The prevalence of orthostatic hypotension during physiotherapy treatment in patients with an acute spinal cord injury

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Study design: Prospective, observational.

Objectives: To investigate the prevalence of orthostatic hypotension (OH), its associated signs and symptoms, and the resulting limitation to treatment during initial physiotherapy treatments involving mobilisation for patients with an acute spinal cord injury (SCI).

Setting: Spinal Injuries Units, Royal Adelaide Hospital and Hampstead Rehabilitation Centre, Adelaide, South Australia.

Methods: Fourteen of 17 consecutive patients admitted with an acute SCI completed the study. Non-invasive measurement of systolic and diastolic blood pressure (BP), heart rate and percutaneous oxygen saturation were recorded during a 10-min baseline period with the patient resting in supine, and then at 1-min intervals for the first 10 min of physiotherapy treatments that involved mobilisation (ie sitting or standing). Following cessation of the treatment and return to the supine position, further measurements were made immediately and following 5 min of rest. Visual signs and patient reported symptoms of OH and any perceived limitation to therapy were also recorded. The study period comprised the first 10 physiotherapy treatments that involved mobilisation.

Results: BP changes indicative of OH occurred during 73.6% of mobilisation treatments. Of these treatments, accompanying signs and symptoms of OH were noted on 58.9% of occasions and were perceived as limiting treatment on 43.2% of occasions. Patients with tetraplegia had a higher prevalence of OH and a greater fall in BP than patients with paraplegia, irrespective of whether their lesion was complete or incomplete.

Conclusion: Blood pressure changes indicative of OH and accompanying signs and symptoms of OH are a common occurrence during the physiotherapy mobilisation of patients with an acute SCI.

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Introduction

Orthostatic hypotension (OH) refers to a significant decrease in blood pressure (BP) on assuming an upright posture. While the precise definition of OH is variable, the American Autonomic Society and American Academy of Neurology¹ achieved consensus in defining OH as a reduction in systolic BP of at least 20 mmHg or diastolic BP of at least 10 mmHg within 3 min of standing or being raised greater than 60° on a tilt table. Orthostatic hypotension may be symptomatic or asymptomatic, with symptoms including light-headedness, dizziness, blurred vision, ringing in the ears, nausea, fatigue, cognitive impairment, palpitations, tremulousness, headache and neck ache.¹

Orthostatic hypotension is described as being a common occurrence in patients who have sustained a spinal cord injury (SCI), as the injury to the spinal cord interferes with the normal transmission of the sympathetic nervous system messages which provide short term regulation of BP.^{2–4} As OH can restrict patients from fully participating in rehabilitation, it has the potential to delay the achievement of rehabilitation goals and to increase the length of hospitalisation.² There is also limited evidence that OH can lead to neurological deterioration in patients with a biomechanically stable SCI.⁵

Despite there being a general belief that OH is a common occurrence in the acute SCI patient population, no published studies specifically investigating the prevalence of OH and the resulting limitation on rehabilitation were found for patients with acute SCI. The aim of this study was to document the prevalence

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of OH during physiotherapy treatment involving mobilisation, its associated symptoms and signs, and the resulting limitation to treatment in patients presenting with an acute SCI.

Methods

Consecutive patients admitted to the Royal Adelaide Hospital Spinal Injuries Unit from May to November 1998 with an acute SCI who gave their written informed consent were eligible for inclusion in the study. Patients unwilling to participate or unable to understand written or spoken English were excluded. Patients transferred to another hospital (apart from Hampstead Rehabilitation Centre) prior to completion of data collection were withdrawn from the study. Patients that were transferred from the Spinal Injuries Unit at the acute hospital setting (Royal Adelaide Hospital) to the Spinal Injuries Unit at the rehabilitation setting (Hampstead Rehabilitation Centre) remained in the study.

Each patient's age, sex, past medical history, level of SCI, American Spinal Injury Association (ASIA) impairment scale level,⁶ additional injuries, spinal reflexes and medications were recorded from the casenotes. In addition, the number of days of bed rest prior to commencement of mobilisation was noted.

Once mobilisation was approved by medical staff, each patient was placed in the supine position. The patency of the urinary catheter was checked to avoid the possibility of bladder distension affecting BP.⁷ Anti-embolic stockings were worn by all patients throughout testing, and abdominal binders were used for patients with complete motor tetraplegia, as per the Royal Adelaide Hospital Spinal Injuries Unit protocol. If directed by medical staff, external spinal bracing was applied. The treatment procedure was explained to the patient prior to commencement of the treatment. As the study was observational, the frequency of treatment varied between patients according to their medical condition. Frequency of treatment ranged from once to twice a day for most patients and did not always occur on consecutive days.

Treatment procedure

The patient lay quietly in a supine position with the bed flat for 10 min while baseline resting data were obtained. The patient was then slowly brought to an upright sitting position on the edge of the bed with the assistance of one or two physiotherapists. Sitting balance, transfers, standing and/or gait re-education was then performed according to the individual patient's capabilities. This treatment continued for the length of time tolerated by the patient, as per usual clinical practice. At the cessation of treatment, the patient was returned to the supine position, with the bed flat, and kept resting in this position for 5 min while further data were recorded.

In separate treatment sessions, the physiotherapist performed stretching and range of movement exercises for patients, and assisted with strengthening exercises, with the patient positioned in supine. Blood pressure was not measured during these separate sessions nor during usual nursing care procedures such as showering and feeding.

Measurement procedure

Measurements recorded for the purpose of the study comprised systolic BP, diastolic BP, heart rate (HR) percutaneous oxygen saturation (SpO₂), the visual signs and patient reported symptoms of OH, and the perceived limitation of treatment due to OH. Measurements were made every 5 min during the initial 10-min baseline resting period and then at 1-min intervals for the first 10 min of treatment (or part thereof if the patient did not tolerate treatment for the full 10 min). Further measurements were taken immediately after cessation of treatment and resumption of the supine position, and then again 5 min later. Measurements were obtained for the first 10 treatments for each patient that involved mobilisation by physiotherapists.

The definition of OH by the American Autonomic Society and American Academy of Neurology¹ was modified for the purpose of this study. As patients with SCI are not necessarily able to stand and, in our Spinal Injuries Unit, do not routinely receive tilting on a tilt table, it was decided to document the occurrence of OH during the change in position from supine to either sitting or standing. Furthermore, rather than documenting OH for the first 3 min of attaining an upright position, this was extended to the first 10 min of treatment as it was anticipated from previous experience that delayed OH may occur in this patient population.⁸ The specific reduction in systolic and diastolic BP required to be classified as OH remained as described by the American Autonomic Society and American Academy of Neurology (ie a reduction in systolic BP of at least 20 mmHg or diastolic BP of at least 10 mmHg).¹

A calibrated DinamapTM 9720 monitor was set up by one of two physiotherapists not involved in treating the particular patient, in accordance to the manufacturer's recommendation, to measure systolic and diastolic BP, HR and SpO₂. To ensure a standard protocol for measuring these data, the brachial artery was palpated and the cuff was placed firmly around the patient's bare left arm. The lower end of the cuff was positioned approximately 2 cm above the cubital fossa. Care was taken to ensure that the correct sized cuff was selected for each patient, based on arm circumference, and this cuff was then used throughout data collection for that patient. The oximeter probe was placed on the patient's left ear lobe and secured in place with tape. A printer attached to the DinamapTM 9720 monitor recorded the measurements.

The treating physiotherapist documented the occurrence of signs of OH that were evident visually,

namely pallor, sweating and loss of consciousness, and questioned patients regularly about symptoms of OH (ie dizziness, light-headedness, nausea, visual changes, ringing or buzzing in the ears). In addition, the treating physiotherapist recorded occasions when she/he perceived OH as limiting treatment. The treating physiotherapist was unaware of the results of the measurements of BP, HR and SpO₂ throughout the study period.

To ensure that the measurements obtained with the DinamapTM 9720 monitor were accurate, systolic BP, diastolic BP and heart rate from the DinamapTM 9720 monitor were simultaneously compared to invasive intra-arterial BP monitoring and ECG recordings. Ten measurements were taken at approximately 1-min intervals on a sample of convenience, comprised of five patients in the Intensive Care Unit who had pre-existing intra-arterial BP monitoring. Intra-class correlation values (1,1) of at least 0.90 were found, indicating high consistency between the two types of monitoring.

Analyses

For the purpose of analysis, mean values for the entire group were calculated. In addition, individual patient data were examined in an attempt to identify features associated with the development of OH. Due to the small sample size, statistical analysis was not considered appropriate.

Results

Seventeen consecutive patients admitted to the Royal Adelaide Hospital Spinal Injuries Unit were eligible for inclusion in the study. Two patients were excluded from the study – both patients could not give informed consent due to a closed head injury for one patient and impaired cognitive ability for the second patient. A further patient was withdrawn from the study as he was transferred to another hospital prior to completion of data collection. Thus, 14 patients completed the study.

Descriptive data for the 14 patient that completed the study are presented in Table 1. As all patients were receiving numerous medications, these have not been listed in the Table, but they commonly included analgesia (morphine, Panadeine Forte or codeine phosphate) and aperients (Micalax and /or coloxyl with senna). Some patients also received medications such as steroids, antibiotics, bronchodilators and/or anticoagulants. Two patients (patient numbers 1 and 4) were discharged home after five mobilisation treatments, and for one patient (patient number 7) data collection was incomplete for one of the 10 treatments. The data from these patients have been included in analyses.

The mean values for systolic and diastolic BP for the 14 patients considered together, averaged over the treatments that comprised the study period, are shown

in Figure 1. As can be seen, the mean systolic BP was stable during the baseline rest period, fell during the mobilisation treatment period and returned to baseline levels soon after resumption of the resting position (Figure 1). The maximum fall in mean systolic BP (21.0 mmHg) during the treatment period from the mean of the three baseline readings, was sufficient to meet the criterion for OH. Mean diastolic BP also fell during the treatment period, however the maximum fall (7.2 mmHg) was not sufficient to be defined as OH. Mean HR was stable during the initial baseline period (73.8 beats per minute), increased by a maximum of 14.9 beats per minute during the treatment period and returned to near resting values on resumption of the supine position. The mean SpO₂ showed little change throughout the study period (mean value: 97.1% during baseline period, 96.6% during treatment period). For the sake of brevity, HR and SpO₂ data have not been presented

In order to simplify data presentation, all the variables collected for each subject during each treatment have not been presented. Instead, a summary of the data collected for individual patients is presented in Table 2. As can be seen, OH as defined by BP measurements occurred for all patients on at least one treatment occasion. Out of the total of 129 treatments where BP was measured, OH occurred during 95 treatments (73.6% of treatments). Of these 95 treatments, visual signs and patient reported symptoms of OH were also noted for 56 treatments (58.9%). Treatment was perceived as being limited due to OH on 43 of the total of 129 treatments (33.3%), which included 41 of the 95 treatments (43.2%) when BP changes indicative of OH were noted. On all occasions when treatment was perceived as being limited by OH, visual signs and symptoms of OH were present. On four occasions (three occasions for patient number 1 and once for patient number 14) visual signs and symptoms of OH were noted, but were not accompanied by a fall in BP indicative of OH. When OH occurred a fall in both systolic and diastolic BP was seen on 73.7% of occasions, with a fall in either systolic or diastolic BP (but not both) relatively evenly spread over the remaining occasions. Only two patients (patient numbers 1 and 4) demonstrated rebound hypertension (of more than 10.0 mmHg) when they were returned to the supine position at the completion of the treatment period.

To ascertain whether patients with tetraplegia were more prone to OH than patients with paraplegia, patients were allocated to one of two groups according to the level of their lesion. Any patient with a cervical spinal cord lesion was considered to have tetraplegia (10 patients) and the remaining patients (4 patients) were classified as having paraplegia. Figure 2 shows the mean BP for patients according to this classification. The patients with paraplegia had a small decrease in systolic BP, not sufficient to classify as OH, and a small increase in diastolic BP during treatment. Patients with tetraplegia however, showed a marked

Table 1 Descriptive data for the 14 patients completing the study

Patient number	Age (years) /Sex	Past medical history	Level of spinal cord injury	ASIA impairment scale	Presence of spinal reflexes	Additional injuries	Number of days of bed rest prior to mobilisation
1	38/M	Nil	C7	D	Yes	Nil	4
2	43/M	Nil	T12	A	No	Nil	10
3	46/F	Oesophagitis	T10	A	Yes	Haemothoraces, fractured acetabulum, mild CHI	45
4	18/M	Drug abuse	L3	D	Yes	Nil	4
5	16/M	Asthma	C5	A	Yes	Nil	24
6	44/M	Asthma	C5	D	Yes	Fractured ribs, pneumothorax liver haematoma, DVT	12
7	45/M	Nil	L2	B	No	Nil	13
8	32/F	Nil	C2	D	Yes	Fractured ribs, L1-3 clavicle, haemothorax, mild CHI	24
9	17/F	Asthma	C6	D	Yes	Perforated bowel	12
10	75/F	Cardiomegaly	C6	D	Yes	Fractured T3-6	3
11	30/M	Thalassemia	C5	A	Yes	Nil	20
12	36/M	Nil	C4	A	Yes	Nil	17
13	19/M	Nil	C5	B	No	Nil	17
14	27/M	Nil	C5	B	Yes	Nil	15

ASIA = American Spinal Injury Association; M = male; F = female; C = cervical; T = thoracic; L = lumbar; CHI = closed head injury; DVT = deep vein thrombosis.

ASIA impairment scale:⁶

A = complete (no motor or sensory function preserved in the sacral segments S4–S5);

B = incomplete (sensory but not motor function below the neurological level and extends through the segments S4–S5);

C = incomplete (motor function is preserved below the neurological level and the majority of key muscles below the neurological level have a muscle grade less than three);

D = incomplete (motor function is preserved below the neurological level and the majority of key muscles below the neurological level have a muscle grade greater or equal to three);

E = normal (motor and sensory function is normal)

Figure 1 Mean systolic and diastolic blood pressure for the 14 patients

fall in systolic BP, which was of sufficient magnitude to classify as OH, and a less marked fall in diastolic BP during treatment. As far as the prevalence of OH was concerned, OH occurred during 78 of the 95

Table 2 Summary of outcome data for the 14 patients completing the study

Patient number	1	2	3	4	5	6	7	8	9	10
1	SL	S	S	S		X	X	X	X	X
2										
3										
4						X	X	X	X	X
5	SL	SL	SL	SL	SL	SL	SL	SL	SL	SL
6	SL	SL	SL	S	S	SL	SL	SL		
7	SL*									
8										
9	SL	SL	SL	SL	SL	SL				
10	SL	SL								
11	SL	SL	SL	SL	SL	SL	SL	SL	SL	SL
12	S	S	S	S	SL	S	S			
13	SL	SL	S	S						
14	SL	SL	SL	SL		S	S	S	S	S

*BP data unavailable. S, visual signs and/or patient reported symptoms of OH. L, perceived limitation to treatment because of OH. Shaded areas represent treatments when OH was present as defined by BP changes. X, no treatment performed – patient discharged from hospital

treatments (82.1%) that the patients with tetraplegia received, compared to 17 of the 34 treatments (50.0%) that patients with paraplegia received. Visual signs and patient reported symptoms of OH were noted during

Figure 2 Mean systolic and diastolic blood pressure, comparing patients with tetraplegia ($n=10$) to those with paraplegia ($n=4$)

60 of the 95 treatments (63.2%) that patients with tetraplegia received, compared to one of the 34 treatments (2.9%) that patients with paraplegia received.

As there appeared to be an association between OH and tetraplegia, it was decided to further investigate the impact of the completeness of the lesion as classified by the ASIA score for the 10 patients with tetraplegia. Of the 10 patients with tetraplegia, three were classified as complete tetraplegia (ASIA A) and the remaining seven patients had incomplete tetraplegia (ASIA B or D). Decreases in systolic and diastolic BP were found for patients with both complete and incomplete tetraplegia, all of which were of a magnitude indicative of OH. However, the fall in BP appeared to be more rapid for the patients with complete tetraplegia.

The impact of the length of time in bed prior to commencement of mobilisation for patients with tetraplegia was also investigated. Patients with tetraplegia were divided into two groups depending on whether they stayed in bed for more or less than 14 days prior to commencement of mobilisation. Fourteen days was selected as the defining period of time because this length of bed rest has been shown to be associated with OH in normal subjects.⁹ Four patients with tetraplegia had less than 14 days bed rest prior to mobilisation (mean 7.8 days) and six patients with tetraplegia had more than 14 days bed rest prior to mobilisation (mean 19.5 days). From Figure 3, it can be seen that patients with tetraplegia who had more than 14 days bed rest showed decreases in both mean systolic and diastolic BP that were indicative of OH. The four patients with less than 14 days bed rest also showed a fall in mean

Figure 3 Mean systolic and diastolic blood pressure for the patients with tetraplegia, comparing those with less than 14 days bed rest ($n=4$) and more than 14 days bed rest ($n=6$) prior to commencement of mobilisation. RIB=rest in bed

systolic BP indicative of OH but only a slight fall in mean diastolic BP. Hence, patients with tetraplegia demonstrated a fall in BP indicative of OH, regardless of the length of bed rest prior to mobilisation.

Discussion

This study found an overall prevalence of OH, as defined by BP changes, of 73.6% over the 129 mobilisation treatments by physiotherapists that comprised the study period for 14 patients admitted to the Spinal Injuries Unit with an acute SCI. Visual signs and patient reported symptoms of OH accompanied these BP changes on 58.9% of occasions. Signs and symptoms were perceived as limiting treatment on 33.3% of the 129 mobilisation treatments. The prevalence of OH was greater for patients with tetraplegia compared to those with paraplegia, irrespective of whether the SCI lesion was complete or incomplete. The length of time that patients with tetraplegia spent in bed prior to commencement of mobilisation did not appear to have a major influence on the prevalence of OH.

The findings of this study confirm and extend those of Mathias *et al*^{8,10} and Kooner *et al*¹¹ who demonstrated similar substantial drops in mean BP for patients with complete chronic tetraplegia during tilting on a tilt bed. However, the rebound hypertension reported by Mathias *et al*⁸ was not seen for most of our patient sample. Further comparison is not appropriate, given that the patient sample in the present study had acute and varied levels of SCI and the positional changes used in the present study were different from those of Mathias *et al*^{8,10} and Kooner *et al*.¹¹

Although a higher rate of OH was anticipated during the initial treatments of the 10 treatments which comprised the study period, this was not the case, with the occurrence of OH evenly spread over the 10 treatments. This may be due to the progression of the mobilisation treatments over the study period increasingly challenging orthostatic responses.

When considering the association between BP changes and the visual signs and patient reported symptoms of OH, there were occasions when OH as defined by BP changes was detected, but signs and symptoms of OH were not present. The possible explanations for this include the possibility that cerebral perfusion was maintained despite a fall in systemic BP or that the modified definition of OH that was used for this study may have over-estimated the prevalence of OH. Additionally, it appeared that some patients with stoical personalities under-reported symptoms of OH. Another consideration is that during data collection it appeared that excessive movement of the patient's arm with the BP cuff attached, or isometric contraction of the upper limb muscles, resulted in large fluctuations of BP between readings. Thus, a series of BP readings may provide a more accurate indication of OH, rather than basing the diagnosis on a single BP reading.

As far as the accuracy of the visual signs and patient reported symptoms of OH is concerned, there were four occasions (three times for patient number 1 and once for patient number 14) when signs and symptoms of OH were noted, but a fall in BP indicative of OH was not seen. Patient number 1 demonstrated a high level of anxiety during treatment which may have contributed to his signs and symptoms. Although it cannot be stated with certainty from the results of this study, the visual signs and patient reported symptoms of OH appeared to offer experienced clinicians an acceptable means of monitoring the occurrence of OH during physiotherapy treatment. It should also be noted that although the treating physiotherapist was unaware of the reading from the BP monitor, no patients lost consciousness, suggesting that the termination of treatment based on visual signs and patient reported symptoms of OH may be sufficient to avoid syncope.

Based on the results of this study, it is recommended that physiotherapists carefully and frequently monitor visual signs and patient reported symptoms of OH during treatments that involve positional change from supine to an upright position for patients with an acute SCI. The routine use of non-invasive BP monitoring may provide additional information, but may also generate spurious readings on a moment to moment basis due to excessive movement or isometric contraction of the upper limbs. As expected, while heart rate increased during mobilisation for all patients, it did not reflect the occurrence of OH. The changes in SpO₂, recorded using an ear probe, were of a small magnitude and did not provide any additional information about OH during mobilisation treatment.

Further study is needed to further identify characteristics that increase the risk of patients with acute SCI developing OH. Additionally, it would be of interest to monitor cerebral blood flow as well as systemic BP during mobilisation to clarify why some patients demonstrated falls in BP without signs and symptoms of OH. The effect of OH on the neurological status of acute SCI patients could also be investigated in future studies. Longitudinal studies may help in the identification of the time required for these patients to accommodate to orthostatic stresses. The most effective means of management of OH also needs to be studied in order to minimise the disruption to rehabilitation that may result from OH.

Conclusion

Orthostatic hypotension as defined by BP changes occurred during 73.6% of mobilisation treatments for 14 patients who had sustained an acute SCI. Visual signs and patient reported symptoms of OH were often, but not always, associated with a fall in BP. Of all the treatments that comprised the study period, signs and symptoms of OH were perceived as limiting treatment on 33.3% of occasions. For this sample of patients, the prevalence of OH was higher and the fall in BP was more severe for patients with tetraplegia, regardless of whether the lesion was complete or incomplete and the number of days of bed rest prior to mobilisation.

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