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Editorial Open Access

Effects of Vagus Nerve Stimulation on Weight Loss and Associated Disorders: A Therapeutic Perspective

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Editorial

The benefits of Vagus Nerve Stimulation (VNS) on obesity and associated disorders are established. Trials of VNS implantable devices have found direct evidence of an effect on weight loss. A study to optimize electrical algorithms conducted in 27 patients in Australia, Norway and Switzerland found that at 6 months, the benefit of VNS applied near the esophagogastric junction was proportional to the number of pulses delivered, without any device-related or medical adverse events [1]. Figure 1 illustrates weight loss in patients treated with an implanted VNS device for the treatment of epilepsy.

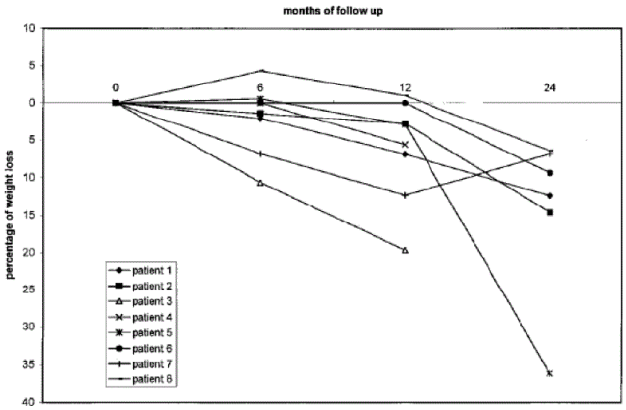

Figure 1: Weight loss in patients treated with VNS for epilepsy. Burneo, Device Based Therapy in Obesity.

With regard to the underlying mechanisms, stimulation of the vagus nerve is associated with a modulation of activity in the regions that mediate craving and addiction, such as the orbitofrontal cortex, hippocampus, cerebellum and striatum. Stimulation of the satiety centre is thought to be related to vagus stimulation. Vagus nerve stimulation has been shown to stimulate the Nucleus Tractus Solitarius (NTS) as demonstrated by direct recordings of the NTS in rats. Nonetheless, some stimulation still persists after vagotomy, indicating that the vagus nerve is not the only component involved in gastrointestinal stimulation; it appears that presynaptic melanocortin-4 receptors in the NST are involved. Nitrergic pathways may also play a role, especially in electrical stimulation of the intestine [2]. Although the mechanism has not been fully elucidated, the data so far suggest that vagus stimulation produces beneficial effects on satiety. It is striking that the comorbidities sometimes associated with obesity, such as hypertension, sleep apnea and inflammation, are all strictly dependent on the activity of the autonomic nervous system.

Treatment of depression by left cervical VNS led to weight loss in 14 patients [3], even though the patients did not diet or exercise. In patients treated by VNS for depression, an immediate effect on the craving for sweets was seen in response to stimulation of the vagus nerve in the neck: turning the device on or off, with patients blind to the on-off status, significantly changed craving ratings for sweet foods.

In diabetes, vagus nerve stimulation leads to a decrease in glycosylated haemoglobin levels in diabetic patients. Insulin-resistant rats showed a large decrease in vagal activity measured by variability in heart rate. Similarly, hepatic vagotomy increased lard intake in streptozotocin-induced diabetic rats. Conversely, chronic vagal stimulation by an implanted microchip in rats on a high calorie diet decreased weight gain and meal size. Vagus nerve stimulation by magnetically driven solenoid electrodes placed near the oesophagealvagus nerve in animals influenced food intake, weight gain and serum leptin concentrations. Vagus nerve stimulation in pigs led to changes in weight without modifying global metabolic activity.

With regard to arterial hypertension, the autonomic nervous system maintains blood pressure constant. Destruction of the Nucleus Tractus Solitarius (NTS) produces fulminant hypertension. Other studies have shown that the NTS is involved in the vasopressor response. VNS produced a large decrease in hypertension, from 185/109 to 155/95 mmHg over a period of 3 months. In humans, renal sympathetic denervation lowered blood pressure without altering the hypotensive vagal response. In rats, vagus nerve stimulation reduced hypertension associated with obesity. The direct effects of VNS on blood pressure have been observed in animals and in humans. Weight gain results in a decrease in this activity whereas weight loss results in an increase in this activity. Obesity itself can cause hypertension and deactivation of the autonomic nervous system. VNS breaks this vicious circle.

Control of the inflammatory pathway by the autonomic nervous system has been described as a reflex (Figure 1). Decreased autonomic activity leads to a heightened inflammatory response and studies of parasympathetic stimulation to reduce inflammation have shown efficacy in gastrointestinal inflammation, as in intensive care medicine. A chronic cholinergic deficiency has been suggested as a possible cause of vascular dementia. Sinoaortic denervation leads to the development of atherosclerosis in rats. Systemic inflammation and endothelial dysfunction are present in resistant hypertension. Neurologic control of inflammation has recently been investigated and found to depend in

particular on the alpha7 subunit of the acetylcholine cholinergic receptor.

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