The effects of positioning after stroke on physiological homeostasis: a review

SIR—There is increasing evidence that several abnormal physiological parameters (pyrexia, hyperglycaemia, hypotension, hypoxia) post stroke are associated with poor outcome [1] and strategies are now being used to maintain physiological homeostasis, although randomised controlled trials are required to measure their effectiveness [2, 3]. Other physiological parameters such as oxygenation, blood pressure, cerebral blood flow and intracranial pressure may be potentially modified by changes in body position after stroke, some beneficial and others not [4, 5]. Normally, assuming an upright position leads to transient hypotension which is compensated by an increase in heart rate and cerebral vasodilatation, thus maintaining cerebral perfusion [6]. After acute stroke, cerebral autoregulation is impaired, thereby risking cerebral hyperperfusion upon standing [7]. Whether sitting out of bed within 24 hours of stroke as part of an early rehabilitation programme reduces early neurological deterioration, because of a complex interaction between improved cerebral perfusion pressure, reduced intracranial pressure and improved oxygenation, is unclear at present [8]. Before evidence-based recommendations can be made on positioning in the acute phase of stroke (within 24 hours to 7 days), information is required about its effects on physiological homeostasis, which may have prognostic significance.

A systematic literature search was therefore undertaken to find clinical studies investigating the effects of different body positions on physiological homeostasis during the first week after stroke.
Methods

A systematic search of Medline/PubMed databases, as well as the Cochrane Database of Systematic Reviews from 1966 to 2003 was undertaken entailing the terms ‘body position’, ‘posture’, ‘oxygen’, ‘cerebral perfusion’, ‘cerebral blood flow’, ‘cerebral artery velocity’, ‘intracranial pressure’ and ‘blood pressure’. We combined these items with the term ‘acute stroke’. In addition, the search strategy included hand searching of reference lists, bibliographies of retrieved papers and contact with authors as suggested by the MOOSE Group [9]. The list of studies identified by the search was independently assessed by two reviewers (AB, VMP) to find those studies which met the inclusion criteria for this review. For further details of the methods used, please see Appendix I in the supplementary data on the journal website (www.ageing.oupjournals.org).

Results

We identified 28 studies on the effects of body positioning on oxygenation, blood pressure, cerebral perfusion, cerebral artery velocity and intracranial pressure during the first week of stroke [4, 10–35]. Four studies only described single case reports [17–20], ten studies examined blood pressure change after 1 week of stroke [24–33] and four studies described cerebral blood flow in patients in the chronic phase of stroke [21–24]. Ten studies met the pre-determined criteria for inclusion in the review [4, 10–16, 34, 35]. Table 1 summarises the univariate associations between the effects of positioning on physiological parameters after stroke.

Oxygenation

Four studies were described [4, 10–12]. Elizabeth and colleagues [4] demonstrated that mean oxygen saturation levels were higher in stroke patients managed in the semi-recumbent position (93.2% versus 91.9%) than those supine. Rowat and colleagues [12] suggested that if patients could tolerate sitting in a chair, this was the optimal position to maintain the highest mean oxygen saturation (≥96%) within 72 hours of stroke, although a small proportion of patients (18%) desaturated when in this position. Pang and colleagues [10] and Chatterton and colleagues [11] demonstrated no significant changes in oxygenation with different body positions within 48 and 72 hours, respectively, of acute stroke.

Blood pressure

Four studies were described [13, 14, 34, 35]. Panayiotou and colleagues [13] demonstrated significant increases in blood pressure in mild to moderate stroke patients when managed supine for 10 minutes and then either sitting or standing for 5 minutes. The incidence of sustained postural hypotension was <10%. Panayiotou and colleagues [34] also demonstrated no significant falls in mean arterial blood pressure and heart rate in stroke patients specifically taking anti-hypertensive therapy after sitting or standing up. Schwarz and colleagues [14] demonstrated significant falls in mean arterial blood pressure with early head elevation (15–30°) from supine (90 ± 1.6 mmHg to 76.1 ± 1.6 mmHg) in 18 patients within 6 days of ischaemic stroke. Asperg [35] demonstrated in a controlled study (non-randomised) that regular early standing up within 48 hours of stroke did not lead to significant orthostatic hypotension (tilting at 70°) at 1 week but was associated with a lower proportion of severely disabled patients at 1 week compared with a control group (no regular standing practice).

Cerebral perfusion and cerebral artery velocity

Three studies were identified [14–16]. Jack and colleagues [15], using single photon emission tomography, showed improved regional cerebral perfusion in the semi-recumbent position (30–45°) compared with supine. Schwarz and colleagues [14], however, demonstrated significant falls in middle cerebral artery velocity and cerebral perfusion pressure with early head elevation (30°) from supine in 18 patients within 6 days of ischaemic stroke. Wojner and colleagues [16] showed that middle cerebral artery velocity was significantly higher by 13.1% in 11 patients who were managed in the supine position compared with the semi-recumbent position within 48 hours of ischaemic stroke.

Intracranial pressure

Schwarz and colleagues [14] demonstrated that intracranial pressure decreased from 13.0 ± 0.9 mmHg to 12.0 ± 0.9 mmHg at 15° and to 11.4 ± 0.9 mmHg at 30° backrest elevation in 18 patients with ischaemic stroke.

Discussion

The research evidence available does not enable us to answer the following clinical questions due to lack of randomised controlled trial data. Should we manage all stroke patients initially in the semi-recumbent position or sitting up? Which patients should be placed in which positions, for how long and at what time after stroke? Studies included in this search were observational in nature, including a narrative description of each study with limited data on outcome. Limitations include number of patients studied, lack of data on stroke type, lack of multivariate analyses and the heterogeneity of the populations studied.

Studies measuring the effects of body position on oxygen saturation levels differed in stroke severity, variations in positions, duration of oxygen saturation measurements and intervals between stroke onset and oxygen saturation measurement. However, there was some evidence that patients nursed in a sitting position or propped up in bed had higher oxygen saturation levels than those in supine positions, although desaturation occasionally occurred in these positions, throwing into question the adoption of this position for all patients in routine practice. The short-term changes in oxygen saturation described in these observational studies may not necessarily reflect what is seen in clinical practice where positional changes are adopted over a long period of time (2–4 hours) [36]. Nursing and therapy practice occasionally advocates the practice of side lying on the affected side [37]; however, potentially this could lead to further hypoxia resulting from increased blood flow to the
Table 1. Effects of positioning on physiological parameters in acute stroke

<table>
<thead>
<tr>
<th>Variable</th>
<th>Positional intervention</th>
<th>No.</th>
<th>Mean age</th>
<th>Stroke type</th>
<th>Timing of intervention</th>
<th>Duration of each position</th>
<th>Stroke severity</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen</td>
<td>Supine, sitting, paretic and non-paretic side lying</td>
<td>20</td>
<td>–</td>
<td>–</td>
<td>&lt;48 hours</td>
<td>20 minutes</td>
<td>MRC grade ≤2</td>
<td>O₂ saturation &gt;90% for all positions</td>
</tr>
<tr>
<td>Oxygen</td>
<td>Supine to semi-recumbent (45°)</td>
<td>19</td>
<td>80</td>
<td>–</td>
<td>&lt;48 hours</td>
<td>One hour</td>
<td>MRC grade ≤2</td>
<td>O₂ saturation on supine 91.9% versus semi-recumbent 93.2%**</td>
</tr>
<tr>
<td>Oxygen</td>
<td>Sitting (70°), sitting in chair, right and left side lying (45°)</td>
<td>24</td>
<td>68.2</td>
<td>83% CI</td>
<td>&lt;72 hours</td>
<td>15 minutes</td>
<td>ESS median 57.5 range (0–100)</td>
<td>Mean arterial O₂ &gt;95% for all positions</td>
</tr>
<tr>
<td>Oxygen</td>
<td>Sitting in chair, propped in bed, supine lying, paretic and non-paretic side, right and left side lying</td>
<td>129</td>
<td>72</td>
<td>TACS 35% PACS 36% LACS 22% POCS 5% Unclassed 2%</td>
<td>&lt;72 hours (median)</td>
<td>10 minutes</td>
<td>MRC grade ≤2 (50% patients)</td>
<td>O₂ saturation ≥96% on sitting in chair compared to other positions*</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Supine to sitting and standing</td>
<td>40</td>
<td>76</td>
<td>45% CI</td>
<td>&lt;72 hours</td>
<td>5 minutes sitting and standing</td>
<td>CNSS median 90 range (70–105)</td>
<td>Increase change in DBP from supine to sitting (5 ± 7 mmHg)***</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Supine to sitting and standing</td>
<td>40</td>
<td>74</td>
<td>100% CI</td>
<td>&lt;72 hours</td>
<td>5 minutes sitting and standing</td>
<td>CNSS median 90 range (65–115)</td>
<td>Increase in MABP from supine to standing (30 ± 9 mmHg)*</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Sit to stand hourly for 12 hours each day for a week (trial group)</td>
<td>30</td>
<td>45–86</td>
<td>70% CI</td>
<td>&lt;48 hours</td>
<td>Tilting at 70° from supine for 6 minutes at one week</td>
<td>10% incontinent 66% hemiparesis 17% dysphasia</td>
<td>During tilting, fall in SBP of 8 mmHg in trial group versus 19 mmHg in control group. At one week, severe disabled patients (Katz’ Index, F and G), trial group 20% versus 52% control group***</td>
</tr>
</tbody>
</table>
### Table 1. continued

<table>
<thead>
<tr>
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<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure</td>
<td>Supine to head elevation (15–30°)</td>
<td>18</td>
<td>61.2</td>
<td>100% CI</td>
<td>&lt;6 days</td>
<td>5 minutes</td>
<td>&gt;2/3 middle cerebral artery ischaemic stroke</td>
<td>Reduction in blood pressure on head elevation (90 ± 1.6 mmHg to 82.7 ± 1.7 to 76.1 ± 1.6 mmHg)****</td>
</tr>
<tr>
<td>Cerebral perfusion</td>
<td>Supine to semi-recumbent (30–45°)</td>
<td>9</td>
<td>–</td>
<td>100% CI</td>
<td>&lt;48 hours</td>
<td>Not presented</td>
<td>Partial anterior circulatory stroke</td>
<td>Increase in cerebral perfusion on semi-recumbency (&gt;50% change)</td>
</tr>
<tr>
<td>Cerebral blood flow</td>
<td>Supine to head elevation (30°)</td>
<td>11</td>
<td>60</td>
<td>100% CI</td>
<td>&lt;48 hours</td>
<td>2 minutes</td>
<td>NIHHS median 8.7 range (4–20)</td>
<td>Increase change in cerebral blood flow from head elevation (51 ± 17.5 cm/s) to supine (58 ± 19.3 cm/s)*</td>
</tr>
<tr>
<td>Cerebral blood flow</td>
<td>Supine to head elevation (15–30°)</td>
<td>18</td>
<td>61.2</td>
<td>100% CI</td>
<td>&lt;6 days</td>
<td>5 minutes</td>
<td>&gt;2/3 middle cerebral artery ischaemic stroke</td>
<td>Reduction in cerebral blood flow on head elevation (72.87 ± 11.3 cm/s to 67.2 ± 9.7 cm/s to 61.2 ± 8.9 cm/s)****</td>
</tr>
<tr>
<td>Intracranial pressure</td>
<td>Supine to head elevation (15–30°)</td>
<td>18</td>
<td>61.2</td>
<td>100% CI</td>
<td>&lt;6 days</td>
<td>5 minutes</td>
<td>&gt;2/3 middle cerebral artery ischaemic stroke</td>
<td>Reduction in intracranial pressure on head elevation (1.6 ± 0.3 mmHg)</td>
</tr>
</tbody>
</table>

*P < 0.05, **P < 0.03, ***P < 0.001, ****P < 0.0001. DBP, diastolic blood pressure; MABP, mean arterial blood pressure; MRC, Medical Research Council; ESS, European Stroke Scale; CNSS, Canadian Neurological Stroke Scale; NIHHS, National Institute of Health Stroke Scale; TACS, total anterior circulatory stroke; PACS, partial anterior circulatory stroke; LACS, lacunar stroke; POCS, posterior circulatory stroke; CI, cerebral infarction; PICH, primary intracerebral haemorrhage.
dependent lung, thus aggravating intra-pulmonary shunting [38]. Whether sitting stroke patients up immediately to improve their oxygen saturation will improve short-term neurological recovery is unknown and requires further evaluation. Larger studies should address the effects of different positions on oxygenation within the first 24 hours of stroke when the ischemic penumbra is potentially salvageable as well in other patients with co-existing cardio-respiratory disease.

The evidence that positional change after acute stroke caused orthostatic hypotension was mixed and was confounded by stroke severity and the positions used. In addition, continuous blood pressure monitoring was not used and therefore beat-to-beat haemodynamic measures were not carried out. Ischaemic strokes as well as older people have been shown to be associated with impairment of orthostatic blood pressure control due to blunting of baroreflex sensitivity resulting from sympathetic nervous system dysfunctions [7]. Orthostatic blood pressure responses after acute stroke might give rise to significant reductions in cerebral perfusion, resulting in neurological deterioration [24]. Whether patients with orthostatic hypotension and concomitant use of antihypertensive agents after stroke should be managed more conservatively with initial supine bed rest is unclear. The effect of the degree of vessel occlusion and territory of stroke (anterior versus posterior) on cerebral haemodynamics also needs to be studied.

The evidence that cerebral blood flow is at risk from semi-recumbent positioning after acute stroke is equivocal. Cerebral blood velocity was measured in these studies using transcranial Doppler, which only provides non-invasive, indirect measurements of cerebral blood flow [39]. The traditional approach of adopting moderate head elevation between 30° and 45° in patients with large hemispheric stroke is tempered by the findings of reduced middle cerebral artery velocity and cerebral perfusion in some studies [14, 16]. Consequently the ischemic penumbra may be exposed to additional risk from reduction in cerebral blood flow mediated through positional changes after cerebral arterial occlusion. What is not clear is the natural history of autoregulation following stroke and how this varies in ischaemic and haemorrhagic stroke.

The practice of early head elevation to reduce intracranial pressure for patients with stroke has been based on studies of head trauma despite differences in pathophysiology [40]. Although there was some evidence that intracranial pressure was reduced in some patients with early head elevation, this was at the expense of reduced blood pressure and cerebral perfusion pressure [14].

Given that stroke is a heterogeneous condition, it is unlikely that one single optimal position will maintain physiological homeostasis in all patients. Systematic evaluation of individual positions is required to assess potential risks and benefits [37]. It is likely that different positional strategies are required for different phases after acute stroke, particularly in agitated and confused patients. Positioning should be tailored to the individual pathophysiological situation. It is currently not known which physiological parameters predict outcome or what is the best target for therapy. If raised intracranial pressure is particularly problematic in the supine position, then head elevation may be appropriate, whereas if control of cerebral perfusion pressure is the priority, the supine position may be an option. Studies are required to examine the effects of positioning on physiological parameters in a wider range of stroke patients than previously studied, particularly at different stages of acute stroke, to ascertain their prognostic significance.

Key points

- There are few published studies demonstrating the effects of positioning after stroke on physiological homeostasis.
- The evidence at present is not robust enough to guide clinical practice for positioning.
- Further trials are required to investigate whether optimal control of physiological parameters through different positions will alter stroke outcome.

Research letters


do:10.1093/ageing/afi106

Religious attendance and 12-year survival in older persons

SIR—In religiosity the question is how religion is manifested in an individual life. Religiosity has many dimensions, e.g. public–private, organisational–non-organisational, intrinsic–extrinsic [1]. Most studies concerning religiosity and mortality have been carried out in the USA, and the measure of religiosity usually used is the frequency of religious attendance [2–4]. In US follow-up studies, those who attended religious events at least weekly had lower mortality rates than those who attended less than once a week [2–4]. In a meta-analysis of data from 42 studies examining the association of religious involvement and all-cause mortality (total n=120,000), religious involvement was associated with lower mortality [5]. The association was stronger in the studies in which women constituted the majority of participants, there was inadequate control of other covariates of mortality, or measures of public religious involvement were used [5].

The aim of this study was to describe the 12-year survival of older Finns according to the frequency of their religious attendance and gender, and to analyse the associations between mortality, the frequency of religious attendance, and the confounding variables describing age, marital status, education, smoking, hypertension, coronary heart disease, functional abilities, depression and number of medications.

Methods

The data for this study come from a population-based follow-up study of 1,080 persons (449 men and 631 women) aged 65 years or over, living in Lieto, a semi-industrialised municipality in south-western Finland.

The frequency of religious attendance (times per year) was asked about in the interview. In the USA, the attendance variable is often dichotomised into once a week or more versus less than once a week [2–4]. In our material...