ORIGINAL RESEARCH

# Effect of Spinal Immobilization on Heart Rate, Blood Pressure and Respiratory Rate

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**Abbreviations:**
ATLS: Advanced Trauma Life Support
ED: Emergency Department
HR: Heart rate
RR: Respiratory rate
SNS: Sympathetic nervous system
SBP: Systolic blood pressure
VAS: Visual analog scale

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## Abstract

**Introduction:** Vital signs remain important clinical indicators in the management of trauma. Tissue injury and ischemia cause tachycardia and hypertension, which are mediated via the sympathetic nervous system (SNS). Spinal immobilization is known to cause discomfort, and it is not known how this might influence the SNS and contribute to abnormal vital signs.

**Hypothesis:** This study aimed to establish whether the pain and discomfort associated with spinal immobilization and the maneuvers commonly used in injured patients (eg, log roll) affect the Heart rate (HR), Systolic Blood Pressure (SBP) and Respiratory rate (RR). The null hypothesis was that there are no effects.

**Methods:** A prospective, unblinded, repeated-measure study of 53 healthy subjects was used to test the null hypothesis. Heart rate, BP and RR were measured at rest (five minutes), after spinal immobilization (10 minutes), following log roll, with partial immobilization (10 minutes) and again at rest (five minutes). A visual analog scale (VAS) for both pain and discomfort were also collected at each stage. Results were statistically compared.

**Results:** Pain VAS increased significantly during spinal immobilization (3.8 mm, $P < .01$). Discomfort VAS increased significantly during spinal immobilization, after log roll and during partial immobilization (17.7 mm, 5.8 mm and 8.9 mm, respectively; $P < .001$). Vital signs however, showed no clinically relevant changes.

**Discussion:** Spinal immobilization does not cause a change in vital signs despite a significant increase in pain and discomfort. Since no relationship appears to exist between immobilization and abnormal vital signs, abnormal vital signs in a clinical situation should not be considered to be the result of immobilization. Likewise, pain and discomfort in immobilized patients should not be disregarded due to lack of changes in vital signs.

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## Introduction

In the management of the trauma patient, vital signs are considered important clinical indicators for the clinician as evidenced by their inclusion in trauma team activation criteria, and much emphasis is placed on them in current trauma training.[1,2] Despite evidence challenging the usefulness of vital signs in the trauma setting, their measurement still remains a core component of early trauma care, when little objective information (other than perhaps the visible injury or mechanism) is known.[3]

Both tissue injury and ischemia cause tachycardia and hypertension mediated via the sympathetic nervous system (SNS) in a manner similar to that of the fight-or-flight reaction.[4] The response to tissue injury attenuates the cardiovascular response to hemorrhage through a reduction in sensitivity of the baroreceptor and depressor reflexes.[4] The result is that relative cardiovascular stability is maintained longer; however, for clinicians this makes hemorrhage more difficult to diagnose when injury is present.[4] Pain only attenuates the vagal component of the baroreceptor reflex, leaving the sympathetic component intact.[5] Sympathetic nervous system activation may therefore result in measurable tachycardia, tachypnea, and hypertension, although these are not usually seen unless pain intensity and duration is sufficiently high.[6,7] The exact details of this relationship remain unclear, but it is possible that sufficient pain may, like injury, obscure the cardiovascular response to hemorrhage,[8] making clinical detection of its presence more difficult.

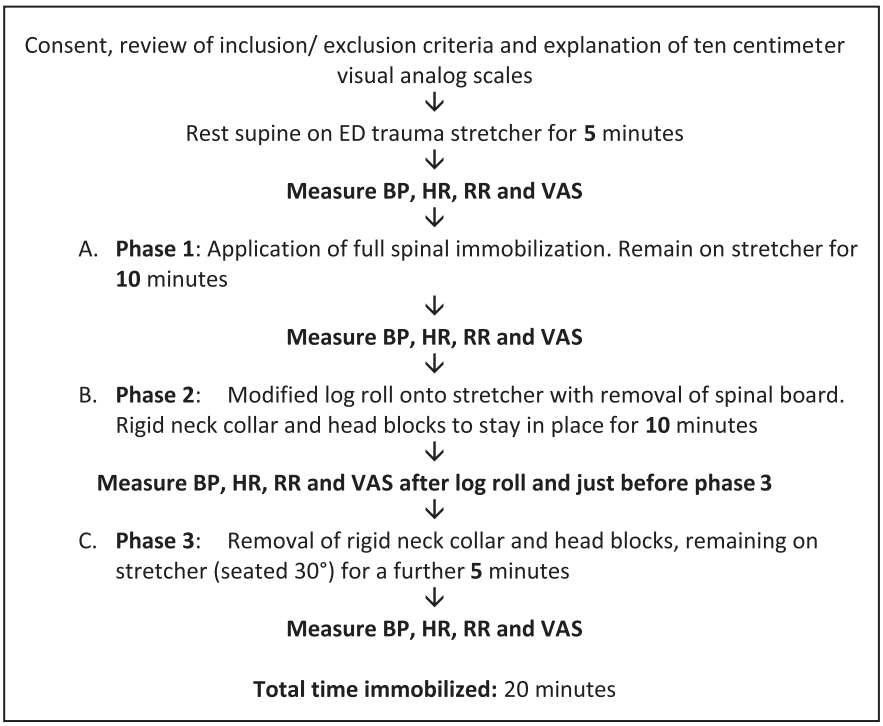


**Figure 1.** Strategy Employed to Collect Data from Healthy Volunteers

Spine immobilization using a firm cervical collar, head restraints and a spinal board is advocated by the Advanced Trauma Life Support (ATLS) course material as a precaution against causing or worsening a spinal cord injury in an injured or potentially injured person.[2] This inevitably results in pain and discomfort.[9-13] Kwan et al reviewed 17 randomized controlled trials (RCTs) in order to evaluate the effect of spinal immobilization on healthy subjects.[9] Of these RCTs, four used pain and another four used discomfort as an outcome measure. All the studies showed that subjects reported a significant increase in pain or discomfort when immobilized.[9] A thorough search of current literature databases (including the British Nursing Index, EMBASE, CINAHL, MEDLINE and Google Scholar) was undertaken, searching for papers that describe an association between vital signs and spinal immobilization. This search revealed five papers of varying quality, describing respiratory restriction associated with spinal immobilization using various devices, though none of these papers included comments on the respiratory rate (RR).[9,14-17] No literature could be found describing the effect of spinal immobilization on heart rate (HR) or systolic blood pressure (SBP).

Given the paucity of literature, it is not known whether sustained pain and discomfort caused by spinal immobilization may be a sufficient stimulus to affect a patient's vital signs. If so, this effect will need to be taken into account during evaluation of vital signs in the injured, spinal immobilized patient, as immobilization may—like tissue injury— make detection of hemorrhage difficult. This study aimed to establish whether the pain and discomfort associated with spinal immobilization and the maneuvers commonly used in injured patients (eg, log roll) affect the HR, BP and RR. The null hypothesis was that there are no effects.

## Methods

A prospective, unblinded, repeated-measure study was used to test the null hypothesis. To power the study to 80% ($\alpha = 0.05$), 52 subjects were required to reject the null hypothesis if the mean differences in HR, RR and SBP were 10 beats per minute, 2.5 breaths per minute and 7.5 mm Hg, respectively (a priori consensus among authors). Uninjured, healthy, adult volunteers were recruited from staff in Derriford Hospital's Emergency Department (ED) (Plymouth, UK) and from paramedic students at Plymouth University. Subjects were excluded from participation if they: (1) had known cardiovascular or respiratory disease; (2) were taking any medications known to affect the heart rate (eg, sympathomimetics or antihypertensive medication); (3) were pregnant; (4) suffered with back problems (including previous back surgery); or (5) developed a symptomatic bradycardia (pulse <60), tachycardia (pulse >120), hypotension (SBP <90) or any hypertension (SBP >180) before or during data collection. Informed consent was obtained from all participants. The study received ethical approval through the NHS South West 1 Research Ethics Committee (REC) (10/H0203/25) and University of Cape Town REC (014/2010).

Outcomes measured were the resting HR, BP, RR, pain VAS and discomfort VAS; these were compared with values 10 minutes after full spinal immobilization (Phase 1), following the log roll (Phase 2), 10 minutes after removal of spinal board (Phase 3), and final resting values. Clinically relevant outcome measures (a priori determined) were mean differences for HR, RR and SBP of ≥ 10 beats per minute, 2.5 breaths per minute and 7.5 mm Hg, respectively. The strategy employed for data collection is shown in Figure 1.

The first author collected all the data, and as subjects were uninjured, performed a modified log roll in Phase 2 of the study.



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| | | | | Range | | 95% CI | |
|---|---|---|---|---|---|---|---|
| | Mean | Median | SD | Lower | Upper | Lower | Upper |
| Age | 37 | 35 | 13 | 18 | 62 | 33.55 | 40.49 |
| Age Males (n = 11) | 35 | 36 | 10 | 20 | 51 | 28.41 | 41.59 |
| Age females (n = 42) | 38 | 35 | 13 | 18 | 62 | 33.41 | 41.69 |
| **SBP at rest (mmHg)** | **114** | **114** | **13** | **84** | **151** | **110.48** | **117.41** |
| SBP fully immobilized | 115 | 112 | 13 | 94 | 150 | 110.82 | 118.16 |
| SBP after log roll | 114 | 114 | 12 | 94 | 142 | 110.65 | 117.31 |
| SBP partial immobilized | 112 | 111 | 12 | 90 | 140 | 108.35 | 114.74 |
| SBP semi seated | 113 | 113 | 12 | 91 | 145 | 110.21 | 116.65 |
| **HR at rest (beats/min)** | **66** | **66** | **11** | **45** | **93** | **62.38** | **68.60** |
| HR fully immobilized | 64 | 62 | 10 | 46 | 83 | 61.10 | 66.37 |
| HR after log roll | 63 | 62 | 10 | 41 | 85 | 60.08 | 65.35 |
| HR partial immobilized | 62 | 64 | 10 | 45 | 88 | 59.76 | 65.10 |
| HR semi seated | 63 | 62 | 9 | 43 | 81 | 60.02 | 65.00 |
| **RR at rest (breaths/min)** | **14** | **14** | **4** | **8** | **22** | **13.01** | **14.99** |
| RR fully immobilized | 14 | 14 | 4 | 8 | 22 | 13.15 | 15.23 |
| RR after log roll | 14 | 14 | 4 | 8 | 24 | 13.13 | 15.36 |
| RR partial immobilized | 14 | 14 | 4 | 8 | 24 | 12.58 | 14.59 |
| RR semi seated | 13 | 14 | 3 | 8 | 20 | 12.41 | 14.16 |
| **Pain VAS at rest (mm)** | **0.35** | **0** | **2.45** | **0** | **18** | **-0.32** | **1.03** |
| Pain VAS fully immobilized | 3.81 | 0 | 9.36 | 0 | 42 | 1.23 | 6.39 |
| Pain VAS after log roll | 0.53 | 0 | 2.54 | 0 | 17 | -0.17 | 1.23 |
| Pain VAS partial immobilized | 1.45 | 0 | 5.48 | 0 | 31 | -0.06 | 2.96 |
| Pain VAS semi seated | 0.13 | 0 | 0.94 | 0 | 7 | -0.13 | 0.39 |
| **Discomfort VAS at rest (mm)** | **0.53** | **0** | **2.90** | **0** | **21** | **-0.27** | **1.33** |
| Discomfort VAS fully immobilized | 17.68 | 15.07 | 17.37 | 0 | 75 | 12.89 | 22.47 |
| Discomfort VAS after log roll | 5.84 | 2.05 | 9.18 | 0 | 46 | 3.31 | 8.37 |
| Discomfort VAS partial immobilized | 8.90 | 2.05 | 17.32 | 0 | 89 | 4.13 | 13.68 |
| Discomfort VAS semi seated | 0.13 | 0 | 0.94 | 0 | 7 | -0.13 | 0.39 |



**Table 1**. Descriptive Statistics of Age and Data Sets (control group in bold)
Abbreviations: SBP, systolic blood pressure; SD, standard deviation; RR, respiratory rate; VAS, visual analog scale

Heart rate and SBP were obtained using the same manual, electronic sphygmomanometer for all subjects, and RR was manually counted over a minute. Full spinal immobilization consisted of a correctly-sized rigid neck collar, rigid spinal board and head blocks. Discomfort and pain were measured separately using a 100-point visual analog scale (VAS). An evaluation of discomfort also was made using a 100-point VAS. This scale has been used previously in spinal immobilization literature as a subjective measure of tissue ischemia.[13,18]

Data were analyzed using SPSS Statistics version 19 (IBM, Armonk, New York USA). Mean, median, standard deviation (SD), range and 95% confidence intervals (CIs) were used to describe the data sets. Friedman's analysis of variance (ANOVA) was used to evaluate SBP, HR, RR, pain VAS and discomfort VAS. A Wilcoxon signed-rank test (with a Bonferroni correction to control for the family-wise error) was used for post-hoc testing for pain VAS and discomfort VAS.[19] Effect size was determined using Pearson's correlation coefficient (r).[19] The correlation coefficient (r) is a useful test, not only to measure strength of a relationship, but also to measure the strength of an experimental effect between two variables (r = 0.10, 0.30 and 0.50 denote small, medium and large effects respectively).[19] Statistical tests were two-sided and a $P < .05$ was deemed statistically significant (with the exception of the Bonferroni correction where $P < .01$ was used to indicate significance).[19] Finally, data were transformed to z-scores to allow expression of values in SD units, thus allowing direct comparison among the vital signs, pain VAS and discomfort VAS data sets.[19]

## Results

Data were collected from 53 subjects (11 male) and there were no missing data points.

Friedman's ANOVA for SBP, HR and RR showed statistically significant differences within their respective data sets ($P < .05$, .01 and .01 respectively), but when compared to outcome measures, these differences were not clinically relevant (Table 1). The pain and discomfort VAS also showed statistically significant differences within their respective data sets (Friedman's ANOVA, $P < .001$ for both). Outcome measures were not set for the pain and discomfort VAS; these were further evaluated using the Wilcoxon signed-rank test and effect size (r) measurement, where post-hoc analysis revealed a significant mean difference. The mean differences among discomfort at rest, discomfort with spinal immobilization, logroll, and partial immobilization



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| | Z | P | r |
|---|---|---|---|
| Pain VAS **at rest** compared to pain VAS: | | | |
| Full immobilization | −3.01 | .003 | −0.29 (small effect) |
| Discomfort VAS **at rest** compared to discomfort VAS: | | | |
| Full immobilization | −5.97 | <.001 | −0.58 (large effect) |
| After log roll | −4.08 | <.001 | −0.40 (moderate effect) |
| Partial immobilized | −4.32 | <.001 | −0.42 (moderate effect) |



**Table 2**. Significant Findings When *At Rest* Control Group Mean Compared to Other Group Means

significant ($P < .001$ each) and effect sizes were −0.58, −0.4 and −0.42 respectively (Table 2). The mean difference between pain at rest and pain with spinal immobilization was the only significant finding on post-hoc analysis ($P = .003$) although the effect was small (0.13).

Figure 2 (supplementary file online) gives a graphical interpretation of the data sets using z-scores (values can be found in Table 4, supplementary file online). These show the significant variation (95% error bars) in the discomfort VAS despite insignificant changes in SBP, HR and RR.

## Discussion

This study is the first in a series of studies to evaluate the physiological effects that confound the prognostic inferences of vital signs in injury. Despite a significant increase in discomfort (moderate to high effect) and pain (small effect), the volunteers' SBP, HR and RR did not show any clinically relevant changes. The z-scores in Figure 2 (online) allow cross-comparison of groups (as demonstrated by the 95% CI error bars) and show that, despite a significant increase in pain and discomfort (also described in Table 2), changes in SBP, HR and RR remained clinically irrelevant. This finding appears to be similar to previous reports on the effect of acute pain observed both in the ED and the prehospital environment.[20-22] These papers have without exception shown that—at least where acute injury is concerned—pain and vital signs showed no meaningful clinical correlation. As described in the introduction, the relationship between acute pain and the autonomic system has not been definitively described, and variability exists as to when a painful stimulus will result in a significant SNS response.[8]

One way to look at this is to consider the mechanism by which pain is induced (Table 3). Pain stimulated in the absence of tissue damage can be seen as physiological (protective or warning), whereas pain stimulated in the presence of tissue damage is pathological, as tissue injury has already occurred.[23] The specific course of the pain is also important (Table 3): phasic pain describes a short duration, high intensity pain, usually as trauma or tissue injury occurs. Acute pain following trauma or tissue injury not only has a phasic component but usually also a tonic component that continues at a lower intensity which can last for hours to days.[23] In spinal immobilization, discomfort and pain worsen with full immobilization, and are reduced when restraints are relaxed or removed (Figure 2, online). In this study there was no phasic component, a relatively low tonic component of pain (median pain VAS = 0), and discomfort (a proxy for tissue ischemia) after ten minutes reached only a median VAS of 15 (out of 100). This appears not to be sufficient to cause a clinically detectable SNS response, and it would seem that a more intense or prolonged stimulus (or both) would be required for the SNS effect to become clinically relevant.[23]

| **Mechanism of pain** |
|---|
| • Physiological pain: painful stimulus in the absence of tissue damage;<br>• Pathological pain: painful stimulus in the presence of tissue damage. |
| **Course of pain** |
| • Phasic pain: short duration, high intensity pain usually at the time tissue injury occurs (eg, phlebotomy);<br>• Acute pain: includes the phasic component and then a tonic phase where pain resolves over hours to days (eg, sprained ankle);<br>• Chronic pain: persists for longer than what is normally expected for healing and recovery (eg, complex regional pain syndrome). |



**Table 3**. Definitions Relating to the Course and Mechanism of Pain[23]

It is thus important to consider other causes of abnormal vital signs, such as hemorrhage, head or spinal injury, associated medical problems or medications.[2] The relationship between hemorrhage and abnormal vital signs has been well described, but even here it should be noted that normal vital signs are not unusual in some cases, despite significant hemorrhage.[2,3] Anxiety, too, may increase serum catecholamine and cortisol levels and decrease baroreflex sensitivity, resulting in abnormal vital signs.[24,25]

## Limitations

There are a few limitations to this study which are important to note. The study took place outside the true clinical setting. The study protocol allowed for a relaxed environment, employing a procedure all participants were familiar with. This was purposeful in order to reduce the possible confounding effect of anxiety. This study was not powered to allow subgroup analysis of gender differences that may have applied, and it is possible that the female predominance may have affected results. It is also possible that a longer period of spinal immobilization would have resulted in abnormalities. However, in the review of randomized controlled trials on the effects of spinal immobilization on subjects, Kwan et al[9] referenced testing times of 10 minutes in three of the 10 studies reviewed. The current study was based on this figure, though in fact the immobilization lasted for 20 minutes (10 minutes fully immobilized and 10 minutes partially immobilized). In planning the study protocol, the authors felt that 10 minutes was a safe duration of exposure to a rigid spinal board. Given the low pain and discomfort scores



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observed following 10 minutes of spinal immobilization, it is questionable whether further study is required to evaluate this. The subjects were uninjured and it is possible that the addition of discomfort from spinal immobilization to that already being suffered as a result of an injury might have more physiological effect than in an uninjured person.

## Conclusion

Health care professionals working in the ED or prehospital environment should be aware that spinal immobilization does not contribute significantly to physiological derangements of SBP, HR and RR. It would follow that when physiological derangements are present, these should be considered to be due to another cause. Likewise it is important to note that since pain and discomfort reported by patients with spinal immobilization do not correlate with vital signs, abnormal values should not be considered a prerequisite for the appropriate treatment of pain or discomfort.

## Supplementary Material

To view supplementary material for this article, please visit http://dx.doi.org/10.1017/S1049023X13000034.


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