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The immediate effect of atlanto-axial high velocity thrust techniques on blood flow in the vertebral artery: A randomized controlled trial[☆]Jonathan W. Erhardt^{a,*}, Brett A. Windsor^b, Roger Kerry^c, Chris Hoekstra^d, Douglas W. Powell^b, Ann Porter-Hoke^e, Alan Taylor^c^a Erhardt Physical Therapy & Sports Medicine, Portland, OR, USA^b Department of Physical Therapy, Campbell University, Buies Creek, NC, USA^c Faculty of Medicine and Health Science, University of Nottingham, Clinical Sciences Building, Hucknall Road, Nottingham, UK^d Therapeutic Associates, Sherwood, OR, USA^e North American Institute of Orthopedic Manual Therapy, Eugene, OR, USA

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ABSTRACT

Background: High velocity thrust (HVT) cervical techniques have been associated with serious vertebral artery (VA) trauma. Despite numerous studies, the nature of this association is uncertain. Previous studies have failed to demonstrate haemodynamic effects on the VA in simulated pre-thrust positions. No study has investigated haemodynamic affects during or immediately following HVT, nor sufficiently controlled for the influence of the thrust.

Objectives: To investigate the immediate effects of HVT of the atlanto-axial joint upon haemodynamics in the sub-occipital portion of the vertebral artery (VA3).

Design: Randomized Controlled Trial.

Method: Twenty-three healthy participants (14 women, 9 men; mean age 40, range 27–69 years of age) were randomly assigned to two groups: an intervention group (MANIP, n = 11) received HVT to the atlanto-axial segment whilst a control group (CG, n = 12) was held in the pre-manipulative hold position. Colour-flow Doppler ultrasound was used to measure VA3 haemodynamics. Primary outcome measures were peak systolic (PSV) and end diastolic velocities (EDV) of three cardiac cycles measured at neutral (N1), pre-HVT (PreMH), post-HVT (PostMH), post-HVT-neutral (N2) positions.

Results: Test-retest reliability for the Doppler measures demonstrated intra-class correlation coefficient (ICC) of 0.99 (95% CI 0.98–1.0) for PSV and 0.91 (95% CI 0.84–0.96) for EDV. Visually, EDV were lower in the MANIP group than in the CONTROL group across the four measurements. However, there were no significantly different changes (at $p \leq 0.01$) between the MANIP and CONTROL groups for any measurement variable.

Conclusions: HVT to the atlanto-axial joint segment does not affect the haemodynamics of the sub-occipital portion of the vertebral artery during or immediately following HVT in healthy subjects.

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[☆] This study was conducted at Erhardt Physical Therapy and Sports Medicine, an outpatient physical therapy clinic in Portland, OR. We, the authors, affirm that we have no financial affiliation (including research funding) or involvement with any commercial organization that has a direct financial interest in any matter included in this manuscript. We received no grant funding. Institutional Review Board approval was obtained from Pacific University, Forest Grove, OR, USA.

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1. Introduction

High velocity thrust (HVT) of the spinal joints has been a part of physical therapy practice since the early 1800's in Europe and the 1920's in America (Pettman, 2006). HVT is included in the pre-professional curricula in the United States, is commonly used in the clinical setting, and its effectiveness has been demonstrated in comparative studies, albeit with a moderate quality of evidence (Miller et al., 2010). HVT of the atlanto-axial joint is utilized by physical therapists to decrease pain and increase range of motion in the upper cervical spine (Yu et al., 2011). Further, effects on the autonomic nervous system, sensory system, and disability levels

have been identified (e.g. [Martinez-Segura et al., 2006](#); [Dunning and Rushton, 2009](#)). Important neural and vertebral structures lie in close proximity to the atlanto-axial joint, theoretically introducing a level of neurovascular risk with HVT of this articulation, e.g. arterial dysfunction leading to central ischaemia. Therefore, use of this technique should be based upon sound clinical reasoning: a thorough physical therapy examination, an evidence-based interpretation of the clinical state and circumstances, patient preferences and actions, and the research literature ([Rushton et al., 2012, 2013](#)).

The estimated risk of serious complications resulting from HVT of the cervical spine has been reported to range between 1 in 4500 ([Dunne et al., 2000](#)) to 6 in 10 million ([Hurwitz et al., 1996](#)). The precise risk of injury is indeterminable due to the unknown number of unreported cases ([Ernst, 2004](#); [Carlesso et al. 2010a](#)) and the paucity of methodologically sound research studies ([Kerry et al., 2008](#)). Although the number of patients experiencing minor to moderate adverse events following manual therapy may be as high as 50%, the risk of major adverse events with manual therapy is reported to be low ([Carlesso et al., 2010b](#)). Nevertheless, given the limitations of known prevalence and the serious nature of the adverse event, HVT of the upper cervical spine remains a contentious issue (e.g. [Cassidy et al., 2012](#); [Wand et al., 2012](#)).

One potential adverse event associated with manual therapy of the atlanto-axial joint is the development of a small tear or lesion (dissection) of the vertebral artery (VA) ([Mann and Refshauge, 2001](#)). Multiple factors may increase the risk of events, including anatomical anomalies or hypoplasia of the vertebral arteries, prior injury, and a history of previous ischaemic episodes ([Taylor and Kerry, 2010](#)). Biomechanically, craniocervical rotation is the movement most likely to be associated with arterial damage, specifically to the VA3 portion of the vessel system ([Mitchell, 2009](#); [Malo-Urriés et al., 2012](#)). The VA3 extends from the point of exit from the axis to its entry into the spinal canal, ([Roberts and Demetriades, 2001](#)) and is reported to be the structure most vulnerable to mechanical insult and injury ([Terrett, 1987](#); [Grant et al., 1994](#); [Refshauge et al., 1995](#); [Macchi et al., 1996](#); [Kuether et al., 1997](#); [Johnson et al., 2000](#)). One underlying concern is that HVT involving rotation may, at least momentarily, alter the velocity of blood flow in one or both of the vertebral arteries. The relationship between alteration of blood flow and adverse event is not fully understood, although flow changes are reported in patients who experience VA stroke ([Sturzenegger et al., 1993](#); [Sengelhoff et al., 2008](#); [Baracchini et al., 2010](#)). The mechanistic reasoning for measuring blood flow in relation to adverse events is principled initially in the idea of altered haemodynamics being a fundamental component of vessel pathology ([Cecchi et al., 2011](#)). Further, flow change is considered to be indicative of the sorts of mechanical stresses responsible for the type of micro-trauma in question ([Mitchell, 2007](#)). Thus blood flow measurement remains a valid procedure to understand vascular tissue stress in the VA.

Clinical relevance is of utmost importance to the practicing physical therapist. In this context, clinical relevance may be described as the net flow volume at or below which patients become symptomatic with vertebrobasilar ischaemia ([Bendick and Glover, 1987](#)). Although blood flow volume is the primary measure affecting brain perfusion ([Ganong, 2005](#)), the measure of interest in this study was blood flow velocity. Changes in blood flow velocity may be used to reflect changes in volume ([Aaslid et al., 1986](#); [Newell and Aaslid, 1992](#); [Mitchell, 2007](#)). Blood flow velocity and volume vary proportionally ([Aaslid et al., 1986](#); [Newell and Aaslid, 1992](#)) in part due to the low pulsatility index of the VA, and during normal laminar blood flow the VA internal diameter does not change ([Mitchell, 2009](#)). Therefore, blood flow velocity changes from spinal movement can be considered a good indicator of related changes in blood flow volume ([Mitchell, 2007](#)).

Previous studies have reported on the nature of blood flow during passive physiological movements such as rotation ([Thiel et al., 1994](#); [Haynes, 1995](#); [Mitchell, 2003](#); [Mitchell et al., 2004](#); [Mitchell and Kramschuster, 2008](#)). Although findings have varied, a meta-analysis of the data reported that normal, physiological rotation compromised blood flow in the contralateral vessel ([Mitchell, 2009](#)). There are very few studies examining flow in positions more closely associated with HVT. Placing the cervical spine in a simulated HVT position has not been shown to adversely affect blood flow in vertebral arteries ([Licht et al., 1998](#); [Licht et al. 1999](#); [Bowler et al., 2011](#)). A recent MRI flow study reported no changes in flow after the execution of HVT ([Quesnele et al., 2014](#)). Information pertaining to blood flow during a cervical HVT technique may enhance the clinical decision making of the manual physical therapist. Therefore, the purpose of this study is to quantify changes in blood flow velocity in the VA during HVT of the atlanto-axial joint. Based on previous findings, it was hypothesized that in healthy adults, HVT of the atlanto-axial joint would not result in altered blood flow velocity in the VA3 compared to the pre-manipulation hold position.

2. Methods

2.1. Trial design

A placebo-controlled, randomized trial was carried out to investigate the effect of a cervical spine HVT on VA blood-flow. This study was approved by the Institutional Review Board at Pacific University, Forest Grove, Oregon.

2.2. Participants

Twenty-three healthy participants (14 females; 9 males) were recruited from the local community by word of mouth for this study over a period of 4 weeks. The principle investigator reached out in person or by phone to friends and family of clinic staff and current or former patients of the clinic. Participants who agreed to take part reported to Erhardt Physical Therapy and Sports Medicine (an outpatient physical therapy clinic) and were screened for exclusion criteria. Participants were excluded if they had a history of known VA anomalies, hypoplasia or previous injury, undiagnosed dizziness, hypertension (140/90 or greater), head or neck trauma within the last six weeks, known upper or mid-cervical instability, recent cervical spine HVT, prior cervical spine surgery, or cerebrovascular events of any kind. Participants were also excluded if they had a history of cervical spinal cord compression, osteoporosis, were currently pregnant, or reported a history of current or previous systemic steroid intake or prescription anticoagulants, chronic upper respiratory infection, cancer, trisomy 21, Klippel Feil, Erlos Danlos syndrome or any other arthritides in its inflammatory state. Finally, participants were excluded if the VA3 could not be visualized on ultrasound. All participants signed a written informed consent form approved by the Pacific University Institutional Review Board prior to participating in this study. [Fig. 1](#) demonstrates the method process.

2.3. Sample size

Based on previous blood flow data as reported in [Bowler et al. \(2011\)](#), it was calculated that the study would require at least 11 subjects per group. The study used a significance level of 5%; power of 80%; a standard deviation in blood flow velocity of 18 cm/s and a mean percentage change in blood flow velocity of 26%, considering Doppler variation, volume flow rate as a function of average velocity and area, and ultrasound machine variability.

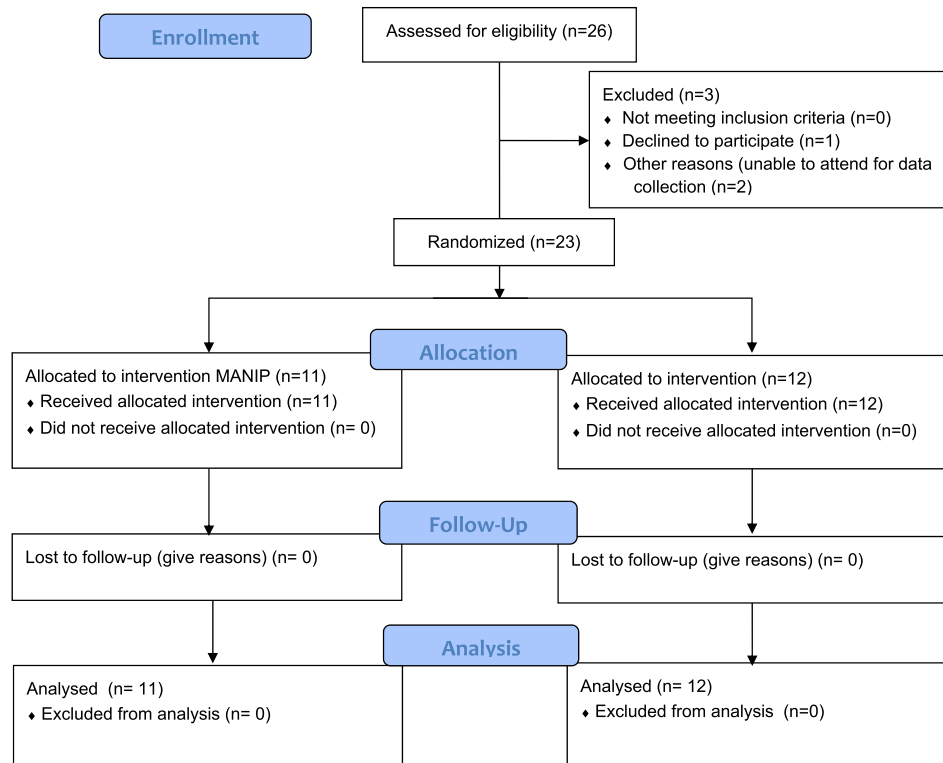


Fig. 1. Protocol flow and method process.

2.4. Randomisation

A research assistant marked 12 cards each with a (1) or a (0) representing MANIP (HVT to the atlanto-axial joint) and Control (No HVT to the atlanto-axial joint) groups respectively. Each of the 24 cards were sealed in an opaque envelop by that person then placed in a bin and shuffled. Participants drew group assignment in the order they arrived at the clinic. The research assistant handed that card to the therapist. Participants were thereby assigned to either the MANIP group or the CONTROL group based on that drawn card.

2.5. Blinding

Due to the nature of the HVT intervention, and the necessary timings of flow data capture, it was not possible to blind the treating therapist or the registered vascular technician (RVT) to group allocation.

2.6. Outcome measures and instrumentation

Primary outcome measures were haemodynamic markers of peak systolic velocity (PSV) and end diastolic velocity (EDV). Secondary measures included mean velocity (V_{Mean}) and a resistance index (RI). These haemodynamic markers indicate mechanical stress of the insonated vessel (Mitchell, 2007). A GE Vivid-I Doppler ultrasound machine with enhanced colour-flow was used to measure VA3 haemodynamics with 2 MHz Sector 3S linear transducer. Doppler settings of 1.7MHz during colour and/or spectral (waveform) Doppler and 3.4MHz while in BMode (black and white image); depth 2–4 cm and angle of insonation range of 18°–60°.

2.7. Interventions

Fig. 2 illustrates the intervention and measurement point flow. Following group assignment, each participant entered a private

room for data collection. Prior to the ultrasound measurements, each participant's resting blood pressure and heart-rate were measured in sitting followed by cervical active range of motion in all planes with rotation held for 10 s while monitoring for signs of hind brain ischaemia. Participants were then asked to lay supine on an examination table with their heads on a folded towel and their legs on a bolster placing the hips and knees in approximately 45° flexion. The physical therapist stood at the head of the table and placed his hands in the proper position to safely control the position, direction, speed and amplitude of movement. The RVT then insonated the right VA3 immediately proximal (inferior) to where the artery entered the transverse foramen of C1 (Fig. 3).

The transducer was held in place for the remainder of the session (for repeated measurements). The angle of insonation remained 60° or less because small changes in this angle may affect absolute velocity readings (Sidhu, 2000). For both the MANIP and CONTROL group, the PSV and EDV of three cardiac cycles were measured four times. For both groups, flow in the neutral spine resting position was taken as the first baseline measure (N1) (Fig. 4).

The patient's cervical spine was then positioned in a pre-HVT hold position (Fig. 5). Clinically, this position deliberately, but gently, takes the “target” joint and surrounding soft tissues slightly beyond the range through which the HVT is performed (Pettman, 2006). In this case, the pre-HVT position was attained by sequentially, 1) side bending the upper cervical spine to the right, 2) slightly rotating the upper cervical spine to the left about a sagittal cranio-vertebral axis, and 3) adding mid-cervical spine right side bending. The position was held for approximately 10 s as the RVT measured VA3 (2nd measurement – PreMH).

2.8. HVT protocol (MANIP)

MANIP group participants received an HVT to the atlanto-axial joint segment through the posterior superior aspect of the right

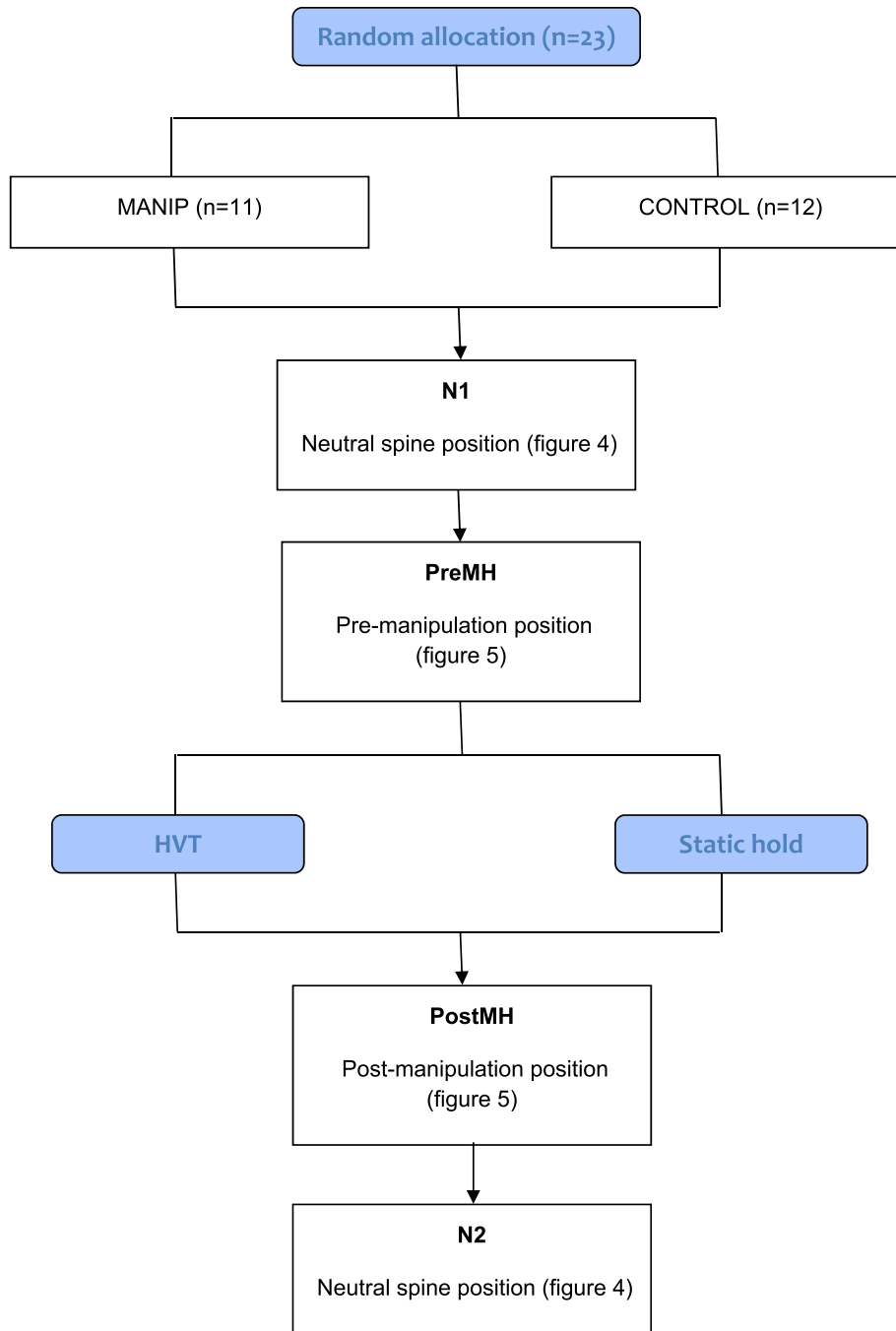


Fig. 2. Measurement point flow.

transverse process of C1 with the physical therapist's right hand in an anterior, inferior and medial direction. This was immediately followed by re-measure (PostMH) by the RVT. The RVT maintained the pulsed-wave Doppler image of the right VA3 during the post-HVT return period as the spine was passively returned to neutral position. A final measurement was taken in the neutral position post-HVT (N2).

2.9. Control group (CONTROL)

Control group participants were measured a second time in the pre-HVT position approximately 3–5 s after the 2nd measurement to simulate the time that would elapse during the administration of

an HVT to the atlantoaxial segment (PostMH). The spine was passively returned to the neutral position where a final measurement was taken (N2).

2.10. Follow up

All participants returned to the clinic five days after participation in the study (5 day follow up visit). During the five-day follow up visit, participants were asked to report any adverse events associated with their manipulation or hold in the pre-manipulation position. An adverse event was functionally defined as any untoward medical occurrence associated with the therapeutic intervention (Carlesso et al., 2010a). "Examples of adverse events would

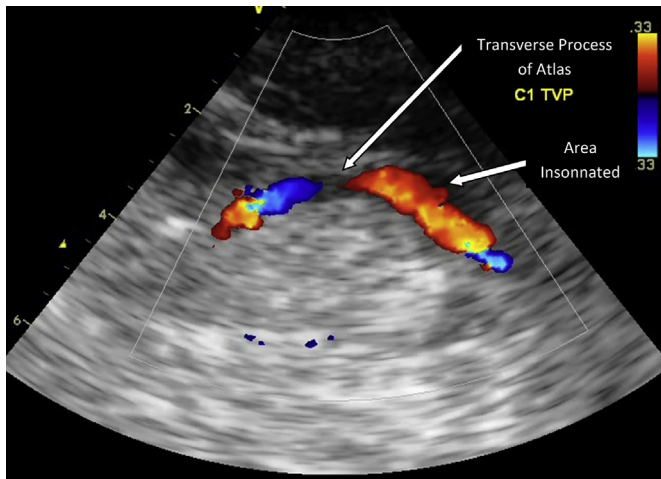


Fig. 3. Sample Doppler ultrasound image of the VA3 from a representative participant illustrating area insonnated.



Fig. 4. Neutral measurement position (N1 and N2) for Doppler ultrasound imaging.



Fig. 5. Pre-manipulation (PreMH) and post-manipulation (PostMH) holds position for Doppler ultrasound imaging.

include transient neurological symptoms, increased neck pain/stiffness, headache, radiating pain, fatigue or other.” (Carlesso et al., 2010b).

2.11. Study reliability

The ultrasound machine was operated by a registered diagnostic cardiac sonographer (RDSCS) and registered vascular technician (RVT) through the American Registry of Diagnostic Medical

Sonographers (ARDMS). The sonographer possessed 6 years of experience in vascular ultrasound. Appropriate images of VA3 were acquired using duplex ultrasound with colour-flow and pulse-wave Doppler imaging to measure PSV, EDV, and calculate RI. The physical therapist administering the HVTs possessed fourteen years of clinical experience; is a Board Certified Orthopedic Specialist (OCS) and a fellowship trained manual therapist (FAAOMPT) through the North American Institute of Manual Therapy (NAIOMT). All measurements were taken at the same physical therapy clinic. Intra-class correlation coefficient analysis was undertaken to examine test-retest reliability.

2.12. Statistical analysis

Vertebral artery blood flow velocity was measured in centimetres-per-second (cm/s). Three measures of PSV and EDV were taken in each of the four experimental positions. These were averaged per individual, and then group PSV and EDV means were used for analysis. Overall mean blood flow velocity (V_{Mean}) was used to calculate an RI based on the formula: $V_{Mean} = [(PSV - EDV)/3] + EDV$ (Mitchell, 2009).

Normality of the study variables was confirmed with a Shapiro–Wilk test. To determine the effects of position on blood flow, a repeated measures analysis of variance (ANOVA) was used to compare all positions, with Tukey's post-hoc tests used to determine the source of any significant interaction. To determine significant differences in blood flow parameters between the HVT and control groups, analysis of covariance (ANCOVA) was used to account for differences in blood flow parameter values in the previous testing position.

Intrarater reliability of measuring flow parameters by ultrasound was estimated by calculating the intraclass correlation coefficient (ICC) for the sole rater, using a one-way random effects model. Three cardiac cycles measured in each position during actual data collection were used for this reliability analysis. To control for Type I errors in view of multiple testing a p value <0.01 was considered statistically significant. All statistical analyses were conducted using SPSS (PASW) 22.0 (IBM Corp., Somers, NY, USA).

3. Results

3.1. Baseline data and measurement

Table 1 lists baseline characteristics of the participants. No significant differences were found. No participants reported or were observed to have experienced adverse events including signs or symptoms of neurovascular compromise at any time throughout

Table 1
Physical and clinical baseline characteristics of the participants.

	MANIP (n = 11)	CONTROL (n = 12)	P value*
Age (years)	41.58 ± 12.6	38.55 ± 9.0	0.590
Sex (n, %)			
Male	3 (25%)	6 (54.5%)	0.265 ^a
Female	9 (75%)	5 (45.5%)	
BMI	30.20 ± 8.23	28.43 ± 5.44	0.567
HR (bpm)	74 ± 14	83 ± 11	0.133
BP (mm/Hg)			
Systolic	119 ± 11	122 ± 10	0.496
Diastolic	74 ± 8	72 ± 6	0.580

Data are reported as mean ± standard deviation (SD) or as frequencies (%).

Note: BMI, Body Mass Index; HR, Heart Rate; BP, Blood Pressure; bpm, beats-per-minute; mm/Hg, millimetres/mercury.

*p-Value = statistical significance of the between-group differences using independent t-tests.

^a As determined by chi square test.

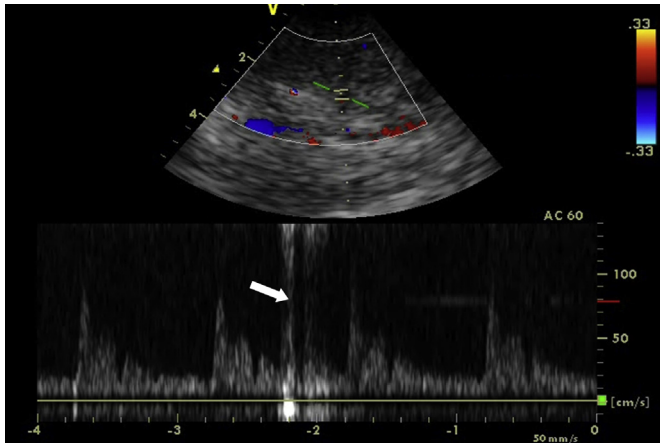


Fig. 6. A representative spectral wave form of the VA3 during spinal manipulation. The arrow indicates the application of the thrust manipulation.

participation in the study. Adverse events were measured by their presence (1) or absence (0). Patients returned to the clinic five days post-study participation for a follow up appointment. The principal investigator assessed the presence or absence of an adverse event at the 5-day follow up visit. The VA3 of each participant was visible using ultrasound, therefore none were excluded on these grounds. No signs of turbulent flow, spurting, high resistance waveform or dampening of the spectral waveform were observed by the RVT. Fig. 6 shows a sample Doppler image, capturing flow before, during, and immediately after intervention points. Test-retest reliability for the Doppler measures demonstrated an intra-class correlation coefficient (ICC) of 0.99 (95% CI 0.98–1.0) for PSV and 0.91 (95% CI 0.84–0.96) for EDV.

3.2. Within-group comparison

Table 2 shows measurements within the MANIP and CONTROL groups across time in all four measurement conditions. No significant measurement effects were observed on PSV (MANIP: $F = 0.279$, $p = 0.840$; CONTROL: $F = 0.433$, $p = 0.724$), nor EDV (MANIP: $F = 0.899$, $p = 0.414$; CONTROL: $F = 1.245$, $p = 0.309$), all at $p < 0.05$.

3.3. Between-group comparison

Table 3 reports differences in all measurement variables in all conditions between the MANIP and CONTROL groups. Figs. 7 and 8 visualize average PSV and EDV in the MANIP and CONTROL groups at the four measurement points. Visually, EDV were lower in the MANIP group than in the CONTROL group across the four measurements. However, there were no significantly different changes (at $p \leq 0.01$) between the MANIP and CONTROL groups for any measurement variable.

4. Discussion

This study investigated potential changes in haemodynamic flow velocity in the VA in response to HVT therapy. Primary findings demonstrate no changes in VA blood flow velocity immediately following HVT and no differences in blood flow velocity between HVT therapy and static hold in the pre-manipulative position.

Previously reported average PSV of normal, neutral VA flow have been between 19 and 98 cm/s (Trattning et al., 1990). The average

Table 2

Effect of cervical spine position on VA PSV (cm/s), EDV (cm/s), V_{Mean} (cm/s), and RI. (Group mean \pm SD).

	N1	PreMH	PostMH	N2	P-value ^a
MANIP					
PSV	86.05 \pm 23.89	88.57 \pm 21.55	88.57 \pm 16.40	90.68 \pm 15.70	0.840
EDV	26.30 \pm 7.05	29.54 \pm 8.78	28.37 \pm 7.94	28.40 \pm 4.79	0.453
V_{Mean}	46.22 \pm 11.46	49.22 \pm 12.32	48.44 \pm 9.76	49.16 \pm 7.17	0.794
RI	0.68 \pm 0.07	0.67 \pm 0.06	0.68 \pm 0.07	0.68 \pm 0.07	0.364
CONTROL					
PSV	84.16 \pm 28.76	80.74 \pm 23.78	84.20 \pm 26.78	80.45 \pm 23.18	0.724
EDV	24.54 \pm 5.76	22.45 \pm 6.25	23.84 \pm 7.48	23.63 \pm 7.27	0.309
V_{Mean}	44.41 \pm 12.63	41.88 \pm 10.75	43.96 \pm 12.98	42.57 \pm 11.82	0.634
RI	0.69 \pm 0.08	0.71 \pm 0.07	0.71 \pm 0.07	0.70 \pm 0.07	0.666

Note: PSV, peak systolic velocity; EDV, end diastolic velocity; V_{mean} , mean velocity; RI, resistance index; N1, neutral position before intervention manipulation; PreMH, pre-manipulation hold; PostMH, Post-manipulation hold; N2, neutral position after intervention manipulation.

^a Tests the hypothesis that there is no difference between any cervical positions ($p < 0.05$).

Table 3

Effect of HVT compared to control on VA PSV (cm/s), EDV (cm/s), V_{Mean} (cm/s), and RI (Group mean \pm SD).

	MANIP	CONTROL	P value ^a
PSV			
N1	86.05 \pm 23.89	84.16 \pm 28.75	0.866
PreMH	88.57 \pm 21.55	80.74 \pm 23.78	0.350
PostMH	88.57 \pm 16.40	84.20 \pm 26.78	0.633
N2	90.68 \pm 28.76	80.45 \pm 23.18	0.202
EDV			
N1	26.30 \pm 7.05	24.54 \pm 5.76	0.518
PreMH	29.54 \pm 8.78	22.45 \pm 6.25	0.036
PostMH	28.37 \pm 7.94	23.84 \pm 7.48	0.315
N2	28.40 \pm 4.79	23.63 \pm 7.27	0.281
V_{Mean}			
N1	103.59 \pm 27.03	100.52 \pm 31.63	0.806
PreMH	108.27 \pm 26.39	22.45 \pm 6.25	0.212
PostMH	107.48 \pm 20.22	23.84 \pm 7.48	0.574
N2	109.61 \pm 17.26	96.20 \pm 27.03	0.198
RI			
N1	0.68 \pm 0.07	0.69 \pm 0.08	0.793
PreMH	0.67 \pm 0.06	0.71 \pm 0.07	0.048
PostMH	0.68 \pm 0.07	0.71 \pm 0.07	0.164
N2	0.68 \pm 0.07	0.70 \pm 0.07	0.962

Note: PSV, peak systolic velocity; EDV, end diastolic velocity; V_{mean} , mean velocity; RI, resistance index; N1, neutral position before intervention manipulation; PreMH, pre-manipulation hold; PostMH, Post-manipulation hold; N2, neutral position after intervention manipulation.

^a Tests the hypothesis that there is no difference between thrust intervention group and control group ($*p < 0.01$).

neutral position PSV (MANIP 86.05 cm/s; CONTROL 85.16 cm/s) fell within this range but were higher than those reported by Bowler et al. (2011) (right VA: 54.06 cm/s). Bowler et al., however, measured flow slightly distal (C2/3 level) to the present study where there is less convolution of the vessel. Bowler et al. reported no changes in response to HVT. No significant flow or velocity changes were reported by Quesnele et al. (2014) with peak velocity at the VA3 section in neutral at 16.7 cm/s. However, this was measured with phase-contrast MRI and calibrated accordingly. Licht et al. (1998) used Doppler to measure PSV following HVT on 20 subjects with biomechanically dysfunctional spines, finding PSV averages at 63 cm/s and no changes 3 min following HVT. Using colour-coded duplex sonography, Licht et al. (1999) again measured flow volume, which is indirectly related to velocity. Once again, no changes following HVT were found.

Although no reference values for minimally clinically important differences in VA velocity have been established, some have attempted to identify clinical relevance of flow and velocity data: a

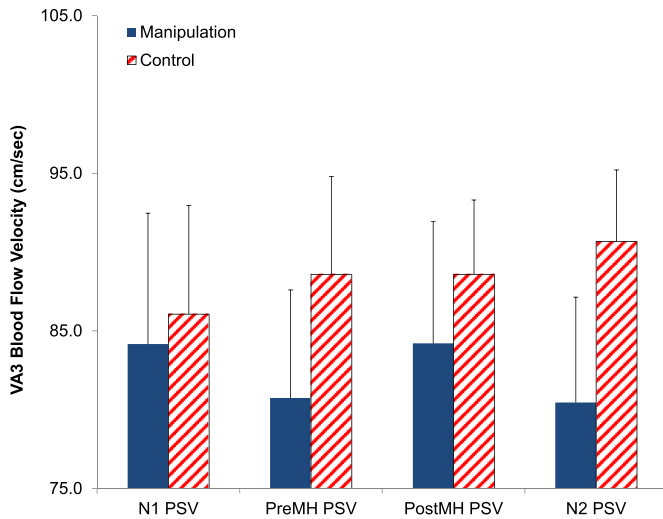


Fig. 7. Average peak systolic blood flow velocities in the VA3.

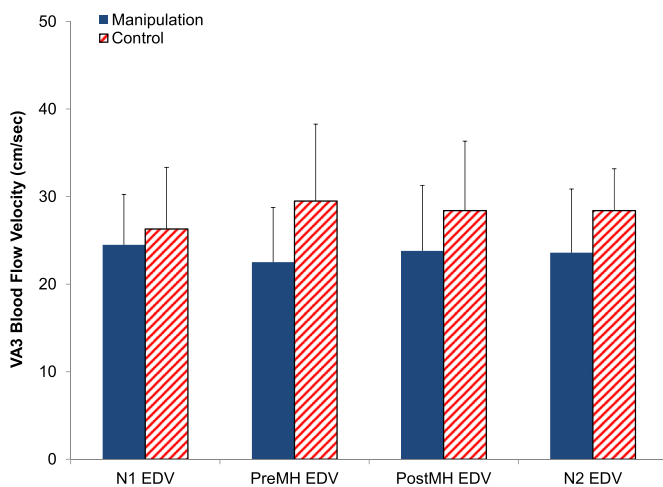


Fig. 8. Average end diastolic blood flow velocities in the VA3.

doubling of PSV from a preceding segment can be representative of a stenosis $\geq 50\%$ (Gerhard-Herman et al., 2006). The present study, however, did not compare VA3 flow to its distal segment. Alternatively, haemodynamically significant stenosis has long been considered as a $>50\%$ diameter reduction (Burns, 1995), which in the VA would yield PSV of >108 cm/s (Yurdakul and Tola, 2011). Quesnele et al (2014) align these data to clinically relevant changes previously suggested by Licht et al. (1998) as being a $>25\%$ change from baseline. Despite one present finding (PreMH between group difference in EDV) being statistically significant, the change is less than a 25% clinically relevant difference. All other measures were statistically not significant, and observation of descriptive trends revealed no clinically relevant changes between cervical positions, or between groups.

Signs of aberrant vascular flow can present in many ways. Abnormally low PSV, high resistance waveform and absence of flow or reversal of flow have been used to identify a potentially abnormal VA (Buckenham and Wright, 2004). A diagnosis of stenosis typically depends on the positive findings of increased velocity and abnormal flow characteristics, while occlusion is associated with the absence of a signal (Cher et al., 1992). More specifically, low grade stenoses result in spectral broadening and spike turbulence over the systolic peak. Higher grade stenoses

result in significant increases in PSV, with turbulence appearing as bi-directional flow over the systolic peak (Colquhoun et al., 1992). Furthermore, a focal velocity >100 cm/s accompanied by disturbed flow can be suggestive of a stenosis (Sidhu, 2000). None of the participants in our study showed those characteristics. Images of colour Doppler and spectral waveforms of VA3 are shown in Figs. 1 and 2 respectively.

Vertebral artery injury linked to HVT usually occurs at the sites of fixation within the suboccipital portion (C1 transverse foramen, the groove on the superior aspect of C1 and at the dura intracranially) (Johnson et al., 2000). The intracranial portion is rarely injured from cervical HVT (Mechler, 1988; Pollanen et al., 1992). It has been suggested that VA blood flow changes associated with cervical rotation would be more accurately measured distal to the point of compromise, likely in the suboccipital or intracranial parts of the artery (Zaina et al., 2003; Mitchell, 2009). According to Bernoulli's Principle (Ganong, 2005), blood flow velocity at and immediately beyond the point of constriction of a vessel increases with either compression or stretching, so at the point of compromise velocity increases (Ganong, 2005). Constriction of the vessel may result in a "spurting" of blood or turbulent flow in the artery immediately distal to the point of distortion. This could present as a decrease in blood flow velocity a short distance distal to this point in the artery with blood flow usually returning to normal in the more distal parts of the vessel (Ganong, 2005). Therefore, best practice is thought to be to measure blood flow of the VA distal to these regions of disturbed haemodynamics, because the part of the VA measured may affect the outcomes (Mitchell, 2009). Logically, the risk of vessel dissection and/or VBI is thought to increase when the VA is subjected to tensile (stretching distortion) forces and/or compressive forces. Simultaneous measurement of flow during a manipulative procedure is most likely to capture haemodynamic changes.

These data suggest that in normal subjects the studied HVT technique does not appear to have an adverse effect on VA haemodynamics. This however, raises the question of what does predispose an individual to a vascular event leading to ischaemia. It remains unknown whether adverse haemodynamic responses would be recorded in the presence of vessel hypoplasia, vessel wall pathology (e.g. existing dissection) or inherent weakness. As such this study indirectly supports a stance taken by IFOMPT (Rushon et al., 2012) that careful subjective and objective risk assessment combined with prudent clinical reasoning may be the way to progress for clinicians.

While this study presents clinically relevant findings, some limitations exist. Neither the PT nor the RVT were blinded to the experimental condition, potentially introducing error. Further, duplex ultrasonography is not without limitations, including: 1) insonation angulation may falsely lead to a drop in diastolic flow (particularly in hypoplastic arteries), 2) shadowing and tortuosity which may lead to difficulty measuring lumen size, and can be highly technician dependent (Sakaguchi et al., 2003; Weintraub, 2003). To reduce the risk of error we maintained an angle of insonation 60° or less. Finally, this study was performed on normal subjects. The haemodynamic effects of a similar study on pathological vessels are unknown.

5. Conclusions

The findings of this study indicate that in normal subjects, peak systolic and end diastolic blood flow velocities of the VA are not affected by HVT to the atlanto-axial joint. Therefore, in apparently healthy vessels, HVT to the atlanto-axial joint does not appear to increase mechanical stress on the VA. It remains unknown whether adverse haemodynamic responses would be recorded in the

presence of vessel hypoplasia, vessel wall pathology or inherent weakness. Clinicians should retain an index of suspicion for potential vascular pathology as a presentation and conduct an appropriate risk assessment as suggested by IFOMPT (Rushton et al., 2012, 2013). Additional research should investigate latent changes in blood flow velocity as a slowly developing thrombus may not be immediately manifested through altered haemodynamics. Further, additional data suggesting a lack of effect of HVT techniques on blood flow can focus attention on alternative hypotheses such as a pathological predisposition to vessel injury.

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