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Significant Changes in Systolic Blood Pressure Post Vectored Upper Cervical Adjustment Vs Resting Control Groups: A Possible Effect of the Cervicosympathetic and/or Pressor Reflex

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ABSTRACT

Objective: To determine whether a vectored adjustment of the atlas in patients identified as demonstrating signs of upper cervical joint dysfunction would cause lowering of blood pressure in comparison with resting controls.

Design: Test 1: controlled clinical trial with a treatment (adjustment) group and a control (resting) group. Test 2: controlled clinical trial with subjects serving as their own controls.

Setting: Private chiropractic practice.

Participants: Test 1: Forty established patients demonstrating signs of upper cervical subluxation/joint dysfunction and 40 established patients without such signs. Test 2: Thirty established patients demonstrating signs of upper cervical subluxation/joint dysfunction.

Intervention: Specific, vectored upper cervical (atlas) adjustment or similarly positioned resting.

Main Outcome Measures: Prerest, postrest, and postadjustment systolic, diastolic, and pulse rates as recorded through use of a digital oscillometric sphygmomanometer.

Results: In test 1, subjects receiving adjustment had a significant ($P < .001$) decrease in systolic blood pressure whereas resting subjects did not. Intergroup comparison of the treatment (adjustment) and control (resting) groups demonstrated a significant difference ($P < .001$). A greater pre/post drop in systolic pressure was associated with greater age and higher initial systolic pressure. In test 2, the pre/postrest change in systolic blood pressure was not significant. The systolic blood pressure changed significantly ($P < .001$) from postrest readings to postadjustment readings.

Conclusion: The results indicate that palpation and vectored atlas adjustment causes a significant decrease in systolic blood pressure in patients with putative upper cervical subluxation/joint dysfunction in comparison with resting controls. Similar results were also demonstrated when subjects acted as their own controls. The lack of randomization, blinding, and a manipulated control group are factors that weaken these findings. The sudden drop in systolic pressure is proposed to be due to stimulation of the cervicosympathetic reflex or moderation of muscle tone and elimination of the effects of the pressor reflex. (J Manipulative Physiol Ther 2001;24:101-9)

Key Indexing Terms: Chiropractic Manipulation; Cervical Spine; Blood Pressure; Reflexes.

INTRODUCTION

The association of manipulative treatment and decreases in arterial blood pressure (BP) has been noted and tested several times in the literature[1-6]; among the investigations have been some controlled trials.[2,5,7] In some cases, reduction in BP has been so dramatic as to induce a call for the monitoring of medicated hypertensive patients lest the pressure fall too low[1]; in other cases, no significant postmanipulative change was noted.[4] One small (n = 8), long-term (2-month) study of hypertensive patients that involved the use of specific upper cervical care and a protocol for determining whether and when to adjust showed an average decrease in systolic BP of 27 mm Hg and in diastolic pressure of 13 mm Hg.[5] Conversely, a long-term, controlled trial found osteopathic manipulation not to be useful in moderating BP in hypertensive subjects.[6]

Crawford et al[7] have suggested that the additive effect of a variety of risk factors for high BP, including subluxation, may influence the sympathetic chain, increasing sympathetic tone, causing peripheral vasoconstriction, and resulting in a rise in systemic BP. Gerber[8] has speculated that musculoskeletal dysfunction of certain spinal segments (upper thoracic, rib cage, and thoracolumbar regions) could modify sympathetic outflow and contribute to the hypertensive state. A study by Mannino[9] found decreases in serum aldosterone after manipulative treatment and no such decreases after sham treatment, indicating that hormonal involvement in BP may be amenable to manipulation. Morgan et al,[6] commenting on Mannino's research, stated, "To the best of our knowledge, no other evidence has been obtained that satisfactorily explains the mechanism(s) by which spinal manipulative treatment might reduce systemic BP in hypertensive patients."

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Elimination of putative joint dysfunction in the upper cervical spine has been shown to be associated with decreases in BP.[5,7,10] Hypothetical explanations forwarded for this association include physical compression of the vagus nerve[10] or superior cervical ganglion[10] and traction on the brainstem.[5] For the most part, this association is not understood.[5,10]

There are 2 additional mechanisms by which manipulation could reduce BP that I would put forward: activation of the cervicosympathetic reflexes and activation of the pressor reflexes. In brief (see the Discussion for an expanded explanation), the cervicosympathetic reflex responds to signals from the muscle spindles/Golgi tendon organs (GTOs) of the suboccipital spine to counteract vestibulosympathetic reflexes. Vestibulosympathetic reflexes act to increase BP and heart rate to offset the effects of postural hypotension.[11] Cervicosympathetic reflexes from the upper cervical spine, when stimulated, act to lower BP, heart rate, and the increased sympathetic stimulation of the vestibulosympathetic reflexes.

The second mechanism involves the pressor reflex, which is initiated with muscle contraction. Contraction causes compression of intramuscular arteries while increasing requirements for oxygen, nutrients, and waste removal. To overcome the restriction in vessel patency, neurologic reflexes that result in increased systemic BP are engaged.

Abnormal muscle hypertonicity has long been associated with putative joint dysfunction, osteopathic lesions, and/or chiropractic subluxation. Electromyographic recordings have shown reductions in muscle activity after manipulation of putative joint dysfunction.[12-15] The upper cervical spine is associated with postural muscle control,[16-18] thought to be signaled by the actions of densely packed, physiologically unique muscle spindles.[16,19,20] Joint dysfunction in this area is suspected of causing global muscle contractions and postural distortions, including pelvic unleveling, functional "short leg," foot rotations, and unequal weight bearing.[21-23] Correction of putative upper cervical joint dysfunction has been shown to cause an immediate reduction or elimination of the postural distortion.[24-27] Other studies have found that manipulation of the cervical and upper cervical spine decreased lumbar muscle tone[28] and increase hip range of motion.[29]

The theoretic association of putative upper cervical subluxation with altered spindle afferentation (the cervicosympathetic reflex), as well as the relationship of global postural muscular contractions/postural distortion to increases in BP (the pressor reflex), sets up a testable hypothesis: there is a significant measurable change in BP after upper cervical adjustment.

METHODS

Test 1

Eighty established patients were recruited for this part of the study and verbally consented to participate. The subjects ranged from normotensive and borderline hypertensive to hypertensive; some were taking medication. To avoid sudden shifts in BP due to extreme temperature changes from outdoors to indoors, the BP was checked when the outdoor temperature was between 70° and 85°F (21° and 30°C), with the indoor temperature held at a constant 78°F (26°C). Subjects in demonstrable acute pain or agitated emotional states were excluded from testing. Each subject was allowed to relax seated for a few minutes before the first BP check (Pre reading) was taken. The BP was taken in the left arm, supported by a table. A digital oscillometric sphygmomanometer (model 82T, Omron Healthcare, Inc; accuracy for pressure ± 3mm Hg, pulse ± 5%) that recorded the systolic, diastolic, and pulse rates was used to avoid any bias in recording auscultory points with a stethoscope. Conversation was avoided during the BP checks, and disclosure of the results did not take place until after the second check (Post reading).

After the Pre reading, the subject was checked for signs of pelvic rotation (high iliac crest) through use of a level clamped on the iliac crest. This was followed by a supine visual examination for leg/foot rotations and by a supine leg check. This leg check involves the subject's standing with back to the table, sitting down, pulling himself or herself evenly up the table, and lying down. The shoes are lightly grasped and squared, and the positioning of the heel/sole interface is compared. The supine leg check has been found to have an overall intraclass agreement among examiners of >0.9, with an overall high (0.7) intraexaminer reliability.[30]

Subjects showing signs of postural distortion were assigned to the treatment group (n = 40); subjects showing no signs of postural distortion were assigned to the control group (n = 40). Each subject in the treatment group was turned on his or her side and positioned and adjusted by hand through use of a specific upper cervical vectored technique; the vector of the adjustment had been previously determined by radiographic study. If the subject was in the control, or resting, group, he or she was turned on the side and the head was positioned, as with the treatment subjects, but no adjustment was given. Positioning involved lightly grasping and slightly moving the head. Subjects in the adjustment group were statically palpated for the position of the atlas transverse process; subjects in the resting group were not.

After the adjustment or an equivalent time period, every subject (in both groups) was turned on his or her back and rested quietly for approximately 2 minutes. The subject was then was helped up and the BP was checked again (Post reading) through use of the method already described.

If there was any error message from the sphygmomanometer—because of underinflation or subject movement—for either the first reading or the second reading, the data for that subject were discarded.

Test 2

Thirty established patients were recruited for this part of the study and verbally consented to participate. The subjects ranged from normotensive to hypertensive; some were taking medication. The outdoor temperature range and areas of

Table 1. *Test 1 results: changes in systolic BP for adjustment vs resting groups*

	Treatment (adjustment) group (n = 40)			Control (resting) group (n = 40)		
		SD	95% CONF		SD	95% CONF
Age (y)	53 (21-83)			54 (20-83)		
Systolic BP (mm Hg)						
Pre reading	140.7	25.3	±7.8	124.0	19.4	±6.0
Post reading	130.4	22.7	±7.0	123.5	22.1	±6.8
Average change	–10.3	8.2	±2.5	–0.5	7.6	±2.4

Intragroup *t* tests: treatment (adjustment) group, $P < .001$ (statistically significant); control (resting) group, $P = .68$ (not statistically significant).
Intergroup difference in change in systolic BP Post reading for treatment (adjustment) vs control (resting) groups: $P < .001$ (statistically significant).
Intergroup difference in systolic BP Post reading for treatment (adjustment) vs control (resting) groups: $P = .34$ (not statistically significant).
BP, Blood pressure; *CONF,* confidence level.

disqualification used in test 1 were observed. Each subject was allowed to relax seated for a few minutes before the first BP check was made (Prerest reading). The equipment was the same as that used in test 1.

As in test 1, the subject was examined after the Prerest reading for signs of postural distortion. Only patients who demonstrated signs of postural distortion were used for this test.

After the supine leg check, the subject was allowed to relax in the supine position for approximately 2 minutes. The subject was then helped to a sitting position and the BP was taken again. After this second BP check (Postrest reading), the patient was positioned on the treatment table and adjusted through use of a vectored specific upper cervical technique. After the adjustment, the patient was turned on his or her back and rested again for a few minutes. The subject was then helped up and seated and the BP was taken for a third time (Postadjustment reading).

This method made the subjects their own controls; the purpose was to eliminate any treatment/control group differences that might have contaminated the results of test 1. As in test 1, any error message from the sphygmomanometer caused the data for that subject to be discarded.

RESULTS

Test 1

The treatment, or adjustment, group consisted of 27 women and 13 men; their average age was 53 years (range, 21-83 years; median, 51 years). The control, or resting, group also consisted of 27 women and 13 men; their average age was 54 years (range, 20-83 years; median, 54 years; Fig 1). The intragroup BP and pulse changes from the Pre reading to the Post reading were analyzed through use of a 2-tailed paired *t* test.

In the treatment group, there was a significant preadjustment to postadjustment decrease in systolic BP of –10.3 mm Hg (± 2.5 mm Hg; all confidence levels were calculated at 95%; $P < .001$), from 140.7 mm Hg (± 7.8 mm Hg) to 130.4 mm Hg (± 7.0 mm Hg; Table 1). There was no statistically significant change in diastolic pressure or pulse rate.

In the control group, there was no significant change in systolic BP or pulse rate from the Pre reading to the Post reading. The control group did demonstrate a statistically significant diastolic pressure change of –2.0 mm Hg (±1.8 mm Hg; $P = .04$), from the Pre reading to the Post reading—from 77.5 mm Hg (± 3.8 mm Hg) to 75.6 mm Hg (± 3.5 mm Hg). However, this finding was not within the reported accuracy of the sphygmomanometer and accordingly is not considered significant.

Intergroup comparisons between the adjustment and resting groups were done through use of a 2-tailed, nonpaired, homoscedastic *t* test. The systolic BP Pre reading for the control group, checked as not needing adjustment, was significantly lower ($P < .002$) than that for the treatment group, checked as needing adjustment. In addition, comparing the changes in systolic BP of the treatment (adjustment) and control (resting) groups found a significant ($P < .001$) difference (Table 1). The adjustment and resting groups did not show a significant difference in systolic BP Post readings (130.4 vs 123.5 mm Hg; $P = .34$). There was no significant difference between the adjustment and resting groups in diastolic BP or pulse rate.

One of the interesting findings in this study was a correlation between age and the magnitude of decrease in systolic BP after adjustment (Fig 1 and Table 2). The decrease in systolic BP up to age 55 years for the adjustment group was –7.6 mm Hg (± 3.3 mm Hg). At age 55 years and older, however, the change in systolic BP after adjustment jumped to an average of –13.8 mm Hg (± 3.6 mm Hg). The difference between the ≤55 age group and the >55 age group was significant at $P = .02$. In the resting group, the systolic BP increased slightly from younger to older (Fig 1).

Furthermore, the decrease in systolic BP after adjustment was larger, both outright and percentagewise, for higher starting systolic pressures (n = 40; Fig 2). This amounts to a fair correlation between higher initial systolic pressures and greater drops in pressure after adjustment ($r = –0.46$), this being statistically significant ($P < .01$).

Test 2

The average age of the participants in test 2 was 54 years (range, 14-83 years; median, 54.5 years), and the group consisted of 19 women and 11 men (Fig 3). This group's Prerest reading-to-Postrest reading and Postrest reading-to-Postadjustment reading BP changes were analyzed through use of a 2-tailed, paired *t* test. The change in systolic BP from the Prerest reading to the Postrest reading was 0.5 mm Hg (± 2.1 mm Hg; all confidence levels were calculated at

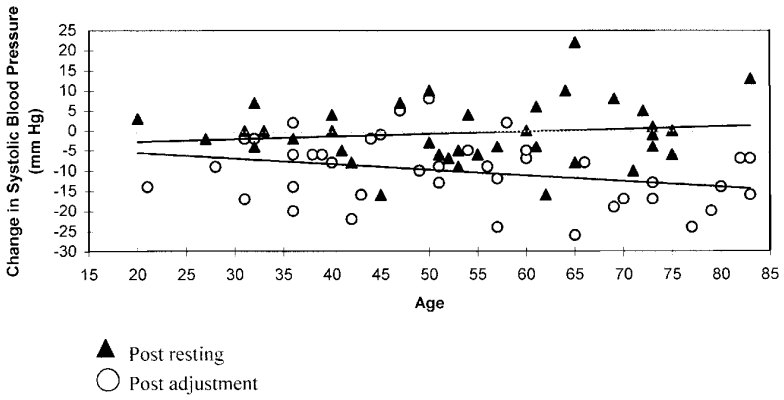

Fig 1. *Graph shows that pre-post manipulation change in systolic BP increased with increased age.*

Table 2. *Test 1 results: changes in systolic BP with age*

Age (y)	Change in systolic BP (mm Hg): Pre reading to Post reading	SD	95% CONF
≤55 (n = 22)	–7.6	7.8	±3.3
>55 (n = 18)	–13.8	7.6	±3.5

Pre reading to Post reading (postadjustment) *t* test (≤55 vs >55): $P = .02$ (statistically significant).

BP, Blood pressure; *CONF,* confidence level.

95%; $P = 0.67$), from 133.1 mm Hg (± 7.2 mm Hg) to 133.6 mm Hg (± 7.5 mm Hg); this was not a statistically significant change (Table 3). There were no significant changes in diastolic BP or pulse rate from the Prerest reading to the Postrest reading.

From the Postrest reading to the Postadjustment reading, the systolic BP dropped from 133.6 mm Hg (± 7.5 mm Hg) to 122.7 mm Hg (± 7.1 mm Hg), a significant change of –10.9 mm Hg (± 2.6 mm Hg; $P < .001$). Again, neither the diastolic nor the pulse rate changes from the Postrest reading to the Postadjustment reading reached statistical significance.

As in test 1, the Postrest reading-to-Postadjustment reading changes in systolic BP were greater as age increased (Fig 3 and Table 4). Up to and including age 55 years (n = 17), the average change was –7.8 mm Hg (± 3.0 mm Hg); after age 55 years (n = 13), the average change was –14.9 mm Hg (± 3.6 mm Hg). This difference between the two age groups in systolic BP drop was significant at $P = .006$.

When the subjects who were adjusted in test 1 (n = 40) were compared with those in test 2 (n = 30), it was found that the average ages were similar: 53 years in test 1 and 54 years in test 2. The group of patients adjusted in test 1 had an average initial systolic BP of 140.7 mm Hg (± 7.8 mm Hg); this was higher than that of the subjects in test 2, which was 133.1 mm Hg (± 7.2 mm Hg). However, the initial systolic BP of the test 1 subjects was not significantly higher ($P = .18$).

DISCUSSION

Test 1 of this study revealed a statistically significant decrease in systolic BP between a treatment (palpation and vectored upper cervical adjustment, the necessity being determined by postural checks) group and a nontreatment (resting) group. Test 2, in which the subjects acted as their own controls, also showed a statistically significant decrease in systolic BP from the Postrest reading to the Postadjustment reading.

Within the nonblinded parameters of this study, it can be said that palpation and vectored atlas adjustment of suspect joint dysfunction in the upper cervical spine has an effect that significantly lowers systolic BP in comparison with what is seen in rested controls. The test 1 study also showed that those patients who, in the judgment of the examiner, demonstrated postural distortion (pelvic torsion/unleveling, "short leg," foot rotations) had significantly higher systolic BP readings than those examined and judged to be free of such distortion.

The lack of blinding of the subjects leaves open the possibility that the decrease in systolic BP was due to a placebo effect. A sham adjustment, perhaps one made through use of an Activator instrument (Activator Methods, Inc) set for no excursion, might help to blind this sort of test. However, neither of the other measurements—diastolic pressure and pulse rate—showed any significant difference between the adjustment and resting groups, which raises the question of why any putative placebo effect would have influenced only the systolic pressure.

Using an adjustive style thrust in a (presumably) noninvolved area of the spine and then checking those results would have helped to control for nonspecific reflex responses. Similarly, having a control group in which each subject was positioned and the atlas transverse palpated would have helped to determine whether the active factor was the thrust of the adjustment or the stimulation of some palpatory reflex. This kind of control is problematic, however, inasmuch as palpation, depending on how forceful it is, might act as an adjustment.

Underinflation of the cuff as a reason for throwing out data could have led to biasing for lowered, not raised, BP after adjustment. However, underinflation happened only rarely, because the sphygmomanometer had a built-in sensor that indicated any need for more cuff pressure while the cuff

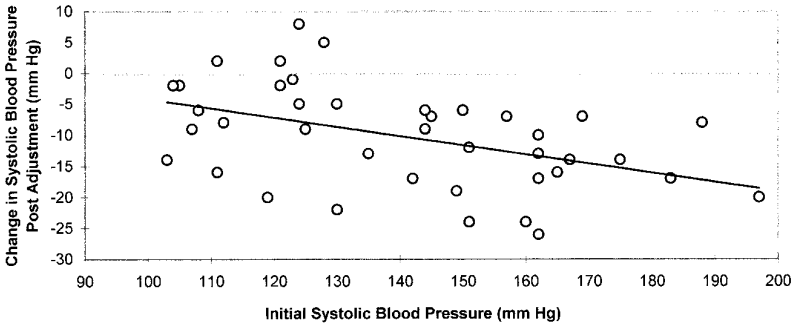

Fig 2. *Graph shows that those with greater initial systolic BP had greater changes in systolic BP after adjustment.*

was being inflated. In addition, although data for some patients were thrown out, the same patients were often examined later and their readings taken without error.

There was a concern in test 1 that inasmuch as the adjustment group's systolic BP Pre reading was significantly higher than the control group's, merely being "hypertensive" might result in a greater decrease in systolic pressure after resting and changing positions from supine to sitting. To check for this possibility, the data for the control group were analyzed to isolate those subjects whose prerest systolic BP values were within the confidence level of the mean or above that of the adjusted group (≥133 mm Hg). There were 11 of these control group subjects, and they registered essentially no change (+0.1 mm Hg) in Pre reading-to-Post reading systolic pressure. In other words, resting of these "hypertensive" control subjects did not cause a significant lowering of their systolic pressure.

Finally, in test 1, although the systolic BP Pre reading of the adjustment group was significantly different from the systolic BP Pre reading of the control group (140.7 mm Hg vs 124; $P < .001$), the postintervention (adjustment or resting) systolic BP values for the two groups were not significantly different (130.4 vs 123.5 mm Hg; $P = 0.34$). This indicates that although the adjustment group's systolic pressure Post reading was still higher than that of the control group, the elevation was not statistically significant.

As a check against the possibility that some unknown difference between the adjustment and control groups was responsible for the change in systolic BP, another test was done. In this test, test 2, the subjects served as their own controls. Three BP checks were done: a Prerest reading, a Postrest reading, and a Postadjustment reading. As in test 1, there was a significant decrease in systolic BP from the Postrest check to the Postadjustment check ($P < .001$). This indicates that the pre/post differences in systolic BP were most likely due to the adjustment and that in test 1 they were not due to some unknown difference between the treatment (adjustment) and control (resting) groups.

Despite the fact that the subjects in test 2 served as their own controls, there are some factors that could have confounded the BP readings, inducing potential error in the tests performed. Studies examining changes in systolic BP from supine to sitting, as opposed to supine to standing (because of concerns about orthostatic hypotension), are few. One study done on hypertensive subjects (110 males aged 16-64 years) found a slight (+3 mm Hg) increase in systolic BP (from 153 to 156 mm Hg) when the position was changed from supine to sitting.[31] However, the subjects in that study were rested far longer in the supine position (30 minutes) and after changing their position to sitting (10 minutes) before BP readings were taken than the subjects in the present study. A study of normotensive patients (22 men and 25 women aged 21-59 years) also found a slight (+3 mm Hg) increase in systolic BP in women when they changed position from supine to sitting.[32] Again, the resting times were longer—10 minutes each for supine and after changing to sitting—than those used in this study.

Another confounding factor is the finding that repeated checks of BP without adequate time for stabilization between the readings result in a decrease in systolic pressure: an average of –3.2 mm Hg was found in one study[33] and –3 to –4 mm Hg was found in another.[34]

How these confounding factors might have affected the results in the studies is not known. Changing the protocol to allow for longer stabilization times after changing positions and use of a second or third BP check might help to eliminate these variables. However, as far as the present study is concerned, the rise in systolic BP that might have occurred in changing positions from supine to sitting may have offset any decrease associated with from taking a second BP reading without sufficient stabilization time.

Table 3. *Test 2 results: systolic BP checked before resting, after resting, and after adjustment (n = 30)*

	Systolic BP (mm Hg)	SD	95% CONF
Prerest reading	133.1	20.1	±7.2
Postrest reading	133.6	21.0	±7.5
Change: Prerest to Postrest	0.5	6.0	±2.1
Postadjustment reading	122.7	19.9	±7.1
Change: Postrest to Postadjustment	-10.9	7.4	±2.6

Average subject age: 54 y (range, 14-83 y).
Prerest reading to Postrest reading *t* test: $P = .67$.
Difference between Postrest reading and Postadjustment reading: $P < .001$ (statistically significant).
BP, Blood pressure; *CONF,* confidence level.

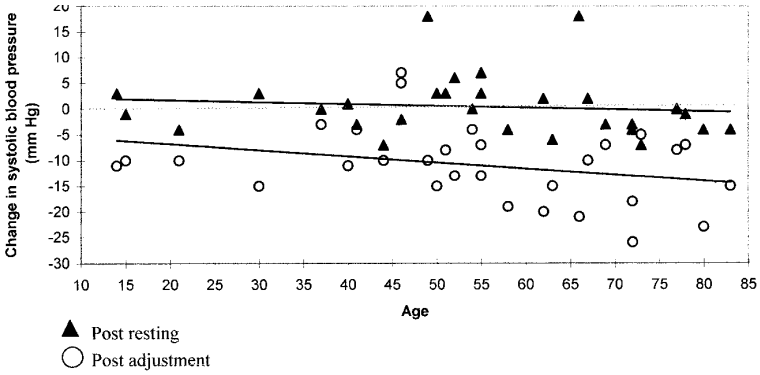

Fig 3. *Graph shows changes in systolic BP after resting and after adjustment.*

Table 4. *Test 2 results: change in systolic BP with age*

Age (y)	Change in systolic BP: Post rest to Postadjustment	SD	95% CONF
≤55 (n = 17)	–7.8	6.3	±3.0
>55 (n = 13)	–14.9	6.7	±3.6

Postrest reading to Postadjustment reading *t* test (≤55 vs >55): $P = .006$ (statistically significant).
BP, Blood pressure; *CONF,* confidence level.

A last critical note: these studies did not examine the long-term effects of vectored adjustment of putative upper cervical subluxation/joint dysfunction on BP, so no conclusions can be drawn as to any general positive health effects of lowering BP. Indeed, the BP changes noted may be only a short-term reaction to the stimulus of the adjustment.

Older subjects versus younger subjects

It is known that with age, arteries progressively stiffen[35,36] and the arterial wall becomes thicker.[35] These changes have a minimal effect on resting BP, but as pressure levels rise the effect becomes more pronounced.[35] White and Carrington[35] found that elderly men had a greater increase in systolic BP to induced pressor reflex and concluded that "the significantly greater rise in systolic BP in these elderly subjects supports the view that this response is exaggerated by increased arterial stiffness in older individuals."

These age-related changes in arteries are the likely reason for the findings noted in the present study. In test 1, as the age of the patient increased, the drop in systolic BP after adjustment increased (Fig 1). The decrease in systolic BP up to the age of 55 years (n = 22) for the adjustment group was –7.6 mm Hg (± 1.2 mm Hg); however, at 55 years and older (n = 18), the change in systolic BP post adjustment group rose to an average of –13.8 mm Hg (± 3.6 mm Hg)—a significant difference ($P = .02$). The figures in test 2 were nearly the same; the postadjustment change in systolic BP up to age 55 (n = 17) was –7.8 mm Hg (± 3.0 mm Hg), whereas after age 55 (n = 13) the change was –14.9 mm Hg (± 3.6 mm Hg)–a significant difference ($P = .006$).

Age and lack of arterial compliance is associated with higher systolic pressure, and this may explain why there was a greater percentage decrease in systolic pressure after adjustment with higher initial pressures (Fig 2).

Age should be taken into consideration when postmanipulation changes in BP are being investigated; a population of students in their 20s and 30s may not demonstrate a statistically significant effect. Such may have been the case in a study of normotensive chiropractic students that found a small but statistically significant drop in both systolic and diastolic BP after adjustment in comparison with what was found in motion-palpated control subjects.[3]

Cervicosympathetic Reflex

I propose here that the decrease in systolic BP was likely due to stimulation or normalization of upper cervical muscle spindle/GTO output. Bolton et al[11] found considerable evidence that in the cat the vestibular system influences the sympathetic and respiratory nerves.[11] The vestibulosympathetic reflexes have been hypothesized to offset orthostatic hypotension in positional changes by raising BP. As a counter to the vestibulosympathetic reflexes, cervicosympathetic reflexes, whose origin seems to be in upper cervical muscle spindles and/or GTOs, act in opposition by decreasing BP.[11] These reflex pathways are complicated, and examination of them, performed in studies of cats, is only recent. However, this explanation is anatomically and physiologically sound and fits what was seen in these 2 studies—a sudden drop in systolic BP after adjustment of putative upper cervical subluxation.

In a similar study of the upper cervical spine, Purdy et al[37] found that touching, massage, or manipulation of the suboccipital muscles lead to "sympathetic dampening," measured by a decrease in the pulse amplitude and height of the dicrotic notch. The effect was greatest with suboccipital manipulation. Again, this may be an effect of the cervicosympathetic reflex. Regardless of mechanism, the Purdy et al[37] study did show decreases in peripheral sympathetic tone with stimulation of the upper cervical spine.

Pressor Reflex

A long-term (2-month) drop in BP (–27 mm Hg systolic, –13 mm Hg diastolic) after specific upper cervical adjust-

ment of hypertensive patients deemed to be atlas-subluxated has been reported.[5] It is proposed here that such long-term BP changes may be due to a postadjustment moderating effect on global muscle tone. The upper cervical spine is associated with postural control,[16-18] and upper cervical adjustment had been noted to cause immediate reductions in postural distortion.[24-27] Muscle contraction causes increased need for blood flow, yet the pressure of contraction can completely close the intrinsic arteries. The intramuscular arteries become completely closed above a 30% maximal voluntary contraction[38]; in back extensor muscles, this occurs above a 40% maximal voluntary contraction.[39] To supply the contracting or hypertonic muscle, BP must increase, which is the function of the pressor reflex. Manipulation that causes a decrease in muscle tone could reduce the pressor reflex and BP.

The pressor reflex involves neural receptors inside the muscle that respond to contraction and are responsible for a series of physiologic effects that act to increase BP, forcing blood through the contracted muscle. According to Rowell et al,[40] the idea that cardiovascular-respiratory responses to exercise originated from chemoreceptors in skeletal muscle originated in 1886. The term *ergoreceptors* was initially used to describe the intrinsic muscle afferents that are sensitive to mechanical and metabolic changes related to muscle work.[41] These muscle afferents include (1) mechanoreceptors that are sensitive to pressure and tension and send signals via myelinated group III fibers and (2) metaboreceptors that are sensitive to chemical substances, the signals transmitted by group IV nonmyelinated fibers.[41]

A high proportion of afferent fibers in the group III and IV range are now known to respond to chemical and mechanical stimuli, which suggests that free nerve endings may be able to provide information about nonnoxious events.[16] As Mitchell et al[42] remark, "These two categories [group III and IV mechanoreceptors and metaboreceptors and group III and IV pain signaling nociceptors] are likely to represent two poles of a continuum, with most of the group III and IV afferents lying somewhere in between."

Muscle contraction (and stretch) immediately stimulates the intrinsic mechanoreceptors, causing an instant increase in cardiac sympathetic nerve activity and resulting in rapid increases in heart rate, left ventricular contractility, and cardiac output.[43] However, the mechanoreceptive group III muscle afferents have a rapid adapting property, and their discharges return to almost control level within seconds after the onset of contraction.[43,44] As the metabolic products of contraction build up, the chemically stimulated group IV metaboreceptors become responsible for the sympathetically mediated physiologic changes.

The physiologic changes induced by the muscle mechanoreceptor and metaboreceptor afferents include all of the following: increased ventilation[40,41]; increased heart rate[40,41,45]; increased sympathetic tone to the blood vessels serving the kidney[42,46] and adrenal glands[43]; changes in vasomotor signal to noncontracting muscles and skin[41,45,47]; increases in glucose production, plasma concentration of glucose, adrenocorticotropic hormone, Met-enkephalin, and B-endorphin; and decreases in plasma insulin.[48] All of these effects act to increase BP[40,49,50] and increase the blood and nutrient flow though contracted muscle(s). The effect of the pressor reflex can be dramatic. An experiment in which the metabolic products of contraction were trapped in the relatively small flexor muscles of the little finger caused an overall increase in systolic BP of 70 mm Hg.[49] Outside the special conditions generated in the laboratory, however, the effects of the pressor reflex may be larger with the involvement of larger muscle groups[40,43,51] and fast-twitch muscle fibers.[36,42]

Long-term/chronic increases in BP may come from renal sympathetic artery stimulation and vasoconstriction. Decreased blood flow to the kidneys causes the body to retain fluid; blood volume increases, cardiac output increases, and BP rises, forcing more blood through the kidney and the contracted muscle(s). The decrease in serum aldosterone noted in one study of manipulation and hypertension may be related to this mechanism.[9]

Thermography

Changes in skin flow patterns have been noted as an effect of the pressor reflex.[41,45,52-54] The sympathetic stimulation associated with the pressor reflex involves complex interactions to control BP and skin blood flow through use of sudomotor and vasomotor stimulation and inhibition. Although the metaboreflex affects peripheral vasculature and may induce vasoconstriction and a rise in BP,[41] this vasoconstriction may not be manifest in skin blood flow.[52]

Skin blood flow pattern changes in response to muscle contraction in normothermia have been found to be under what is called *central command*—ie, the sympathetic centers in the brain[52,53] or the muscle mechanoreceptor afferents.[54] In hyperthermic conditions, muscle metaboreceptor output seems to inhibit the active skin vasodilator system.[45]

If joint dysfunction causes a pressor reflex significant enough to involve changes in central command, abnormal skin thermal patterns may present because of sympathetic vasomotor activity. Such abnormal skin thermal patterns may be noted by means of thermography and other heat-sensing instrumentation. Although such altered cutaneous heat patterns could be indicative of joint dysfunction, they would not likely be segmentally related. Central command could alter sympathetic tone in the skin of the upper thoracic spine as a result of a pressor reflex from muscle contraction and joint dysfunction in the lumbopelvic spine.

Peer-reviewed literature on the use of thermography as an aid in determining chiropractic subluxation is limited[55-57]; discussion of theoretic physiologic models relating subluxation to thermographic changes even more so.[56] Some models postulate thermographic changes segmentally related to chiropractic subluxation[56]; other models postulate thermographic patterns that are not necessarily related to any segmental dysfunction, according to a written communication from members of the Chiropractic Institute of Thermography

and Diplomats of the International Chiropractors Association College of Thermography. An explanation for the nonsegmental thermographic patterns associated with subluxation may be skin vascular blood flow changes due to pressor reflex.

Alterations of Visceral Physiology with Manipulation

Nansel and Szlazak[58] argued persuasively that sustained somatic sympathetic discharge to segmentally related viscera in putative joint dysfunction is not likely to occur, let alone cause frank visceral pathosis. On the other hand, the pressor reflex response to muscle contraction has been shown to involve sympathetic stimulation to the heart,[40,41,45] lungs,[40,41] kidneys,[42,46] adrenal glands,[43] muscles and skin,[41,45,47] and glucoregulatory system.[48] Elimination of putative joint dysfunction and moderating abnormal muscle contraction(s) may have positive effects, normalizing the physiology of these organs and hormonal regulatory systems. Although this line of thought is a logical extension of the argument for the involvement of a pressor reflex in joint dysfunction, it is speculative and needs study.

CONCLUSION

Palpation and vectored adjustment of subjects (n = 40) with putative upper cervical joint dysfunction diagnosed by postural distortions significantly lowered systolic BP both from pretreatment to posttreatment ($P < .001$) and in comparison with a similar resting control group (n = 40; $P < .001$). Another test in which subjects (n = 30) were used as their own controls also showed a significant decrease in systolic BP from resting to postadjustment values ($P < .001$). I propose that the sudden decrease in systolic BP noted in both of these tests was due to stimulation of cervicosympathetic reflexes or possibly to moderation of muscle tone and elimination of the effects of the pressor reflex. This study also found a greater decrease in systolic BP after adjustment in subject patients with increasing age. Associations between the effects of the pressor reflex and thermographic findings and the potential for alterations of visceral physiology in joint dysfunction have been discussed. Further studies involving (1) blinding, (2) testing for direct connections between joint dysfunction, muscle hypertonicity, and the pressor reflex, and (3) the possibility of long-term reduction in systolic BP, are recommended.

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