Vestibular stimulation: A simple but effective intervention in diabetes care

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Abstract

Despite the complexities of the relationship between vestibular stimulation and endocrine disorders being well known, research efforts to understand these complexities are lacking. Interestingly vestibular stimulation may potentially prevent/delay development/progression of diabetes. Here we review the science behind this concept and highlight the need for necessary translational research in this area. Current evidence supports the use of vestibular stimulation not only as a potential intervention to prevent or delay the development of diabetes mellitus in at-risk population, but also to use it as supplementary therapy for diabetic patients management. We urge clinicians to recommend vestibular stimulation by simple means like swing as a goal in maintaining a healthy lifestyle.

Keywords: Diabetes mellitus, swing, vestibular stimulation

INTRODUCTION

The incidence of diabetes is increasing in India with >62 million Indians currently diagnosed with diabetes mellitus. Poor adherence to diabetes medications and treatment and lack of awareness in identifying disease complications and its management are the prime reason for the increasing morbidity due to diabetes. It was only in the middle of 19th century that the vestibular system was recognized as a separate entity. Fibers from all five sense organs travel to the brain stem and terminate in the vestibular nuclei. The vestibular apparatus is a membranous structure consisting of three semicircular canals connected at their base to the utricle, saccule and endo-lymphatic duct. Autonomic nervous system (ANS) is one of the important regulators of the endocrine system. Stimulation of the parasympathetic system stimulates insulin secretion, whereas sympathetic stimulation inhibits insulin secretion. Several studies have demonstrated the synaptic connections between vestibular otolith organs and ANS and its contribution to the control of blood pressure during movement and changes in posture. Synaptic connections in the vestibule-sympathetic reflex are functional at birth and require the involvement of the ventrolateral medulla in adult mammals. Further caloric stimulation of the vestibular apparatus can inhibit noradrenergic neuronal activity in the locus coeruleus (LC), thus supporting the concept of the ability to modulate the vestibular and ANS synaptic connections. One such event is the central regulation of decrease in insulin secretion following stimulation of the sympathetic system. Interestingly cholinergic neurons are associated with
noradrenergic neuronal inhibition during the vestibulo-autonomic reflex, independent of the histaminergic neuron system.[7,8]

Of interest caudal medial vestibular nucleus and inferior vestibular nucleus (IVN) is connected bilaterally to nucleus tractussolitarius (NTS) and dorsal motor nucleus of the vagus nerve (DMX).[9] The NTS and dorsal motor nucleus of the vagus nerve (DMV) constitute sensory and motor nuclei of the dorsal vagal complex, respectively.[10] Single shock vestibular stimulation evokes response from the ipsilateral vagus nerve. Stimulation of the vagus nerve can cause a considerable increase in insulin secretion and a decrease in C-peptide secretion with no significant change in glucagon secretion.[10]

Here we review the possible mechanisms by which vestibular stimulation may affect diabetes, and suggest necessary translational research in this area, for the benefit of diabetic patients and to the society in general.

**MATERIALS AND METHODS**

A detailed review of published literature from [http://www.google.com](http://www.google.com), [http://www.pubmed.com](http://www.pubmed.com), British medical [http://www.journal.com](http://www.journal.com), Medline, ERIC, [http://www.frontiersin.org](http://www.frontiersin.org) and other online journals was performed and analyzed.

**Vestibular stimulation prevent/delay development of diabetes by modulating autonomic activity**

The vestibular system contributes to autonomic regulation that have clinical implications.[11] Patients with peripheral and central vestibular abnormalities manifest both symptoms and signs of autonomic dysfunction presumably via compromised vestibulo-autonomic connections. Hence, vestibular-autonomic connections may form the basis for an association between vestibular dysfunction and panic attacks.[12] Nevertheless autonomic dysfunction are suggested to be an underlying mechanism in the development of vertigo.[13] Interestingly constriction of the stomach is observed following stimulation of peripheral labyrinth. The solitary tractus nucleus (SN) neurons, where the vagal nerve (VN) cell bodies exist, fired spikes with short latencies, following electrical stimulation of peripheral labyrinth. Electrophysiological and histological studies indicates existence of (i) vestibular organs-VN-SN-stomach, (ii) Posterior semicircular canal solitary tractus nuclear neurons (stomach, associated with the vestibulo-autonomic reflex.[14] The neurons of the vestibular system that involves in cardiovascular and respiratory control integrates a variety of sensory signals and receive direct inputs from visual, muscle, skin and visceral receptors.[15,16]

**Vestibular stimulation prevent/delay development of diabetes by increasing insulin secretion through vagal stimulation**

Neuro-anatomical studies in the rabbit and in the cat have identified descending vestibulo-autonomic pathways from the caudal portion of the medial vestibular nucleus and the IVN to the dorsal motor nucleus of the vagus nerve, the nucleus of the solitary tract, and some brain stem medullary sympathetic regions.[17] Single shock vestibular stimulation evokes response from the ipsilateral vagus nerve. Branches of right vagus nerve innervates the pancreatic islets, and stimulation of this parasympathetic pathways causes increased insulin secretion via M4 receptors (atropine blocks the response and acetylcholine stimulates insulin secretion). The effect of acetylcholine like that of glucose activates phospholipase C, with the released IP3 releasing the Ca^{2+} from the endoplasmic reticulum, which facilitates the insulin release process.[18]

**Vestibular stimulation prevent/delay development of diabetes by increasing insulin secretion through sympathetic inhibition**

Vestibular sensory inputs to the vestibulo-sympathetic pathway terminates on cells in the vestibular nuclear
complex, which project to brainstem sites involved in the regulation of cardiovascular activity, including the rostral and caudal ventrolateral medullary regions.[19] Caloric vestibular stimulation inhibits locus coeruleus (LC) nor adrenergic neurons. Noisy vestibular stimulation increases release of gamma-aminobutyric acid (GABA) from substantia nigra and GABA inhibits LC nor-adrenergic neurons.[20] Stimulation of the sympathetic nerves to the pancreas inhibits insulin secretion. This inhibition is produced by released norepinephrine acting on alpha-adrenergic receptors. However, if alpha adrenergic receptors are blocked, stimulation of sympathetic nerves causes increased insulin secretion, which is mediated by Beta-2 adrenergic receptors. Hence, catecholamine's have a dual effect on insulin secretion. Nevertheless the net effect of epinephrine and norepinephrine is usually inhibition of insulin secretion.[18]

**Vestibular stimulation prevent/delay development of diabetes by regulating food intake**

Obesity of nongenetic origin is a consequence to the dis-regulation of food intake, energy balance and overall nutrition. Development of insulin resistance increases is directly correlated with an increase in body weight. Hence, hyperinsulinemia and dyslipidemia and accelerated development of atherosclerosis are associated with obesity. This combination of findings is commonly called the metabolic syndrome or syndrome X. Some of the patients with the syndrome are prediabetic, whereas others have frank type 2 diabetes. Single shock vestibular stimulation evokes response from the ipsilateral vagus nerve and long term VN stimulation decreased food intake and body weight.[21,22]

**Vestibular stimulation prevent/delay development of diabetes by inhibiting hypothalamo-pituitary-adrenal axis**

The onset of diabetes was linked with prolonged sorrow by English physician in 17th century.[23] Stress activates hypothalamo-pituitary-adrenal (HPA) axis, leading to various endocrine disorders such as high cortisol and low sex steroid levels, which antagonize the actions of insulin.[24] Controlled vestibular stimulation inhibits HPA axis,[25] which further supports the role of vestibular stimulation in the regulation of diabetes. Although these are only association, it would be necessary to explore such associations for further therapeutic development.

**Vestibular stimulation prevent/delay development of diabetes by promoting sleep**

Chronic lack of sleep may contribute to the risk of type 2 diabetes mellitus. Adequate sleep and good sleep hygiene should be included among the goals of a healthy lifestyle, especially for patients with diabetes.[26] Vestibular system has extensive interactions with hypothalamus, dorsal raphe nucleus, NTS, locus coeruleus and hippocampal formation all of which promotes sleep.[20] These could also be essential in reducing the general stress levels.

In summary, the current evidence suggests the role for vestibular stimulation as a potential intervention to prevent or delay the development of diabetes mellitus in at-risk population and may also used as supplementary therapy in management of diabetic patients. The benefits from vestibular stimulation are achieved through many well-established physiological pathways nevertheless well-designed translational studies are necessary to rigorously establish these findings into evidence-based clinical practice. Nevertheless, we urge clinicians to recommend vestibular stimulation as a simple means to maintain a healthy lifestyle.

**Footnotes**

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**Conflict of Interest:** None declared.

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