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The influence of cervical spinal cord compression and vertebral displacement on somatosympathetic reflexes in the rat

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Abstract

BACKGROUND CONTEXT: One theory within chiropractic proposes that vertebral subluxation in the upper cervical region induces spinal cord compression sufficient to alter spinal cord efferent output. We report on the feasibility of three different experimental approaches to test this theory.

METHODS: A high threshold electrical-evoked somatosympathetic reflex was recorded in adrenal or renal nerves of 10 anaesthetized adult male rats before and after (1) graded pressure was applied directly to the C1/C2 spinal cord segment in eight rats by the use of either direct compression or inflation of an extradural balloon and (2) displacement, less than a dislocation applied posterior to anterior, to the C2 vertebra in two rats. The latency and amplitude of the pre- and postintervention reflex responses were compared.

RESULTS: The reflex amplitude was not significantly changed by pressure (26 mmHg) from an extra-dural balloon or direct compression of the dura mater onto the dorsal spinal cord. Additional pressure, at least sufficient to occlude the dorsal vessels, induced a significant reduction in the amplitude of the reflex, and this reduction persisted for 20 minutes after removal of the pressure (Dunn's method for all pairwise multiple comparison Q stat=3.437; critical value for k=6 with α=0.05 is 2.936). Maximal vertebral (C2) displacement (4 mm), without dislocation did not induce significant changes compared with the control period.

CONCLUSIONS: Although this feasibility study suggests it is unlikely that upper cervical vertebral subluxation, displacement less than a dislocation, compromises the sympathetic outflow in the adrenal or renal nerves, further vertebral displacement studies are necessary to formally test this.

Keywords: Rat; Somato-sympathetic reflex; Spinal cord; Vertebral subluxation; Chiropractic

Introduction

Central to the use of spinal manipulative therapy by chiropractors is the theory that vertebral subluxation, that is, displacement of a vertebra less than a dislocation, compromises health [1]. One proposed consequence of such vertebral displacement, particularly in the upper cervical vertebral column, is alteration of the descending neural activity from the brain caused by compression of the thecal sac or spinal cord and thereby dysfunction in dependent organs [2,3]. It is well established that compression sufficient to damage the spinal cord can not only result in a loss of somatic sensation and voluntary motor function but also loss

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of descending regulation of the autonomic outflow, resulting in autonomic dysreflexia and significant disease [4]. In contrast, there is only indirect evidence that health is compromised by cervical spinal cord dysfunction in the absence of spinal cord damage or demonstrable pathoanatomical lesions such as spondylotic spinal stenosis or ossification of the posterior longitudinal ligament. It has been shown that after some whiplash events in which there is an absence of an identified pathoanatomical lesion, peripheral sympathetic vasoconstriction reactivity may be reduced [5]. Interestingly, although a causal relationship has not been established, a more recent study has suggested that this reduced cutaneous sympathetic reactivity is associated with posttraumatic stress disorder after the whiplash event [6].

The spinal cord compression component of the chiropractic vertebral subluxation theory, and consequent justification for the use of spinal manipulative therapy of the cervical vertebral column, would predict that (1) interference with spinal cord function can occur with quite modest degrees of compression, (2) such compression occurs with displacement of vertebrae less than a dislocation, and (3) the effects of spinal cord interference are ameliorated by the removal of the vertebral displacement. We are unaware of any study that has specifically tested this paradigm. Therefore, we have undertaken a feasibility study to determine the relative amount of spinal cord compression necessary to modulate nerve activity from the spinal cord to organs and to determine whether vertebral displacement, less than a dislocation, can modulate nerve activity. Here we define dislocation as a loss of apposition of the facet surfaces of one or both zygapophysial joints of adjacent vertebra.

We have chosen the rat as a model to investigate this chiropractic paradigm because there is good evidence that the vertebral canal in the upper cervical region of the rat is similar to that of the human [7]. Furthermore, it is known that electrical stimulation of peripheral nerves in anaesthetized cats and rats elicits at least three reflex components in sympathetic efferent peripheral fibers [8,9]. The first is a short latency response involving spinal neural circuitry and referred to as the ''early A-reflex'' because it is elicited by relatively low (electrical) threshold stimulation that activates Aβ somatic afferents. This is followed by a longer latency ''late A-reflex,'' resulting from increased electrical stimulation that activates Aβ and Aδ somatic afferent nerves and involves supraspinal projections and processing in the medulla [10,11]. The third component has an even longer latency and is referred to as the ''C-reflex,'' because it is elicited by electrical stimulation of unmyelinated C-fibers that have the greatest threshold for electrical stimulation and the slowest conduction velocities [12]. A ''very late A-reflex'' response, involving projections to even more rostral centers, can also be elicited in lightly anaesthetized animals [9,11]. In this study we explored the vertebral subluxation ''compression'' hypothesis [2] by eliciting the late A-reflex in anaesthetized rats. Then, we either compressed the upper cervical spinal cord in a controlled manner or displaced the upper cervical vertebra to determine if these procedures modulate the evoked sympathetic efferent reflex activity in adrenal and renal nerves. Preliminary data have been published elsewhere [13].

Methods

Experiments were performed on 10 adult male Wistar rats ages 10 to 13 weeks, weighing 445 ± 54 g (mean ± SD). The study involved a nonrecovery protocol in which the rats were anesthetized with urethane. All procedures were performed in accordance with protocols approved by the Institute's Animal Care and Ethics Committee and conformed to the National Code of Practice for the use of animals in experiments.

Surgical preparation

Each rat was anesthetized by intraperitoneal injection of urethane (1.3 g/kg). Once anesthesia was achieved, as judged by the absence of withdrawal and corneal reflexes, a rectal probe was used to determined body temperature, which was then maintained at 37 to 37.5°C with the use of a heating blanket and infrared lamp. The right jugular vein was catheterized for administration of additional anesthetic (1/10 of initial dose) and fluids as necessary. The left carotid artery was catheterized to allow continuous measurement of blood pressure (BP-1; World Precision Instruments, Sarasota, FL, USA), and the rat's trachea was intubated and the rat allowed to breath spontaneously. The rat's head was then fixed in a stereotaxic frame (model 1730; Kopf Instruments, Tujunga, CA, USA). The spinous process of the first thoracic vertebra was exposed and held fixed in space with a clamp attached to the stereotaxic frame. Signals from the blood pressure transducer were passed to a data-acquisition system (A-D conversion 10 kHz, Spike 2; CED, Cambridge, UK) connected to a computer for monitoring and recording.

Branches of the left sciatic nerve were exposed, isolated from adjacent tissue, placed on silver-wire stimulating electrodes, and covered with warm paraffin oil. A square wave (duration 0.2 ms) stimulus was applied to determine the voltage stimulus threshold for muscle twitch. The nerve was then ligated and cut so only the central cut end remained on the silver wire stimulating electrode, which was then covered with warm paraffin oil. A left retroperitoneal surgical approach was used to expose the adrenal or renal nerves, which were gently teased apart with fine forceps to allow recordings of whole nerve or filaments of these nerves. The isolated nerves or filaments were ligated with fine suture and cut, and their central cut ends were placed on a bipolar silver-wire hook electrode and covered with warm paraffin oil. Signals from these nerves were amplified (×1000; model 1700 amplifier, A-M Systems, Carlsborg, WA, USA), passed to an oscilloscope for online observation