and biomedical sciences. The 14 studies contained 31 hypothesis tests that evaluated the efficacy of vestibular stimulation as a form of sensory enrichment designed to facilitate various developmental parameters.[9] One of the newest and most popular adjuncts to therapy for developmentally delayed children is vestibular stimulation.

The need for vestibular stimulation can be observed throughout the life from newborns and infants in the cradle to the aged in a rocking chair. Rocking is soothing because it is similar to the movements *in utero*.

The purpose of this article is to review

research reports related to vestibular stimulation and its role on relieving stress, with the intent of clarifying the present knowledge base in this area, and suggesting future research needs.

Materials and methods

Searches of the review study register articles from google.com, pubmed.com, British medical journal.com, Medline, ERIC, frontiersin.org and online standardized journals.

Vestibular system has been shown to have connections with the HPA-axis (Figure 1).[10] Electrical and caloric stimulation of vestibular pathways results in a response in PVN (para ventricular neurons) neurons in the guinea pig [11,12] and an increase in plasma AVP (arginine vasopressin) in the rat.[13] Retrograde viral tracing in the rat brain has demonstrated the presence of a direct vestibulo-paraventricular projection[14] and similarly a paraventricular–vestibular pathway has also been described.[15]

Neuro-anatomical pathways

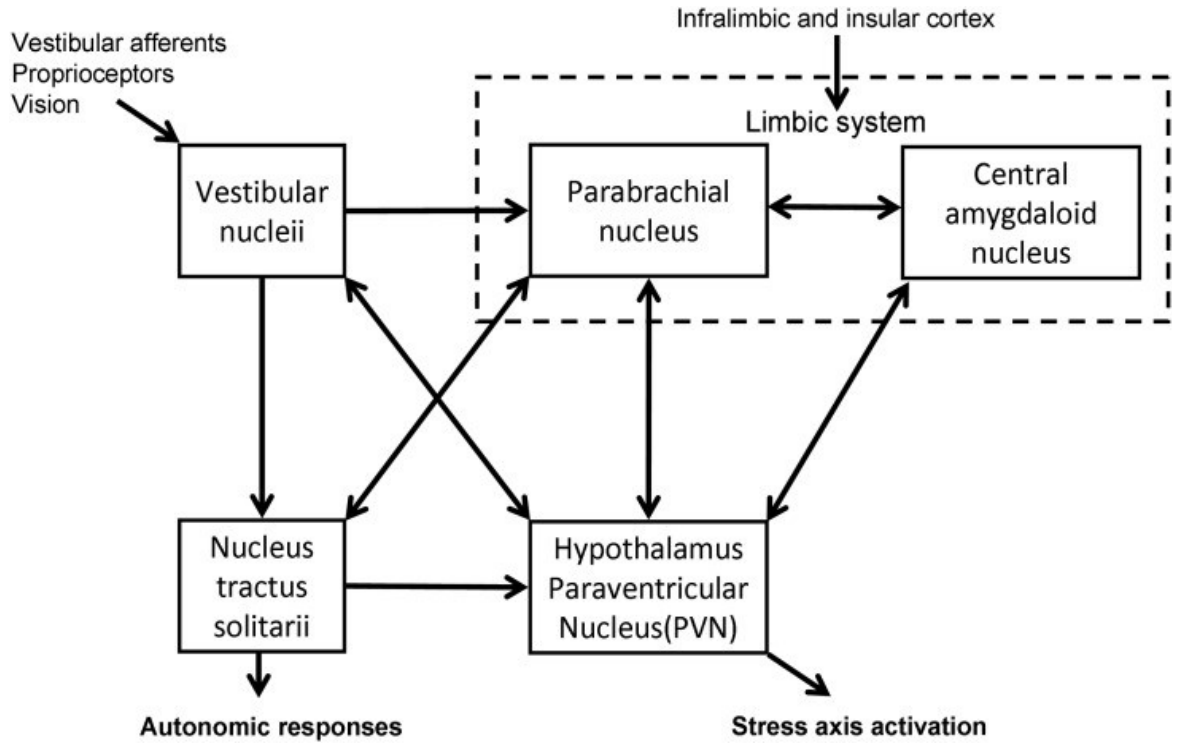

Figure 1.| Diagrammatic representation of the interconnections between vestibular system and hypothalamus and autonomic axes and other central nervous system structures

The presence of inferior vestibulo hypothalamic connections are testified.[16] Vestibular system is also having projections to Suprachiasmatic Nucleus and raphe nucleus.[17] The neurobiological correlates of these phenomenological interrelations may be projections from vestibular nuclei to cortical and sub-cortical brain regions that are also involved in the regulation of mood states.[18]

Most Vestibular signals are not consciously perceived and are usually appreciated through effector pathways classically described as the vestibule-ocular, vestibule-spinal, vestibule-colic and vestibule autonomic reflexes.[19]

Autonomic responses to vestibular stimulation are regionally selective and have defined a 'vestibulosympathetic reflex' in animals.[20] There is substantial evidence that anatomical connections exists between vestibular and autonomic nuclei. Numerous animal studies have shown functional interactions between vestibular and autonomic systems.[21,22,23] However, relatively few studies have examined vestibular-autonomic interactions in humans.[24,25] The mechanisms and underlying physiological basis of vestibular-autonomic interactions are not fully defined.[26,27] Over the last 2-3 years there have been a number of direct electrophysiological demonstrations that vestibular stimulation affects head direction cells in the anterior thalamic nuclei and place cells in the hippocampus. These studies demonstrate the importance of vestibular-hippocampal interactions for hippocampal function but also raise the possibility that the hippocampus may be important for compensation of vestibular function following peripheral or central vestibular lesions.[28] Over the last 2-3 years there have been a number of direct electrophysiological demonstrations that vestibular stimulation affects head direction cells in the anterior thalamic nuclei and place cells in the hippocampus. These studies demonstrate the importance of vestibular-hippocampal interactions for hippocampal function but also raise the possibility that the hippocampus may be important for compensation of vestibular function following peripheral or central vestibular lesions.[29]

Controlled vestibular stimulation directly inhibits the HPA axis

Vestibular symptoms after unilateral vestibular de-afferentation (UVD) activated stress axis. swaying appears to decrease salivary cortisol levels in African elephants.[30] Auditory, tactile, visual and vestibular intervention may reduce infant stress in infants, as the infants who received these interventions showed a significant steady decline in cortisol.[31] vestibular stimulation is performed twice a day for ten days by using infant water bed in infants, decreased urinary cortisol levels significantly when compared with control group.[32] salivary

samples are collected before and after vestibular stimulation and significant steady decline in cortisol after vestibular stimulation is observed.[33]

Twenty-three healthy adult volunteers (male & female) were subjected to rotational (yaw, pitch, roll) and translational vestibular stimulations (surge, heave, sway) which were performed on a hexapod. They observed slight increase in the mean salivary cortisol level towards end of rotational vestibular stimulation and decrease in mean salivary cortisol level towards end of translational vestibular stimulation.[34] The soothing effects produced by rocking and other forms of stimulation may be related to brainstem inhibitory mechanisms.[35] Markia B et al., (2008) reported that vestibular stimulation modulates HPA-axis.

Controlled vestibular stimulation inhibits HPA axis by increasing GABA release

Noisy galvanic vestibular stimulation promotes GABA release in the substantia nigra in animals.[36] GABAergic inhibition controls the activity of the hypothalamic-pituitary-adrenal (HPA) axis , which mediates the body's response to stress. In addition to the actions of stress-derived steroid hormones on GABA(A)Rs, GABA(A)Rs reciprocally regulate the production of stress hormones. [37]

Neuro-anatomical and pharmacological studies have established GABA-mediated inhibition of the HPA axis at the level of the PVN. The origin of this innervation is a series of local hypothalamic and adjacent forebrain regions that project to stress-integrative hypophysiotropic CRH neurons. [38]

It was reported that GABAergic neurons in the bed nucleus of the stria terminalis, preoptic area, and hypothalamus can directly inhibit PVN outflow and thereby reduce ACTH secretion. These inhibitory and PVN-projecting neurons are controlled by descending information from limbic forebrain structures, including glutamatergic neurons of the ventral subiculum, prefrontal cortex, and GABAergic cells from the amygdala and perhaps septum.[39]

Controlled vestibular stimulation may inhibit HPA axis by influencing hippocampal formation

High frequency electrical stimulation of specific vestibular sensory regions of the right labyrinth in anaesthetized guinea pigs induced an evoked field potential in the hippocampal formation bilaterally with a latency with a latency of about 40ms following stimulation onset.[40] Caloric vestibular stimulation in vestibular dysfunction activated hippocampal formation and activated hippocampal formation inhibits stress axis.[41] There is considerable, although not entirely

consistent, evidence that the hippocampus inhibits most aspects of HPA activity, including basal (circadian nadir) and circadian peak secretion as well as the onset and termination of responses to stress. [42] Hippocampal lesions are associated with hypersecretion of glucocorticoids during stress-induced activation of the HPA axis (Figure 2).[43,44,45,46,47,48,49] Whereas stimulation of the hippocampus inhibits the adreno-cortical stress response.[50,51,52] The hypothesis that the hippocampus plays a role in tonic neuronal inhibition of HPA axis. Ventral subiculum plays a particular role in the mediation of the hippocampal formation inhibitory control of the HPA axis.[53]

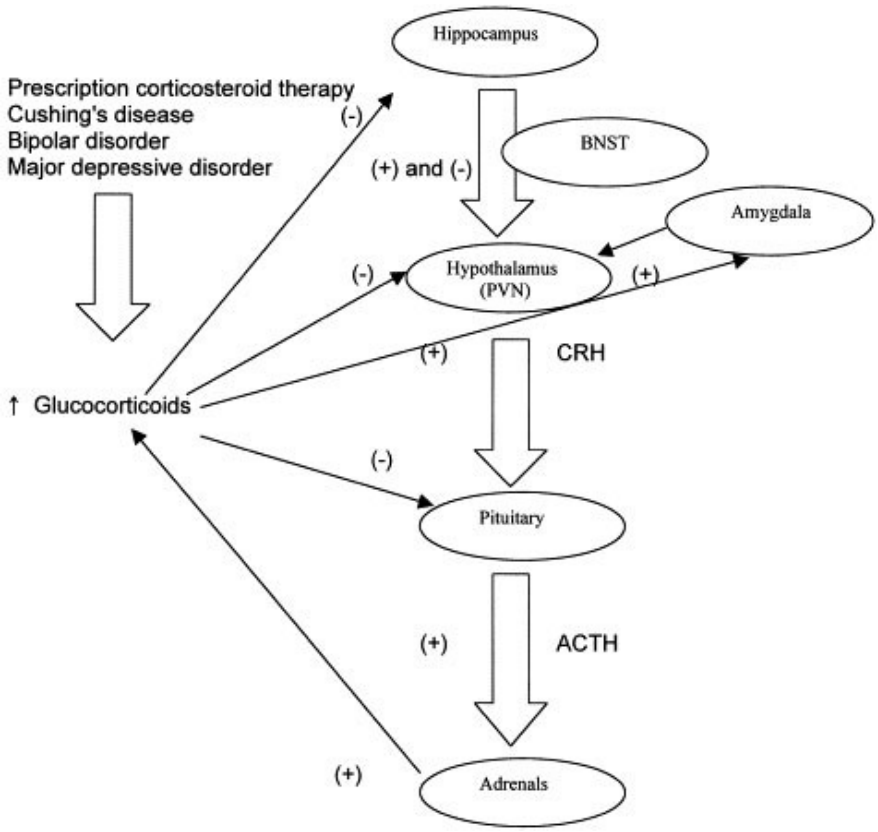

Figure 2. Diagrammatic representation of the interactions between hippocampus, amygdala, glucocorticoids and hypothalamo-pitutary-adrenal axis.

Controlled vestibular stimulation inhibits SAM axis; controlled vestibular stimulation reduces heart rate and blood pressure

Stress responsiveness is primarily regulated by two neuro endocrine axes: the hypothalamic-pituitary-adrenocortical (HPA) and sympathetic adreno medullary (SAM) systems .[54,55,56]

In decerebrate, paralyzed cats, vestibular stimulation resulted in increased rate and depth of respiration and marked elevation of blood pressure. When the stimulation strength was reduced (controlled stimulation) and the evoked respiratory effect is weak or questionable, the blood pressure declined.[57] Sandra Jan Edwards MA *et al* (2010) described the effects of a programmed vestibular stimulation on heart rate change in two children with Down's syndrome exhibiting congenital heart defects. The stimulation programme was applied twice weekly for eight weeks. The total amount of controlled rotatory vestibular stimulation provided within each trial was approximately 1.5 minutes, and the average intensity of the stimulation was at a frequency of about 0.5 Hz. Heart rate was recorded before and after each programmed stimulation. The two children responded physiologically to stimulation: their heart rate decreased but remained within normal limits. [58] Vestibular stimulation has been consistently found to reduce blood pressure in animals by reducing sympathetic activity.[59] Two factors namely decrease in the heart rate controlled by vagus nerve and decrease in heart tone controlled by vaso regulating centre, determine the effect of blood pressure drop after vestibular stimulation.[60]

Controlled vestibular stimulation decreases salivary α amylase

Recently salivary alpha amylase (sAA) has emerged as a novel biomarker for psychosocial stress responsiveness within the sympathetic adreno-medullary (SAM) system.[61]

Lotta Winter *et al* (2012) observed slight decrease insalivary alpha amylase at the end of vestibular stimulation on a motion simulator. However this effect was not statistically significant.

Discussion

A thorough review of literature revealed that vestibular stimulation inhibits both HPA axis and SAM axis and decreases cortisol level and heart rate and blood pressure within normal limits and brings to stress-less condition. The presence of inferior vestibulo hypothalamic connections is testified. Vestibular stimulation can directly inhibit the HPA axis and decreases cortisol levels. Vestibular stimulation inhibits HPA axis by increasing GABA release, and increased GABA inhibits HPA axis. Vestibular stimulation also inhibits HPA axis by activating hippocampal formation. controlled vestibular stimulation decreases heart rate and blood pressure within normal limits. Vestibular stimulation decreases

salivary alpha amylase levels slightly by inhibiting SAM axis. In our review, we are suggesting the controlled vestibular stimulation as vestibular under stimulation, do not have any effect or mild effect and over stimulation causes nausea, vomiting and radical fluctuations in pulse and respiration.

Limitations

Our hypothesis has not been tested experimentally. Work is in progress in our Centre to prove or disprove this hypothesis.

Conclusion

From the above discussion we conclude that controlled vestibular stimulation reduces stress. It is the need of time to identify the importance of vestibular stimulation and to start translational research for the well being and peak performance of human being and also for patient care and treatment.

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Controlled vestibular stimulation- A physiological treatment for stress induced diabetes mellitus. International Conference and Exhibition on Traditional & Alternative Medicine. Kumar Sai Sailesh1and Mukkadan J K.Â Vestibular system is having extensive interactions with hypothalamic nuclei, autonomic system, dorsal and median raphe nuclei, substantianigra, hippocampal formation and modulates pancreatic secretions. The purpose of this article is to explore the role of vestibular stimulation in treatment of diabetes mellitus and to suggest the translational research in this area. Key Words: Controlled vestibular stimulation; Diabetes mellitus; Pancreatic secretions. Biography. Sai Sailesh Kumar has completed Msc in Medical Physiology from KMC Manipal in 2007.