Autonomic Nervous System Function Among Individuals With Acute Musculoskeletal Injury

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Abstract

Objective: To determine differences in peripheral and cardiovascular autonomic function between individuals with acute musculoskeletal injury (<1 week) and healthy controls.

Methods: Autonomic cardiovascular modulation, baroreceptor sensitivity, skin conductance, and peripheral skin temperature were obtained in 6 subjects with acute musculoskeletal injury and 6 age- and sex-matched controls. Power spectral analysis was performed on both beat-to-beat R-R intervals and continuous systolic blood pressure (SBP) peaks. Baroreceptor sensitivity was derived using both heart rate and blood pressure spectral analysis components.

Results: The SD of R-R intervals was significantly different for the acute injury group relative to controls (49.8 $\pm$ 10.5 vs 76.8 $\pm$ 12.7 ms; $P < .01$). Continuous SBP peaks and skin conductance (sympathetic vasomotor and sudomotor indices, respectively) were significantly higher (59.6 $\pm$ 6.7 vs 23.8 $\pm$ 6.4 mm Hg^2/Hz, and 3.87 $\pm$ 1.04 vs 2.19 $\pm$ 0.3 mhos; $P < .01$, respectively) and baroreceptor sensitivity lower (0.97 $\pm$ 0.07 vs 1.10 $\pm$ 0.08 mm Hg; $P < .02$) in the acute injury group compared with controls. Regression analysis revealed a significant relationship between skin conductance and continuous SBP peaks ($r = 0.75$; $P < .01$).

Conclusions: These findings suggest that interaction between cutaneous and vasomotor sympathetic neurons in response to acute musculoskeletal injury, reflected as increased afferent input from sensitized nociceptors and other sensory neurons, results in alterations in autonomic function. (J Manipulative Physiol Ther 2005;28:44-51)

Key Indexing Terms: *Autonomic Nervous System; Musculoskeletal Physiological Phenomena; Musculoskeletal Injury; Power Spectral Analysis*

One of the central hypotheses of traditional chiropractic is that dysfunction of somatic structures, chiefly the musculoskeletal components of the human vertebral column, may have significant impact on regulation of the nervous system, specifically the autonomic nervous system, and hence influence visceral function and health. Although the historical origins of this tenet are rooted in less scientific theories, the foundation of its modern interpretation was based on research performed by the osteopathic investigator Irvin M. Korr.[1] More recent references lend support to the above statement including chiropractic textbooks used at many chiropractic colleges today.[2,3]

Although often the emphasis is placed on the treatment of the spine in chiropractic health care, it is well established that nociceptive and other aberrant neurological input from dysfunctional musculoskeletal structures of any component of the human frame influences the autonomic nervous system.[4] While it has been shown that noxious as well as innocuous stimuli of somatic structures results in changes in the autonomic nervous system,[5,6] no definitive human research has shown a clear relationship between articular and muscular dysfunction of the spine and other musculoskeletal structures and autonomic perturbations (particularly beyond the immediate insult). Evaluation of autonomic nervous system function in subjects presenting with acute musculoskeletal soft tissue injury may shed light on such relationships.

It is generally understood that consequences of somatic tissue injury extend beyond the site of insult and include both spinal and supraspinal changes in neuron excitability and activity.

Injury to the somatic tissues of the musculoskeletal system results in heightened afferent input from sensitized nociceptors and other sensory neurons, and in some chronic conditions (ie, those associated with nerve damage), remodeling of the spinal dorsal horn has been reported.[7] Although supraspinal pain modulatory systems were originally considered solely inhibitory of spinal nociceptive sensory input, stimulation of supraspinal sites can also facilitate spinal nociceptive transmission, both of which have

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been shown to produce autonomic and behavioral effects such as those associated with the 'fight or flight' response.[8]

Long-term activation of spinal and extraspinal nociceptive afferents contributes to stimulation of the autonomic nervous system that may in turn have a deleterious effect over time on visceral function and homeostasis with the potential to negatively impact health and wellness.[9-11] Experimental evidence exists demonstrating a relationship between somatic structures and the autonomic nervous system, mainly via neurological reflex mechanisms.[12,13] In contrast, no relationship has been shown between short-term activation of spinal afferents, as found in nonexperimental human subjects with acute musculoskeletal injury, and the autonomic nervous system. The intent of this observational study was therefore to determine whether differences exist in peripheral and cardiovascular autonomic function between individuals with acute musculoskeletal injury and healthy controls.

Materials and Methods

Subjects

Twelve subjects participated in this study, 6 with acute musculoskeletal injury and 6 age- and sex-matched healthy controls. Inclusion criteria for the injury group were acute musculoskeletal injury to the low back or 1 of the extraspinal articulations less than 1 week in duration and level of pain assessed using a Visual Analogue Scale (VAS) between 3 and 6 (Table 1). Before testing, the musculoskeletal injury group received a routine chiropractic physical examination. For both groups, exclusion conditions included known coronary heart and/or artery disease, hypertension, renal function abnormalities, diabetes mellitus, obesity, current cigarette smoking, and medications known to affect the autonomic and/or cardiovascular systems. The institutional review board of New York Chiropractic College granted approval for the study, and informed consent of each subject was obtained before the investigation.

Testing occurred between 2:00 and 4:00 PM in a private and thermo-controlled autonomic laboratory. Subjects were required to rest quietly (prone position) for 30 minutes followed by 5 minutes of peripheral and cardiovascular autonomic data collection. All subjects refrained from beverages containing caffeine and alcohol during the day of the study, and data were collected 2 hours postprandial. Control subjects abstained from exercise 24 hours before study.

Data Collection

Dependent variables obtained included cardiovascular autonomic modulation: beat-by-beat systolic blood pressure (SBP) and R-R intervals (RRIs) of the electrocardiograph (ECG) QRS complex both measured using power spectral analysis, baroreceptor sensitivity, skin conductance, and peripheral skin temperature (Fig 1). To assess peripheral and cardiovascular autonomic modulation, data acquisition was performed on cardiovascular autonomic data, skin conductance, and peripheral skin temperature at a sampling rate of 250 Hz per channel with a 12-bit analog-to-digital converter. R-R intervals and SBP were measured beat-by-beat using lead V_5 of the ECG and a continuous tonometry and oscillometric blood pressure instrument (Colin, Medical Instruments Corp, San Antonio, Tex). R-R intervals and SBP data were acquired and spectral decomposition performed using a customized program created with LabVIEW software (National Instruments, Austin, Tex) as previously described[14]; a more detailed description of the methodology, physiological interpretation, and clinical use is provided in Ref. 16.

Table 1. *Profile: acute injury group*

Subject	Injury type	Duration	Pain scale (VAS)
1	Low back	3 d	3/10
2	Low back	2 d	6/10
3	R shoulder	2 d	4/10
4	R knee	24 h	5/10
5	Low back	6 d	6/10
6	L knee	4 d	3/10

R, right; *L*, left.

For peripheral autonomic assessment skin conductance was recorded by using a pair of Ag/AgCl electrodes, approximately 0.8 cm^2 in contact area, filled with conductivity gel placed on the volar surface of the distal phalanges of digits I and II of the hand and then attached with a Velcro strip.[15] Data was collected with a Grass model CSA1 Skin Conductance Adaptor then channeled into a Grass P122 amplifier (Astro-Med Inc, W Warwick, RI) and sampled and digitized as previously described. Peripheral skin temperature was obtained by a thermocoupled temperature probe (YSI, Yellow Springs, OH) secured with medical tape to the volar surface of the distal phalanx of digit IV of the hand. The signal was channeled into the computer via an interface module converter (Deban Enterprises, Yellow Springs, OH), and data for all measurements were analyzed off-line.

Signal Processing

All signals were visually inspected for artifact and anomalies and peak detection was performed on all QRS complexes and systolic peaks. The standard deviation (SD) of RRIs (a time domain variable representing global cardiac parasympathetic input[16]) was calculated. R-R intervals and systolic peaks were then interpolated to provide continuous wave forms. The data were transformed into frequency spectra using discrete Fourier algorithms, and the spectral estimates smoothed by applying a Hamming window function to produce the power spectra.[17,18] The standard spectral bandwidths for RRI and SBP parameters each