The effect of limited and unlimited chairnursing on post-operative recovery in elderly orthopaedic patients

PhD Thesis
KS Gebhardt
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Elderly post-operative orthopaedic patients are often sat in chairs for long periods of time (chairnursed), as part of mobilisation programmes, with the intention of preventing the complications of bedrest. A literature review revealed little research evidence to support this practice which causes significant distress to at least some patients. Considering the circulatory system, it was postulated that lengthy chairnursing was likely to encourage some complications rather than prevent them.

Patients on one orthopaedic ward were limited to no more than 2 hours chairnursing at a time until they were mobile (limited chairnursing group - LCG). Chairnursing was unlimited on another (unlimited chairnursing group - UCG). 51 and 53 >60 year old subjects undergoing fractured hip repair or hip or knee replacement were recruited to LCG and UCG respectively. The regimes were then crossed over between wards and a further 49 and 50 subjects were recruited. Pulse rate, blood pressure, lower leg volume and self reported fatigue were assessed pre-operatively and on days 3, 5, 7, 10 and 14 post-operatively in all subjects unless they dropped out or were discharged earlier. Four assessments were made on each day at 9-10am and 12-1, 3-4 and 6-7pm. Time to independent mobilisation and discharge and negative outcomes were also recorded.

Subjects developed tachycardia (pulse > 100 beats per minute), leg oedema (an increase of 20% or more over baseline), severe fatigue (7-9 arbitrary units) pressure sores and constipation more frequently in UCG than LCG. There was no statistically significant difference in the incidence of hypotension (blood pressure < 100mmHg) and urinary tract or chest infection. LCG subjects attained independent mobility and were discharged more quickly than UCG. There were no differences in the mean pulse rate, blood pressure and leg volume between the two groups 9-10am, but in the afternoon there was less tachycardia, hypotension and oedema in the LCG after 12 noon.

The results of this study do not support the practice of lengthy chairnursing. It is suggested that patients who are not able to mobilise independently should not be sat for more than 2 hours per session.
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1 INTRODUCTION

This study developed as a result of the observation that many elderly patients were being left to sit in chairs for long periods of time following surgery. The health care professionals caring for them believed that this was beneficial. The phrase 'chairnursing' has been coined to describe the policy of prolonged sitting in chairs. However, the patients themselves often said they found this fatiguing and that they would have preferred the sitting to be less prolonged. Many found the degree of fatigue they were experiencing distressing even to the point of tears. This study was designed to examine the potential patient outcomes of chairnursing. Since the feeling of fatigue may be related to poor perfusion and sitting has profound implications for blood distribution and breathing, the study focused on the effects of chairnursing on the cardiovascular and respiratory systems.

Before the beginning of this century, hospital patients were nursed almost exclusively in bed. A survey of the Mary Evans Picture Library (Greenwich) revealed, of a total of 37 pictures of hospital wards prior to 1900, 27 with no chairs at all (table 1.1, figures 1.1, 1.2) and 9 where chairs were provided for the use of visitors and staff only (table 1.2, figure 1.3). There were no chairs between the beds for patients’ use, as one would observe in a modern ward. The only exception was one illustration by Small (1887) which showed patients on an accident ward convalescing from limb injuries with their arms or legs in plaster on chairs and wheelchairs (figure 1.4). The non-provision of chairs did not appear to be a matter of financial constraint or lack of space. A well appointed private nursing home with sumptuous seating for visitors and chairs for staff does not have chairs for patients in its large private rooms (Brown 1800’s; figure 1.5). In another illustration, a view of the balcony of St Thomas’ Hospital, as many convalescent patients are seen in bed as in chairs (Renouard 1890; figure 1.6).

Indirect evidence that patients were generally not nursed in chairs is found in the contemporary discussion of pressure sores by Paget (1873). It is notable that ischial sores, which are associated with sitting, are not mentioned. Paget catalogues the sacrum, posterior superior spine of the ilium, the trochanters and the ends of the spines of the vertebrae as common sites for pressure sores and then mentions the heels, ears and nose as less common ones. It therefore seems unlikely that ischial sores are not present in this discussion through simple omission. MacDowell having carried out an oophorectomy in 1817 was astonished to find the patient making up her own bed 5 days after the operation. "I gave her particular caution for the future", he says, suggesting that he believed this was an act of foolishness (MacDowell 1817).

Surgical patients were nursed for long periods in bed following surgery (eg after clean laparotomies and herniorrhaphies for three weeks or more) until the beginning of this century (Boldt 1907). This was thought necessary to ensure good wound healing (Billroth 1882). Immobility of the wound was thought to be so important that some surgeons, at least, applied plaster spicas after herniorrhaphy (Bloodgood 1898-99 cited in Newburger 1943). In 1902, Rehn recommended respiratory and muscular activity as well as sitting up in bed for postoperative patients because he believed that the dangers of immobilisation to the patient’s overall health outweighed the dangers of mobility to the surgical wound. Since then numerous problems and complications associated with bedrest have been recognised, some of them, such as pulmonary embolus and chest infection, life-threatening (Dittmer & Teasell 1993, Teasell & Dittmer 1993).
Table 1.1 Illustrations of wards with no chairs

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<tr>
<td>1</td>
<td>Clement I (1888) The surgical ward. Scriveners.</td>
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<tr>
<td>2</td>
<td>Durand S (C19) Charing Cross Hospital: Alexandra Ward for children.</td>
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<td>3</td>
<td>Anon (pre 1914) Harrison Ward, The London Hospital.</td>
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<td>5</td>
<td>Anon (1868) The Prince of Wales visiting the persons at St Bartholomews Hospital wounded by the Clerkenwell explosion.</td>
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<tr>
<td>7</td>
<td>Anon (1891) Gordon Ward. Boys Own.</td>
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<tr>
<td>9</td>
<td>Laurentie A (1835) Une clinique. France Pittoresque.</td>
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<tr>
<td>10</td>
<td>Bosse A (C17) D'infirmiere de l'hospice de la Charite.</td>
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<td>11</td>
<td>Blanchard B (1865) Visite de l'Empereur a L'Hotel-Dieu de Paris: S.M. visitant les choleriques dans la salle</td>
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<td></td>
<td>Sainte-Anne.</td>
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<td>13</td>
<td>Anon (c1885) La visite de l'interne - Les Grandes Ecoles</td>
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<td>14</td>
<td>Minne (C18) Paris Hotel-Dieu. La Medicine Populaire.</td>
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<tr>
<td>15</td>
<td>Anon (1867) Lille - visite de SM l'Imperatrice a l'hopital Saint-Saumur.</td>
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<tr>
<td>16</td>
<td>Anon (1888) Surgical ward at Bellevue, New York.</td>
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<tr>
<td>17</td>
<td>Anon (1855) The sick deck of &quot;The Belleisle&quot; hospital ship in Faro Sound.</td>
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<td>18</td>
<td>Anon (1891) Imperial University Hospital, Japan.</td>
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<td>19</td>
<td>Anon (1914) Hopital Anglaise, Nevers.</td>
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<tr>
<td>20</td>
<td>Anon (1863) Visit of the Queen to Netley Hospital</td>
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<tr>
<td>21</td>
<td>Nazzaro (1876) Sala dell ospedale militare di Belgrado.</td>
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<tr>
<td>22</td>
<td>Neumier (C19) L'interieur de la grande salle de l'Hospice d'Angers.</td>
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<td>24</td>
<td>Anon (C19) Hospital scene</td>
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<td>25</td>
<td>Anon (1877) Nurses being trained at a military hospital, Russia. Graphic.</td>
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<td>27</td>
<td>Thomassin P (1526-1622) Hopital de l'ordre de Malte.</td>
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Table 1.2 Illustrations of wards with chairs for visitors (Mary Evans Picture Library)

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<tr>
<td>1</td>
<td>Pape E (1894) Night in the womens ward. Scriveners.</td>
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<tr>
<td>2</td>
<td>Anon (c 1880) A hospital scene.</td>
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<tr>
<td>3</td>
<td>Durand S (C19) Charing Cross Hospital - operation room.</td>
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<td>4</td>
<td>Johnson (C19) Guys Hospital - the clinical ward.</td>
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<td>5</td>
<td>Small W (1887) Visiting day at Guys Hospital - in the accident wards.</td>
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<tr>
<td>7</td>
<td>Anon (1886) &quot;One of the wards&quot; - A ward at Whitechapel Hospital. Illustrated London News.</td>
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<tr>
<td>8</td>
<td>Rowlandson, Pugin (1808) Middlesex Hospital.</td>
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<tr>
<td>9</td>
<td>Johnson H (1887) The clinical ward.</td>
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Figure 1.1 Hotel Dieu, Paris by Bourguemou (15th century)

Figure 1.2 The Prince of Wales visiting the persons at St Bartholomews Hospital wounded by the Clerkenwell explosion (anonymous illustrator, 1868)
Figure 1.3 The London Hospital by E Slocombe (1873)

Figure 1.4 Visiting day at Guys Hospital - in the accident wards by W Small (1887)
Figure 1.5 Home hospitals - Fitzroy House, Fitzroy Square by Brown (19th Century)

Figure 1.6 Patients convalescing on the balcony of St Thomas' Hospital by Renouard (1890)
Ries (1899) was probably the first to get patients out of bed 24-48 hours after surgery and it is probably no coincidence that the first pictures of hospital wards with chairs for patients appear at this time (Anon 1900; figure 1.7). By 1911, Kelly (1911) reported that Ries' example had been followed in Britain "by others too numerous to mention". Much debate about the benefits and risks of 'postoperative walking' followed. However, the reported cases of wounds bursting (Koenig in Madelung 1905 cited by Newburger 1943), claimed to have been caused by early walking, may have been the result of poor suturing technique and materials (Brieger 1983). By 1941, however, lengthy postoperative bedrest had become the exception rather than the rule (Newburger 1943).

Frail elderly patients continued to be nursed in bed for long periods until the 1940's and 1950's, it would appear mainly due to custom and practice rather than any specific perceived benefit (McEwan & Laverty 1949). Marjory Warren, who founded the specialty of geriatrics in the 1940's, advocated getting "elderly folk" up "as soon as their physical condition warrants......." (Warren 1946), as part of her drive to introduce rehabilitation into the process of caring for the sick elderly (Bennet & Ebrahim 1992).

The pictorial evidence from the period 1900-1950 (for example pictorial archives of St Bartholomew's Hospital) shows a trend towards the availability of chairs and their use by patients. However, unstable fractures of the lower limb and pelvis in orthopaedic patients necessitated many months of bedrest to allow the fractures to heal under traction. Even the introduction of surgical repair by internal fixation in the late 1930's did not allow walking within 48 hours of surgery as it was thought necessary to allow the repair to become stabilised (Horton 1983). Orthopaedic patients were nursed in bed for a number of weeks postoperatively to prevent breakdown of the repair. Partly due to improved
operative procedures and materials, this period has been gradually reduced and it is generally accepted in modern texts that patients should be mobilised by the second or third postoperative day at the latest (Maher et al 1998, Evans 1997, 1997a, Poston 1996, Charnley 1979, Muckle 1977).

Writing in 1943, Newburger defined 'early postoperative walking' as:

"The daily postoperative continuation of bodily activity, including walking (not just being placed in a chair), self care in matters of toilet, dressing and feeding and even actual gymnastics..."

This definition seems equally appropriate to the modern term 'mobilisation', which describes the maintenance or improvement in the physical function of the individual and the prevention of problems and complications through movement. Whilst the importance of post-operative mobilisation is acknowledged by nearly all modern authors (eg Haas 1998, Maher et al 1998, Dillerud 1989), particularly for elderly patients (Eckert et al 1979), there is scanty research to support evidence-based practice. For example, a recent Danish review of treatments for the prevention of atelectasis and pneumonia after abdominal surgery found no evidence for the efficacy of mobilisation and recommends this be researched (Lie et al 1998). Similar conclusions are reached by a German review on the prophylaxis of thrombosis after orthopaedic surgery. The authors state that "Basic forms of prophylaxis are physiotherapy and early mobilisation. However, there are no data on the safety and efficacy of these methods" (Pauschert et al 1998).

There is little information of how different mobilisation regimes impact on patient outcomes such as their experience of pain, fatigue, general 'well-being' and the incidence of complications such as deep vein thrombosis (DVT) and chest infection that they are supposed to prevent. Studies which focus on isolated physiological changes, whilst valuable in their own right, often provide contrasting evidence which does little to aid clinicians' decision making. This difficulty is illustrated using the following two studies, as an example. Ashby et al (1995) found that a subject lying supine in bed has a more rapid blood flow rate through the legs than one who is semi-recumbent and therefore they deduce that the semi-recumbent position is likely to help prevent DVT in post-surgical patients. Mynster et al (1996), on the other hand, found that peripheral arterial oxygen saturation increased on sitting up for 10 minutes in the semi-recumbent position following lying supine for 30 minutes and therefore the semi-recumbent position may be preferable for the prevention of cerebral, wound and cardiac dysfunction and complications. Clinicians do not know if, in reality, these positions have any significant impact on the complications in question and, if so, how much. Therefore they cannot weigh up the relative risks and benefits of the two positions and design mobilisation schedules based on this information.

A further difficulty in examining the quality of evidence to support mobilisation of elderly post-operative patients is the lack of clarity over what a mobilisation regime actually consists of. In many cases it simply means that the patient is got out of bed (eg Moiniche et al 1995, Bardram et al 1995). However, there is no information on what they are supposed to do when they are out of bed or how long for. The programme of active mobilisation (ie walking and exercise) envisaged by Newburger and others (eg Charnley 1979) is likely to be labour intensive and difficult to carry out in practice. This may be the reason why the warnings of Newburger that early post-operative walking was not "just being placed in a chair" and those of more modern authors (Charnley 1979, Evans 1987) have been largely ignored and mobilisation has frequently become synonymous with sitting in chairs (Middleton 1983, Muckle 1977, Royal Marsden Hospital 1984, Wilson 1985, Anon 1991). To distinguish sitting in chairs from an active and planned mobilisation it will be referred to as 'chairnursing' throughout this thesis. Unlike 'bed nursing' or 'bedrest' which was (and in some cases still is, eg for back pain, some fractures) a deliberately chosen therapy, chairnursing appears to be the result of getting patients out of bed for as long as possible without a practicable plan of mobilisation.
Warren was not in favour of getting elderly patients into chairs at all costs, as her use of the proviso "as soon as their physical condition warrants" suggests. Bliss (1992) cites her as grading patients by physical ability and only getting patients up into chairs when she thought they were fit enough. However, her colleagues, such as Asher coined the phrase "bed is bad" (Asher 1947) and vigorously implemented a policy of getting patients out of bed, often without considering their condition or wishes. This tradition has continued with the result that on medical, geriatric and orthopaedic wards today, one frequently sees very ill, unhappy (Bliss 1992) elderly patients sitting for many hours in chairs (Gebhardt 1987) next to their beds which may even be elevated to prevent the patients from getting back into them.

Dzielski (1999), a patient who spent 22 weeks in hospital following complications of surgery, septicemia, cardiac arrest, renal failure, respiratory failure and MRSA infection among other problems, gives a powerful personal account of what it can be like to be subjected to lengthy chairnursing whilst unable to get out of the chair independently.

"The time spent in the armchair was the worst experience I remember and even now I still think of it with great unhappiness. I was set down in one place, and because there was no strength in my hands or arms - they were simply skin and bone - I had no way of bracing myself to alter my position. I sat there for several hours at a time unable to move and my backside was so sore I was reduced to tears on more than one occasion. Always I was told it was extremely important to sit up and I spent several hours a day in discomfort increasing to ache, thence to pain."

In orthopaedic wards, patients who may be suffering postoperative fatigue (Vogele & Steptoe 1986), are further subjected to sessions of up to 15 hours in a chair with an average of 6-7 hours (Gebhardt & Bliss 1993). It is not surprising that they complain: "I was so utterly exhausted I wanted to cry" (Gebhardt & Bliss 1993) and "I don't want to be out of bed - ever!" (Gebhardt, unpublished data) are typical comments. In a survey conducted on two orthopaedic wards in a district general hospital, 14 (67%) of a sample of 21 orthopaedic nursing staff (ranging from students to experienced ward sisters) and completing a questionnaire, believed that patients at risk of pressure sores were left sitting too long (appendix 1). The main reason for this, they felt, was a shortage of staff.

Perhaps because it was considered a therapy, many investigations have been carried out concerning the physiological, clinical and psychological effects of bedrest (Browse 1965, Dittmer & Teasell! 1993, Teasell & Dittmer 1993). In comparison, chairnursing has been subjected to hardly any research. Even serious discussion of the topic is scarce. Probably because of this, it is difficult to establish from the literature how much chairnursing does in fact occur in various clinical settings (and this deserves some research). However, in the author's experience it appears to be universal in British hospitals.

Not only is lengthy chairnursing distressing and fatiguing for many patients, the physiological effects of lengthy chairnursing may actually increase the incidence of complications and the length of convalescence - the very things that it is carried out to prevent. Therefore, more knowledge is needed to understand the effects of chairnursing on patients and to guide the design of rehabilitation programmes for patients who have undergone surgery.

The research study presented here was designed to enhance knowledge in this area and to answer some specific questions identified from clinical experience and from the review of existing literature which follows in the next section.
2 LITERATURE REVIEW

The literature review was planned to cover four main areas of knowledge relating to the design of the study. Section 2.1 attempts to identify the amount of chairnursing that takes place in the hospital environment. Section 2.2 goes on to explore the impact chairnursing might have on the physiology of the cardio-pulmonary system while section 2.3 attempts to identify the possible outcomes of these physiological processes in the target population. Section 2.4 surveys possible methods of measuring the outcomes discussed in section 2.3 and attempts to identify the methods of choice for use in this study.

2.1 CHAIRNURSING

It is difficult to quantify from the literature the amount of chairnursing that occurs. One study by Gebhardt (1987) showed that elderly patients on long-stay wards who were unable to walk independently spent up to 6 hours sitting in chairs per session with a mean of 3.5 hours per session. In this study, 85% of a sample of 26 patients sat over 2 hours in chairs at least once during a 27 day study period. Unpublished data by Bliss et al showed that in a study of pressure relieving equipment conducted in 1991-1993 in a small District General Hospital (under 400 beds), mean chairnursing time was 5.4 hours per session in a population of 50 geriatric, 89 orthopaedic, 43 intensive care, 42 medical, 5 oncology patients and 1 surgical patient. As the subjects were selected for the study if they were at risk of pressure sores, it is possible that the mean was even higher in the general population. The patients were admitted to the study within 2-3 days of hospital admission, observed daily and followed through till discharge so the survey is likely to be comprehensive. The mean length of time for orthopaedic patients was 6.5 hours per session. 83% of orthopaedic chairnursing sessions lasted over 2 hours. Gebhardt & Bliss (1994) observed post-operative orthopaedic patients sitting up to 15 hours continuously in a chair and averaging 6-7 hours continuous sitting per session (see chapter 4 and appendix 2).

There is some anecdotal evidence of chairnursing from personal accounts of patients (eg Dzielski 1999) and from personal views expressed by clinicians such as the geriatricians Bliss (1992) and Mulley (1993), Wells (1979) a surgeon, Kaldcar (cited in Bliss 1992) a researcher into deep vein thrombosis and Roper (1988) a nurse. Bliss describes, among others, "many sick old people" who "remain white, dizzy and confused, propped up in their chairs". Her article questions the popular wisdom of getting elderly sick patients into chairs regardless of their condition. Mulley suggests that the "enthusiasm for the chair may be overdone", speaks of "prolonged immobility" in chairs and describes what are, in his view, the inherent dangers. Wells describes his clinical experience in 1946. He describes early ambulation as "getting-out-of-bed-and-sitting-in-a-chair" and becoming the "in thing". In Kakkar's view "early ambulation equals early angulation", a reference to the angulation of major blood vessels which occurs when patients sit in a chair with their feet dependent. Roper describes post-operative patients "lying like logs" due to exhaustion caused by sitting in chairs. Many nursing staff surveyed by Gebhardt (appendix 1) thought that patients at risk of pressure sores sat out too long.

Further supporting evidence might be inferred from some recommendations for clinical practice. Muckle (1977) states that patients can sit out of bed within 2-3 days of surgery. Middleton (1983) describes helping the patient to sit under the heading 'ambulation' although he does caution against being "over-enthusiastic". The Royal Marsden Hospital Manual of Clinical Procedures (Royal Marsden Hospital 1984) recommended sitting in chairs for long periods of time as a means of preventing DVT. Wilson (1985) recommends active mobilisation for post-operative patients but then
continues to describe procedures for nursing patients in chairs rather than walking. Maher et al (1998) suggest patients be "mobilised into a chair" in the first post-operative days after hip replacement.

Perhaps the main difficulty lies in the lack of precision in the literature about what is meant by mobilisation and early ambulation. Chantley (1979) was very specific about the rehabilitation regime he recommended for patients who had undergone a hip replacement. Walking was started from the 3rd post-operative day. How this should be implemented is described in detail. Sitting, on the other hand, was "not permitted till the seventh post-operative day". After instruction in the lavatory on how to slide out the operated leg, the patient was permitted half to one hour in the morning and the same in the afternoon. The duration of sitting was to be increased daily to 6 hours per day by the 9th day (with sitting interrupted by 1-1.5 hour’s bedrest after the mid-day meal). The feet were not supposed to be dependent for more than 1 hour at a time, being supported on a stool for an equal period.

Other authors have rarely been so precise. For example Bardram et al (1995) indicate that patients in their study were 'mobilised' for a mean of 12 hours per day or more from the 3rd post-operative day following colonic resection. However there is no definition of what this means ie whether it is time out of bed, time sitting in a chair or time spent walking. Bearing in mind the mean of 12 hours and maxima of 14-15 hours the last seems unlikely. Similarly, Moiniche et al (1995) give no indication what 'early full mobilisation' means although it is one of the central planks of their uncontrolled study of a new approach to colonic surgery. It is notable that in the results section Moiniche et al (1995) use 'hours of ambulation' in the figure describing mobilisation implying that the two were synonymous. Given a pre-operative median of 16 hours and a 3rd day post-operative median of 11 hours (which the authors describe as indicating the patients being 'fully mobilised'), it seems unlikely that the patients really spent this amount of time walking as the authors imply.

Hanuncharurnkui and Vinya-nguang (1991) clearly confuse ambulation with some other activity when they describe patients ambulating 1-4 hours on the first day, 2-8 hours on the second and 4-12 hours on the third post-operative day. Many more such examples of imprecise use of the terms 'ambulation' and 'mobility' can be quoted both in the medical and nursing literature, whether as variables or outcomes of research (eg Johnson et al 1996, Johnson et al 1995, Jenkins et al 1989) or as recommendations for clinical practice (eg Kumta et al 1998, Torance & Servigson 1997, Flanagan et al 1995). The most likely meaning of 'mobilisation' in these instances is 'time out of bed'. This confusion of 'ambulation' with the absence of bedrest has become so widespread that it occasionally appears in medical dictionaries (Spraycar et al 1995). The term 'early activation' has also been used in this context in the care of elderly acutely ill patients (Hulter Asberg 1986).

If the patients are not actively walking, it is likely that much of this 'time out of bed' will be spent sitting in a chair or being 'chairnursed'. Possibly, even when there is every intention of either moving the patient back to bed or walking with them, other priorities may intervene or there simply are not enough staff to carry this out. This view is supported by nursing staff surveyed in 1994 by Gebhardt (appendix 1) many of whom felt patients spent too much time sitting and that this was due to too few staff to assist with walking and getting in and out of bed.

If it is accepted that unless otherwise stated, mobilisation and ambulation generally mean 'time out of bed' which equates to chairnursing for the most part, then the evidence for chairnursing is plentiful. In a survey of 940 surgical centres in West Germany of 564 responders (60% response rate) representing 1,200,000 operations per annum all claimed to use early mobilisation to prevent DVT and pulmonary embolism (Klempa et al 1992). The examples quoted in the above paragraphs give a good indication of the lengths of time involved. The following sections of this thesis attempt to describe what the possible physiological consequences and potential outcomes of lengthy chairnursing might be for elderly post-operative orthopaedic patients.
2.2 THE EFFECTS OF CHAIRNURSING

Being seated for a long period of time places various demands and limitations on the human body. The causes of these demands and limitations can be broadly summarised as the effects of gravity on a circulatory system, parts of which are at different heights to each other (orthostasis); of the seated posture and alignment of body parts during sitting; and of the overall effects of immobility.

The existing body of knowledge on orthostasis is mainly limited to studies of subjects undergoing passive tilt and standing experiments whilst knowledge of the effects of immobility is mainly based on studies of subjects in bed. Such studies that have been carried out on subjects in the seated posture tend to be confined to healthy, mobile workers as opposed to elderly post-operative patients - the focus of this thesis. Extrapolations, therefore, have to be made from the body of knowledge of the effects of long-term bedrest, passive tilt experiments and studies of healthy individuals to make theoretical predictions about the likely effects of chairnursing in this patient group.

It is likely that the specific condition of elderly post-operative orthopaedic patients will influence the effects of orthostasis, immobility and posture. It is therefore necessary to discuss age related changes to circulatory mechanisms and the effects of surgery on the circulation (and respiration) to obtain a fuller picture of the possible outcomes of prolonged chairnursing in this group. However, it should be noted that while there is a significant body of literature on the effects of surgery on breathing and respiration, the subjects of the research that this literature is based upon have usually undergone thoracic or abdominal surgery. It is possible that the respiratory effects observed may be reduced or absent in patients who have undergone limb surgery which does not directly affect the thoracic cavity and therefore the respiratory and circulatory organs.

Although in the interests of clarity the various aspects of chairnursing elderly post-operative patients will be considered separately, it is clear that they are intimately interlinked, as indicated by the simplified model (figure 2.1).

2.2.1 The effects of orthostasis

Although orthostasis literally means 'standing straight', in this thesis it will be used to describe any posture in which parts of the body are above and/or below the level of the heart, thus necessitating compensatory activity of the body to maintain equal circulating pressure in all parts of the system. Macro- and micro-circulatory changes occur when an individual is in an orthostatic position as opposed to supine (Winkel 1985). Humans are particularly sensitive to different directions of the gravitational field relative to the body due to their normally erect posture, large body mass and large blood volume below the level of the heart, according to Blomquist & Stone (1983).

Because of the effect of gravity, on standing from supine to upright there is a shift of blood, mainly to the legs (Sjostrand 1952). The venous volume of the legs increases by about 500 ml (Gauer & Thron 1965). Other areas which gain blood volume are the veins in the buttocks and pelvis (Blomquist & Stone 1983). It is thought that most of this translocated blood volume comes from the intrathoracic compartment of the low-pressure system (Gauer & Thron 1965, Sjostrand 1953). This introduces some cardiac strain as the heart attempts to maintain output despite a reduced input volume.

The downward shift of blood during orthostasis causes the venous and arterial pressure in the dependent legs to increase and approximately equal the hydrostatic pressure of a column of blood to the heart in a standing individual (Arnoldi & Linderholm 1966). Using a tilt table, Katkov & Chestukhin (1980) found increases of over 100 mmHg in systolic and diastolic arterial pressure and over 60 mmHg in mean venous pressure measured in 10 adult humans. During sitting, the hydrostatic pressure will clearly be less than it is on assuming the standing posture. Nevertheless, it will be higher than when supine and similar physiological outcomes are to be expected, though to a lesser degree.
Figure 2.1 The physiological effects of prolonged chairnursing

- Surgery
  - Reduced movement of limb due to pain
- Old Age
  - Postmenopausal hormonal effects
  - Drugs (1)
    - Reduced fluid intake
    - Dehydration
- Orthostasis
  - Shift of blood volume to legs
  - Increased venous pressure in legs
  - Loss of plasma in the legs to NRBCIC and/or interstitial spaces
  - Reduced venous return
  - Reduced cardiac output
  - Homeostatic mechanisms:
    - a) tachycardia
    - b) increased circulatory efficiency
    - c) increased peripheral resistance
    - d) reduced blood supply to non-essentials
    - e) reduced diuresis
  - Pathologies (2)
  - Opioid analgesics
  - Drugs (3)
  - Normotension
- Immobility
  - Reduced calf muscle pump activity
  - Reduced foot/leg activity
- Posture
  - Increased thoracic volume
  - Increased tissue compression and shear
  - Reduced lung function
  - Fixed tissue deformation
  - Local tissue ischaemia

Key:
Increases
Decreases
May increase or decrease
(1) oedema as potential side effect
(2) e.g. diabetes
(3) hypotensive e.g. diuretics, vaso-dilators
NRBCIC = non red blood cell intracellular compartment
FRC = functional residual capacity
Over time, if high hydrostatic pressures are maintained, it is likely that there will be loss of plasma fluid through the blood vessel walls by the process of capillary filtration (Starling 1896). Waterfield (1931) observed a mean 15% plasma loss in the general circulation in 8 healthy volunteers standing for 40 minutes. Bjerkhoel & Lundvall (1994) and Lundvall & Bjerkhoel (1994, 1995) observed plasma losses of 400-700ml after 15 minutes standing. Part of the plasma is lost in the lower leg, where the pressure is highest. Waterfield (1931a), for example, observed swelling of the lower leg of 43-120 cc in 16 normal subjects standing for 40 minutes. However, the bulk of the fluid loss occurs in the thighs and buttocks, probably because the large tissue mass and capillary beds of the thighs and buttocks can allow the transfer of large volumes more readily than the smaller ones of the lower leg. Bjerkhoel and Lundvall (1995) found an increase of 391 ±23ml in the extravascular volume in the thighs and buttocks, compared with 192 ±17ml in the lower leg in 7 healthy men standing still for 15 minutes.

Similar processes appear to occur in the lower limbs on sitting, although at a somewhat slower pace. Winkel and Jorgensen (1986a) observed 6.3% loss of plasma volume in seven healthy subjects over 8 hours of seated work without leg movements. Greenleaf et al (1980) found a gradual loss of plasma volume in 4 healthy young adult males sitting for 8 hours, despite an overall positive water balance. Although they did not observe overt oedema, it is likely that some of the fluid passed into the non red blood cell intracellular compartment and/or interstitial spaces due to hydrostatic pressure. This effect has been observed in many studies which measured foot volume in healthy sedentary workers, with foot volume increasing 2-7% following prolonged sitting (Whitney & Gear 1965, Pottier et al 1969, Winkel 1981, Winkel 1985, Noddeland & Winkel 1988). Regardless of where the fluid goes, blood volume is further reduced and cardiovascular strain increased by the gradual loss of circulating volume.

The main task of the macrocirculatory orthostatic regulating mechanisms is to maintain an arterial blood pressure which is sufficient to supply the brain with blood despite the reduced central venous return flow caused by the shift of blood volume to the legs and loss of plasma volume. The initial compensatory mechanisms on standing up from supine are tachycardia, followed by an increase in peripheral resistance.

On standing from supine, the pooling of blood in the legs causes a 20% reduction in venous return to the heart and a similar reduction in cardiac output (Brobeck 1973). This causes unloading of arterial baroreceptors. These receptors are found in the walls of the arteries and their function is the short-term regulation of arterial blood pressure. As the blood pressure drops there is a reduction in arterial baroreceptor firing. This stimulates the sympathetic and dampens the parasympathetic systems. The net results of this are tachycardia and constriction in the peripheral veins and the arterioles. The increase in heart rate and vasoconstriction (which increases venous return to the heart) produce an increased cardiac output, while arteriole constriction increases total peripheral resistance (Vander et al 1998).

Tachycardia and raised peripheral resistance during orthostasis has been demonstrated in tilt-table experiments (Jahan et al 1996, Stevens 1966, Asmussen et al 1940). Despite the tachycardia and increased venous return, cardiac output drops and this is compensated by a further increase in peripheral resistance so that blood pressure may actually rise (eg Asmussen et al 1940, Bevegard 1962). Tachycardia then subsides, is followed by a brief rebound bradycardia and in healthy young adults this process is complete in a matter of seconds (Colbert 1993). Oxygen uptake is kept constant by an increase in the oxygen partial pressure difference between the venous and the arterial circulation (Asmussen et al 1940) which serves to increase the circulatory efficiency of the reduced volume of circulating fluid. In addition, some other functions of the circulation are not fully carried out such as thermo-regulation, digestion and renal function (Asmussen et al 1940), so that blood can be shunted to the central circulation.
During prolonged upright orthostasis on tilt tables, however, there is a progressive increase in heart rate and arterial blood pressure (Kilbom 1971, Stevens 1966, Asmussen et al 1940). This may be due to the ongoing plasma loss through capillary filtration continuously reducing blood volume. Although fluid loss is partially counteracted by increasing blood concentrations of anti-diuretic hormone, aldosterone and renin (eg Brown et al 1966), which reduce diuresis, this can only be effective if adequate input is maintained. Balance is not always fully restored.

A change from supine to sitting produces qualitatively similar though quantitatively somewhat smaller haemodynamic adjustments. Thadani and Parker (1978) found mean heart rate and blood pressure to be significantly higher in 10 men aged 32-58 during sitting as compared with lying supine. These findings have been confirmed by many other researchers (eg Bevegard et al 1963, Saltin et al 1968).

What happens during prolonged sitting is less clear. Schwartz et al (1982, 1983) found blood pressure to rise but heart rate to fall during 5 hours of sitting. Greenleaf et al (1980) also found a lower mean heart rate during 8 hours of quiet sitting as compared with ambulatory controls. It is not certain why this happens but there are a number of possible interpretations. One is that during prolonged sitting the changes in blood volume are slow and the baroreceptors are more sensitive to sudden as opposed to more gradual changes (Fox 1999). Furthermore heart rate is not just controlled by baroreceptor activity. Skeletal muscle activity also has a tachycardic effect - the muscle-heart reflex (Colbert 1993). It is possible that during quiet sitting, there is less muscular activity than when standing (perhaps even on a tilt table) and therefore less cardiac stimulation from the muscle-heart reflex.

Another possible explanation is that in very fit young individuals such as athletes the heart responds to cardiovascular demands initially by increasing the stroke volume and only later by increasing heart rate. In seated orthostasis, the cardiovascular demands may be less than in standing and fit young individuals may be able to fulfil them without the need for tachycardia. Petitt et al (1999) found that in 8 men and nine women, there was an increase in stroke volume and decrease in heart rate during two hours of sitting at rest. However, this study was carried out under conditions of cold-air exposure at 5°C. These findings are therefore confounded by the effect of cold which also tends to have a bradycardic effect (Tipton et al 1998) and so should not be taken as representative of sitting under room temperature conditions.

It is also possible that the slower reduction in plasma volume during sitting can be effectively compensated by the reduction in diuresis provided input is adequate. Greenleaf et al (1980) observed that there was reduced diuresis during chair sitting despite input. When their subjects went on bedrest following 8 hours of sitting, there was an increased diuresis, particularly during the first two hours following chair rest. These observations contrast sharply with those obtained when the subjects were immersed up to their necks in water and therefore not subject to orthostatic stress as the back pressure of the water counteracts the effects of orthostasis. During 8 hours of immersion diuresis was almost double that during chair rest. Conversely, during the first two hours of bedrest following immersion, diuresis was one tenth of that observed after chair sitting. This suggests that the orthostasis, which occurs during chair sitting but not during immersion, is responsible. The fifth explanation may lie in the habituation of the system in general and of the baroreceptors in particular to a condition of lowered blood pressure, so that a lowered blood pressure is tolerated if orthostatic stresses are prolonged in a uniform fashion. In the absence of vigorous activity and with a shift of circulatory volume from non-essential activities, this may be of limited consequence in the short term.

In summary, the primary effects of orthostasis are to shift some of the blood volume from the thorax to the legs. The immediate result is a reduction in cardiac stroke volume and the body attempts to compensate with a tachycardia, an increase in peripheral circulatory resistance and decreased diuresis. If this does not fully restore arterial blood pressure, there is a shift of blood volume to the vital organs at the expense of other functions such as thermoregulation, digestion and renal function. The effects of prolonged sitting are less clear, however. The increases in blood pressure in the legs due to the downward shift cause a gradual loss of blood volume, probably into the intracellular and
interstitial compartments of the legs which causes a progressive reduction in venous return. This is compensated to some extent by reduction in fluid excretion and, if input is adequate, may fully replace the volume lost. The heart rate tends to become bradycardic. The reasons for this are not fully understood but reduced muscular activity and habituation may play a part.

2.2.2 The effects of immobility
The normal state of the body when not at rest, is thought to be one of motion (Lentz 1981). Because of reduced sensory input, after prolonged inactivity a stable level of autonomic activity cannot be maintained and the body becomes less able to cope with even small stresses such as changes of posture or increased cardiovascular demands (Vallbona 1982). Thus, during periods of immobility there is a tendency towards tachycardia and loss of cardiac output.

For example, in a study of healthy volunteers, Halar & Bell (1988) found that the resting pulse rate increases by one beat per minute for every two days of bedrest. This effect is probably due to increased sympathetic nervous system activity (Dittmer & Teasell 1993). The result is less diastolic filling time and thereby a reduced systolic ejection volume. The coronary circulation is also affected, as some coronary arteries are only filled during diastole. As a result, cardiac output, stroke volume and left ventricular function decline (Halar & Bell 1988, Holmgren et al 1960). These effects of bedrest are common to healthy as well as ill individuals (Holmgren et al 1960). It should be noted that an elevated heart beat is common among all sedentary individuals as well as those on bedrest (Vallbona 1982), and thus is likely to be an outcome of immobility rather than consequence of recumbent posture or lack of orthostatic challenges.

Lack of exercise causes a general physical deconditioning. One measure of physical condition is obtained by comparing heart rate with the magnitude of a task determined by measuring the oxygen uptake required to complete it. Physical condition is then expressed as the heart rate at a standard oxygen uptake. Various studies have shown significant deterioration in this measurement following bedrest in healthy volunteers (eg Saltin et al 1968) and orthopaedic patients (eg Bassey & Fentem 1974). Greenleaf et al (1980) similarly found a reduction in oxygen uptake as compared with ambulatory controls in volunteers who had either sat in a chair or been immersed in water for 8 hours, which suggests this too is an effect of immobility rather than posture.

Another benefit of activity as opposed to immobility is that during walking, contractions of the gastrocnemius and soleus muscles of the lower leg squeeze blood up the major leg veins which are equipped with non-return valves so that the whole system acts as a pump (the calf muscle pump; Amberton 1943). The calf muscle pump is important in maintaining a good circulatory return from the lower limbs (Vander et al 1998). This is necessary for the maintenance of an adequate central venous return flow to the heart and consequently adequate diastolic filling pressure. This, in turn, is needed to maintain stroke volume and therefore adequate cardiac output.

The calf muscle pump also has a role in the prevention of oedema during orthostasis. In very fit individuals, this system is sufficiently efficient to prevent any leg oedema despite prolonged walking (Vaananen et al 1997). During sitting, Winkel (1981) for example, showed that there was little foot swelling if individuals interrupted their sitting by taking a short walk every 15 minutes as compared with an uninterrupted sitting of 8 hours duration. Even modest plantar flexions of the foot proved beneficial reducing foot swelling to 2.5% after 8 hours sitting as compared with 4.8% during the same period of constrained sitting (Winkel & Jorgensen 1986a). Another likely reason why sitting promotes oedema is that immobility also reduces lymphatic drainage, thus allowing greater oedema formation. These effects will be discussed in more detail in section 2.3.4.

To carry out its required functions, the circulating fluids must also be able to exchange gases at an appropriate rate. Physical activity is necessary to maintain the principal organ of gaseous exchange, the lungs, working to their full capacity (Teasell & Dittmer 1993) and clear of secretion build-up and
infection (Browse 1965). Immobility reduces nearly all lung volumes (Jagmin 1998). Loss of respiratory muscle strength and failure to fully expand the chest wall results in a 25-50% reduction in respiratory capacity (Teasell & Dittmer 1993). Respiratory rate increases to compensate for this.

Immobility (which is often coupled with motor weakness of the respiratory muscles) reduces the ability to clear secretions and these accumulate in the dependent parts of the bronchial tree and interfere with gaseous exchange (Judd 1989, Browse 1965). The distribution of blood flow in the lungs is also affected. In an upright position, for example, blood flow to the apex is less than that to the base of the lung (West 1966, cited in Blomqvist & Stone 1983). Thus, blood flow is likely to be mainly directed to the dependent areas which are also most likely to be compromised by the pooling of secretions. The result of this is that in dependent areas of the lung the resulting poor ventilation and overperfusion causes arteriovenous shunting and reduced arterial oxygenation. The effects of immobility are likely to be independent of posture, although, of course, position will determine which parts are dependent and therefore affected.

The global effect of cardio-respiratory and physical deconditioning following immobility is a loss of efficiency which can be observed as reduction in maximal oxygen uptake. This has been demonstrated in numerous studies in which subjects were placed on bedrest for a period of time and then exercised (eg Saltin et al 1968, Convertino et al 1975). These effects can be ameliorated to a degree by exercise regimes in bed (Stremmel et al 1976). Because reduction in cardio-pulmonary efficiency is seen in subjects sitting in chairs (Lamb et al 1965) as well as lying down, it is suggested that they are caused by immobilisation and/or confinement rather than by elimination of orthostasis during recumbency. Chairnursing, therefore, apart from introducing exercise when the individual gets into the chair and then out of it, cannot be considered an effective substitute for active ambulation in this respect.

2.2.3 The effects of posture

Even during sleep, the calf muscle pump is kept at least partially active by movements of the legs (Sforza et al 1999). This reflex activity can be further enhanced in post-operative patients by encouraging them to carry out ankle flexion and rotation exercises during any periods that they are confined to bed. Although there is little or no evidence of this in the literature, it is likely that foot movements are more difficult to carry out when the patient sits in a chair with his/her feet on the ground or foot support. This may be the reason for the very marked reduction in blood flow in the lower limbs in the seated posture as opposed to any other. Ashby et al (1995) found that in 8 healthy adult volunteers, mean blood velocity in the femoral vein in the seated position was significantly less than in any other posture studied (supine, legs up 6° and horizontal; head up 35°, legs up 6° and horizontal; standing). The much lower mean peak velocity in the sitting (1.3 cm/s) as opposed to the standing posture (9.2 cm/s) suggests that this is not purely a gravitational effect. The likelihood is that the inactivation of the calf muscle pump and vein flexion in the seated posture have a major role to play in the reduction of blood flow in the legs during sitting although their relative importance is not known.

The importance of venous stasis is that firstly the overall oxygen carrying capacity of the blood is reduced due to a reduced intracardiac packed cell volume (Simpson 1990). Secondly, an inadequate circulation is likely to lead to intra-cellular oedema because underperfused cells have an impaired ability to pump out ions that enter them. Water follows the ions (especially sodium ions) due to the osmotic gradients that are created (Guyton 1991). Intra-cellular oedema is likely to be more persistent because it cannot be directly removed by the lymphatic system. Also, where there is particularly poor perfusion, such as in an ischaemic leg, intracellular oedema can be severe with tissue volume doubling or more, often as a prelude to widespread tissue death (Guyton 1991). These effects are independent of orthostasis, being a result of reduced blood flow rather than of increased blood pressure.

Posture impacts upon lung function and through lung function also upon circulatory efficiency. If gaseous exchange is less efficient, the blood carries less oxygen and more carbon dioxide and waste
products. Therefore a more rapidly circulating blood volume is required to satisfy cellular requirements. Functional residual capacity (FRC - defined as the volume of gas in the lungs and airways at the end of a spontaneous expiration) is decreased in the supine as opposed to the sitting position in healthy volunteers (Craig et al 1971). An improvement in gas exchange in the seated posture, thought to be due to the observed increase in FRC has been demonstrated in post-operative patients who had undergone abdominal surgery (Vaughan & Wise 1975). Meyers et al (1975) showed in a sample of 28 post-abdominal surgery patients that FRC is greater when sitting in a chair as opposed to sitting upright in bed. Hsu and Hickey (1976) demonstrated greater FRC in the seated (whether in chair or bed is not stated) as opposed to supine position in a small sample (n=6) of patients (whose age range is not given) who had operations performed on their extremities.

The reason why an upright posture increases FRC is often attributed solely to the increase in thoracic volume which results from the downward movement of the diaphragm (Harper & Lyles 1988). However, Sjostrand (1951), using a corset plethysmograph to measure trunk volume and young volunteers as subjects, found that only 40% of the increase in FRC was due to an increase in thoracic volume. He concluded that the remainder of the increase must be due to a reduction in thoracic blood volume. Thus, any benefits of increased lung capacity, may be undermined by a reduction in pulmonary circulation and therefore less efficient gaseous exchange. The matter is further complicated by the relationship between FRC and closing volume (CV). Closing volume is a measurement of volume at which forces of gravity, elasticity of lung tissue and airway properties can close off small airways. When CV exceeds FRC some airways are closed off and the ventilation of the dependant lung zones is decreased (atelectasis). This has little or no effect on blood flow, so a ventilation/perfusion mismatch occurs which leads to inefficient gaseous exchange (Jagmin 1998).

With increasing age and disease, the lungs become less pliable and CV increases. By the age of 35-44 CV exceeds FRC in the supine position (Craig et al 1971, Leblanc et al 1970). CV does not change with position but continues to increase with age (Craig et al 1971a) so that by the age of 65 it exceeds FRC in the seated as well as the supine position (Milic-Emili 1970). Therefore in elderly patients, such as the subjects of this study, the seated posture may offer few or no advantages over supine in terms of atelectasis and gaseous exchange. A direct measurement of oxygen and/or carbon dioxide saturation during sitting could resolve these issues.

Mynster et al (1996) studied arterial oxygen saturation (SpO₂), using an adhesive finger probe, in 11 patients the day before major abdominal surgery and on days 1, 4 and 7 after. They found that on all days SpO₂ was higher when sat up in bed than when lying supine and higher still when standing upright. On days 1 and 4 post-operatively, these differences were statistically significant. It should be noted, however, that the measurements taken were a mean of ten minutes continuous monitoring in the chosen position directly after the patient had rested 30 minutes in bed and then moved into the desired position. The increase in peripheral SpO₂ may well be the result of the exercise involved in getting up into a sitting or standing position from bedrest and of the tachycardic activities of the muscle-heart reflex rather than of the position itself. Therefore this study provides little insight into the long-term effects of sitting in chairs for post-operative orthopaedic patients especially when the small sample size and the abdominal as opposed to orthopaedic nature of the surgery to the sample is taken into account. However, if data is harvested over a longer time-span representative of the actual times patients sit or actively mobilise, this may prove to be a valuable methodology, particularly if data on pulse rate, blood pressure and other variables is collected at the same time.

It is impossible to discuss the effects of the seated posture without considering the interaction between the body and the surface the individual is seated upon. Because the body is supported by a relatively small area (compared with bedrest for example) large interface pressures are generated even if specialised 'pressure reducing' cushions are used (Medical Devices Agency 1997). Vertical shearing within the tissues occurs because the ischial tuberosities press through the soft tissues and compress small areas of tissue more than the surrounding areas. The degree to which this occurs depends partly on the alignment of the knee and thigh (figure 2.2). Horizontal shear occurs as the individual tends
to slide forward in the chair to make him/herself more comfortable (Goosens et al 1993). The combined shearing and compressive forces tend to stretch, angulate, compress and kink blood vessels within the affected tissues. This interrupts blood flow and leads to ischaemia of the tissues supplied by those vessels. If ischaemia is sufficiently prolonged, a pressure sore may occur. This issue is discussed in section 2.3.7.

Figure 2.2 Pelvis tilt as a function of trunk-thigh angle (after Keegan 1953)

Furthermore, when the skin is subjected to pressure, the muscle tissue beneath is also compressed. Muscle tissue is richly supplied with nerve endings and numbness or even painful sensations in the muscles of the buttocks have been reported after prolonged sitting (Babbs 1979, Jurgens 1969 cited in Eklund 1986, Floyd & Roberts 1958). Another interaction of under-thigh pressure is the reduction of venous return from the legs (Pottier et al 1969). This is likely to further increase lower leg congestion and therefore oedema.

The complex implications for the general musculature and skeletal posture of prolonged sitting in a chair are beyond the scope of this thesis, however reviews are given by Eklund (1986) and Ernst (1992). In summary, sitting for prolonged periods of time can cause a number of problems such as back pain (of muscular or disc prolapse origin) and muscular pain of the neck and shoulders. Ernst (1992) makes the point that since sitting is invariably associated with unfavourable statics, 'correct' seating is a contradiction in terms. At best, well-designed seating can reduce the incidence of problems. However, the design and condition of much hospital seating is generally poor (McAlpine et al 1993, Lowry 1992). For example it has been observed that slightly forward inclined chairs are more comfortable and cause fewer neck and back problems (Naqvi 1994). Hospital chair seats are, however, usually inclined backwards to help prevent patients slipping out of them. Many hospital chairs have a backward tilting back rest which is likely to increase shearing on the buttocks (Goosens & Snijders 1995). These factors suggest that prolonged sitting in hospital is likely to be uncomfortable. It is interesting to note that there is thought to be a connection between seat comfort and reduced fatigue for car drivers (Fubini 1997). This is also likely to be true for hospital patients, and therefore it is reasonable to assume that prolonged sitting in uncomfortable seats is likely to promote fatigue.
While not strictly an effect of posture, it is convenient to discuss here the interaction of the seated individual with the chair he/or she is sitting on. Sweat evaporates from the skin. If the upholstery of the chair is not permeable enough, the skin will become wet with condensed sweat, which is a commonly experienced unpleasant feeling. The seating within the hospital environment will have little or no permeability due to the requirements for infection control and protection of the upholstery from incontinence and other bodily fluids. The problem of moisture accumulation increases with increasing ambient temperature (Andren et al 1975), bearing in mind that the local temperature of the thighs, seat and back areas of the body are likely to be higher than ambient due to insulation (Holmer et al 1995). Hospital wards are often kept at a high temperature (21-25°C) so the problem of moisture accumulation is likely to be another source of discomfort which may contribute to overall feelings of discomfort and fatigue.

In summary, apart from orthostasis, the main effects of the seated posture are a reduction in calf muscle pump activity combined with angulation of major blood vessels and a reduction in venous return due to under-thigh pressure. This serves to reduce blood flow rates in the legs and the venous return from them, potentiating the effects of orthostasis on cardiac stroke volume. There is, however, an increase in thoracic volume partly due to posture which results in increased vital capacity and FRC of the lungs. The benefits of these, if any, are unknown. The poor design of many if not most hospital chairs and non-vapour permeability of their upholstery is likely to make prolonged sitting an uncomfortable experience even in the absence of circulatory failure induced by orthostasis. The high loading on the tissues of the thighs and buttocks is likely to increase discomfort further and possibly lead to local tissue ischemia.

2.2.4 The effects of old age
Much of the preceding discussion of the likely effects of chairnursing has been derived from data obtained from young and healthy subjects or mixed patient populations. The target population of this thesis, however, consists of elderly patients who have undergone orthopaedic surgery. Consequently the effects of old age and surgery on the circulatory system need to be explored as well as those of orthostasis, immobility and posture.

The mechanisms described in sections 2.2.1 - 2.2.3, whose function is to maintain effective circulation despite the challenges of orthostasis, the seated posture and immobility are examples of homeostatic mechanism. Homeostasis can be defined as the maintenance of a constant internal environment despite external changes. Under resting conditions the healthy old person can maintain a constant internal environment within physiological parameters similar to those of young people (Exton-Smith 1985). However, there is a diminished ability to react to stress. Typically, when the homeostatic environment is challenged in the elderly, there is a greater swing away from equilibrium and it takes longer to return to normal parameters than in the young (Dunbar 1996).

As has been stated earlier, sitting challenges homeostasis, with particular demands being placed on the cardiovascular system. In the old there is a decline in blood pressure control. Even in the fit and healthy elderly, who appear to withstand standing up well, the mechanisms by which this is achieved are different to the young. The tachycardic response is attenuated. For example, one study compared 4 samples (n = 10 in each sample) of 10-15 year old boys; 60-69 year old driver’s license applicants and two samples of elderly men, 70-79 and 80-89 years old, who were participating in vigorous social activity such as folk dancing in a community centre (Dambrink & Wieling 1992). In the young there was a pronounced tachycardia on standing up and a lesser one in head up tilt experiments. In all the elderly samples, the response was similar, but much attenuated. In the young systolic blood pressure remained stable and diastolic rose. In contrast, in the elderly there was no rise in diastolic pressure and a fall in systolic.
It is of interest, that in the elderly, head up tilt on a tilt table caused a greater drop in blood pressure than standing, whereas there was little difference in the young. This suggests, perhaps, that in the elderly orthostatic cardiovascular control is more reliant on either calf muscle pump activity or the muscle-heart reflex than in the young. This may have implications for the prediction of the likely effects of chairnursing because both calf muscle pump and muscular activity are reduced during sitting as compared with standing. These findings of reduced tachycardic response have been found in other studies (Wieling et al 1992, Wieling et al 1982, Collins et al 1980) and Wieling et al (1992) who examined haemodynamics using pulse contour analysis suggests that in the elderly, orthostasis is achieved predominantly through increased peripheral vasoconstriction.

It is notable that in the Dambrink & Wieling (1992) study the samples were of healthy and active elders and excluded all individuals with known major systemic disease or those taking medication. No blood pressure drops of 20 mmHg or greater were noted. Rudas et al (1996), on the other hand, found a mean systolic blood pressure drop of 22 ± 13 mmHg in 12 young volunteers and of 34 ± 18 mmHg in 10 elderly volunteers. The discrepancy between the two studies may lie in the older age of the 'young' volunteers (26-40 years) in the Rudas et al (1996) study and the fact that the elderly volunteers were recruited from an outpatients department of a hospital rather than from a healthy and active group. In the general elderly population, which includes individuals with cardiovascular disease, Caird et al (1973) found a fall of blood pressure on standing of 20 mmHg or more in 16% of a sample of 65-74 year olds living at home and 30% in those over 75 years of age. Exton-Smith (1977) later found similar results in the general elderly population (14% having a drop of 20 mmHg or more). It seems reasonable, therefore, to assume that orthostatic control relies on different mechanisms and tends to become less effective with increasing age and decreasing condition.

This change in orthostatic control is thought to be mainly due to a decline in the functioning of the autonomic nervous system. Gribbin et al (1971) found a statistically highly significant trend of age-related reduction of baroreflex sensitivity in a sample of 61 subjects. Others (eg Mancia et al 1984) have shown that the speed of baroreceptor responses is slower in sixty year olds compared to 20 year olds. As baroreceptors are stretch receptors which respond to the distension of the arterial walls (Fox 1999) loss of sensitivity may be due at least partly to reduced arterial distensibility caused by atherosclerosis (Wollner & Collins 1992). It has also been suggested that not only do the autonomic responses tend to deteriorate with age, but increased rigidity of blood vessels caused by atherosclerosis may impair blood vessel response to autonomic reflexes (Collins et al 1980) as well. However evidence for this is limited (Wing & Tonkin 1997). Whatever the precise reasons, the elderly are particularly prone to baroreceptor deconditioning following recumbency (Campbell & Rinken 1985).

In the longer term in the healthy young, there is a day-to day reconstruction of the vascular system to meet metabolic needs. Lack of oxygen or increased blood pressure causes capillaries to expand in size and to sprout new capillaries (Guyton 1991). In the elderly, this response is reduced (Khan et al 1992). This may be advantageous to the seated individual because in conditions of continuous orthostatic pressure an increase in the amount vasculature would simply increase surface area for filtration and fluid loss, while increasing the capillary network will do little to counteract anoxia caused by circulatory stasis.

There is also significant evidence, which will be discussed in detail in section 2.3.4, that the mechanisms which are reasonably effective in preventing stasis oedema in the young are less so in the elderly. This suggests that the losses in plasma volume into the interstitial spaces which are relatively small during sitting in the young (section 2.2.1) may be quite large in the elderly. This is likely to increase the problem of hypovolaemia induced by orthostasis, particularly if it is prolonged. A further limitation to an effective response to orthostatic challenge in the elderly is the lowered maximum heart rate and diminished muscle contractility which together reduce stroke volume (Flanagan et al 1995). Therefore, even if adequate venous return is provided by vaso-constriction, the heart is less able to pump blood to the brain and other vital organs. In addition, vaso-constriction
too may become impaired in some elderly (Lee et al 1966). That is probably why as many as 20% of individuals over 60 years of age have an attenuation of cardiovascular efficiency sufficient to cause symptomatic cerebral hypoperfusion on standing - 'ageing-related orthostatic hypotension' (AOH) which is a significant cause of mortality and morbidity among the elderly (Wing & Tonkin 1997). Hypotension and its effects will be discussed further in section 2.3.3.

Age related deconditioning is compounded in the elderly by pathology and drugs. Diabetes is the most common condition that gives rise to orthostatic hypotension (Wollner & Collins 1992) but a wide range of pathologies can affect the autonomic nervous system such as degeneration of the intermediolateral columns of the spinal cord (Oppenheimer 1980). Caird et al (1973) found two or more of the following factors - the presence of varicose veins, urinary tract infection, anaemia, hyponatraemia, absent ankle jerks and the use of potentially hypotensive drugs to be positively correlated with orthostatic hypotension. Cardiovascular disease is common in the elderly and may affect the ability to respond to cardiovascular challenge. Heart failure, for example, blunts the cardiovascular reflexes to standing from supine (Sanderson et al 1996).

The elderly are prescribed many more drugs than the young (Audit Commission 1994, Roberts & Harris 1993, Nolan & O'Malley 1988) and many drugs in common usage for the elderly can cause orthostatic hypotension: thiazide and loop diuretics, vaso-dilator hypotensive agents, phenothiazines and tricyclic anti-depressants are most commonly the culprits but butyrophenones, benzodiazepines, levodopa, bromocriptine, barbiturates, anti-histamines, glyceryl trinitrate, adrenergic beta-blocking agents and insulin are also frequently involved (Wollner & Collins 1992). Some hypotensive drugs can also contribute to oedema formation, nifedipine being particularly associated with leg swelling (Dubiel et al 1990, Schnapp et al 1987).

In summary, age related deconditioning is likely to make the elderly individual less able to respond to the challenges of orthostasis than a younger one. This is likely to be compounded if the individual is suffering from illnesses which can cause orthostatic hypotension and/or is taking hypotensive, or oedema inducing drugs.

2.2.5 The effects of surgery

During surgery there is inevitable loss of blood and in major surgery of the lower limbs such as hip replacement surgery (An et al 1991, Sculco 1985, Ling 1984) and total knee replacement (Cushner & Friedman 1991), the blood loss may be severe. Typically, there is a drop in haemoglobin level often requiring the transfusion of blood following surgery (Birgegard et al 1991). There tends to be a residual deficit, which is slowly made up over a period of days. The anaemic state of the patient is likely to make him or her tachycardic and a little anoxic and thus even less able to respond to the challenges of orthostasis during prolonged sitting.

While blood loss obviously involves fluid loss, this is a relatively small compared with the fluid deficits caused by pre-operative withdrawal of oral fluids. This is done to prevent fluid inhalation during anaesthesia. While debate continues regarding safe margins and what should be done to prevent dehydration, the fact remains that patients are usually deprived of fluids for many hours prior to surgery, especially as the operation may be re-scheduled, sometimes more than once (Audit Commission 1995). Following surgery, patients may be unable or unwilling to take adequate fluids for some time. Thus, in the immediate post-operative phase, patients are likely to be dehydrated to some degree. This is likely to exacerbate cardiac strain and postural hypotension during orthostasis since the effectiveness of anti-diuretic reflexes are dependent on an adequate fluid input for their effect in increasing circulating volume.

Another likely result of surgery is post-operative pain. Actual pain and fear of pain are likely to reduce patient mobility generally and contribute to unwillingness to mobilise the affected extremities. This will result in less calf muscle pump activity, with increased pooling of blood in the affected limb.
or limbs. It is also likely that this will lead to increased oedema in the affected limbs additional to any swelling that may occur due to operative trauma. Furthermore, some pain relieving drugs such as opioids are known to cause hypotension in some patients (Joint Formulary Committee 1998), thus potentiating any hypotensive effects of orthostasis. Pain itself can affect heart rate and blood pressure (Brand & Abraham-Inpijn 1996). In orthopaedic patients, Vogele & Steptoe (1986) found only blood pressure to be correlated with pain scores. However, their results should be treated with caution, as the small sample size (n = 8) did not allow for analysis of possible confounding factors such as blood loss and medication.

Following abdominal or thoracic surgery, there is a restrictive pattern of breathing with severely reduced inspiratory capacity, vital capacity (VC) and reductions in FRC (Ali et al 1974, Meyers et al 1975, Alexander et al 1973). Vital capacity is the maximal volume of air that a person can expire after a maximal inspiration and inspiratory capacity is the maximal volume of air that can be taken in during deepest inspiration (Vander et al 1998). However, VC is particularly affected by upper abdominal surgery as opposed to lower abdominal and there is no reduction in VC following surgery of the extremities (Ali et al 1974, Alexander et al 1973). FRC is thought to be "the most significant of all lung volumes" (Beecher 1932, Meyers et al 1975) because of its functional consequences (Alexander et al 1973; section 2.2.3). However there is no demonstrable loss of FRC following limb surgery (Alexander et al 1973, Hsu & Hickey 1976) and therefore increasing FRC by a seated posture may be less beneficial than following abdominal surgery.

Another effect, though not strictly surgical, but which is part of the process of surgical treatment, is the ambient temperature of the ward. This is often kept high. An informal survey of 5 hospitals including the one in which the study described took place, revealed that temperatures were kept at 21-25°C in winter, while in the summer they could rise higher - to 28-30°C at times. At high ambient temperatures, blood flow is optimally distributed to increase heat loss. This involves vasodilation in the blood vessels near the surface of the skin (Rowell et al 1969). The need for thermoregulation, therefore, competes with the needs of orthostasis. As overheating appears to be more dangerous than falling, thermoregulation tends to take priority. The large peripheral blood volume that is created, means there is less blood available centrally, which intensifies the orthostatic challenge (Rowell 1993). That is why soldiers standing still on parade are more likely to faint in hot weather than in cold (Vander et al 1998). There is also likely to be increased oedema, as the increased blood volume in the dilated vessels increases filtration pressures further.

In conclusion, orthopaedic limb surgery leads to blood and fluid loss and is likely to cause pain which may lead to a reduction in mobility of the affected limbs. In the seated posture, these may exacerbate the effects of orthostasis by further reducing plasma volume and therefore cardiac stroke volume, because limb immobility reduces lymphatic drainage and calf muscle pump activity. If opioid analgesics are used for pain relief, the hypotensive effects of orthostasis are likely to be potentiated. Another response to pain, tachycardia, may speed up heart rate further. However there is little or no evidence in the literature that orthostatic and pain responses are additive. Although there is an increased FRC in the seated position, which may be beneficial, the benefits are unclear, since there appears to be no reduction in FRC following limb surgery as compared with normal pre-operative values. Benefits will only be gained if the FRC can rise sufficiently to exceed CV. This may not always be possible in an elderly individual in the seated posture.

2.2.6 Conclusions
The elderly individual sitting in a chair following orthopaedic surgery of the lower limbs will be subject to orthostatic pressures tending to reduce cardiac stroke volume and to reduce arterial blood pressure with concomitant increase in blood pressure and fluid loss from the plasma in the legs. The normal immediate responses are tachycardia and peripheral vaso-constriction which are followed by bradycardia and an elevated blood pressure. Reduced renal filtration and shunting blood away from non-essential functions completes the response.
In post-surgical elderly patients, these responses are likely to be less effective due to age-related deconditioning. Swelling of the legs is likely to be greater due to less efficient anti-oedema mechanisms in the elderly, by medication, heat and hormonal changes in women. Immobility, under-thigh pressure of the seat and blood vessel angulation due to the seated posture is likely to cause venous stasis. This tends to promote intracellular oedema and an overall reduction in the efficiency of the circulating blood. Reduced excretion can only help prevent loss of fluid if there is an adequate fluid input. Surgical patients, however, are usually dehydrated before and immediately post-operatively due to nil per orum regimes. Furthermore oral intake by elderly patients tends to be poor.

Paradoxically, at the same time a greater circulating volume is required in the post-surgical patient because of post-operative anaemia and the demands of wound healing. Thus the circulating volume is likely to become inadequate. Some potential consequences of this failure of the circulation to meet demand will be discussed later in section 2.3.

While there is an increase in lung capacity in the seated as opposed to supine posture, this is probably achieved primarily by a reduction in thoracic blood volume. The benefits of increased lung capacity may, therefore, be counterbalanced by reduced gaseous exchange and the effects of prolonged sitting on oxygen perfusion are not known. The potential effect of reduced circulation to the kidneys and gut will be discussed in sections 2.3.8 and 2.3.9 respectively.

Finally, on a local level, poor chair design and non-permeable chair coverings are likely to make prolonged sitting unpleasant. Compression and shearing forces exerted on the sacrum and buttocks coupled with the already depleted blood supply may lead to ischaemia in these areas of skin and subcutaneous tissue.

2.3 POTENTIAL OUTCOMES OF CHAIRNURSING

This section reviews some of the possible outcomes of prolonged chairnursing. The most common complaint that patients seem to have is a feeling of extreme fatigue or tiredness. Fatigue has, therefore, been used as a starting point and has been discussed in some depth. This is partly because fatigue is an abstract concept which is difficult to define and which therefore has no universally accepted definition and partly because an understanding is required of the varied concepts of post-operative fatigue aetiology in order to develop a theory of how chairnursing may affect this outcome. Furthermore, despite over 20 years of study, there have been few if any comprehensive reviews of the definition and the theories of aetiology of post-operative fatigue that could be referred to.

As well as fatigue, other potential outcomes of the effects of chairnursing on the circulation have been discussed. The same format has been used for discussing each outcome. This consists of a definition, brief aetiology, the importance of the outcome, incidence within the target population and a discussion of how the outcome might be affected by chairnursing.

2.3.1 Post-operative fatigue

2.3.4.1 Definition
Fatigue has been defined differently depending on the area of interest and outcomes being measured. At the physiological level it can be considered as "a loss of maximal force-generating capacity that develops during muscular activity" (Lewis & Haller 1991) and can be measured relatively objectively both in vivo (Christenssen et al 1985, Plaitakis et al 1988) and in vitro (Brass et al 1993, Berger et al 1991). It can also be viewed as a social phenomenon and described in terms of a whole population - in other words as an issue of public health or ergonomics. Examples include measurement of fatigue
in high school students (e.g. Takakura 1993), research carried out into effects of pollution (e.g. Scarborough et al. 1989) or stress at work (e.g. Konishi et al. 1991, Makowska 1989), accidents in motoring (e.g. Corfitsen 1993, Zhou 1991) and military and civil aviation (e.g. Caldwell 1997, Rosekind et al. 1996) and the incidence of work related diseases (e.g. Jin et al. 1990). In the latter cases fatigue is often equated with sleep deprivation (Alfredsson et al. 1991).

Because it is an everyday experience, in colloquial usage, 'fatigue' is often used as a synonym of lethargy, tiredness, exhaustion, weakness or sleepiness (Walsh & Dement 1993). This confusion extends to the medical and nursing literature. 'Fatigue' can denote diverse concepts such as loss of muscle strength (Critchley & Richardson 1998), tiredness, or a disease in its own right - chronic fatigue syndrome (Holmes et al. 1988) which Straus (1991) identifies as synonymous with conditions historically known as 'febricula' (Manningham 1750), 'nervous exhaustion' (Flint, early 1900's, quoted in Beard 1869) later renamed 'neurasthenia' (Beard 1869), 'Dacosta's syndrome' (Dacosta 1871) later known as 'effort syndrome' (Wood 1941), 'chronic brucellosis' (Evans 1947) and other more modern syndromes. Fatigue can thus be a subjective, personal experience which either has its roots in physiological processes but is modified by the psychological state of the individual experiencing it or, some would argue, vice versa.

To differentiate between the fatigue of muscles or muscle groups and the whole body feeling of fatigue or tiredness the distinction has been made between peripheral and central fatigue respectively (Wilson & Maughan 1992, Leith 1990). Further differentiation has also been made within the concept of central fatigue between physical and mental fatigue (Chalder et al. 1993, Wood et al. 1990).

Fatigue is usually a normal response to physical or mental activity. This type of fatigue is usually described as 'acute fatigue' in the literature and is defined as being related to an identifiable cause and being rapid in onset, of short duration and relieved by a good night's sleep (Piper 1988, McFarland 1971). The purpose of this form of fatigue (whether central or peripheral) is thought to be protective. The feeling of fatigue indicates the need for rest because if the body or its parts are over-exercised it can be damaged. This is illustrated by the overtraining syndrome encountered in athletes (Parry-Billings et al. 1990).

It has also been suggested that fatigue develops when the body either needs all its resources to fight infection such as influenza or is not functioning to its normal ability. It is postulated in the latter case, that our ancestors, if they continued normal activities whilst not functioning optimally, had a lower chance of finding or catching food and a greater chance of becoming another creature's food themselves. There may be a selective advantage in feeling fatigued and staying out of harm's way in a protected environment. The observation that some patients with chronic fatigue respond to surgery which cures nasal problems interfering with efficient olfaction is used to support this theory (Chester 1993). However, in the absence of controlled trials a psychological explanation of this observation cannot be discounted.

Depending on level or circumstances however, fatigue can become a symptom of illness (Shwartz et al. 1993). In fact, excessive fatigue may be the most common symptom of both acute and chronic illness (Ffrench 1960). For example, 18-34% of respondents in one community survey reported always feeling tired in the past month (Cox et al. 1988). Incidence, characteristics and severity of symptoms of fatigue vary depending on where, in what context, and in which population fatigue is assessed (Ridsdale & Mandalia 1993, Lawrie & Pelosi 1993). This type of fatigue, described as 'chronic fatigue' by many authors is induced by a disease or treatment, is insidious in onset and is rarely dissipated by sleep, while rest or diversion gives only temporary relief (Hart et al. 1990).

While there is no universal definition of fatigue in this context, Ream and Richardson (1996) suggest a definition of fatigue based on concept analysis methods suggested by Walker and Avant (1995) as follows:
"Fatigue is a subjective, unpleasant symptom which incorporates total body feelings ranging from tiredness to exhaustion, creating an unrelenting overall condition which interferes with the individuals' ability to function to their normal capacity."

With improvements in the control of pain and other symptoms, many patients report fatigue to be the most or one of the most distressing symptoms of conditions such as cancer (Smets et al 1993), silicone poisoning (from silicone breast implants) (Dowden 1994), rheumatoid arthritis (Crosby 1991), fibromyalgia (Waylonis & Perkins 1994, Yunus et al 1989), postoperative brain damage (Aberg et al 1987), lung disease (Gugger 1991), cardiac failure (Shafer & Shober-Potylycki 1993, Mayou et al 1991), intensive care (Gipson 1991), lupus erythematosus (Krupp et al 1990), ankylosing spondylitis (Calin et al 1993) multiple sclerosis (Hubsky & Sears 1992), HIV (Darko et al 1992), and hyperparathyroidism (Kristoffersson et al 1992).

Many patients complain of postoperative tiredness (Reid 1992) and when this persists for an unusually long period it is known as the postoperative fatigue syndrome which has recognisable characteristics of chronic fatigue (Cordova-Martinez & Marcen 1992). The definition proposed by Ream and Richardson (1996) is a good starting point for defining post-operative fatigue. However, this definition, by including the phrase "an unrelenting overall condition", is only applicable to post-operative fatigue which has a chronic course. While there is plentiful evidence that in many patients who have had abdominal or thoracic surgery fatigue persists over a significant period of time: 1-2 months (eg Christensen & Kehlet 1993), the evidence for the presence of such chronic fatigue is less compelling in patients who have had limb or other peripheral surgery. In any case, the presence of chronic fatigue does not exclude the possibility of acute fatigue being superimposed on and affecting levels of fatigue already elevated by chronic fatigue.

For the purposes of this thesis the following definition therefore will be used, based on Ream and Richardson (1996), which is not confined to chronic fatigue:

Fatigue is a subjective, unpleasant symptom which incorporates total body feelings ranging from tiredness to exhaustion.

2.3.4.2 Theories of aetiology
If there is still much debate about a definition of post-operative fatigue, there is even less agreement about the possible causes - "a quagmire of complexity" as described by Pepperman (Reid 1992). While most authors agree that the causes are multifactorial (eg Schroeder & Hill 1991), there is little agreement about which factors are the dominant ones. Broadly, there are those who believe that post-operative fatigue is principally a side-effect of the physiological response to surgical trauma and/or anaesthesia while others propose that the social and cultural conditioning and psychological state of the patient pre and post-operatively are more significant than purely physical responses.

Proponents of the psycho-social theory of post-operative fatigue genesis point out that despite over 20 years of research focusing on the physical causes of fatigue, results are conflicting and inconsistent (Salmon & Hall 1996). For example some studies have found postoperative supplemental feeding to reduce fatigue (Keele et al 1997) while other have not (Watters et al 1997) and, in general, single modality interventions to reduce post-operative fatigue have not been very successful (Kehlet 1997). It has been claimed that many patients only experience post-operative fatigue when they return to work, long after their surgical wound has healed and the metabolic and endocrine responses to surgical trauma have subsided (Editorial 1979). A long convalescence does not always prevent it (Moss et al 1957).

Other general observations in support of the psycho-social theory are that post-operative fatigue is a uniquely human experience - domestic animals after major surgery quickly return to vigorous activity (Allam & Martin 1958); fatigue as a symptom is much more commonly seen in developed Western
societies which actually do less physical work; the offer of monetary reward is a more potent in enhancing physical performance in endurance exercise than correction of metabolic disturbance (Felig et al 1982) (ie fatigue is a matter of motivation rather than a physical state) and that physical exercise can reduce feelings of fatigue rather than increase them (Thayer 1987).

There are a number of difficulties with some of these arguments. The assumption seems to be made that because a subject, in this case post-operative fatigue, has been studied for a long time, it has been studied exhaustively. This is not the case. Relatively little research has been carried out into how postoperative fatigue may be affected by the effect of surgery on perfusion and the central nervous system and little or no research has been conducted on the effect of various post-operative mobilisation regimes. Even if uni-modal interventions such as effective pain control (Schulze & Thorup 1993) are not successful this does not mean that multimodal approaches may not be more so. Although there is no controlled clinical trial data to support this at present (Kehlet 1997), approaches combining various anti-fatigue therapies seemed to reduce post-operative fatigue after colonic surgery in un-controlled studies (Bardram et al 1995, Moinische et al 1995) as compared with levels expected from the literature.

The observations of animals post-surgery are not based on careful ethological studies of total behaviour, but observations of occasional activity. Therefore they do not prove that animals do not suffer post-operative fatigue, merely that they respond to some stimuli which are highly motivating despite having had recent surgery. The assumption that fatigue is more common in 'developed' countries is an assumption and one that is not necessarily borne out by surveys carried out in 'underdeveloped' communities. Finally the fact that a fatigued individual can temporarily overcome their fatigue when given a strong motivating stimulus does not ipso facto mean they are not and have not been fatigued. Rather the needs of tissue repair and recuperation have been superseded by a more pressing need. Similar situations have been observed in humans under extreme situations. For example severely wounded soldiers on the battlefield may continue to make great exertions while it is necessary for survival (Beecher 1936). No one would suggest that this proves they are not wounded. This argument also applies to the animal model described previously.

The psycho-social theory of post-operative fatigue appears to centre on three propositions. The first is that patients do not feel physical fatigue, rather they feel anxiety and depression and that these are relabeled in the physiologically oriented context of a surgical ward as fatigue (Salmon & Hall 1996a). The direct evidence quoted in support of the above proposition appears to be that patients in one study who reported mental fatigue post-operatively tended to be those who had reported more negative mood pre-operatively (Aarons et al 1996). This finding has not been corroborated by other workers (Schroeder & Hill 1993). Supporting evidence is found in the correlation between state-anxiety pre-operatively \( r = 0.68 \) and the increase in fatigue postoperatively correlated with increase in anxiety \( r = 0.65 \) in one study (Christensen et al 1986) and in a correlation found between mental and physical fatigue and anxiety and depression measured at the same time in another (Pick et al 1994). Further supporting evidence is found in the observations that anxiety and depression can remain elevated for long periods following surgery (Vogele & Steptoe 1986, Johnston 1980, Chapman & Cox 1977), similarly to fatigue.

The main weakness of the basic proposition is that while subjects may indeed express a single internal state in emotional or physical terms (Pannebaker 1982) they can do so in either direction depending on the social context. Thus, while anxiety and depression may be relabeled as fatigue in the physiologically orientated context of a surgical ward, equally, fatigue may be relabeled as anxiety or depression in the psychologically orientated context of a psychologist's or psychiatrist's research project. The supporting evidence, too is open to many different interpretations. Correlation does not imply that two variables are one and the same. It is perfectly feasible that patients can be fatigued and anxious and depressed at the same time. A reasonable alternative interpretation might be that patients are anxious and depressed because they are feeling fatigued and this has lead them to believe that there is something wrong with them.
The second proposition is that fatigue is the response to the patient's perception of the surgical stimulus. Evidence comes from the observation that patients who undergo joint arthroplasty do not experience postoperative fatigue (Aarons et al 1996), while patients having abdominal surgery do (eg Zeidermann et al 1991). It is argued that abdominal surgery generally signifies a threat to well-being or life, while arthroplasty "usually presages a marked improvement in function and well-being". It seems, however difficult to see how this distinction can be made between limb arthroplasty and cholecystectomy, both of which are treatments for generally non-life threatening conditions, albeit very painful ones, with excellent prognoses (Levy et al 1995). Like all abdominal surgery, cholecystectomy is usually followed by post-operative fatigue (Persson et al 1990). Therefore, it seems equally plausible, that surgery of the abdomen and thorax may have a physical effect which predisposes to fatigue which surgery of the limbs does not. A more useful comparison, perhaps, might have been between hip replacement surgery and fractured hip repair which carries a poor prognosis (Karpman & Del Mar 1995). What this theory is unable to explain, however, is why different methods of carrying out the same operation have different outcomes on fatigue - for example laparoscopic as opposed to open cholecystectomy (Hill et al 1993). To this end another theory is constructed - the third proposition.

The third proposition is that post-operative fatigue occurs because it is "a cultural expectation held by both patients and staff responsible for their care, that fatigue is an inevitable consequence of major surgery and that it will prevent a rapid return to normal activities" (Salmon & Hall 1996). Because people tend to behave in ways that reflect their expectations (Rosenthal 1966), patients expected to feel fatigue will feel it, or at least say they do. Thus, in a study comparing open cholecystectomy with laparoscopic cholecystectomy, patients undergoing laparoscopic cholecystectomy feel less fatigued because the researchers and staff looking after them expect them to be so.

The main evidence offered in support of this premise is the comparison between man and animals, specifically greyhounds, which could return to racing 3 weeks after hysterectomy (Allam 1958) and therefore (presumably) were not fatigued. The weakness of this argument has already been touched upon. It is also incorrect to equate speed of recuperation in a relatively small animal with a higher metabolic rate and therefore likely to have a shorter healing time to that of man. It is also dangerous to make predictions about the behaviour of one species based on that of another with a different developmental history. Ancestral dogs may have had a need to keep up with their pack to survive and therefore fatigue would have been a disadvantage. Ancestral humans, on the other hand, may have had a selective advantage in fatigue encouraging them to remain in their protected environment.

However, it is the basic proposition that has some fundamental weaknesses. Firstly there is little evidence that clinicians, in general, expect patients to be as profoundly fatigued as they are, nor is there evidence that they convey such expectations to their patients. A striking feature of personal accounts of the experience of post-operative fatigue is that it was wholly unexpected (Smith 1992). Raper (1992) interviewed a number of patients experiencing post-operative fatigue after day surgery. All her interviewees had assumed that the surgical intervention was not of major significance (as it did not involve hospitalisation) and that they therefore ought to make a rapid recovery. If return to work was discussed with general practitioners or practice nurses, the advice given was that they should go back as soon as they themselves felt fit. There is no suggestion that any of the patients had been told that post-operative fatigue was likely to occur. Indeed, there is little to suggest that the majority of clinicians are acutely aware of the significance of post-operative fatigue or that they are expecting their patients to develop it. In many, if not most studies of post-operative recovery, fatigue is not considered at all (eg Attwood et al 1992, Peters et al 1991). In others it is not discussed in the introduction but appears as a significant outcome in a questionnaire such as POMS (McNair et al 1971) which has been applied in unrelated research (eg Oberst & Scott 1988, Galloway & Graydon 1996).

Secondly just because clinicians expect certain psycho-social outcomes, does not necessarily mean they will occur, ie clinical prophecies are not necessarily self-fulfilling. For example, until recently
it was assumed that older women would have a lower quality of life and more symptomatology than younger ones if breast cancer surgery was followed by adjuvant therapy as compared with surgery alone (Wyatt and Friedman 1998). Probably as a consequence, an inverse relationship between age and aggressiveness of treatment exists in the USA (Clark 1992). However research has not borne out clinicians’ expectations. There appears to be no difference in quality of life outcomes in older women who have had adjuvant therapy and those who have not (Wyatt & Friedman 1998, Fleming & Fleming 1994, Morrow 1994).

The psycho-social theory of post-operative fatigue is relatively new and to date the evidence to support it is weak. More research may be beneficial to establish what role psycho-social factors have to play in post-operative fatigue and whether patients would benefit from psychological interventions post-operatively. The benefits of some pre-operative psychological interventions on post-operative outcome have been established (Johnston & Voge 1993) and therefore continuing this process into the post-operative period would appear to be a promising line of enquiry. However, at present this theory does not offer a plausible explanation of why chairnursing appears to be so fatiguing.

The central assumption of the \textit{physiological} theory is that fatigue is one of the symptoms of the complex response to injury. This response, which is common to any major trauma such as burns and haemorrhage as well as surgery consists of metabolic, hormonal, haematological and immunological events. These are reviewed by Hall (1994). There are a number of further assumptions made by some authors to guide research and development of practice. One is that if correlates of fatigue can be found and corrected for, fatigue will be reduced (Hill et al 1993). Another is that if postoperative fatigue is a response to a stimulus, then the size of the response should be related to the size of the stimulus. Therefore, the greater the surgical insult to the body, the more post-operative fatigue should be expected (Christensen & Kehlet 1993).

The metabolic response to surgery has been extensively studied for its potential to influence post-operative fatigue. Cuthbertson (1932) divided the response into an early ‘ebb’ phase during which there was hypovolaemia and adrenal response, followed by a ‘flow’ phase which involved a mobilisation and loss of protein at an accelerated rate. The length of flow phase depended on the size of the injury. The flow phase was then gradually replaced by an anabolic phase, during which proteins and reserves lost in the previous phases were replenished. There is a loss of weight during the flow phase and because protein is mobilised from the muscles, there is a loss of muscle function. This loss of weight and muscle function has been correlated with reported feelings of fatigue (eg Shroeder & Hill 1991, Christensen & Kehlet 1984). Furthermore, those patients who have the lowest protein reserves before surgery are most likely to suffer prolonged fatigue (Shroeder & Hill 1993). It was hypothesized from these results that fatigue may simply be the result of reduced voluntary muscle function. However, the administration of pre-operative enteral nutrition, which preserves body protein stores post-operatively, did not abolish post-operative fatigue (Cruickshank et al 1990, Jensen & Hessov 1997). More recently, however, the administration of growth hormone treatment prior to and for 7 days after abdominal surgery \textit{did} significantly reduce fatigue in a randomised, controlled, double-blind trial (Kissmeyer-Nielsen et al 1999). Therefore, it seems that only reversal of the catabolism of the flow phase can prevent the degradation of muscle mass. It is likely that these recent findings will renew interest in this area.

Other studies concentrated on skeletal muscle enzyme activities (eg Christensen et al 1990), pain reduction (eg Lanz et al 1982, 1984, Hjortso et al 1985) and the reduction of the surgical insult, most notably the introduction of laparoscopic surgery (Schulze & Thorup 1993). Of these only laparoscopic surgery has proven to be consistently successful in reducing post-operative fatigue (Stage et al 1997, Hill et al 1993). Therefore the premise that the amount of post-operative fatigue has some relation to the size of the surgical insult appears to be largely correct. However, like psychosocial theories, the metabolic theories too have limited potential for explaining fatigue induced by chairnursing, since sitting is an inactive process which does not appear to increase metabolic demands greatly.
In contrast to the metabolic and endocrine aspects, relatively little attention has been paid, to date, to the central nervous system. This is, perhaps, surprising since post-operative fatigue (Davis & Bailey 1997) is likely to be a 'central' fatigue and it would seem intuitive to seek central reasons for central problems. Fatigue has been extensively investigated by sports physiologists and a number of theories of central fatigue have been developed and investigated (see Maclaren et al 1989 and Newsholme et al 1992). The finding that accumulation of ammonia in the blood and brain may increase fatigue may be of significance to this thesis, as orthostasis involves a reduction in renal function.

Tryptophan is converted in the brain into the neurotransmitter 5-hydroxytryptamine (5-HT) which is thought to cause not only fatigue (Fernstrom 1990) but also anxiety and depression (McNaughton 1993). This suggests that the relationship between fatigue, anxiety and depression found in a number of studies (Christensen et al 1986, Pick et al 1994) may be due to all three being subject to the same central controls. This is supported by the finding that giving high dose steroids such as glucocorticoids (which are known to have a mood enhancing effect) 90 minutes before major abdominal surgery was found to reduce post-operative fatigue (Shulze et al 1990). The higher levels of fatigue despite better control of pain with patient controlled analgesia (PCA) found by Passchier et al (1993) may well be the result of the central effect stimulated by larger doses of morphine in the PCA group. Similar results were found with epidural anaesthesia (Zeiderman et al 1991). Therefore it is possible that the relationship between fatigue, anxiety, and low mood in post-operative patients may be the result of one or more agents acting to increase all three simultaneously.

A recent finding that there appears to be an increase in the amount of free tryptophan post-surgically in coronary artery bypass graft patients (Yamamoto et al 1997) is likely to stimulate further interest in the study of central fatigue in post-operative patients. It is known that increased levels of tryptophan in the brain increase the rate of formation of 5-HT in animal models (Blomstrand et al 1989). Branched chain amino acids (BCCAs) (leucine, isoleucine and valine) enter the brain on the same amino acid carrier. Therefore it is likely that the two types of amino acid compete for entry into the brain (Fernstrom 1990). There is some evidence including two controlled trials (Blomstrand et al 1991) that adding BCCAs to the diet can reduce fatigue and improve physical and mental performance in sportsmen. There are clear implications in this for the management of surgical patients which require investigation.

However, tryptophan is released from the muscles during activity and therefore is considered to be a factor in exercise-induced fatigue. Therefore this theory of fatigue is unlikely to explain the inactivity induced fatigue occurring after prolonged chairnursing. It is possible that there are other centrally active fatigue-stimulating processes other than the effect of ammonia already discussed, which may be affected by inactivity or posture. However, to date there is insufficient evidence to postulate such a hypothesis in respect of fatigue induced by chairnursing.

Little attention also, has been paid to the cardiovascular and perfusion aspects of fatigue although oxygen deficiency and accumulation of metabolites is thought to be the cause of physiological fatigue by many authors (Shmidt & Thews 1989, Griffiths 1981, French 1960) and low blood pressure has been shown to have a relationship with fatigue (Pilgrim et al 1992, Wessely et al 1990). Furthermore, fatigue appears to be a symptom of nearly all respiratory and cardiovascular diseases such as constrictive pericarditis (Higgins & Ports 1982), lung disease (Gugger 1991), inappropriate pacemaker settings (Heldman et al 1990), heart failure (Baligadoo et al 1990), chronic lung disease (Morgan 1991) and impaired capillary blood flow (Simpson 1992). It seems, therefore, likely that cardiovascular disturbance post-operatively may have an effect on post-operative fatigue.

Studies have shown that there is a reduction in exercise capacity after surgery and that post-operative fatigue is related to this reduction (Christensen et al 1985). Treatment with amino acid and improvement in nitrogen balance (which maintain muscle tissue) does not improve exercise capacity (Guess et al 1984). This may be because the cardiorespiratory system is delivering oxygen
suboptimally due to disturbances caused by the surgical process. Under these circumstances the body, although with adequate metabolic substrates and with intact musculature, can only function to the level that the diminished oxygen supply will permit. This view is supported by the observation that mortality and morbidity was significantly higher in patients who were undergoing abdominal surgery with empty iron stores (measured by assuming low serum ferritin equated to empty iron stores) as compared with those with normal stores (Harju 1988).

Rose & King (1978) surveyed likely causes of post-operative fatigue. They listed many alterations of cardiovascular functioning that occur post-operatively and after trauma generally. These included altered heart rate, reduction of maximum aerobic power and impaired response to orthostasis. These are familiar effects of immobility which have already been discussed (sections 2.2.1-2.2.3). They all contribute to a reduction in perfusion of the body and the brain.

If aspects of perfusion are considered, they provide a potential answer to some of the questions that the literature poses - for example the apparent paradox that major limb surgery causes little or no fatigue (Aarons et al 1996) while abdominal surgery does (Keele et al 1997). It has been shown that following abdominal surgery there is a restrictive pattern of breathing which is absent in limb surgery (Hsu & Hickey 1976). This pattern of breathing significantly reduces lung capacity which is likely to lead to reduced efficiency of the circulation. The reduction in fatigue in laparoscopic cholecystectomy as compared with open cholecystectomy appears to be mirrored by reduced interference with respiratory muscle function (Da Costa et al 1995).

It is possible also, that one of the reasons why progress in post-operative fatigue research been limited is that attention has been focused exclusively on the surgical procedure itself rather than on the whole process of treatment, including post-operative rehabilitation. If it is accepted that the feeling of fatigue is affected by cardiothoracic and lung function, then physical activity of the patient after surgery becomes an important variable. Bedrest, chairnursing and active ambulation and exercise all are likely to have different impacts on the levels of tissue perfusion. For example, chairnursing is likely to be associated with venous pooling. When venous pooling occurs, the reduced venous return is associated with a lowered intracardiac packed cell volume which results in sub optimal oxygen delivery to the tissues (Simpson 1990). To date few or no studies of postoperative fatigue have controlled for patient activity. This may be a significant confounding factor.

Supporting this view are the results of studies in which immobility and inactivity have been identified as primary causes of fatigue in chronic conditions such a rheumatoid arthritis (Belza et al 1993) and end stage renal disease (Brunier & Graydon 1993). Exercise programmes have been shown to reduce fatigue in a number chronic conditions such as rheumatoid arthritis (Stenstrom 1994) and in healthy individuals (Thayer 1987). Exercise may therefore, also be effective in relieving post-operative fatigue. This suggests that there is a need for research into the effects of different mobilisation regimes on post-operative fatigue.

2.3.4.3 Significance and prevalence

If for no other reason, post-operative fatigue should be a matter of concern because when it occurs it causes very real and disabling distress to patients (Smith 1992). Possibly because the fatigued patient becomes less mobile, fatigue can cause muscular weakness (Schroeder & Hill 1991) and a reduction in performance capacity (Barofsky & Legro 1991) necessary for rehabilitation (Belza et al 1993, Morris 1982), thus prolonging hospital stay with consequent cost to the health service and the general economy due to loss of productive labour (Linden 1968, Fleming 1958).

Fatigue was shown to be a barrier to mobilisation by a number of studies (eg Moiniche et al 1994, Holas & Faulborn 1993). Conversely, it has been suggested that reduced fatigue levels are likely to be a factor in the more rapid mobilisation and quicker discharge of patients who have undergone laparoscopic as opposed to open cholecystectomy (Schulze & Thorup 1993). Possibly because it
interferes with muscle control (observable, for example in eye pupil function; Cheng & Catalano 1989, Yoss et al 1970), fatigue interferes with self care (Rhodes et al 1988) and socialisation (Jamar 1989). Muscle control problems may also explain the connection between fatigue and lower back pain (Redeker 1993). It has been shown that fatigue can be associated with potentially dangerous arrhythmias even in healthy adults (Stamler et al 1992) with prolonged fatigue being associated with cardiac death (Appels & Otten 1992). In the latter case, however, a causal relationship has not been demonstrated and it is fairly likely that the failing cardiovascular system is responsible for the fatigue and not the other way around.

Fatigue has been directly associated with depression (de Bleucort et al 1993) although, as has been discussed above, it is possible that both fatigue and depression are the end results of the same central processes. Nevertheless, if that is so, factors increasing fatigue are also likely to increase depression and vice-versa. Depressed elders are particularly susceptible to the effects of fatigue where multiple skills need to be used (Hayslip et al 1990) and multiple skills are needed for rehabilitation.

Extreme fatigue may also cause a drop in skin blood perfusion. A drop in skin blood flow has been observed on physical exhaustion in collapsed fun runners and exercising volunteers. Hales et al (1986) suggest this is a reflex response to reduced central venous pressure caused by cutaneous vasodilation which in turn is caused by the need to lose heat and is compounded by dehydration in fun runners. However, a drop was also observed to occur in three healthy volunteers who exercised to exhaustion in conditions of cold and wet and who were not dehydrated. Wiles et al (1986) suggest this is therefore likely to be a non-specific consequence of acute fatigue rather than of heat stress.

It could be argued that if such a reduced skin perfusion occurs in fatigued post-operative patients, it is likely to make the patient more susceptible to pressure damage and have deleterious effects on wound healing. However it must be borne in mind that the effects reported by Hales et al (1986) and Wiles et al (1986) occurred under extreme temperature conditions and following very vigorous muscular exercise. They may well not occur in patients who are exhausted by inactivity rather than activity and under no great temperature stress.

Numerous studies have shown that many patients who undergo abdominal surgery are likely to suffer severe and protracted fatigue which may last up to 3 months (Hill et al 1993). The usual pattern is for mean fatigue to increase to a maximum at 2-5 days postoperatively and then to tail off gradually over the next month to pre-operative levels (Buxton et al 1992, Christensen et al 1982). It is not clear whether patients who undergo limb surgery have similar fatigue processes. Vogele & Steptoe (1986) observed a pattern of fatigue similar to that found in abdominal/thoracic patients, although of shorter duration. This finding was not confirmed by the more recent and larger study by Aarons et al (1996) (n = 63) which found that post-operative fatigue tended to remain at pre-operative levels. These studies may, however, not be contradictory. Vogele & Steptoe (1986) assessed patients pre-operatively and on days 1-7 postoperatively and observed a peak of fatigue on day 2, with fatigue levels virtually returning to pre-operative levels by the seventh day. Aarons et al (1996) studied the patients pre-operatively and at one week and seven weeks postoperatively. If there was a similar day 2 peak of fatigue in their sample, Aarons et al would have missed it. Moiniche et al (1994) also found a rise in fatigue between days 1-2 in 20 patients undergoing hip replacement which fell off to pre-operative or even below pre-operative levels depending on the analgesia regime. The 22 total knee replacement patients in the same study appeared to fluctuate considerably in their perception of fatigue over the first 7 post-operative days. In other studies of post-operative fatigue involving orthopaedic patients (Ritter et al 1995 and Cleary et al 1991), the first assessment was carried out at 3 months and 6 months respectively and so gives little information on the immediate post-operative period.

2.3.4.4 Fatigue and chairnursing
If it is accepted that a reduction in effective perfusion is likely to increase fatigue, then clearly chairnursing is likely to be a fatiguing experience. Blood pooling and volume loss due to orthostasis, immobility and posture are likely to affect perfusion adversely. Furthermore, since renal function is
likely to be reduced during orthostasis there is likely to be an increase of circulating ammonia which is thought to have a centrally fatiguing effect.

The view that chairnursing increases fatigue is supported anecdotally by complaints of severe fatigue following lengthy chairnursing by postoperative patients (Gebhardt & Bliss 1994, Radford & Wright 1980) and healthy individuals who have sat for prolonged periods during long-haul flights (Wooldridge 1990). Some of flight fatigue may be attributable to disturbance of circadian rhythms during east/west flight causing shortening/lengthening of day and changes in time zone (Wooldridge 1990). However individuals flying north/south also experience fatigue (Humphries 1981) and exercise designed to increase circulation (preferably in a standing position) is thought to be effective in reducing post-flight fatigue (Anon 1994). There is little or no direct research evidence, however, of the effect of chairnursing on fatigue.

2.3.2 Heart rate

The resting heart rate (i.e. the number of contractions of the heart in a given time) is around 60-80 beats per minute (bpm) and varies between individuals and between men and women as a whole. The resting heart rate tends to rise with age (Colbert 1993), during bedrest (Halar & Bell 1988) and in individuals leading a sedentary lifestyle (Valbona 1982). However, if the cardiovascular system is stressed either by reduction in functional blood volume through shifts (such as those caused by orthostasis as described in 2.2.1) or through loss of total blood volume, as in haemorrhage or loss of fluid or if greater than normal demands are placed on the body due to exercise for example, the heart rate increases so that the loss of volume or greater demand can be met by a faster circulation of blood. During vigorous exercise such as running, the heart rate can rise to up to 190 bpm or more in young adults or 165 bpm in 60 year olds (Colbert 1993). Adaptive tachycardia of this nature is usually of short duration and rapidly returns to baseline levels when the stress is removed.

Tachycardia can also be used to describe any condition where the heart exceeds a given rate. In this context tachycardia is usually defined as a pulse rate > 100 bpm (Spraycar et al 1995, Anderson 1994, Cowell 1989). It is with this latter definition that this part of the thesis is concerned.

Each cycle of heart contraction consists of the contraction (systole) and a relaxation (diastole). During the diastole the heart fills with blood, the heart muscle rests and it is only during the diastole that the coronary blood flow to the subendocardial portions of the left ventricle occurs (Gannong 1997). Although both systole and diastole can decrease in duration when the heart rate increases, the systole is much more fixed than the diastole. Therefore, tachycardia is only possible if there is a proportional loss of time in diastole. At 75 bpm diastole comprises 66% of the cycle (Barger & Richardson cited in Gannong 1997). At 200 bpm this drops to 47%. At the same time, due to the increased muscular activity and reduced resting time there is a rise in myocardial oxygen consumption (Colbert 1993). The paradox, therefore, arises of increased cardiac oxygen requirement coupled with reduced blood supply. Very high rates (180-220 bpm) tend to be ineffectual because there is insufficient filling time to fill the heart with blood before the next contraction (Colbert 1993). If the venous return is reduced, such as occurs during orthostasis, the rate at which the heart becomes ineffectual is lowered because of reduced filling pressure to the heart.

There is little to suggest that short term, adaptive tachycardia is harmful in an otherwise healthy individual. However, it is thought that where tachycardia occurs at rest and for prolonged periods of time or in an individual with heart disease it may precipitate cardiac failure, cardiac arrhythmias, angina or even a myocardial infarction which may prove fatal (Colbert 1993). It is difficult to establish what the incidence of tachycardia - induced morbidity might be in the target population. However, mortality from cardiac conditions is high among orthopaedic patients accounting for 23% of all deaths following hip fracture repair (Perez et al 1995). Vogele & Steptoe (1986) measured heart rate following hip replacement surgery and found peak levels at day three following surgery, with heart rate remaining high up to the 11th post-surgical day. However, since the regime of mobilisation
was not recorded, nor the patients' Hb levels post-operatively and the subject population was small (n = 8), these results have to be treated with caution and do not offer an explanation of the phenomenon.

Heart rate tends to rise with age and therefore individuals in the target population are likely to have a slightly raised heart rate at baseline (Lye & Walley 1998). Before undergoing surgery members of this population are likely to have had at least partial loss of mobility for some time due to osteoarthritis (hip replacement and knee replacement patients) or complete loss of mobility for a shorter period of time (fractures). This is followed by at least a complete day of bed rest on the day of surgery and often for a few days after. Mobility is limited for some time following surgery by the post-operative regime aimed at successful union of the repair and by pain. The heart rate tends to rise in individuals with a sedentary life and this rise can be quite rapid during complete immobilisation (Halar & Bell 1988).

In addition to a likely higher baseline, extra demands are placed on the cardiovascular system by surgery. Blood loss occurs during surgery and patients are likely to be anaemic post-surgically (An et al 1991, Cushner & Friedman 1991, Bierbaum et al 1999). Because of the reduced haemoglobin level, blood has to be circulated around the body more rapidly to compensate for the loss of carrying capacity. Paradoxically, the demands of wound healing and other trauma related processes increase energy expenditure in the body after surgery (Watters et al 1992) and further increase the demands on the circulation.

It is difficult to determine from the literature what the possible effects of chairnursing might be on the heart rate in the target population. On the one hand data from healthy young volunteers suggests that quiet sitting elicits a lowered heart rate and the immediate response to orthostasis in the elderly tends to be one that involves less tachycardia than in the young (section 3.2.4). However it must be noted that the subjects of chair sitting experiments in young people are otherwise mobile individuals. The effects of daily immobility in a chair coupled with increased bedrest and a recent history of reduced or absent mobility may be different.

The long-term response of the elderly to orthostasis is not known and the long term demands may be greater than in the young due to the likelihood of plasma loss in the lower limbs and oedema (section 2.3.3 below). Furthermore, when venous pooling occurs, the reduced venous return is associated with a lowered intracardiac packed cell volume which results in sub optimal oxygen delivery to the tissues (Simpson 1990) and this is likely to be compounded by the effects of post-operative anaemia.

Tachycardia, is therefore a possible outcome in post-surgical orthopaedic patients sitting for long periods. However there is little or no research data to support this hypothesis at present, nor to determine how significant a part lengthy chairnursing may play, if any.

2.3.3 Orthostatic hypotension

Blood pressure is considered by some to be an index of some of the most fundamental physiological processes (Stafford & Batchelor 1992). Hypotension is generally considered to be an abnormal condition during which blood pressure is not adequate for normal perfusion and oxygenation of the tissues (Anderson et al 1998). Few English medical dictionaries or texts on human physiology define it in terms of actual blood pressures. Partly this may be because blood pressure varies between individuals, between sexes and between different age groups and a blood pressure that is adequate for one individual may not be for another, and indeed the requirement may vary in the same individual at different times. Where an actual blood pressure is quoted to define hypotension, then it is either a blood pressure which is systolic < 110 and diastolic < 70 bpm (Blanchards Scientific Publications 1997) or a systolic blood pressure < 100 bpm (Fox 1999).
While many of the symptoms of acute hypotension are unpleasant such as dizziness, fatigue (Nozawa et al 1996) and nausea, probably the main danger is the risk of falls. Cerebral blood flow tends to be very stable at medium and high blood pressure ranges. However if the mean blood pressure (see section 2.5.2 for definition) falls below about 60 mmHg, cerebral blood flow falls off very rapidly (Lassen 1959, cited by Guyton 1991). This can result is loss of consciousness and a fall (fainting, syncope). Fainting has been considered to be adaptive by some (Vander et al 1998) because once on the ground, orthostatic stresses are removed, blood pressure levels are restored to normal and the subject usually quickly recovers. However, in the elderly, falls can be the cause of serious injury and mortality. In the United States 15% of falls in the elderly are reported to result in physical injury requiring medical intervention, including head injuries and broken bones, most commonly fractured neck of femur (Tinetti & Speechley 1989). Falls often lead to a fear of further falling which can decrease mobility and confidence. In one study 50% of elderly people who had fallen admitted to avoiding activities because of fear of further falls (Nevitt et al 1989). A fall could therefore be an important setback in the rehabilitation of an elderly individual following arthroplasty. Furthermore, in a prospective study of postprandial hypotension (that is an acute hypotension which occurs after eating due to blood pooling in the splanchnic region) in nursing home residents, Aronow and Ahn (1997) found that maximal decrease in postprandial systolic blood pressure was an independent risk factor for falls, syncope, new coronary events, new stroke and total mortality. While this does not necessarily imply a causal relationship, such a possibility cannot be rejected either. The most common form of acute hypotension in the elderly is orthostatic hypotension which has a similar aetiology to post-prandial hypotension (ie, it is caused by a pooling of blood in one region, thus reducing the amount available to the central circulation) and thus may pose similar risks.

Orthostatic hypotension is associated with orthostatic disregulation in the elderly. It is defined as a fall in arterial pressure on assuming the erect position or on maintaining it for prolonged periods (Walton et al 1994). Even in asymptomatic elderly individuals, there is a tendency to respond to orthostatic cardiovascular stress with a reduced tachycardia and compensation with increased peripheral vasoconstriction (Wieling et al 1992) as compared with young individuals. However, this response is not as efficient as a mainly tachycardic one particularly if the peripheral vaso-constrictive one is also impaired, and the transient drop in blood pressure on standing from supine tends be greater in the elderly (Rudas et al 1996).

In a proportion of the elderly, cardiovascular response becomes inadequate to orthostatic challenge and the affected individuals develop syncope and fall on standing up. Estimates of prevalence among the elderly are varied, but it is thought to be 4%-33% depending on the population sampled (Ward & Kenny 1997). Most falls due to loss of postural tone without loss of consciousness (sometimes referred to as drop attacks) are probably also caused by cardiovascular disorders. For example, in a series of 35 patients referred with drop attacks to a dedicated 'syncope and falls centre', diagnosis of the reason for falling was established in 25 (71%) of whom only one did not have a diagnosis of cardiovascular disorder. 5 had orthostatic hypotension (O'Mahoney & Foote 1998).

As well as old age, bedrest is also thought to make individuals more prone to orthostatic hypotension because of cardiovascular deconditioning. According to Dittmer & Teasell (1993) this occurs after about 3 weeks of bedrest in young individuals and earlier in the elderly. However Melada et al (1975) observed this phenomenon after 5-14 days of bedrest in young, healthy volunteers. The reasons for this are not altogether certain, but are likely to be a combination of factors including vasovagal disturbances caused by immobility.

In the young, administration of propranolol reduces tachycardia and the incidence of syncope on standing after bedrest (Melada et al 1975, Chobanian et al 1974). This suggests that the tachycardia after bedrest is abnormally high and ineffective due to inadequate diastolic filling time under conditions of low venous return due to orthostasis. Slowing the heartbeat with propranolol may be effective by improving cardiac output.
Another known effect of bedrest which may be relevant is the loss of total body water, plasma volume and total blood volume (Hyatt et al 1969). Although the amount of fluid lost does not seem to correlate with orthostatic intolerance in healthy young volunteers (Miller et al 1964), in postoperative patients, fluid loss may be severe due to surgery, pre-operative nil by mouth regimes and poor oral intake postoperatively (Audit Commission 1995). The use of diuretics and low postoperative fluid intake has been linked with postoperative hypotension in a series of 99 total knee replacement patients receiving epidural opiate analgesia (Olson et al 1992) which suggests loss of fluid may have a role to play in the aetiology of falls in post-operative patients. Regardless of the cause, in another study, post-operative patients were found to be at increased risk of falling (Plati et al 1992). It is possible that patients with fractured neck of femur may be particularly likely to have a higher incidence of orthostatic disregulation, because a fall is often what has caused the fracture.

While the dangers of acute hypotension are generally recognised, there is debate about the significance of chronic low blood pressure. In continental Europe and French Canada, constitutional hypotension is accepted as a disease entity (Pemberton 1989). Symptoms are considered to be tiredness, sleep disturbances, giddiness, blackouts, fainting, anxiety or depression, consciousness of the heart beat and racing of the heart and sweating (Vosschulte et al 1979 cited in Pemberton 1989). The criterion of a systolic blood pressure of <110 mmHg or diastolic <60 mmHg is usually used to define hypotensive disease. In contrast, in the English speaking countries, blood pressures about or below 110/60 are generally thought to be beneficial (Braunweld 1987) and the disease entity is not recognised in English medical textbooks (Pemberton 1989).

Many of the symptoms above appear in the general population without hypotension and in a large population survey in Australia no correlation was found between low blood pressure and palpitations, dizziness or fainting. However there was a correlation with fatigue in women (National Institute of Occupational Health and Safety Blood Pressure Study 1987). However, another survey, this time of over 100,000 London civil servants found a correlation between low blood pressure and symptoms such as dizziness, tiredness and low mood (Pilgrim et al 1992). Evidence of an association between low blood pressure and fatigue has also been found by Wessely et al (1990). Therefore the long term effects of having a low blood pressure may not be quite as benign as is thought in the English-speaking countries. It is not known how long hypotension needs to persist before the development of symptoms, but it cannot be ruled out that onset may be rapid and occur in a matter of hours.

The elderly tend to have a higher resting blood pressure (Diem & Lentner 1970) and in post-operative patients, there is likely to be some alteration of baseline blood pressure due to pain. Vogele & Steptoe (1986) found pain to peak on the first day following hip replacement and to drop off to a stable level from day three onwards. There was a close correlation between pain scores and blood pressure in their studies. However the mobilisation regime in their study is unknown and therefore it is impossible to deduce the relative influence of the various factors on blood pressure level.

It has been argued that sitting may decrease orthostatic intolerance associated with bedrest (Harper & Lyles 1988), although little supporting evidence is given. Sitting may increase the level of stimulation by increasing range of sight and the use of some anti-gravity muscles, which may reduce autonomic lability. It may also help to recondition cardiovascular response to gravity. However there is little or no evidence to support these views. Anecdotal evidence suggests that elderly patients sitting for long periods of time do suffer from orthostatic hypotension (Mulley 1993, Bliss 1990) and studies of young men sitting in space cabin simulators for prolonged periods displayed orthostatic intolerance similar to that following bed rest (Lamb et al 1964). Indeed their deconditioning seemed to occur within 4-10 days (Lamb et al 1995), more rapidly than some of the periods quoted for bedrest. Consideration of all the above suggests that exposure to prolonged orthostasis and immobility in elderly post-operative patient is likely to cause hypotension. This may increase the likelihood of the patients falling, being dizzy, fatigued and perhaps low in mood. However, at present there is little or no research evidence to support this hypothesis.
2.3.4 Lower limb oedema

Oedema is the presence of excess fluid in the tissues of the body (Guyton 1991) and can be intracellular or extracellular. Intracellular oedema occurs in any part of the body where the local blood flow is depressed and delivery of oxygen and nutrients is too low to maintain normal tissue metabolism. This depresses the activity of ionic pumps in the cell membrane (particularly sodium ion pumps) and therefore when sodium ions leak into the cells, they are not removed and water follows them by osmosis. This can increase the global tissue volume by as much as two or more times than normal. Extracellular oedema is caused by abnormal leakage of fluid from capillaries and/or inadequate removal of interstitial fluid by the lymphatic system or due to renal retention of salt and water (Guyton 1991).

Using the formula for capillary filtration of Starling (1896), and an estimation of capillary pressure from their study of foot swelling in office workers, Winkel (1985) calculated that without oedema-preventing mechanisms feet are likely to swell 20%-25% during 8 hours sitting. That they do not do so is consistent with the existence of oedema-preventing mechanisms. The observation by Winkel & Jorgensen (1986a) that during 8 hours of sitting, foot volume increased by 3.5% in the first 4 hours and only 1.9% during the rest of the study, suggests that some of these mechanisms develop slowly.

What these mechanisms are and their precise function is not fully understood. There are local and systemic mechanisms. Locally, in young adults, the acral skin (ankles, forearms) is stiffer and less distensible but becomes more elastic in the evening (Gniadecka et al 1994). A similar adaptation of very stiff lower leg skin exists in the giraffe and places a mechanical resistance to swelling of the limb under great pressures generated by the animal’s height (Hargens et al 1987). The veno-arteriolar reflex is another important local anti-oedema mechanism. This is a mechanism by which when vein and venules swell in a microcirculatory unit (that is, an arteriole, venule and the capillaries and preferential channels, or shunts which connect them [Bliss 1998]) due to distention with blood, the arteriole that feeds into the unit constricts under sympathetic control to reduce fluid overload (Michel & Gillott 1990). Other mechanisms are related to changes in hydrostatic and colloid osmotic pressures of capillary blood flow and interstitial fluid which oppose further filtration (eg Aukland 1984, Noddeland et al 1984).

Systemically, the most important anti-oedema mechanism is the lymphatic system which drains off excess interstitial fluid and returns it to the arterio-venous circulation. Some factors which increase lymphatic flow, relevant to this discussion, are increases in venous capillary pressure (Landis and Gibbon 1933, cited in West 1991), increased functional activity of glands and muscles and tissue movement (West 1991).

Oedema interferes with the oxygenation of cells and the anatomical function of the affected part, predisposes to infection and can be unsightly (Colbert 1993). Swollen legs and feet can prevent the wearing of normal footwear (appendix 8, figure 1) and interfere with ambulation and also potentially make it unsafe to walk (Tinetti & Speechley 1989, Kellog International Work Group 1987). Even the relatively small amount of swelling of the feet and legs experienced after lengthy sitting by healthy young individuals is frequently associated with discomfort (eg Winkel 1981, Winkel & Jorgensen 1986). Long standing oedema in the legs, it is thought, can cause tissue breakdown and ulceration (Bliss & Schofield 1995, Mulley 1993). However, even gross oedema of this aetiology can be rapidly reversed by a period of bedrest, suggesting that orthostasis is the primary cause (Bliss 1990, Bliss & Schofield 1995).

The incidence of dependent oedema (that is oedema caused by dependency of limbs) in hospital patients in the United Kingdom does not appear to be known. However, many patients are referred for investigation of leg swelling in the United States. In one study of referrals for venous duplex scanning, 719 patients were referred in one hospital over a period of two years. The scans of the majority of these proved completely normal, which suggests dependent oedema as a possible cause. Leg oedema is recognised as a common problem in the United States population and venous stasis is
the most common cause of leg oedema in the elderly, forming 63% of cases reviewed in one study of attenders at an oedema clinic (Ciocon et al 1995).

When elderly individuals who have undergone surgery of the lower limbs are sat in chairs the following are likely to happen. The gravitational and postural effects on circulation described in sections 2.2.1 and 2.2.3, raise the capillary pressure and promote pooling of blood in the legs. The volume of the legs increases rapidly initially, because of venous distention. It is thought likely that this distention is sufficient to cause the venous valves to become incompetent (Keane & Fegan 1969). This contributes to venous stasis because the valves are part of the calf muscle pump mechanism and prevent backflow down the veins when the calf muscles are contracted. Other factors associated with sitting which may promote venous stasis are under-thigh pressure of the seat and angulation of the blood vessels at the knee and in the pelvis and reduced calf-muscle pump activity. This may lead the development of intracellular oedema over time as local tissue perfusion becomes less than optimal.

There then follows a gradual increase of oedema due to slow filtration of fluid through the capillary membranes into the interstitial space. In young individuals the feet increase by up to 5-7% in volume after sitting for 8 hours (Winkel 1985). In the elderly, this is likely to be more, as at least some of the oedema-preventing mechanisms deteriorate with age. Gniadecka et al found that the properties of acral skin were different in the elderly. However these differences mainly related to the diurnal changes, with elderly skin not becoming more elastic in the evening. This suggests that the difference may affect the rate of reduction in size of the leg when the supine posture is assumed for night time sleep rather than the amount of swelling that actually occurs. However another local oedema-preventing mechanism that appears to be affected by age is the venoarteriolar reflex. Laser Doppler flowmetry showed 16.6 reduction in cutaneous blood flow in 21, 17-27 year olds challenged with orthostasis compared with pre-challenge blood flow. In subjects aged 75-100 years old, the reduction was only to 29% (Gniadecka et al 1994a). This suggests that filtration is likely to proceed at a much faster rate in the old than in the young.

Older women (likely to make a significant proportion of arthroplasty patients due to their liability to femoral fracture [Versluysen 1986]) are thought to be particularly prone to leg oedema due to hormonal changes associated with the menopause (Kroemer 1971). Another factor predisposing the elderly to oedema is the common prescription of drugs which may cause oedema as a side-effect. Nifedipine, for example, is commonly prescribed for the elderly (Nolan & O'Malley 1988) and has been strongly linked to leg swelling (Dubiel 1990, Shnapp et al 1987).

Because muscular activity and tissue movement is important to the maintenance of good lymphatic drainage, inactivity is likely to reduce its efficacy. Seated elderly postoperative patients are likely to have reduced mobility generally because of the constraints of the seat and difficulty with or impossibility of ambulation. Movement of the operated limb, in particular is likely to be constrained by the needs of postoperative management for the prevention of dislocation. Pain or the fear of pain is likely to reduce movement further. Therefore lymphatic drainage is likely to be less effective than in a fully active individual.

High temperatures appear to hasten leg swelling, possibly because of the dilatory effect on the circulation. For example Turner et al (1930) found that leg swelling in women standing for 15 minutes was doubled from 1.9% to 3.4% when measurements were taken in summer as opposed to winter. Hospital wards tend to be kept at a high temperature, so this factor too is likely to promote oedema in hospitalised patients.

The only factor that may promote reduction of post-operative oedema is likely to be the wearing of anti-embolic stockings, which are commonly used for the prevention of deep vein thrombosis. It has been suggested, based on laboratory observations of interface pressure exerted by such stockings and air-plethysmography, that they provide an effective edema preventive effect (Veraart & Neumann 1996) and the application of light to medium compression over 5 days was found to reduce dermal
oedema, without reducing the diameter of the limb (Gniadecka et al. 1998). However, the long-term benefits of compression hosiery have been questioned as they seem to be effective for only a short period of time and may not be useful for individuals with disproportionately large thighs at all (Galinda Ciocon 1995, Liehr et al. 1992).

Consideration of the above factors - orthostatic pressure, age-related deconditioning of anti-oedema mechanisms, angulation and compression of blood vessels, oedema promoting drugs, reduced mobility, high temperatures and the doubts cast on the practical effectiveness of wearing anti-embolic stockings as an oedema preventing strategy - suggests that post-operative elderly patients sitting in chairs for long periods of time are likely to develop oedema of the legs. However, there appears to be no controlled trial evidence to support this view.

2.3.5 Thrombosis and embolism

Deep vein thrombosis (DVT) is a serious postoperative complication. It is defined as a clot of blood constituents (thrombus) that forms in deep veins of the pelvis and legs such as the iliac, popliteal, femoral, posterior tibial or peroneal (Judd 1989). The presence of a clot is often asymptomatic (Barnes 1982) but in some cases there is pain in the calf, particularly on dorsiflection of the foot (Homans’ sign). The risk of DVT associated with hip surgery and knee reconstruction ranges from 40% to 74% (Clagett et al. 1995). Patients who sustain a fracture or have major orthopaedic surgery of pelvis, hip or lower limb are particularly at risk (Evans 1993). DVT has been the most common reason for emergency readmission following elective hip replacement (Seagroatt et al. 1991). In recent years there is thought to have been a reduction in DVT in orthopaedic patients, probably as a result of the introduction of low-molecular-weight heparins and routine anti-coagulation. In the context of clinical trials it has been shown that incidence rates can be reduced to 10-12% (Planes et al. 1998).

The particular importance of DVT is that in about 45%-51% of cases it leads to pulmonary embolism (Partsch et al. 1997). Although it is asymptomatic in the majority of patients (Partsch et al. 1997), in a small percentage the embolus can cause major obstruction of the pulmonary circulation which is reported to cause 10% of deaths in hospitalised patients (Sandler & Martin 1989). Perez et al. (1995) found pulmonary embolus to be the cause of death in 14% of 581 necropsies of patients who had died following fractured neck of femur.

Thrombo-embolism has been studied extensively and an in-depth discussion of the mechanics of thrombus formation and aetiology is outside the scope of this thesis. However, Risberg (1988) provides a comprehensive review. Some authors believe that DVT is primarily caused by increased venous stasis due to decreased contraction of the gastrocnemius and soleus muscles which normally occurs during walking (the calf muscle pump) (Dittmer & Tease 1993). However, animal studies suggest it is unlikely that venous stasis alone causes large thrombi (Wessler 1962). However when it is combined with hypercoagulability of the blood following trauma or surgery, it becomes a major factor in thrombus formation (Paramo & Rocha 1985) and the incidence of DVT has been shown to be related to the length of post-operative bedrest (Micheli 1975). The higher incidence of DVT following hip surgery compared with other types of surgery is attributed to direct compression of the veins during operation (Risberg 1988), venodilation due to endothelial injury during surgery (Stewart et al. 1987) and the old age of the majority of the patients. Old age is an important risk factor with risk increasing exponentially after 50 years of age (Sue-Ling et al. 1986).

The most effective nursing intervention for the prevention of DVT and pulmonary embolism, is considered to be early mobilisation of the patient (eg Maher 1998). Lassen & Borris (1991) found in a group of 70 orthopaedic patients that patients ‘mobilised’ within 4 days of surgery had an incidence of DVT of 21-23%. Those whose mobilisation was delayed to the 9th day had an incidence of 75%. Unfortunately Lassen & Borris do not specify what their mobilisation regime entailed. It would appear that apart from this study little or no other data is available on the efficacy and safety of mobilisation as preventive treatment for DVT (Pauschert et al. 1998).
Sitting alone is unlikely to be effective. Indeed, in healthy adults, rate of blood flow in the legs is decreased by over 21 times when sitting as compared with lying supine with feet elevated 6° (Ashby et al 1995). This is likely to be partially due to the blood having to be pumped against a gravitational gradient. However inactivation of the calf muscle pump and vein flexure (and in obese individuals angulation and compression of the veins in the groin) causing obstruction are probably more important causes since the standing posture, which produces a steeper gravitational gradient than sitting, reduces blood flow by three times only (Ashby et al 1995). The kinking of blood vessels is likely to disturb the pattern of blood flow and turbulent flow is thought to predispose to platelet deposition - the start of a thrombus (Browse 1965). Furthermore, it is likely that when the feet are placed on the floor as in a sitting posture they cannot be moved as freely to partially exercise the calf muscles as they might be in bed.

While there are no clinical trials' data comparing chairnursing with bednursing or ambulation there is both anecdotal and epidemiological evidence that DVT is associated with activities that involve sitting. Wells (1979) reports his clinical impression of an increase in DVT following the introduction of chairnursing in postoperative patients. One fifth of all sudden deaths in passengers arriving at Heathrow airport are due to pulmonary embolus following long spells of sitting in chairs (McIntosh 1990). This observation has been supported by reported increased incidence of DVT in passengers in airplanes and other modes of transport such as cars, trains and buses in which patients remain seated, compared with pedestrians (Homans 1954, Haegar 1957). Ferrari et al (1999) carried out a case-control study of 160 patients presenting with thrombo-embolism in one cardiology department. Patients with thrombo-embolism were almost 4 times as likely to have travelled over 4 hours in a seated position (train, airplane, car) in the preceding 4 weeks. An increased risk of DVT has also been associated with prolonged television viewing - an activity usually carried out in a seated position (Naide 1957).

The evidence thus suggests that prolonged chairnursing is likely to increase the incidence of deep vein thrombosis in post-operative patients rather than decrease it as suggested by the Royal Marsden Hospital (1984). However, the direct evidence of this is anecdotal (Wells 1979) and needs to be confirmed by carefully designed, controlled studies.

2.3.6 Pulmonary complications - chest infection
Infection is a state resulting from the process whereby pathogenic organisms become established and multiply in or on the body of the host (Konigsberg et al 1998). In the case of chest infection, the target areas of the body are the lower respiratory tract. Depending on where the infection is situated, infections of the respiratory tract are often given separate names such as bronchitis (infection of the bronchi), tracheobronchitis (trachea and bronchi) (eg McGeer et al 1991). Pneumonia, it should be noted, strictly refers to a condition of infection of the lungs, which is accompanied by a radiologically documented pulmonary infiltration. Lower respiratory tract (LRTI) is often used do describe chest infection without pneumonia (Tapson & Kussin 1995).

The development of chest infection in post-operative patients is usually attributed to the effects of the surgery itself (in the case of thoracic and abdominal surgery) and to immobility and bedrest. An accumulation of secretions at the base of the lungs is associated with immobility, eventually causing atelectasis and hypostatic pneumonia. These conditions provide an ideal environment for bacteria to grow, leading to infection (Lansing & Jamilson 1963). Doyle (1999) gives a review of the risk factors and preventative techniques useful for prevention and management of post-operative chest infection.

The incidence of chest infection is high in post-operative patients. For example, in a study of 127 patients undergoing elective upper abdominal surgery, there was an incidence of 20.5% (Dilworth & White 1992). Smoking and smoking-related diseases were found to be the most significant predisposing factors with non-smokers having an incidence of only 7.1%. It has been suggested that pneumonia is particularly dangerous to the elderly as they are thought to be less able to mount an
effective immune response due to age-related reduction in immune function and attenuation of inflammatory response. This theory is supported by the findings of reduced lymphocyte function (eg Murasko et al 1986). However, Albazzaz et al (1994) demonstrated an adequate inflammatory response in 41 elderly individuals with chest infection. Nevertheless, bronchopneumonia is the leading cause of death following hip fracture with 46% of all deaths following hip fracture being attributable to this cause (Perez et al 1995).

Apart from mobilisation, recommended physical treatments for the prevention of pulmonary complications include chest physiotherapy (deep breathing and coughing, vibration, postural drainage) and 'spirometry' (Tease11 & Dittmer 1993, Johnson et al 1995, 1996) (it should be noted that in this context 'spirometry' refers to the activity of blowing into a spirometer rather than to actual measurement of air flow). However, the evidence for the efficacy of physical treatments is limited (Lie et al 1998). Although it is believed that the seated posture is beneficial (appendix 1) the evidence for this is also limited. Although the sitting position makes it easier to contract some ventilatory muscles (Svanberg 1957), the displacement of organs in the abdominal cavity when the individual sits with a 90° angle between thighs and trunk may cause a reduction in space and increased pressure on the lungs, particularly if the spine is kyphotic (Burandt 1970, Garner 1936). It should be noted that musicians playing wind instruments increase their performance and capacity by increasing the angle between trunk and thighs (Eklund 1986).

The increases in lung capacity during orthostasis are likely to be primarily due to a reduction in blood volume and the effects on oxygenation have not been adequately investigated. The deposition of secretions and pulmonary blood flow being directed to the most compromised area are a product of immobility not of posture. However it should be noted that in the upright position, deposition takes place in the areas of the lung furthest away from the point of exit. It is probably no coincidence that chest physiotherapy tends to be carried out in the prone or lateral position. There is little or no evidence that chairnursing reduces the incidence of atelectasis and chest infection. On the contrary, in a small sample, Gebhardt & Bliss (1994) found a greater rate of clinically diagnosed chest infection among patients sitting for prolonged periods in chairs as opposed to those limited to no more than two hours per session. Because of the clinical importance of chest infections, investigations should be carried out to determine if mobilisation programmes can reduce the incidence of chest infection (Lie et al 1998) and if so, how they can do this most effectively.

2.3.7 Pressure sores
Pressure sores occur when soft tissues are deformed either by being supported on an uneven surface, being compressed between the skeleton and the support or by shearing. Shearing occurs, for example when an individual is sliding down a bed from a semi-recumbent position. The skeleton pulls the soft tissues in one direction, while frictional forces between the tissues and the support surface pull in another. When tissue is deformed the blood vessels that run through it are stretched, angulated compressed and sheared and blood flow through them is occluded. The tissues that those blood vessels supply become ischaemic, and if this ischaemia is prolonged they die, forming a pressure sore.

In a healthy individual, pressure sores do not occur despite tissues being constantly subjected to compression and shearing, because there are defence mechanisms to prevent damage. These mechanisms are most likely to consist of reflex activity which initiates changes of posture such as those that occur during sleep (Exton-Smith & Sherwin 1961), and should that prove inadequate, the individual will experience discomfort and pain in the ischaemic tissues (such as the pain experienced when carrying a heavy bag that is cutting into the hand) which will also encourage him/her to relieve the pressure (Gebhardt 1994). Therefore, an individual only becomes susceptible to pressure sores if these defence mechanisms fail.
Patients with fractured neck of femur are particularly prone to pressure sores (Versluysen 1986) and orthopaedic patients generally have a higher incidence than general surgical ones (Dunford 1994). So much has been written elsewhere about the costs of pressure sores personally, socially and financially (Dealey 1997, Bader 1990) that it would be beyond the scope of this thesis to discuss these issues in detail. However it is clear that pressure sores are a source of significant morbidity and even mortality and their estimated cost to the health services is high (Department of Health 1993). When small they are unpleasant and painful and possibly discourage the patient from moving (appendix 8, figures 2,3). Large sores can be a major setback which can delay hospital discharge (Allman et al 1999), may affect future life and can be fatal (Bliss 1994).

The aetiology of why some patients become susceptible and others do not is not always clear although there are many possible causative factors (Bliss 1993, Bridel 1993). Possible causes in orthopaedic patients are the loss of sensation induced by anaesthesia and analgesia and poor health of some of the patients (Bliss 1993).

Once an individual is susceptible, how readily they will develop pressure sores is likely to depend on three factors which interact with each other: time, tissue resistance to pressure and the magnitude of the applied force. During sitting, the time factor depends on how long the individual is sat, but also on how effectively they can alter posture while sitting to relieve compression of the tissues. Tissue resistance to pressure is probably determined by the efficacy of complex homeostatic mechanisms of the macro and micro-circulation. Small postural changes, for example will only effectively prevent ischaemia if the hyperaemic response can rapidly revascularise tissues previously devascularised by compression or shearing (Bliss 1998). This requires an adequate supply of blood. The size and nature of the applied distorting forces will determine how quickly damage will occur and how large an area will be affected. Reswick and Rogers (1976) showed that in their sample of patients pressure sores occurred more quickly the larger the force that was applied. In other words, tissues could tolerate high pressures for short periods only, while smaller loading could be tolerated longer. The actual figures they give for 'acceptable' and 'unacceptable' time/pressure applications should be considered with great caution as the data consists of "subjective comments by physicians, nurses and therapists relating to actual tissue breakdown occurrences; actual pressure measurements in situations where the patient's skin showed clinical signs of potential or actual breakdown following known times of pressure application and ability to measure pressure which was acting; controlled tests on volunteers where pressures were applied for times sufficient to produce clinical signs of potential breakdown". However, the overall premise has an intuitive face validity and is supported by animal studies (Daniel et al 1981).

When an individual sits in a chair, all three of the above key factors above are magnified, in comparison with lying in bed, for example. Whilst the amount of time the individual spends in a chair is determined by how mobile they are and by policy and practice of the healthcare institution they are in, chairnursing is also likely to have an impact on the ability to carry out small bodily shifts. When lying, even very frail individuals can often move themselves. This is probably more difficult in a chair if the patient has no strength in their arms (Dzielski 1999), although the relative amount of movement in beds and chairs has not been studied. Alternating pressure devices can be used in bed with good effect to move support areas cyclically from one part of the body to another, effectively reducing the time of compression for any part of the body to less than 6 minutes (Gebhardt et al 1996, Bliss 1995). However there is little evidence of their effectiveness in chairs (Clark & Donald 1999).

The issue of tissue resistance to pressure, despite its importance is, as yet, little understood. However, chairnursing will affect the skin circulation through the effects of orthostasis and immobility which have been discussed in preceding sections (2.2.1, 2.2.2). Orthostasis tends to cause peripheral vasoconstriction through sympathetic activity while orthostatic oedema in the dependent legs also triggers local arterial constriction because of the veno-arteriolar reflex. Blood flow in the skin is significantly reduced in the upright posture (Rowell 1977, Johnson 1986). The effects of posture on tissue
Compression and shearing have been discussed in section 2.2.3. When seated, the compressive and shearing forces which are applied to the sacrum and buttocks, are probably greater than in any other position (Goosens & Snijders 1995).

Despite belief even now by some practitioners that sitting in chairs somehow prevents pressure sores (Haynes et al 1997), most authors who have studied this aspect of pressure sore aetiology agree that patients of equal sickness nursed in chairs are more likely to develop pressure sores than those in bed (Barbenel et al 1977, Nyquist & Hawthorn 1987, St Clair 1992). Gebhardt & Bliss (1994) found a significant positive association between the incidence of pressure sores and length of chairnursing in a study of pressure sore mattresses in the orthopaedic wards. A further study showed that pressure sore incidence could be reduced to 7% if chairnursing was limited to no more than two hours at a time in comparison to 63% incidence on another ward where chairnursing was unlimited (Gebhardt & Bliss 1994). However, because of ethical constraints on continuing a control (unlimited) regime with statistically significant deleterious effect, the sample size of this study was small and therefore the confidence interval was wide.

In summary, sitting in chairs should be considered a high risk activity with regard to pressure sore formation in elderly, post-operative orthopaedic patients. Although the precise aetiology and differential diagnostic signs and symptoms of those who are and who are not at risk of pressure sores is not fully understood, the known incidence in this patient group is high. Those who are at risk are likely to develop sores quickly in the seated position because of difficulty with moving from one position to another when in a chair; high compressive and shearing forces and lowered skin perfusion due to cardiovascular responses to orthostasis. There is weak direct evidence from one controlled study, with small sample and wide confidence intervals, of the potential benefits of controlling the length of chairnursing and supporting evidence from surveys which have found pressure sores to be associated with chairbound patients. As the complication can be a significant cause of mortality, morbidity and potentially unnecessary use of health care resources this potential 'negative' outcome of chairnursing should be examined with further studies.

2.3.8 Constipation
A common, medical definition of constipation is defecation less frequently than every third day (Clinch & Hilton 1998). This broad definition reflects the wide variation of bowel habit in the general population (Connell et al 1965). It should be noted, however, that in colloquial usage constipation is most often used to describe straining rather than infrequent defecation. Less frequently, it is also used by lay people to describe a feeling of incomplete evacuation or firm stool consistency (Whitehead et al 1989).

Hospitalised patients are thought to suffer frequently from constipation. The reasons for this are complex. It has been suggested that social reasons (lack of privacy, delays in assistance, use of bedpans) (Harper & Lyles 1988) and change in diet on entering hospital (Browse 1965) play a part. However reduced mobility of the gut is probably the major factor. Factors which affect gut motility in the hospital population are likely to be a reduction in activity and introduction of drug therapy. In the elderly, mass propulsion by the colon correlates with physical activity (Brocklehurst & Khan 1980) and the rate of movement of radioactive markers through the colon varies with the level of somatic activity (Holdstock et al 1970). Medication which has a constipatory effects such as opioids is commonly prescribed for post-operative analgesia after orthopaedic surgery (Olson et al 1992). The constipatory effects of opioid analgesics have been extensively studied and are reviewed by De Luca & Coupar (1996).

Constipation has been associated with a range of complications in the elderly. These include cardiovascular and cerebral consequences such as syncope, cardiac arrhythmias, coronary insufficiency and cerebrovascular accidents. Severe bowel consequences include faecal impaction, rectal prolapse, haemorrhoids, sigmoid volvulus, ischaemic colitis and bowel perforation. Faecal
impaction often leads on to urinary retention and acute confusion. Therefore, constipation can precipitate life-threatening complications and significant morbidity (Wald 1990, Hyams 1974).

Although it is generally believed that constipation is more common in older people, there is little real evidence that that is the case (Clinch & Hilton 1998). Only 2% of males and 7% of females were found to have constipation in one survey of 159 people aged 65-93 living at home and selected from census records by identifying a census tract with a high proportion of elderly residents (Whitehead et al 1989). Nearly a quarter of the sample either refused or were unable to be interviewed due to shielding by relatives or illness. The non-participants may have been significantly different in some ways such as being sicker or more psychologically distressed (both factors positively associated with constipation) than the study population and therefore the results may be skewed towards less constipation. However, the authors conclude that incidence of constipation does not increase with age although the elderly report more constipation which they tend to define differently to physicians.

The effects of chairnursing on gut mobility have not been researched. It could be considered that sitting may assist movement of faeces into the rectum by gravity. However, it is likely that the problem of constipation in immobile individuals lies with the slow filling of the rectum not stimulating emptying in the way that faster filling does and the recta of constipated patients are usually full of faecal matter (Browse 1965). It is the rate of passage down the bowel that is important not the position. During orthostasis, there is a shift of blood volume away from the gut. Asmussen et al (1940) identified the gut circulation to be one of the systems likely to become less well supplied with blood as the body attempts to maintain normotension in the brain during orthostasis. There is a reflex-induced splanchnic vasoconstriction with an approximately 40% reduction in splanchnic blood flow in response to 75° head-up tilt (Culbertson et al 1951). This reduces peristalsis and thus the rate of movement of matter down the gut. This is probably why sitting has been reported to be associated with impairments of the digestive system in workers. Some authors have suggested that this may be due to prolonged pressure on the abdominal cavity and slackening of abdominal muscle due to posture (Grandjean et al 1980). Others have believed that general immobility related to sitting is the primary cause (Holdstock et al 1970). Whatever the cause or causes, there is a slowing of gut motility during sitting which has also been linked to the increased risk of colonic cancer in individuals doing sedentary work (Gerhardsson et al 1986).

Gebhardt & Bliss (1994) found a significantly higher rate of constipation among patients allowed unlimited chairnursing as compared with those who had been restricted to sitting for no more than two hours at a time. As the consequences of constipation may be severe, including life-threatening complications, this aspect of rehabilitation programme design requires investigation.

2.3.9 Urinary tract infection (UTI)

Like chest infection UTI is the result of the process whereby pathogenic organisms become established and multiply in or on the body of the host, in this case in the urethra, bladder, ureters and the kidneys in any combination. Many individuals, particularly the elderly, have bacterial populations within their urinary tract but do not display any symptoms (McGeer et al 1991). For this reason UTI is generally considered to exist only if a bacteriuria is accompanied by other signs and symptoms.

It is thought that reduced urinary flow predisposes to UTI (Judd 1989). One of the body’s normal defences against bacterial invasion via the urinary tract is the flushing and scouring mechanism of micturition which is reduced with low urine flow. Increased excretion of calcium caused by immobility tends to cause deposits of calcium in the renal pelvis or the ureters (Vallbona 1982). These restrict the flow of urine and cause foci for infection, as bacteria can flourish in the stagnant urine. The emptying of the kidneys and bladder is assisted by an upright posture. In the supine position peristaltic activity of the kidney pelvis is not sufficient to overcome gravitational resistance and urinary stasis occurs in the kidney, while the bladder too is not fully emptied in the recumbent position (Olson & Schroeder 1967). An upright position, therefore, may be beneficial.
Urinary tract infection is common in hospitalised patients although the reported incidences of urinary tract infection after hip surgery vary (Ling 1984). Probably due to the availability of antibiotics, UTI rarely leads to sepsis and death. In the Perez et al (1995) study of 581 autopsies, 24 patients (4%) died from sepsis of all causes following surgical repair of fractured neck of femur. However, UTI symptoms which can include burning pain on micturition, suprapubic pain, pyrexia and acute confusion (McGeer et al 1991) are unpleasant for the patient and are probably likely to delay mobilisation and hospital discharge.

There are three potential effects on urinary dynamics of sitting. Firstly, there is a likelihood of improved drainage due to the gravitationally more advantageous upright position. Secondly there may be a reduction in calcium excretion if sitting causes enough stresses on the skeletal system to significantly reduce bone demineralisation, the cause of the problem (eg van der Wiel et al 1991). However this is speculative. Thirdly, overall urinary flow is likely to be considerably reduced due to orthostasis. Reflex vasoconstriction in the kidneys significantly reduces blood flow. For example, in one study, renal blood flow was reduced by 32% in a 60° head-up tilt (Ring-Larsen et al 1982). Aside from the reduced renal circulation, diuresis is also diminished by the anti-diuretic response of the body to orthostasis (Brown et al 1966). Greenleaf et al (1980) observed a reduced diuresis during sitting as did Guite et al (1988). In the latter study, the output of 4 elderly catheterised patients was observed for 6 consecutive days and night. For 3 days the patients were on complete bedrest and during the other 3, were sat out in chairs for 8 hours. Urinary output was significantly less during the day during chairnursing and increased at night. During complete bedrest there was no difference in water excretion between day and night.

Because of these competing effects of posture and orthostasis it is difficult to predict the effects of chairnursing on the risk of UTI. The preliminary study (Gebhardt & Bliss 1994) showed no difference in incidence between patients in the limited and unlimited chairnursing groups. However, the sample size was unlikely to have been sufficient to detect any difference in the incidence of this rarer complication if one actually existed. A larger study was required to make a more informed decision.

2.4 THE EFFECTS OF CHAIRNURSING ON MOBILITY

2.4.1 Independent walking
It is believed by some that it is preferable for patients to spend as much time as possible sitting in chairs because they can get up more easily and thus become more independent. However this proposition is not supported by published research. Conversely, the preliminary study found that more patients who had chairnursing limited to no more than two hours at a time became independently mobile (ie could get out of the chair and walk about) at two weeks than those who were permitted unlimited chairnursing (Gebhardt & Bliss 1994). However, the difference was not statistically significant and mean lengths of time before walking are not known as patients who were still immobile at 14 days were not followed up.

While use of Early Discharge Schemes (ie schemes which provide nursing and physiotherapy care within the patient’s own home to enable earlier discharge) may hasten the process of returning the patient home (and must be borne in mind as a confounding factor), even there, a minimum degree of independent mobility will be required before the patient can be discharged.

2.4.2 Discharge from hospital
Hospital stay has been considered an overall measure of post-operative morbidity (Kehlet 1988). This appears to be a somewhat simplistic view. Before a patient is discharged from hospital following surgery they must be deemed fit enough by the multi-disciplinary team caring for them and this is a

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subjective decision. In the absence of significant postoperative complications such as a major wound infection or pulmonary embolus or any other untoward medical events, the primary deciding factors are the patient's level of mobility, their abilities in self care and the support available for them wherever they are discharged to.

This means that social considerations can significantly affect when a patient is discharged irrespective of their physical condition or fitness. For example, one study in the United States (Falcone et al 1991) found that in one month 3,111 elderly patients in 80 Carolina hospitals were awaiting discharge despite being considered medically fit to go. Average delay was 16.7 days, though this is likely to be an under-estimate as patients who were still awaiting discharge on completion of the study were not followed up. Although some of the variables associated with delayed discharge were related to patients' medical conditions, such as the need for domestic nursing care, others were not, such as financial considerations and the patient's race. The reasons for the latter are unclear, but may have included social and cultural factors.

Furthermore, the clinician's decision on whether or not a patient should be sent home may be swayed by the need to vacate the bed for another patient who needs it more urgently. It has been documented that pressure on beds is associated with faster discharge from specialist areas such as critical care units (Dawson 1993). Finally the patient's views may also be important. It has been suggested that it is patients who fear that they may not be able to cope at home who are most likely to have a protracted hospital stay (Fenton-Lewis 1986). Therefore it is likely that when a patient is discharged will be influenced by many factors other than simply their medical condition.

Prolonged hospital stay is undesirable both for the patient and the healthcare institution. It has been suggested that the longer patients stay in hospital, the more likely they are to lose the skills necessary for independence (Editorial 1982). This may be particularly true of patients who have lost their confidence due to a fall resulting in femoral fracture. While a detailed analysis of hospital economics is beyond the scope of this thesis, it is clear that when a patient remains in hospital longer than necessary, this increases the costs of care as well as denying the use of the hospital bed for another patient. This incurs a 'lost opportunity cost' (Hibbs 1990).

There is little or no research evidence of how chairnursing affects length of hospital stay following surgery. If the effects are detrimental overall, then it seems intuitively logical that directing the patient's energy to maintaining a seated posture as opposed to improving self-care and mobility will reduce the amount available for the latter activities. Therefore it is possible that long periods of chairnursing may in fact slow the process by which the minimum levels of ability necessary for discharge are achieved by the patient. As length of stay in hospital is an important economic consideration, this should be assessed in any study of the outcomes of chairnursing.

2.5 NON-INVASIVE METHODS FOR ASSESSING THE PHYSIOLOGICAL AND CLINICAL EFFECTS OF CHAIRNURSING

This section examines techniques which are available for the measurement of variables which may be affected by prolonged chairnursing. Only non-invasive methods will be considered in this section as there appears to be little ethical or practical justification for the use of invasive methods in this study. All invasive procedures cause discomfort and carry serious health risks, some potentially fatal (sepsicaemia, blood-borne infection). Other methods such as cardiac catheterisation, also carry serious risks of mechanical injury. The gains in accuracy of measurement are too small to warrant such risks. Furthermore the sampling error involved in comparing measurements in different individuals at different times means small gains in accuracy of measurement will reduce overall error to only a very limited degree.
The only exception is the taking of blood samples for screening for markers of DVT. There are likely to be significant improvements in sensitivity and specificity over non-invasive methods and the presumed small risks to the subject are compensated by the benefits of a screening programme which is not generally available to patients.

2.5.1 Self perceived fatigue
This section has been pursued in some depth because, possibly due of the abstract nature of the phenomenon, the design of measurement methods depends on definition and perception of aetiology. Because there are different ways of defining and understanding fatigue (section 2.3.1), many methods have been proposed and used for its measurement. Furthermore, there appear to be no comprehensive reviews of measurement of post-operative fatigue that could be referred to or used as a basis for further study.

At the turn of the last century considerable attention was focused on the study of fatigue (Rabinbach 1991). It was thought that artificial reduction of fatigue was possible and would bring great social and military benefits. By the 1920's disenchantment with these ideas had set in. One of the main stumbling blocks proved the difficulty of defining what fatigue was. Without definition, it was virtually impossible to measure it. In 1921 Muscio, a leading researcher into fatigue, stated in despair "that the term be absolutely banished from scientific discussion, and consequently that attempts to obtain a fatigue test be abandoned". Interest dwindled and the many fatigue laboratories closed down (Forbes 1943).

Because of the distressing nature of postoperative fatigue for patients, potential harmful effects and the assumption that fatigue delays convalescence, surgeons increasingly seek techniques which reduce postoperative fatigue. In the 1980's, renewed interest in measuring the subjective feeling of fatigue by psychologists (eg Burke et al 1989), led to the development of new instruments for measuring this phenomenon (eg Piper et al 1989, Rhoten 1982, Christensen et al 1982). Indeed, a department of fatigue research again exists to concentrate on the study of this phenomenon (Aaronson et al 1999). This has enabled an increasing amount of work to be carried out to measure fatigue in post-surgical patients in the last 20 years.

A literature search (appendix 9), identified 4 main methods that have been used previously for measuring post-operative fatigue. These are the symptom checklist, questionnaire, visual analogue scale (VAS) and numerical scale. They are discussed in more detail in the following sub-sections.

2.4.4.1 Symptom checklist
Fatigue being expressed as present or absent on a symptoms checklist has the advantage of simplicity and is used widely in studies of medication, and its side-effects (eg Santandrea et al 1993, John et al 1993, Tsaveris et al 1993). However side-effects commonly ascribed to drugs are common in the general population. Khosla et al (1992), for example, found that in a questionnaire survey of 236 healthy volunteers only 26% stated no experience during the past three days of the side-effects commonly ascribed to drugs. Fatigue is also very commonly experienced in the general population. Hannay (1978) interviewed patients from one practice at home and found that nearly a quarter felt more tired or 'run down' than usual. It is, therefore, difficult to distinguish between fatigue induced by surgery and "background noise" on a symptom checklist (Kristoffersson et al 1992).

Furthermore, nearly all patients who have undergone major surgery are likely to experience some fatigue whether as a result of the endocrine and metabolic processes which constitute the surgical stress response (Kehlet 1988a) or psychological state as suggested by Salmon & Hall (1996). It is the degree of fatigue experienced by the patient and how this changes over time that is of interest to most researchers of fatigue in postoperative patients, who wish either to show which treatment modalities reduce fatigue levels or to detect the abnormally high fatigue levels associated with post operative fatigue syndrome. A simple yes/no response cannot give this information and for these reasons this technique has only rarely been used to measure postoperative fatigue.
Significantly perhaps, of the 13 examples in appendix 9, 6 studied the effect of oral medication on postoperative recovery and 4 others were studies of patients who had undergone minor surgery which is not thought to be usually followed by increased fatigue (Christensen et al 1985).

2.4.4.2 Questionnaires
It is less clear why the questionnaire approach has been little favoured. It is very popular among psychologists (eg Fujii 1993), psychiatrists (eg Shwartz et al 1993) and researchers into chronic fatigue syndrome (eg Wood et al 1992), fatigue in patients with cancer (Pickard Holley 1991) and other chronic illnesses (Schaefer & Shober-Potylycky 1993, Crosby 1991). Questionnaires have been validated in non-surgical (Pickard Holley 1991a) and surgical populations (Buxton et al 1992) including populations undergoing hip replacement (Cleary et al 1991).

The most plausible explanation is that the longer time and greater effort needed by the subjects to complete questionnaires (McDowell & Newell 1987) may be a deterrent to their use. This is especially likely to be so in the peri-operative situation where the subjects are stressed, may be semiconscious due to sedatives and anaesthetics, possibly in pain and there is a lot of nursing care or patient monitoring going on. Questionnaires may also be disadvantageous when frequent measurement is required. For example Buxton et al (1992) blame the low test-retest reliability of fatigue measurement by the Profile of Mood States (POMS) on carelessness or indifference of patients confronted by a long list of adjectives. These authors note that many subjects complained about having to complete the POMS twice for the purpose of establishing test-retest reliability. Completing a long questionnaire may be tiring in itself, raising the subject's level of fatigue, thus creating a measuring artefact. This may partially explain the curious finding by Jacobs & Blandino (1992) that the scoring of fatigue by 246 students differed depending on the colour of paper the questionnaire (POMS) was printed on. No other subscale was affected. The colour may affect how difficult the questionnaire is to read or may have some impact on subject motivation.

There are however, advantages in using a questionnaire when fatigue is only one of many aspects of physical or psychological status being measured at the same time. Of the 20 references identified in the literature review (appendix 9), two (Buxton et al 1992, Schroeder & Hill 1993) used questionnaires partly to evaluate validity of various methods to measure post-operative fatigue; these and the remaining references describe studies of aspects of patients' quality of life, social or psychological adaptation to surgery or the relationships between psychological factors and physical outcomes, with fatigue being one of many parts of these investigations.

2.4.4.3 Visual analogue scales
If fatigue is the main focus of a study, it is advantageous to use the simplest available method, provided it is valid and reliable. The VAS (figure 2.3) fulfils the criteria of simplicity and utility as it is easy to understand, quick to complete, requires little effort on the part of the subject and even those with a slight visual impairment can complete it easily (Gift 1989). Aitken (1969) found it preferred by subjects as compared with adjectival scales while Eastwood et al (1984) used it daily over 9 months with good compliance. Christensen et al (1982), who can be considered the pioneers of modern post-operative fatigue measurement, introduced a constructed analogue scale which has been used by many researchers in this field (appendix 9, table 3).

Validity of the VAS
Validity has been established for the VAS using a variety of techniques, mainly concurrent validity (Gift 1989) - that is by comparing the results obtained using the VAS with those from another instrument which has been previously validated and which has been used simultaneously in the same group of patients. Evidence of concurrent validity of the VAS, for measuring postoperative fatigue, has been provided by comparison with the POMS (McNair et al 1971) which has been validated extensively, both in post-surgical patients (Buxton et al 1992) and in other patient groups. Schroeder & Hill (1993) found a highly significant correlation of fatigue scores on the Christensen et al (1982) VAS and fatigue subsection of POMS ($r = 0.767, p < 0.0001$). Buxton et al (1992) also found...
significant correlations between the fatigue subscale of the POMS and the Christensen et al (1982) VAS. Supporting evidence can be found from fatigue measurement using VAS’s and POMS in patients with other conditions (Blesch et al 1991) and healthy volunteers (Wood et al 1990). Wood concluded that VAS’s showed sufficient short-term stability to be used in more extensive studies, both of well being and of physiological correlates.

Figure 2.3 Constructed Visual Analogue Scale (after Christensen et al 1982; not to scale)

Concurrent validity can also be demonstrated through comparison with objective physiological correlates of fatigue such as loss of body weight (Buxton et al 1992, Christensen et al 1988), reduction in triceps skin fold (Christensen et al 1988), change in effort in the cycle ergonometer test (Buxton et al 1992) and increased heart rate in response to exercise (Christensen et al 1989).

Discriminant validity is demonstrated through comparing the results in populations which are expected to have differing outcomes. All studies of patients undergoing open abdominal surgery have observed differences in fatigue score before and after surgery (eg Schroeder & Hill 1993, Christensen et al 1982). As it involves less trauma, laparoscopic cholecystectomy is expected to cause less fatigue than open cholecystectomy. This has been found in many trials (eg Jakeways et al 1994, Hill et al 1993). The elderly and those who suffer from cancer would be expected to have more fatigue post-operatively. This too is supported (Schroeder & Hill 1993).

The necessity to demonstrate construct validity is proportional to the degree to which a phenomenon is abstract or concrete according to Nunnally (1978). Since individual perceptions such as fatigue are abstract, verbal responses are often subject to distortion and misinterpretation. Since the VAS is free of the constraints imposed by words it appears to be a viable tool for measuring these constructs (Mottola 1993), if it is accepted that fatigue is ‘what the patient says it is’.

As has been discussed previously, fatigue is a multi-dimensional phenomenon (central or peripheral, mental or physical, tiredness or weakness). Some authors (eg Wewers & Lowe 1990) have argued that the single-item VAS is inappropriate for measuring multi-dimensional phenomena as it is impossible to discern which dimension of the construct the subject is evaluating. Although this objection can be overcome by using multiple VAS (eg VAS-F, Lee et al 1991) this approach negates many advantages that make the VAS useful in the first instance, ie ease of use, speed and simplicity. In any case, there is little evidence to suggest that there is much benefit from using multi-dimensional scales or questionnaires to determine a global outcome. On the contrary, highly significant correlations have either been shown between single item measurements and multidimensional ones for

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such complex constructs as a feeling of health (Jenkinson et al 1994) or claimed (Forsberg et al 1996). Buxton et al (1992) suggest that a VAS seems to measure both tiredness and weakness equally, unlike the questionnaires they used. They therefore recommend the VAS if time and space permit only one measure.

**Reliability**
Buxton et al (1992) found a high test-retest reliability (0.81) of the Christensen VAS (higher than POMS). Numerous studies involving patients who have undergone abdominal surgery have shown consistent patterns of fatigue with most findings having a peak of fatigue between days 1-3 postoperatively followed by a gradual reduction to preoperative levels over 30-40 days. The magnitude of preoperative ratings is very consistent, with nearly all reported mean measurements being between 2.2 and 3.8 on the ten point scale.

**Sensitivity**
There are little data on the sensitivity of the VAS in measuring post-operative fatigue. A VAS has been found to be a more sensitive measure of subjective feelings than a graphic rating scale or a 4-point rating scale (Joyce et al 1975). VAS’s have also been shown to be sensitive to changes in symptoms over time (Gift 1989, Priestman & Baum 1976)

**Other issues in the use of the VAS**
The VAS has traditionally been used as a horizontally oriented scale without markings (Aitken 1969). However, more recently, it has been used as a vertical scale with or without gradations. The Christensen scale (1982) used extensively in the measurement of post-operative fatigue is an example of a graduated scale. Horizontal and vertical scales have been found to be equally valid, but the vertical scale is more sensitive, produces higher scores and is claimed to be easier for subjects to use (Gift et al 1986, Scott & Huskisson 1979, Sriwatanakul et al 1983). The use of graduations on the scale is thought to reduce sensitivity, however (Gift 1989).

**2.4.4.4 Numerical scales**
These scales appear to have been used only in the last 4 years. They are usually used as part of a more extensive questionnaire examining different parts of the patients’ post-operative experience. Their reliability and validity has not been established in the post-operative population.

**2.4.4.5 Confounding factors in measuring fatigue**
Whatever the method of measurement, validity of the results may be compromised by confounding factors. Fatigue levels in some chronic conditions appear to be affected by weather (de Bleucort et al 1993), gender (Ridsdale et al 1993, Belza et al 1993), psychological factors such as anxiety (Radvila 1991) and depression (Radvila 1991, Schroeder & Hill 1991), muscle use (Gift & Pugh 1993), pain (Belza et al 1993, Crosby 1991), drug abuse (Radvila 1991) and sleep quality (Belza et al 1993, Crosby 1991). However, there is little or no evidence, that these factors play a significant role in the response to surgery (eg Hjortso et al 1985, Schroeder & Hill 1993, Christensen & Kehlet 1993). Patients who present for surgery already fatigued, are most likely to remain fatigued longer postoperatively, especially if elderly, suffering from cancer or who have few extra reserves of body protein ( Schroeder & Hill 1993).

Measurement of fatigue in healthy and sick individuals has shown significant differences between morning and evening scores (Lee et al 1991, Wood et al 1990, Blesch et al 1991) but this effect may be absent in some diseases (van Hilten et al 1993). Elderly subjects tend to have higher fatigue scores than young ( Mangione et al 1993), oral contraceptives may increase fatigue in some women (Schanzer 1991) and a noisy environment has been shown to be related to fatigue in healthy individuals (Putt 1975).
2.4.4.6 Conclusions
The design of any study which aims to measure fatigue needs to be careful to take into account possible confounders (e.g., age, contraception, amount of surgical trauma) fatigue levels before surgery and nutritional status. It may be prudent to exclude patients with cancer from the samples. Fatigue should be measured at the same time of day and on the same preoperative and postoperative days in all subjects.

Although not tested extensively in orthopaedic patients, the VAS has been shown to be valid and reliable when used for measuring fatigue in post-surgical and other patients. It measures both weakness and tiredness (Buxton et al. 1992), is quick to administer and does not make excessive demands on the subjects’ time, effort and concentration. While it has been suggested that POMS is an improvement on the VAS (Salmon & Hall 1997), there is little or no evidence of that in other literature. If the aim of the study is the measurement of global fatigue, then existing literature supports the VAS as the instrument of choice, particularly if multiple measurements are to be made (Buxton et al. 1992).

2.5.2 Heart rate
The heart rate is defined as the number of ventricular contractions within a given time interval and is usually described in terms of beats per minute (bpm) (Wilson et al. 1989). As the heart expels a bolus of blood with every beat, this is thrown into the arterial system. This causes an increase in blood pressure within the arteries which elastically expand and then contract after the wave has passed. This is called a pulse and can be manually felt at various points of the body with every heartbeat. The simplest and most common method of heart rate measurement involves the manual palpation and timing of the pulse for a set period of time. This is usually recommended to be a period of 1 minute (Maslen 1995) to ensure levelling out of the sinus arrhythmias - the continuous acceleration during inspiration of breath and deceleration during expiration. The main advantages of the manual method are minimal equipment and training and convenience for the researcher and the subject. The main disadvantage is the interaction between subject and measurer which may potentially alter the heart rate.

There are also a number of automated techniques which include electrocardiography (ECG), echocardiography, impedance cardiography (ZCG) and the less common oscillometric measurement of pressure fluctuations, plethysmography and phonocardiography. These methods are reviewed in Wilson et al. (1989) and all rely on measuring some physical manifestation of each beat. For example ECG relies on electrical activity of the heart, while nuclear cardiac imaging observes blood flow through the heart. All the automated techniques measure other variables as well as the heart rate itself such as heart sounds (ZCG), valve motion (ECG, Echo, ZCG) or cardiac volumes (Echo).

The main advantages of automated methods are their greater precision and unobtrusiveness. Once the appropriate transducers are fixed to the patient, measurements can be made over a prolonged period of time and without the researcher present, thus reducing the 'white coat effect' a recognised confounding variable in clinical measurement (e.g., Carek et al. 1995). Disadvantages include the need for expensive equipment, training of the researcher, the inconvenience of attaching transducers to the patient and, most importantly, if the patient remains attached to the machinery for some of these methods, their ability to mobilise will be restricted, thus confounding any study of mobility.

Overall the disadvantages of automated methods over the simple palpation of pulse appear to outweigh the advantages in a sampling study. The main disadvantage of 'white coat effect' can be minimised if the same individual carries out all the measurements.
2.5.3 Blood pressure

The pressure in the arteries (defined as the force exerted by the blood on the walls of the vessels within which it is contained) is one of the most widely measured cardiovascular variables (Maslen 1995). Arterial blood pressure has a characteristic waveform caused by the pumping action of the heart. There is an initial rapid rise followed by an early systolic peak (percussion wave), followed by a second slightly smaller late systolic peak (the tidal wave) and then a smaller still notch (the incisura) which corresponds to the closing of the cardiac valves. During diastole, the pressure gradually decreases to baseline. The peak pressure is labelled the 'systolic pressure' and the baseline, the 'diastolic'.

Despite efforts to find a better method, the Korotkoff method described first in 1905 is still the most widely used today (Pickering & Blank 1989). A cuff is placed around the arm, inflated to above arterial closing pressure and then slowly deflated. On auscultation, when the systolic blood pressure is reached a regular thumping sound can be heard. As the cuff continues to be deflated, the sound disappears then reappears, becomes muffled at about 7 mmHg above true diastolic pressure and disappears at around 2 mmHg above true diastolic (Short 1976). The cuff is inflated by an air pump via a sphygmomanometer on which the cuff pressure can be read off a scale. The most commonly used is a mercury sphygmomanometer, in which pressure is indicated by a column of mercury. Another type are aneroid sphygmomanometers, where pressure is measured using a coiled spring but these appear to be less accurate (Burke et al 1982). The Korotkoff sounds are thought to be caused by pressure-induced movements of the arterial wall (Dock 1980), although there is some debate (Pickering & Blank 1989).

The Korotkoff method using a mercury sphygmomanometer gives an accuracy to within 0.9 mmHg as compared to 'gold standard' intra-arterial catheter measurements. However, there are significant potential sources of methodological, intraobserver and interobserver error. There is no full agreement on whether the muffling phase or the final disappearance of sound should be used for the determination of diastolic pressure, although the American Heart Association (Kirkendall et al 1981) and the British Hypertension Society (O'Brien et al 1997) recommend the disappearance of sound.

There are a number of potential errors of technique. Cuffs that are the wrong size for the diameter of the arm lead to significant error (Pickering & Blank 1989). If the arm is held down rather than being supported at mid-sternum, there can be an artificial rise in systolic and diastolic pressure of up to 20 mmHg (Netea et al 1999). If the arm is unsupported, the muscular contraction required to keep it in place raises systolic pressure by up to 8 mmHg (Webb 1980). The positioning of the column of mercury other than at the level of the heart, the column not being set to zero when no pressure is applied and blockage of the air exit hole are other common mechanical problems (Pickering & Blank 1989). Observer error and bias are also important sources of error. Wilcox (1961) found wide inter- and intraobserver variation. Digit preference is also common, with most observers recording 5 or 0 ending numbers (Pickering 1968). The ambient temperature may also affect the results by changing the amplitude of the Korotkoff sounds and thereby the audible range (Rabbany et al 1993). Finally the recorded level can be affected by psycho-social interactions between the observer and the subject. The 'white coat effect' - that is the artificial rise of blood pressure by up to 20 mmHg when it is measured by a physician was first observed in 1940 (Ayman & Goldshine 1940). Physicians also record higher pressures than do technicians or nurses (Mancia et al 1983). Comstock (1957) found that men tended to have higher blood pressure when it was measured by a woman whereas the opposite was true for women.

Apart from aneroid sphygmomanometers mentioned earlier, a number of devices have been constructed as an alternative to standard mercury sphygmomanometers to overcome specific types of error, to allow continuous monitoring and to allow measurement without a cuff. These are reviewed by Pickering & Blank (1989) and Kenner (1996). They do not seem to offer any advantages in general blood pressure measurement and some give significantly less accurate results.
A number of automatic and semiautomatic devices are also available, which use Korotkoff sound detection, oscillometry or ultrasound. Their main advantages are the ability to measure and record blood pressure over a period of time and reduce placebo and 'white coat effects'. Ambulatory models are now available which have minimal effect on patient mobility (Portaluppi 1996). This has led to the suggestion that mercury manometers will soon be obsolete (O’Brien 1995). However, for the purposes of a study sampling blood pressure in two populations of patients, the extra expense and inconvenience to the patient of automatic devices is not justified. White coat error would be small if the researcher is not a physician and/or not seen as such. The decrease in 'white coat error' which would be the main benefit of automatic techniques is therefore unlikely to affect overall error significantly, the main component of which would be sampling error. A recognised procedure such as the British Hypertension Society recommendations (1990) should be followed to minimise methodological error. Interrater error can be eliminated if only one researcher carries out the measurements.

Another issue relating to the measurement of blood pressure is the presentation of the results. Various measures of blood pressure can be derived from the initial systolic and diastolic determinations. Two common ones are the 'pulse pressure' and the 'mean arterial pressure'. Pulse pressure reflects the stroke volume of the heart and is calculated by subtracting diastolic from systolic blood pressure. Mean arterial pressure represents the average arterial pressure throughout the cardiac cycle. Its relevance lies in that the difference between mean arterial pressure and venous pressure is what drives blood through the capillary beds. It is estimated by adding 1/3 pulse pressure to the diastolic pressure (Colbert 1993). However, since these measurements can be easily derived from the basic systolic and diastolic blood pressures, this seems to be the method of choice for presenting the data as other. This approach has been taken by most studies investigating the effects of orthostasis on blood pressure (eg Dambrink & Wieling 1992, Katkov & Chestukhin 1980) which gives this method the added benefit of comparability.

2.5.4 Lower limb oedema
The 'gold standard' for measuring lower leg volume is the measurement of displaced fluid when the limb is placed in a container of water (Wilson et al 1991). However, orthopaedic patients may not be able to lift their legs into a container following hip or knee repair/replacement and the method is very inconvenient and time-consuming for ward use. Patients who have undergone knee replacement are likely to have their wound dressings made damp. This might increase the risk of wound infection as the dressings would need to be changed more frequently than otherwise would be necessary, thus exposing the wound to contamination. The 'gold standard' method of volume measurement is also not without its own error. The back-pressure of the column of water the leg is placed in, reduces the volume of the leg by pushing some of the blood back up.

An alternative method is to measure the calf at its widest point, the ankle at its narrowest and the distance between the two. These measurements can be used to calculate the volume of the leg using the formula for the frustum of a cone (Persson et al 1989). Persson et al (1989) compared the frustum method with water displacement volumetry in 10 patients (20 limbs) and found a good correlation, (correlation coefficient $r = 0.97$) even though the frustum method did not include the foot.

For repeated measurements in multiple patients, the water displacement method appears to have too many practical drawbacks to justify the small increase in precision. Therefore the frustum method is the method of choice.

2.5.5 Deep vein thrombosis
Clinical diagnosis of deep vein thrombosis (DVT) has been shown to be both insensitive and non-specific (Barnes et al 1975) in comparison with contrast venography (Gauer 1940) which is generally recognised nowadays as the "gold standard" for the diagnosis of this condition (Lensing et al 1992). Up to 80% of patients with classical signs and symptoms of DVT (calf swelling, redness, pain,
Homans' sign) do not have this condition (Barnes et al 1975) and in some groups such as postoperative patients DVT is usually asymptomatic (Kakkar et al 1969). Most authors, therefore, advocate objective testing for DVT (Koopman et al 1994) since treatment of all patients suspected of DVT on clinical grounds with anticoagulants would unnecessarily expose large numbers to the potentially life-threatening side-effect of bleeding (Levine 1992).

Unfortunately, contrast venography, is an invasive procedure involving the injection of contrast medium into the veins (Rabinov & Paulin 1972). This may exclude up to 20% of patients who either do not have patent veins or cannot be injected on medical grounds (Heijboer et al 1992). In up to 22% it is associated with side-effects such as allergic reaction, local pain, nausea, vomiting, dizziness, skin reactions, oedema and thrombophlebitis (Lensing et al 1990). It is also highly likely that the procedure itself may cause thrombus formation in some patients (Hull et al 1981). Furthermore contrast venography involves considerable expense, disruption to the patient and the services of a radiology suite, special equipment and skilled staff (Baker & Bick 1994). Because of these limitations, numerous other non-invasive techniques have been developed. Impedance plethysmography (IPG) is of value in the diagnosis of symptomatic patients but it has a low sensitivity (24%) (Agnelli et al 1991) in asymptomatic postoperative patients. This makes the technique unsuitable as a screening tool as even with the additional use of $^{125}$I-fibrinogen leg scanning sensitivity is only 50% (Paiement et al 1988).

Real-time B-mode ultrasonography (US) visualises the deep veins of the leg and it has been shown that non-compressibility of the affected vein by gentle pressure with a probe is the most accurate diagnostic criterion (compression ultrasonography - CUS; Lensing et al 1989). Other US techniques include Doppler US, colour Doppler US and a combination of real time B-mode US with colour Doppler (duplex scanning). None of these techniques are sufficiently sensitive in asymptomatic patients to be used as a screening test (Davidson et al 1992). $^{125}$I-fibrinogen leg scanning has a low sensitivity for small calf thrombi (Paiement et al 1988) and is time consuming, expensive and requires the services of a nuclear medicine department. However the main reason for its discontinuation as a diagnostic technique is the risk of transmission of viral, blood-borne disease as the fibrinogen is of human derivation and cannot be pasteurised (Koopman et al 1994). Other scintigraphic methods such as $^{99m}$Tc red blood cell venography have been found to be inaccurate (Lisbona et al 1982) and have not been introduced into clinical practice (Koopman et al 1994).

Thermography is a noninvasive method to detect temperature differences on the surface of the human body and was introduced by Soulenuet et al in 1972. The test for DVT is based on the principle that the affected leg is likely to have an increased skin temperature. A temperature difference between a patients' legs greater than 0.7°C is considered abnormal. The initial telethermography has been more recently replaced by liquid crystal contact thermography (LCCT; Pochaczewski et al 1982). The liquid crystal detector consists of latex sheets impregnated with cholesteric crystals which change in colour with temperature. A polaroid photograph can be taken of each thermographic image as a permanent record.

The main advantages of this technique are that the equipment is portable, can be used on the ward and does not require much training to use allowing use by a layman. In symptomatic patients it has a high sensitivity (97% - 100%) as compared with ascending venography (Pochaczewski et al 1982, Jensen et al 1983, Sandler & Martin 1985) but low specificity (50-97%). This is due to false positive thermograms generated by other conditions such as ruptured bakers cysts, cellulitis, muscular or joint trauma and superficial thrombophlebitis. Further investigation is necessary, therefore, where a positive result is obtained. Bounamaux et al (1989) found that because of many bilateral emboli, the method had a low sensitivity (55%) in a study of symptom free, high risk patients (digestive surgery).

Elevated levels of D-dimer (DD), a fibrin derivative, are found in individuals with DVT. Measurement of plasma levels of DD is sensitive to the presence of DVT in symptomatic patients (95-100% sensitivity) (Heaton et al 1987, Rowbotham et al 1987, Ott et al 1988, Bounamaux et al 1989).
It is also sensitive in asymptomatic patients (89% when a cutoff DD level of 3,000 ug/l was taken) (Bounameaux et al 1989). For this reason, although it involves taking 5ml of blood for analysis from the patients, DD assay is the method of choice for screening asymptomatic patients. However specificity is low (48%) (Bounameaux et al 1989), so that positive tests need confirmation by ascending venography.

2.5.6 Chest infection
Fundamental to the concept of measuring the incidence of a condition is a precise definition (Nixon 1990). For purposes of research or audit the criteria should be unambiguous and as objective as possible as comparisons can only be made where terminology is clear and recognised (Gordon & Reid 1986). Therefore vague criteria which have been used in the past such as "attending physician's diagnosis of infection" (Garner et al 1988) are not very useful. However reliance on laboratory data only can be equally unhelpful. For example sputum samples are frequently contaminated with oropharyngeal bacteria (Lode et al 1993). Therefore if definition is based solely on the presence of bacteria in sputum, there will be a large over-estimation of the problem. A compromise is needed, and this has been achieved, mostly using the consensus group approach, by combining laboratory and clinical observations into formulae that allow reasonably precise definition for research/audit purposes (eg Garner et al 1988, McGeer et al 1991, The Steering Group of the National Prevalence Survey of Hospital Acquired Infections 1993).

It should be noted, however, that all these definitions still rely on subjective analysis of data such as x-rays and patient signs and symptoms, and therefore cannot be described as objective. Another problem with identifying infections in post-operative patients is the possibility of symptoms of infection being masked by the surgical response (Machiedo & Suval 1988). The usual signs of infection - fever, leukocytosis, malaise and symptoms specific to the site of infection (cough, chest pain) are clear in a patient with no other medical condition. However in the post-operative patient many of the general signs (elevated temperature and white blood cell count, malaise) are the normal responses to surgical stress. The same might be true of local responses such as chest radiographic changes being confused with atelectasis. Therefore findings should be treated with some caution.

The various definitions differ mainly in structure and emphasis and there is little or no research to determine their reliability and validity. Therefore, for research purposes, choice is probably best guided by what is most familiar to the individuals who will make the clinical decisions of whether a patient has or has not got a chest infection. In the case of studies in the UK, this is most likely to be the criteria adopted by the Second National Prevalence Study of Hospital Acquired Infections (Steering Group of the National Prevalence Study of Hospital Acquired Infections 1993).

2.5.7 Pressure sores
The presence of pressure sores is thought to be relatively easy to determine objectively as they have a physical presence and are clearly visible on the skin surface. However there are a number of areas of possible confounding. Some studies (of prevalence for example; eg O'Dea 1994) have included persistent erythema, which is considered a marker of pressure damage. This usually presents as redness or other discolouration, without an actual skin break. Diagnosis of persistent erythema has been found to be unreliable (Barbenel et al 1977) and is probably best excluded, especially in the context of an incidence study where clinically relevant tissue damage is likely to be revealed as a skin break before the end of the study period.

It is also important to exclude tissue damage of other aetiologies. Therefore, blisters are probably best excluded as they may be caused by incorrect patient movement techniques (St Clair et al 1995) and so are any wounds smaller than 0.5cm, as the aetiology of such wounds may be questionable. It seems logical, also, to exclude pressure sores on areas such as the back of the heels which are not subject to pressure while sitting.
It is also important to ensure, as much as possible, that the results are not confounded by pressure sores developed during bedrest when the patient is not sitting in a chair. To this end the bed should be provided with an effective pressure relieving or reducing mattress. Large-celled alternating pressure mattresses (Bliss et al - unpublished data) and foam mattresses (Gray & Campbell 1994) have been found to be effective in preventing pressure sores in orthopaedic patients. There does not appear to be any difference in the effectiveness of these two types of support in this patient group (Gebhardt & Bliss 1993).

2.5.8 Constipation
A definition of constipation has been discussed previously (section 2.3.8). Although the definition of constipation being present if there has not been a bowel movement for three days appears to be based on tradition rather than a calculation based on population norms (Clinch & Hilton 1998) it has the advantages of being unambiguous, readily recognised, and comparable with studies of prevalence (eg Whitehead et al 1989).

2.5.9 Urinary tract infection (UTI)
The general considerations regarding measurement of the incidence of UTI are similar to those for chest infection described in section 3.5.6. Therefore, here too the best choice appears to be the criteria adopted by the Second National Prevalence Study of Hospital Acquired Infections (Steering Group of the National Prevalence Survey of Hospital Acquired Infections 1993).

2.5.10 Measurement of mobility
As has been previously discussed (section 2.1), mobility has seldom been precisely defined or measured. Most commonly it appears to be measured as hours of not being in bed (Moiniche et al 1995, Bardram et al 1995). Where it has been specified as an activity, mobility has generally been measured in terms of endpoints which are meaningful for that particular study or in the particular environment that the study is conducted in. For example Krantz et al (1990) devised a scoring system for post-operative patients which took into account daily fluid and food intake, bowel and bladder function, washing, mobility and mental needs. These activities are scored daily and a global score is calculated for each patient. Simpler assessments require the patient to achieve certain goals such as weightbearing with support or walking the length of the ward (Bastow et al 1983). The length of time to the achievement of these goals from the day of surgery is the unit of measure. In the absence of commonly agreed approaches or data on reliability and validity of these systems, the simpler system is attractive in that it is less labour intensive and therefore appears to be the method of choice.

2.5.11 Study design
There are a number of possible approaches to studying the effects of an activity on physiological processes and their potential outcomes. The main division is between observational studies and experimental studies (Bland 1992). In observational studies, the effects of the activity, in this case chairnursing, could be observed by chairnursing patients or volunteers for periods of time and recording observations of (for example) blood pressure, pulse rate fatigue and similar during the study or before and after. Many studies of this nature have been carried out in the past, including studies of the effects of prolonged sitting (eg Lamb et al 1964, 1965, Greenleaf et al 1980). These studies can give a lot of data on the processes that occur during the activity, but cannot establish causative relationships between these processes and clinical outcomes.

A clinical trial needs to be carried out to establish connections between a healthcare intervention (such as prolonged chairnursing) and an outcome (such as the development of pressure sores). Clinical trials are a type of experimental study (Bland 1992). A number of bodies have suggested that randomised, controlled trials are the best method of assessing health care interventions in all cases (Bergstrom et
al 1994, Royal College of Nursing and Midwifery 1998). This does not seem to be a defensible position. The best methodology for any given research question is one which has the fewest threats to validity and reliability built into it.

A useful approach to studying the effects of chairnursing might be to limit the amount of chairnursing in one group of patients and compare the results with another group in whom chairnursing was unlimited. This type of study is effectively the study of the effect of a policy (in this case of limiting chairnursing) on a patient population. A body of literature exists on the study of the implementation of policies which is reviewed by Davies et al (1994). It is believed that in this type of research, the randomised controlled trial is subject to a range of biases such as the contamination of the control by protocol or vice-versa when the same clinician cares for patients in both groups, which limit its usefulness (Russell & Grimshaw 1992).

Randomisation by ward to either limited chairnursing policy (LCP) or standard management has the disadvantage that the wards allocated to LCP are subject to greater Hawthorne effects (the beneficial effects on performance of taking part in research) (Moser & Kalton 1971) and so the benefits of LCP may be overestimated.

One of the most reliable trial designs for the implementation of a policy is considered to be the balanced incomplete block design based on two or more clinical conditions (Davies et al 1994). For example, in the case of chairnursing, patients on one ward who have emergency surgery could be allocated to LCP while those undergoing elective surgery could be allocated to standard management. On another ward this would be reversed. However, since this particular intervention affects basic care there is the possibility of contamination through learning effects (for example). Furthermore in this particular context this design is complex to carry out in practice. There is the possibility that patients would receive wrong interventions due to staff being confused as to which policy they are to follow with regard to which patient.

Another design considered to be powerful is the cross-over trial (Davies et al 1994). Here the clinicians implement different regimes in random order and therefore act as their own controls. The main benefit is the reduction in Hawthorne effects. There may however be contamination across periods due to, for example, learning effects which may lead to an underestimation of the effect of the policy. The contamination across periods can be reduced by allowing a period of 'washout' between phases of the study. These are periods during which no data collection takes place so that the clinicians have time to return to normal patterns of activity. The relative practical simplicity of this approach coupled with the likelihood of good reliability suggest this is the method of choice where resources are unavailable for carrying out a complex or multi-centre trial.

2.6 OVERALL DISCUSSION AND CONCLUSIONS

When patients are chairnursed for prolonged periods, they experience challenges to their circulatory homeostasis. These challenges are likely to be orthostatic, postural and due to immobility. It is possible that the circulatory systems of elderly post-operative patients may not be able to meet these challenges fully and, therefore, the patients may become less well perfused than optimal. It was postulated that as a result, patients in this group were likely to become fatigued, tachycardic and develop pressure sores. Furthermore, it was postulated that as a result of orthostatic and postural forces and due to immobility they were also at increased risk of hypotension, lower limb oedema, DVT, chest infection, constipation and UTI.
A cross-over study appeared to be a useful methodology for determining whether a policy of limiting
chairnursing could be used to reduce the incidence of negative outcomes for the variables itemised
above compared with standard practice, that is unlimited chairnursing. The literature review suggested
various methods of choice for measuring these outcomes.

A preliminary study was carried out to determine the practical usefulness of the study design and of
some of the measurement methods. This preliminary study is described in the next chapter.
3 PRELIMINARY STUDY

3.1 INTRODUCTION

This study had two main aims. The first was to determine if the two-ward, crossover trial was a useful study design in practice. The second was to test the null hypotheses that a policy of limiting chairnursing to no more than 2 hours per session would have no effect on the length of chairnursing sessions and the incidence of pressure sores. Pressure sores were selected as the model to test the study methodology, as, based on the results of a concurrent study of pressure relieving equipment, it appeared likely that there would be a large difference in outcomes if a policy of limited chairnursing could, in fact, reduce the length of chairnursing sessions.

A secondary aim of the study was to identify other factors which may be affected by the policy and to identify differences in outcomes for those factors, to enable calculation of sample sizes for the main study.

3.2 STUDY DESIGN

3.2.1 Location and subjects

The study was conducted in two orthopaedic wards, 1 and 2, which were identical in design and shared the same consultants. The only difference in policy was that ward 2 had the benefit of a consultant geriatrician to assist with medical management and discharge planning of elderly patients; however, since even early discharge took place more than 2 weeks postoperatively, this was unlikely to affect the trial seriously. All patients >65 years who had had a fracture or major orthopaedic surgery of the pelvis, hip or lower limb within the previous four days were recruited to the trial. To prevent the development of pressure sores in bed, subjects were given a large celled alternating pressure air mattress (APAM). This type of equipment has been shown to be effective in randomised controlled trials (Gebhardt et al 1996, Bliss 1995, Bliss et al 1967).

3.2.2 Method

In the experimental ward, chair nursing was restricted to two hours per session. There was no restriction on the number of sessions per day but patients had to have at least two hours in or on the bed between sessions. Patients in the control ward were nursed according to the normal routine of that ward with the amount of time they spent in chairs decided by the ward staff. All patients were allowed physiotherapy and encouraged to walk with assistance if possible. The times that the patients were sat out in chairs and put back to bed were recorded by the staff with the assistance of the researcher.

Baseline data consisted of age, sex, body build (estimated by researcher), grip strength (measured using grip strength dynamometer), smoking history, mental test score (Quareshi & Hodgekinson 1974), Norton score (Norton et al 1962; see appendix 10), associated diagnoses, type of surgery (emergency or elective) and condition of pressure areas. The patients' mental test score, Norton score, drug therapy and pressure areas were further assessed every 3-4 days throughout the trial. Pressure sores were defined as actual skin breaks. Other outcomes (e.g., deep vein thrombosis, urinary tract infection, constipation and ankle oedema) were identified through medical or researchers diagnosis. Patients remained in the trial for two weeks or until they could walk independently; they were discharged/transferred to another ward; they died. No patient was entered into the study more than once.
A power calculation (80% power, p = 0.5 significance level) based on the results of the previous study of pressure relieving equipment (Bliss et al unpublished data) and allowing for 10% dropout, suggested 114 subjects would be needed in each group to detect a difference of 20% from a base of 5%. Records of patients admitted to the orthopaedic wards in 1992 showed that a total of 8 months would be necessary to achieve this sample size. To obviate differences in ward management, a crossover between the experimental and control wards was planned after 4 months with an intervening month’s adjustment period.

3.2.3 Analysis
The results were analysed to assess the comparability of the two patient groups and of any association between the number of chair hours and the development and progress of complications. Demographic, health characteristics and treatment variables were compared using: Chi square test for categorical variables; t-tests for normally distributed and the Mann Whitney test for non-normally distributed continuous data. Significant differences were accepted at the p = 0.5 level. The Chi square test was used to compare the incidence of complications in the experimental and control groups, significant differences being accepted at the p = 0.5 level.

3.3 RESULTS

3.3.1 Subjects
Analysis of the data after three months showed that the difference in the incidence of pressure sores between the limited and unlimited groups was almost three times greater than expected, so on ethical grounds it was decided to reduce the study period to 6 months, ie 3 months followed by crossover, a months adjustment period, and another 3 months data collection (7 months in total).

Fifty seven patients were recruited to the trial, 30 in the limited and 27 in the unlimited group with more patients being admitted to ward 2 in both groups (table 4.1), possibly due to the consultant geriatrician input.

<table>
<thead>
<tr>
<th>Table 3.1. Patients and chairnursing</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Chairnursing</strong></td>
</tr>
<tr>
<td><strong>Patients</strong></td>
</tr>
<tr>
<td>total</td>
</tr>
<tr>
<td>Ward 1</td>
</tr>
<tr>
<td>Ward 2</td>
</tr>
<tr>
<td><strong>Days in trial</strong></td>
</tr>
<tr>
<td>total</td>
</tr>
<tr>
<td>in chair (%)</td>
</tr>
<tr>
<td><strong>Chair sessions:</strong></td>
</tr>
<tr>
<td>total</td>
</tr>
<tr>
<td>no per day (%)</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3-8</td>
</tr>
<tr>
<td>hours per session (%):</td>
</tr>
<tr>
<td>&lt;2</td>
</tr>
<tr>
<td>&gt;2-3</td>
</tr>
<tr>
<td>&gt;3-6</td>
</tr>
<tr>
<td>&gt;6-9</td>
</tr>
<tr>
<td>&gt;9-12</td>
</tr>
<tr>
<td>&gt;12</td>
</tr>
<tr>
<td>median hours per patient per session (range)</td>
</tr>
</tbody>
</table>
3.3.2 Extent of lengthy chairnursing

The number of days spent on full bedrest was similar in the limited and unlimited groups, mean 2.9 (SD± 2.3) and 3.2 (SD± 3.0) days respectively. However, on the days when patients sat out, 83% of sessions in the limited group were restricted to two hours or less and only 8% lasted longer than 3 hours. In contrast, in the unlimited group, only 18% of chair nursing sessions were for under 2 hours and 73% were for more than 3 hours, including 21% for over 9 hours. The majority of the unlimited patients were sat out for long periods once daily, compared with patients in the limited group who on 16% of days got up twice and on 14% between 3 and 8 times. The median length of chair nursing session in the limited and unlimited groups was 2 (range 0.25-12.0) and 6 (range 0.25-15.25) hours respectively.

Table 3.2 Patient characteristics

<table>
<thead>
<tr>
<th></th>
<th>Limited</th>
<th>Unlimited</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total patients</td>
<td>30</td>
<td>27</td>
</tr>
<tr>
<td>Age in years (mean and range)</td>
<td>78 (65-94)</td>
<td>79 (65-92)</td>
</tr>
<tr>
<td>Sex: Male/Female</td>
<td>5/25</td>
<td>4/23</td>
</tr>
<tr>
<td>Build: no (%) emaciated</td>
<td>4 (13)</td>
<td>5 (19)</td>
</tr>
<tr>
<td>average</td>
<td>11 (37)</td>
<td>10 (37)</td>
</tr>
<tr>
<td>obese</td>
<td>15 (50)</td>
<td>12 (44)</td>
</tr>
<tr>
<td>Grip strength in Kg (mean and range)</td>
<td>13.1 (0-30)</td>
<td>13.0 (5-28)</td>
</tr>
<tr>
<td>Smokers: no (%)</td>
<td>4 (13)</td>
<td>3 (11)</td>
</tr>
<tr>
<td>Mental Test Score &lt;8: no (%)</td>
<td>9 (30)</td>
<td>8 (30)</td>
</tr>
<tr>
<td>Norton Score (median and range)</td>
<td>11 (7-16)</td>
<td>11 (7-16)</td>
</tr>
<tr>
<td>Associated diagnoses: no (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arthritis</td>
<td>13 (43)</td>
<td>11 (41)</td>
</tr>
<tr>
<td>Circulatory disease</td>
<td>7 (23)</td>
<td>2 (7)</td>
</tr>
<tr>
<td>Bowel disorders</td>
<td>2 (7)</td>
<td>2 (7)</td>
</tr>
<tr>
<td>Dementia/confusion</td>
<td>4 (13)</td>
<td>3 (11)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1 (3)</td>
<td>3 (11)</td>
</tr>
<tr>
<td>Infections</td>
<td>1 (3)</td>
<td>3 (11)</td>
</tr>
<tr>
<td>Breast cancer</td>
<td>1 (3)</td>
<td>1 (4)</td>
</tr>
<tr>
<td>Surgery - acute planned</td>
<td>18 (60)</td>
<td>18 (67)</td>
</tr>
<tr>
<td></td>
<td>12 (40)</td>
<td>9 (33)</td>
</tr>
<tr>
<td>Catheterised</td>
<td>16 (53)</td>
<td>14 (52)</td>
</tr>
<tr>
<td>Anticoagulated during trial</td>
<td>14 (47)</td>
<td>14 (52)</td>
</tr>
</tbody>
</table>

Table 3.2 shows the characteristics of the patients in the two groups. There was no significant difference in the patients’ mean ages, sex or physical or mental state.

3.3.3 Incidence of complications

7% patients in the limited chairnursing group developed pressure sores compared with 63% in the unlimited group (p = 0.000007, 95% CI of the difference: -77 to -36%)(table 3). No patients in the limited chairnursing group developed chest infections compared with 3 in the unlimited group. Incidence of urinary tract infection was similar, 10% and 11% respectively but patients in the limited group were significantly less constipated than those in the unlimited group (33 and 59%; p = 0.05). In addition, despite the fact that the limited group had more patients with cardiovascular disease,
ankle and foot oedema occurred almost exclusively in the unlimited chairnursing patients (3 and 26%; p = 0.02). One patient in the unlimited group had swelling to the knees and two patients were unable to wear their slippers which may have delayed mobilisation.

Patients nursed for long periods in chairs frequently complained of exhaustion. Examples include "I was out too long, I couldn’t get back into bed quick enough. I was so utterly exhausted I wanted to cry"; "I was up all day yesterday - it wore me out"; "My bottom feels sore from sitting." In contrast, some of the patients on limited chair nursing became very active. One got in and out of bed 8 times a day. They did not feel that this was difficult, some seeing it as "breaking up the monotony of the day" and "good exercise".

3.3.4 Independent walking
Sixteen (53%) patients in the limited group became independently mobile before the end of the trial compared with only 9 (33%) in the unlimited group although this was not statistically significant (p=0.07)

Table 3.3 Results

<table>
<thead>
<tr>
<th>Chairnursing group</th>
<th>95% confidence interval of the difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients Total</td>
<td>Limited</td>
</tr>
<tr>
<td>Pressure sores:</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>number</td>
<td>2 (7)</td>
</tr>
<tr>
<td>mean diameter (cm)</td>
<td>2.3</td>
</tr>
<tr>
<td>Other complications:</td>
<td>no (%)</td>
</tr>
<tr>
<td>deep vein thrombosis</td>
<td>1 (3)</td>
</tr>
<tr>
<td>urinary tract infection</td>
<td>4 (13)</td>
</tr>
<tr>
<td>chest infection</td>
<td>0 (0)</td>
</tr>
<tr>
<td>constipation</td>
<td>10 (33)</td>
</tr>
<tr>
<td>ankle oedema</td>
<td>1 (3)</td>
</tr>
<tr>
<td>Outcome: died</td>
<td>-</td>
</tr>
<tr>
<td>transferred</td>
<td>5</td>
</tr>
<tr>
<td>walking at two weeks</td>
<td>17</td>
</tr>
</tbody>
</table>

3.4 DISCUSSION

The results of the study showed that implementation of a policy of limited chairnursing, did reduce the incidence of prolonged chairnursing. When a regime departs from usual clinical practice it is inevitable that despite the best efforts of the researcher and ward staff, it will not be adhered to at all times. Some patients in the experimental wards sat out for longer than two hours, for example, two demented patients who refused to comply with the limited regime and accounted for 31% of sessions longer than 2 hours, 52% longer than 3 hours and all sessions longer than 6 hours. However, overall, particularly if the data from 2 demented patients was excluded, there were few sessions over 2 hours.

Another concern with cross-over trials is that when the experimental ward becomes the control ward after cross-over, the behaviour of the staff may be influenced by the experimental regime that they were under in the first half of the study (learning effect). There was no evidence of a large effect in this case. Although there appeared to be slightly fewer chairnursing sessions over 7 hours following cross-over, there was no significant difference either in the number of sessions over 4 hours overall, or in the incidence of pressure sores.
Thus, overall, the trial methodology seemed to be appropriate and practicable for assessing differences between two policies. Some methods of measurement were not found to be either adequate or useful. Assessing body build by researcher estimate was felt to be highly unreliable in practice. It would have been better to have used a more objective method of comparison such as the body mass index (Bray 1978). Nurses’ recollections of how long patients had been sitting in chairs were found to be unreliable. Only time-sheets filled in by patients or by the researcher were useful guides to the length of time patients had been up. For future research it was felt that either comprehensive daily assessment of chairnursing time would have to be undertaken or chairnursing time should be assessed by sampling. Observation of outcomes through clinical diagnosis also appeared unreliable and it would have been better to have had fixed criteria for the recording or otherwise of medical conditions. Ankle oedema in particular was difficult to identify clearly and an objective measurement of leg size or volume appeared to be a better approach, with hindsight. Finally, it was observed that demented patients were occasionally unable to comply with the limited chairnursing regime, thus significantly skewing the findings. They should probably be studied separately as their management is likely to have to be different in important ways to that of alert patients.

The difference in pressure sore incidence between the two groups was in accordance with those of other pressure sore surveys (Petersen & Battmana 1971, Barbenel et al 1977, Nyquist & Hawthorn 1987, St Clair 1992) which have shown that patients being nursed in chairs are more likely to have sores than patients in bed. Although the sores seen in this study were small (mean maximum diameter 1.4cm), had it not been for the presence of the researcher and the raised awareness of pressure area care generated by the study, it is likely that at least some would have deteriorated further. Even the smallest sores were often very painful, once general anaesthetic had fully worn off. "My arse is sore as hell! " as one patient put it.

The results of the pilot study generally supported the hypothesis postulated at the end of chapter 2. The complications being monitored either showed no difference of incidence (DVT, UTI) or had a greater incidence in the group chair-nursed for long periods (chest infection, constipation, ankle oedema, pressure sores). Patients nursed in chairs for long periods also appeared to be more fatigued and significantly fewer of them became independently mobile by two weeks.

3.5 CONCLUSIONS

This work has been published (Gebhardt & Bliss 1994) -a copy is attached as appendix 2. The method was found to be practicable in this patient population. Introduction of the policy seemed to produce an environment which was sufficiently different from the control regime to influence outcomes. Elderly orthopaedic patients who were supplied with large celled alternating pressure mattresses on their beds and in whom chairnursing in the early postoperative period was restricted as far as possible to <2 hours per session, rarely developed pressure sores compared with similar patients who sat out for longer periods. They also suffered from fewer chest infections, were less constipated and appeared less fatigued and more active and motivated. They appeared to mobilise earlier than patients subjected to prolonged chair nursing, although the difference only approached significance.

However, the population studied was small, primarily because the incidence of pressure sores in the control group made continuation of the study unethical. Therefore, the results for complications other than pressure sores are of borderline significance and/or have wide confidence intervals. For this reason, a larger, more detailed study was indicated to explore in more detail, whether a policy of limiting chairnursing would have a beneficial effect on these other outcomes and on patient fatigue levels.

Having established the practical feasibility of this approach, the main study was begun 2 years later in a large teaching hospital. The aims and objectives, methodology, results and discussion of this study are the subjects of the remaining chapters of the thesis.
4 AIMS AND OBJECTIVES

4.1 AIMS

The aims of this study were:

i) to determine if limiting chairnursing is related to a reduction in the incidence of post-operative complications
ii) to determine if limiting chairnursing is related to a reduction in immobility and hospital stay
iii) to determine if limiting chairnursing is related to differences in observed pulse rate, blood pressure, lower leg oedema and self-assessed fatigue

4.2 OBJECTIVES

4.2.1 Part 1.
To compare the incidence of severe fatigue, tachycardia, hypotension, leg oedema, chest infection, pressure sores, constipation, urinary tract infection and deep vein thrombosis between a limited and unlimited chairnursing group.

4.2.2 Part 2
To compare the time from surgery to independent mobility and discharge from hospital between a limited and unlimited chairnursing group.

4.2.3 Part 3
To observe mean pulse rate, blood pressure, lower leg oedema and self-assessed fatigue after different lengths of chairnursing on different days post-surgically and at different times during the day in a limited and unlimited chairnursing group.
4.3 NULL HYPOTHESES TO BE TESTED

The following null hypotheses were tested:

4.3.1 Part 1
1. There will be no difference in incidence of severe fatigue between the limited and unlimited chairnursing groups.
2. There will be no difference in incidence of hypotension between the limited and unlimited chairnursing groups.
3. There will be no difference in incidence of tachycardia between the limited and unlimited chairnursing groups.
4. There will be no difference in incidence of leg oedema between the limited and unlimited chairnursing groups.
5. There will be no difference in incidence of deep vein thrombosis between the limited and unlimited chairnursing groups.
6. There will be no difference in incidence of chest infection between the limited and unlimited chairnursing groups.
7. There will no difference in incidence of pressure sores between the limited and unlimited chairnursing groups.
8. There will be no difference in incidence of constipation between the limited and unlimited chairnursing groups.
9. There will be no difference in incidence of urinary tract infection between the limited and unlimited chairnursing groups.

4.3.2 Part 2
1. There will be no difference in the time taken to achieve independent mobility between the limited and unlimited chairnursing groups.
2. There will be no difference in length of post-operative hospital stay between the limited and unlimited chairnursing groups.

4.3.3 Part 3
1. There will be no change in self-assessed fatigue associated with different days post-surgically and different times of day in the limited and unlimited chairnursing groups.
2. There will be no change in pulse rate associated with different days post-surgically and different times of day in the limited and unlimited chairnursing groups.
3. There will be no change in blood pressure associated with different days post-surgically and different times of day in the limited and unlimited chairnursing groups.
4. There will be no change in lower leg volume associated with different days post-surgically and different times of day in the limited and unlimited chairnursing groups.
5 STUDY DESIGN AND METHODOLOGY

5.1 METHOD

This is a study of experimental design and compares the effect of a new mobilisation policy in one group of patients (the experimental group) against the effect of the existing policy in another (the control group). The study was carried out on two orthopaedic wards. The experimental group consisted of patients admitted to one ward and the control group of patients admitted to the other.

The wards were identical in design and shared the same consultants and the same senior nurse. There were no known differences in either medical or nursing policy regarding the general care of the patients. However neither the senior nurse nor the consultants were involved in the prescription of rehabilitation programmes for the patients, which was left primarily to the ward nursing staff and to the physiotherapists who were assigned to each ward. There could, therefore be effective separation of policy regarding mobilisation regimes. Patients scheduled for lower limb surgery which was either planned or required following a recent fracture were admitted depending on the availability of beds, to either ward. If beds were available on both wards, patients were admitted one per ward in strict rotation. Differences in quality of care and personal preferences of staff for different ways of managing patients were controlled for by a break half-way through the study (washout period) followed by a crossing over of the experimental and control regimes between wards. The experimental ward became the control ward and vice versa. The purpose of the washout period was to reduce the influence of the policy used in the first part of the study on staff behaviour during the second part.

On the experimental ward (which will be referred to as the limited chairnursing group, LCG), subjects had chair nursing restricted to two hours per session until they became independently mobile. Independent mobility was defined as the ability to stand up from the chair and move onto the bed independently of staff (with Zimmer frame or walking stick(s) if necessary). There was no restriction on the number of sessions per day provided that there were at least two hours of bedrest or walking between sessions. Once subjects were mobile, all restrictions on chairnursing were lifted.

Table 5.1 Cushions used in the study

<table>
<thead>
<tr>
<th>Cushion</th>
<th>Manufacturer</th>
</tr>
</thead>
<tbody>
<tr>
<td>ROHO High Profile</td>
<td>ROHO Inc (Belleville)</td>
</tr>
<tr>
<td>Bed pillow</td>
<td>Various</td>
</tr>
<tr>
<td>Propad</td>
<td>Medical Support Systems (Cardiff)</td>
</tr>
<tr>
<td>Bodipillo Aquaflow</td>
<td>First Technicare (London)</td>
</tr>
<tr>
<td>Flowtech Plus</td>
<td>Park House Ltd (Batley)</td>
</tr>
<tr>
<td>Qbitus</td>
<td>Qbitus Ltd (Halifax)</td>
</tr>
</tbody>
</table>

Subjects on the control ward (unlimited chairnursing group, UCG) were nursed according to the normal routine of that ward with the amount of time they spent in chairs decided by themselves and/or ward staff. The results of the preliminary study suggested that lengthy chairnursing was particularly hazardous to the pressure areas of this patient group. The risks, being known, it was felt to be unethical to simply permit the development of sores. To attempt to protect their pressure areas, patients on the control ward were issued with one of a range of cushions (see table 5.1) for their chairs. These cushions are thought to reduce the risk of pressure sores by spreading the weight of the patient more evenly and thus reducing point pressures and therefore tissue deformation, over the ischial tuberosities (Medical Devices Agency 1997). Efforts were made to provide patients with cushions which they preferred and every effort was made to provide a chair that was of the appropriate height and shape (Collins 1998), taking the height of any cushion used into account. Correct height of the chair is important as chairs which are either too high or too low increase loading.
on the thighs and buttocks respectively. Having taken these precautions, if fewer sores developed in the LCG compared to the UCG, this would give particularly strong evidence of the efficacy of limiting chairnursing.

5.2 ETHICAL ISSUES

Ethical approval was obtained from the Nursing Research Ethics Committee. Patients were asked to sign a written consent if they agreed to participate in the trial (see appendix 3).

In accordance with the guidelines for Application for Ethical Approval, patients will not be identified by name or case notes' number on the computerised data base, or in any publication. Data collection forms will be stored in a locked cabinet in a locked room. In accordance with the recommendations of the Royal College of Physicians, the data will be retained and be available for inspection for 10 years following the completion of the study.

5.3 SUBJECTS

Consenting patients who satisfied the inclusion and exclusion criteria (table 5.2) were admitted to the trial either the day before or on the day of surgery. They were provided with either a pressure reducing foam or a large-celled alternating pressure air mattress on admission to the ward (fracture) or on day of operation (planned surgery) as part of normal hospital policy. As discussed previously (section 3.5.7) there is no evidence of differential benefits of these two mattress types in the study population.

Table 5.2 Inclusion and exclusion criteria

<table>
<thead>
<tr>
<th>Inclusion criteria:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aged ≥64 years last birthday</td>
</tr>
<tr>
<td>Admitted to the experimental or control ward</td>
</tr>
<tr>
<td>Admitted for arthroplasty of hips or legs</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Exclusion criteria:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dementia (mental test score &lt;8)(Quareshi &amp; Hodgkinson 1974)</td>
</tr>
<tr>
<td>Diagnosis of cancer</td>
</tr>
</tbody>
</table>

The primary aim of the study was to detect the difference between the experimental and control groups in the incidence of a number of outcomes of interest which will be collectively referred to as 'negative outcomes'. There were little or no data on the likely incidence of these outcomes in chairnursed and non-chairnursed populations in the literature. Therefore data obtained from the preliminary study were used as a basis for estimating the sample size required to detect statistically significant differences in the incidence of negative outcomes.

In the preliminary study (chapter 3), the differences in incidence of 4 complications (pressure sores, chest infection, constipation and ankle oedema) were statistically significant or approached significance. To ensure a high probability of detecting differences in the incidence of all complications, the smallest difference (chest infection, 0% and 11% incidence in LCG and UCG respectively) was used to estimate the sample size. Because multiple outcomes were to be assessed, a higher level of significance (1%) than the usual 5% was aimed at, to reduce the probability of 'fishing error' - that is the probability of a difference being detected where none exists (Type I error)
through chance. A Bonferroni adjustment which is often used in similar circumstances to limit the probability of this error (Grove & Andreasen 1982) could not be applied as the outcomes are dependent, thus violating a basic assumption of the adjustment. In any case, the usefulness of Bonferroni adjustments is questioned in the context of assessing evidence about 'a priori' hypotheses and increases chances of Type II error (Perneger 1999).

A power calculation (85% power, 1% significance level) using ARCUS Quick Stat (Addison Wesley Longman Ltd, Cambridge) based on an 11% difference, suggested 128 subjects needed to be admitted into each group. Allowing for 10% dropout, this was increased to a total of 141 patients in each group. Surgical records from 1992-93 and 93-94 showed that 10 months would be necessary to achieve these numbers. This allowed five months of data collection followed by a crossover of the experimental and control wards, a months 'washout' period with no data collection and another 5 months of data collection (11 months for data collection in total). However this time frame had to be adjusted later due to changes occurring within the study site (see 5.8 below).

5.4 DATA RECORDING

Baseline data were recorded pre-operatively, on admission to the study with the following additions. A post-operative baseline lower leg volume was recorded on the 3rd post-operative day, 9-10am and before the subject got out of bed. Postoperative haemoglobin level was recorded when results of analysis of the first post-operative blood sample, taken 1-3 days post-operatively, were received. Otherwise, data were grouped in three parts. Parts 1 and 2 were concerned with comparing the incidence of negative outcomes and postoperative mobilisation and discharge respectively between LCG and UCG. Only one measurement per outcome per subject was analysed in parts 1 and 2. Part 3 was concerned with the effect group (LCG or UCG) had upon pulse rate, blood pressure, lower leg volume and self assessed fatigue. Multiple measurements (assessments) were taken from each subject for this part. Data for each patient was recorded on a data collection proforma (appendix 5).

5.4.1 Baseline data
This consisted of subject number, experimental group and the following:

Demographic variables - age in years at last birthday, sex, body build category (based on body mass index obtained from comparison of subject weight and height)(Bray 1978), below-knee volume of the operated and un-operated legs in cubic centimetres (on admission and on day 3 post-operatively);

Health characteristics - diagnosis of any pathalogy or recent surgery recorded in the medical and/or nursing documentation, smoking history, pre-operative and post-operative haemoglobin level in g/dl, Norton score (Norton et al 1962), systolic and diastolic blood pressure in mmHg on admission, pulse rate on admission (beats per minute), self assessed fatigue on visual analogue scale (VAS) on admission, the level of mobility prior to admission to surgery or fracture (emergency surgery) as reported by the patient (full mobility [able to carry out shopping], restricted mobility [needing assistance with shopping], housebound, chair/bed bound);

Treatment variables - type of surgery (elective or emergency), type of operation, drugs taken at any time during the course of the study, which have fatigue, tachy- and bradycardia, hypo and hypertension, constipation, diarrhoea and reduction of resistance to infection listed as potential side-effects (Joint Formulary Committee 1998).

5.4.2 Part 1: Incidence of negative outcomes
The subjects' medical and nursing notes and the subjects themselves were assessed on days 3, 5, 7, 10 and 14 following surgery, counting day of surgery as day 1. The following negative outcomes (which are defined in section 5.6) were recorded if present: tachycardia, hypotension, lower leg
oedema, severe self perceived fatigue, chest infection, pressure sores, constipation and urinary tract infection (UTI). It was not possible to assess the incidence of DVT as funding was not made available to carry out the necessary laboratory analysis of blood samples. Negative outcomes were recorded as either present or absent in the 14 day post-operative period and only counted once per subject. Only new development of a negative outcome during the study was counted. Data from all patients were included on an intention-to-treat basis.

5.4.3 Part 2: Independent mobility and discharge
The post-operative day the patient first became mobile (defined in 5.1) and the day of discharge following surgery were recorded, counting day of surgery as day 1. Also recorded, were the circumstances of discharge ie discharge home independently; with the early discharge team; to a nursing/residential home; to another acute hospital ward or to long-term hospital care. Subjects who dropped out of the trial due to major medical or surgical complications were excluded from the analysis of this part of the study.

5.4.4 Part 3: Pulse, blood pressure, leg oedema and fatigue
With the day of surgery being counted as day one, pulse rate, blood pressure, lower leg volume and self reported fatigue were assessed on days 3, 5, 7, 10 and 14 post-operatively. Four assessments were made on each day at 9-10am and 12-1, 3-4 and 6-7pm. The time spent in a chair prior to the assessment being made was recorded as none (patient in bed), up to 2 hours, 2-4 hours, 4-6 hours, 6-8 hours or more than 8 hours. A record was also kept of any medication the patient had taken within the past 24 hours of 9-10am on the day of assessment. Data from all subjects were included on an intention-to-treat basis.

5.5 MEASUREMENT METHODS

5.5.1 Self-assessed fatigue (SAF)
Fatigue was self-assessed using the VAS after Christensen et al (1982). Patients were asked to indicate how fatigued they felt at that moment in time by placing a horizontal line through a vertical visual analogue scale (appendix 4).

5.5.2 Pulse rate
The radial pulse was palpated for two minutes and the mean rate/minute recorded. Pulse rate was not recorded within one hour of meals to avoid recording during potential postprandial hypotension (Jansen et al 1995).

5.5.3 Systolic and diastolic blood pressure
Blood pressure was measured using the method recommended by the British Hypertension Society (1990) with a desk top mercurial sphygmomanometer with plastic column (Hospital Model 0010, Accosan, UK) and a single head stethoscope (Model 110, Kenzmedico Co Ltd, Japan). The patient’s left arm was supported horizontally at the level of the mid-sternum. Tight clothing was removed and an appropriately sized cuff (bladder length at least 80% and width at least 40% of the arm circumference) applied just above the anti-decubital fossa. The manometer was positioned with the column of mercury vertical. The brachial artery was felt with the fingers and the cuff was inflated until the pulse disappeared. The diaphragm of the of the stethoscope was then placed over the brachial artery and the cuff inflated a further 30mm. The cuff was then deflated at a rate of 2-3mmHg per second. The appearance of the first sound was recorded as the systolic blood pressure and the complete disappearance of sound was recorded as the diastolic blood pressure. The pressures were
recorded to the nearest 2mmHg. Where possible, blood pressure was not recorded if the patients were cold, had a full bladder or within 1 hour of having a meal or cigarette as these events can have a significant confounding influence on the results (Kendrick & Luker 1995). The manometer was zeroed at the beginning of each day and underwent routine maintenance every six months.

5.5.4 Lower leg volume
The circumference of the leg was measured with a tape at the narrowest part of the leg in the gaiter area (c) and at the thickest part of the calf (C). The exact sites were marked on the first assessment to ensure measurements were taken in the same place. The volume (V) of the lower leg was calculated according to the formula for the frustum of a cone:

\[ V = \frac{h}{12n} \left( C^2 + c^2 + Cc \right) \]

where h is the distance between C and c.

5.6 DEFINITION OF NEGATIVE OUTCOMES

The following negative outcomes (signs, symptoms and complications) were recorded as present if any of the criteria below were met. An outcome was considered 'new' if it was preceded by an assessment during which the outcome was absent.

5.6.1 Severe self perceived fatigue
A score of 7 or more arbitrary units on the fatigue VAS after Christensen et al (1982) was arbitrarily defined as severe fatigue. This coincides with the highest group on the scale (Tired - Fatigued) as designated by Christensen et al (1982) and other users of the scale (eg Troidl et al 1992).

5.6.2 Tachycardia
Tachycardia was arbitrarily defined as a pulse rate > 100 beats/minute in line with existing medical definitions (Spraycar et al 1995, Anderson 1994, Cowell 1989).

5.6.3 Hypotension
Hypotension was arbitrarily defined as a systolic blood pressure of < 100 mmHg, in line with existing definitions (Booker 1996).

5.6.4 Lower leg oedema
Baseline volume was measured after a night's bedrest on the 3rd postoperative day, between 9-10am prior to the subject getting out of bed. Lower limbs were defined as oedematous if any recorded lower leg volume exceeded the baseline volume by 20%.

5.6.5 Chest infection
A diagnosis of chest infection was recorded if the subject experienced new or increased production of purulent sputum and/or fever of > 38°C with appropriate chest signs and/or new progressive x-ray evidence of chest infiltrates not attributable to embolus or heart failure (Steering Group of the National Prevalence Study of Hospital Acquired Infections1993). Appropriate chest signs were defined...
as: wheezing, crepitations, breathlessness, reduced chest movement, bronchial breathing and pleural rub (Macfarlane 1996).

5.6.6 Pressure sores
Skin breaks > .5cm on (excluding any on the back of the heels, which are unlikely to be caused by sitting) were counted as pressure sores.

5.6.7 Constipation
No bowel movement for three days and/or the prescription of laxatives as a treatment for constipation (as opposed to prevention) was counted as constipation.

5.6.8 Urinary tract infection (UTI)
The presence of UTI was counted if the subject had a mid-stream specimen (or catheter specimen) of urine with a bacterial count of $> 10^5$ organisms per ml accompanied by one or more of the following: dysuria, urgency, loin pain, suprapubic tenderness, pyrexia ($> 38^\circ$C) or pyuria (Steering Group of the National Prevalence Survey of Hospital Acquired Infections 1993).

5.7 ANALYSIS AND STATISTICAL METHODS
The results were analysed using the STATA 5.0 (Stata Corporation, Texas) and MLwin (Multilevel Models Project, London) software packages.

5.7.1 Baseline data
The comparability of the experimental and control groups was assessed. Demographic, health characteristics and treatment variables were compared using: chi square test for categorical variables; t-tests for normally distributed data and the Mann Whitney test for non-normally distributed continuous data. The numbers of patients whose haemoglobin level, lower leg volume, blood pressure and pulse rate fell below, into and above normal adult values were also compared. Normal values are given in table 5.3.

Table 5.3 Normal values for the whole population

<table>
<thead>
<tr>
<th>Variable</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin level (Mourad 1991)</td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>14.4-16.6 g/dl</td>
</tr>
<tr>
<td>Women</td>
<td>12.7-14.7 g/dl</td>
</tr>
<tr>
<td>Lower leg volume (based on Diffrient et al 1974)</td>
<td></td>
</tr>
<tr>
<td>Men*</td>
<td>1411-2328 cc</td>
</tr>
<tr>
<td>Women*</td>
<td>1220-2067 cc</td>
</tr>
<tr>
<td>Systolic blood pressure (Egan et al 1998)</td>
<td>100-140 mmHg</td>
</tr>
<tr>
<td>Diastolic blood pressure (Egan et al 1998)</td>
<td>60-90 mmHg</td>
</tr>
<tr>
<td>Pulse rate (Ferguson et al 1999)</td>
<td>60-80</td>
</tr>
</tbody>
</table>

*These ranges should be taken as an approximation, based on the US population. The calculation is based on 2.5 percentile height and calf circumferences for the lower end of the range and 97.5 percentile height and calf circumferences at the highest using the formula in 5.4.3. It was assumed that h was proportional to overall height and that calf circumference was linearly associated with height.
5.7.2 Length of time sat in chairs by mobile and immobile patients
The incidence of observations after bedrest and 0-2, 2-4, 4-6, 6-8 and > 8 hours of chairnursing was compared between mobile and immobile patients (see 6.2 for definition of 'mobile') using z-test and chi^2 test, in LCG and LCG separately and in combination.

5.7.3 Part 1: Incidence of negative outcomes
The effect, if any, of group (LCG and UCG) was compared univariately using the z-test for the comparison of two proportions. The effect of group and the explanatory variables listed in 5.3.1 on the incidence of negative outcomes (listed in 5.3.2) was then compared using univariate logistic regression models. Variables significantly associated with each negative outcome were entered into a multivariate logistic regression model to determine which factors were independently predictive of development of negative outcomes. Here the effect an explanatory variable had on the outcome in addition to the effect of another variable was sought. Terms were added to the model sequentially until the additional term made no significant improvement to the model. The order of the adding terms was decided by the strength of the explanatory variable's univariate association with the outcome.

An issue to take into account with this study was the large number of significance tests being performed. There are 9 outcomes which are not independent and approximately 40 explanatory variables. In the absence of using a Bonferroni correction (only applicable when the outcomes are independent) it was decided to test at a significance level of p = 0.01 and use 99% confidence intervals to make some adjustment for multiple testing.

Another issue to take into account was the reduced probability of detecting a new occurrence of a negative outcome in a subject who had the outcome on admission. To examine this potential effect, the data was re-analysed with those subjects excluded from the analysis.

5.7.4 Part 2: Independent mobility and discharge
The effect, if any, of group (LCG and UCG), the variables listed in 5.4.1 and destination after discharge on time to independent mobility (defined in 5.1) and discharge was compared univariately using the t-test. Variables significantly associated with time to independent mobility and to discharge were entered into a multivariate regression model to determine which factors were significant after adjusting for all variables.

5.7.5 Part 3: Pulse, blood pressure, leg oedema and fatigue
These outcomes were repeated measures at 4 time points on 5 days after operation. A complete dataset, therefore, consisted of 20 assessments per patient. Any statistical analysis had to take into account that the measurements taken from the same subject are likely to be more similar than measurements from different subjects. The data were analysed by two methods: summary statistics and multi-level models.

5.7.5.1 Summary statistics
A mean pulse rate, systolic and diastolic blood pressure, lower leg volume and self-assessed fatigue overall for each subject independent of day of assessment was calculated. This was compared univariately with the patient characteristics listed in table 5.4. Analysis of variance was used for the association with the categorical variables: group (LCG and UCG), surgery (elective or emergency), type of operation, build, mobility prior to surgery/fracture. Linear regression was used in order to assess the relationship between outcomes and continuous variables (baselines, Norton score, operated and unoperated limb volume, SAF on admission). Univariately significant variables were entered into a multivariate regression model in order to identify those which remained significant whilst adjusting for confounding variables.

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Table 5.4 Patient characteristics

<table>
<thead>
<tr>
<th>Patient characteristic</th>
<th>Variable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group (LCG or UCG)</td>
<td>Pulse rate</td>
</tr>
<tr>
<td>Sex</td>
<td>Blood pressure</td>
</tr>
<tr>
<td>Age</td>
<td>Lower leg vol</td>
</tr>
<tr>
<td>Type of surgery (elective or emergency)</td>
<td>Fatigue</td>
</tr>
<tr>
<td>Operation</td>
<td></td>
</tr>
<tr>
<td>Haemoglobin level before and after surgery</td>
<td></td>
</tr>
<tr>
<td>Norton score on admission</td>
<td></td>
</tr>
<tr>
<td>Pre-operative mobility</td>
<td></td>
</tr>
<tr>
<td>Build</td>
<td></td>
</tr>
<tr>
<td>Taking of drugs which may cause tachy-</td>
<td>Pulse rate</td>
</tr>
<tr>
<td>or bradycardia (at any time)</td>
<td></td>
</tr>
<tr>
<td>Taking of drugs which may cause hypo-</td>
<td>Blood pressure</td>
</tr>
<tr>
<td>or hypertension (at any time)</td>
<td></td>
</tr>
<tr>
<td>Taking of drugs which may cause oedema</td>
<td>Lower leg vol</td>
</tr>
<tr>
<td>(at any time)</td>
<td></td>
</tr>
<tr>
<td>Taking of drugs which may cause fatigue</td>
<td>Fatigue</td>
</tr>
<tr>
<td>(at any time)</td>
<td></td>
</tr>
<tr>
<td>Respiratory/circulatory conditions</td>
<td>Pulse rate</td>
</tr>
<tr>
<td>Smoking history</td>
<td>Blood pressure</td>
</tr>
<tr>
<td>Heart failure</td>
<td>Lower leg vol</td>
</tr>
<tr>
<td>Recent coronary artery bypass surgery</td>
<td></td>
</tr>
<tr>
<td>Conditions which may cause fatigue</td>
<td>Fatigue</td>
</tr>
</tbody>
</table>

Findings were confirmed by repeating calculations using mean variable over mean rate per day for each subject.

5.7.5.2 Multilevel models
Multilevel models can be used to analyse hierarchies of data and incomplete data sets. In this case an assumption was made that correlations exist between measures taken on the same day and measures taken from the same patient. A three level model was used with patients taken as level 3, post-surgical day at level 2 and measurement at each time point as level 1. In all the models effects of days and time points were fitted and also the group effect (LCG or UCG) at each time point. Associations with baseline variables were also investigated.

5.7.5.3 Pulse rate, blood pressure, lower leg volume and fatigue after various lengths of chairnursing
The difference between each observation and the pre-operative baseline for the subject was calculated. Depending on the distribution of differences, a mean or median value was calculated for each of the above variables, separately for each chairnursing timeband (bedrest, 0-2 hours, 2-4 hours, 4-6 hours, 6-8 hours, 8 or more hours). Initially these were calculated separately for LCG and UCG. If there were no significant differences between LCG and UCG (suggesting there was no group effect), the data from the two groups were combined to observe if different lengths of chairnursing were associated with different mean changes in these variables.

5.8 SAMPLE SIZE ADJUSTMENT
Shortly after commencement of the study, the health authority which purchased services from the acute hospital trust in which the study was taking place, went into severe financial difficulties. One result was a reduction in funding for elective orthopaedic surgery. This reduced the throughput of suitable candidates (to about 10-15 per month) and thus the rate of recruitment to the trial. To complete the study within a reasonable time, a decision was made to reduce the sample size to 100 subjects in each group. The effect of this reduction was to reduce the power of the study to 72%.

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6 RESULTS

6.1 SUBJECTS

Due to the reduction in numbers of suitable subjects on the research site described in part 5.8, data collection took longer than originally expected: 12 months for each group, 26 months in total including 2 month’s washout period at cross-over (the washout period was extended in an attempt to provide more time for the influence of the policy to be reduced after the extended time there had been for the policy to be absorbed by the staff during the first part of the study).

203 patients were recruited to the trial, 51 to the limited chairnursing group (LCG) and 55 to the unlimited chairnursing group (UCG) before crossover, 49 and 48 respectively after crossover giving totals of 100 to the LCG and 103 to the UCG. Of this total one subject in the UCG developed serious complications during surgery and was unable to take any further part in the study. A further 2 subjects in LCG and 4 in UCG dropped out of the trial after undergoing at least the day 3 assessment due to either dislocation of prosthesis (2 subjects) or medical complications - haemorrhaging gastric ulcer (1 subject), acute dementia (1 subject) and cardiac events (2 subjects). Thus data were available for 100 LCG and 103 UCG subjects for parts 1 and 3 of the study (on an intention to treat basis) and 98 subjects for both groups for part 2 of the study.

Main demographic and treatment variables and health characteristics are given in tables 6.1, 6.2 and 6.3 respectively. Diagnoses on admission are given in table 6.4. The patients’ characteristics were compared between the two regimens using t-tests for the comparison of the continuous variables (haemoglobin level pre- and post-operatively; age, operated and unoperated lower leg volume, systolic and diastolic blood pressure and pulse rate on admission) and chi square tests for the categorical ones (sex, build, smoking history, mobility prior to surgery/fracture, type of operation, diagnoses on admission; the taking of drugs with potential side-effects affecting the outcomes of interest at any time during the study). Because of the small frequencies at some of the cells, appropriate collapses between categories with ‘similar’ meaning were carried out (hemiarthroplasty with revision of total hip replacement and with revision of total knee replacement; emaciated with underweight; housebound with chairbound). Norton score and SAF on admission were compared using both tests, i.e. they were treated as continuous and as categorical.

The results showed close agreement between the two regimens with regard to the above characteristics. This re-assures that any difference found between the two groups regarding the outcomes of interest could not be attributed to baseline imbalances.
Table 6.1 Main demographic variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>LCG (SD)</th>
<th>UCG (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (yrs at last birthday)[SD]</td>
<td>75.1 (6.8)</td>
<td>74.5 (7.5)</td>
</tr>
<tr>
<td>Sex (% male/female)</td>
<td>38/62</td>
<td>35/65</td>
</tr>
<tr>
<td>Build (% number of subjects)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- emaciated</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>- underweight</td>
<td>9</td>
<td>14</td>
</tr>
<tr>
<td>- normal</td>
<td>41</td>
<td>43</td>
</tr>
<tr>
<td>- overweight</td>
<td>36</td>
<td>30</td>
</tr>
<tr>
<td>- obese</td>
<td>10</td>
<td>12</td>
</tr>
<tr>
<td>Mean lower operative leg volume on admission(cc).</td>
<td>1407(329)</td>
<td>1356(387)</td>
</tr>
<tr>
<td>% missing data [2]</td>
<td>16</td>
<td>15</td>
</tr>
<tr>
<td>% subjects volume &lt;95% range</td>
<td>32</td>
<td>38</td>
</tr>
<tr>
<td>% subjects volume within 95% range</td>
<td>48</td>
<td>43</td>
</tr>
<tr>
<td>% subjects volume &gt;95% range</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Mean lower unoperative leg volume on admission(cc).</td>
<td>1404(313)</td>
<td>1378(407)</td>
</tr>
<tr>
<td>% subjects volume &lt;95% range</td>
<td>40</td>
<td>44</td>
</tr>
<tr>
<td>% subjects volume within 95% range</td>
<td>49</td>
<td>45</td>
</tr>
<tr>
<td>% subjects volume &gt;95% range</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

[1] Unable to weigh subject
Insufficient time (2)
[3] Patient refused (9) Insufficient time (2) Various (7)

Table 6.2 Main treatment variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>LCG</th>
<th>UCG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surgery (% number of subjects):</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- elective</td>
<td>78</td>
<td>67</td>
</tr>
<tr>
<td>- emergency</td>
<td>22</td>
<td>33</td>
</tr>
<tr>
<td>Operation (% number of subjects):</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Total hip replacement (THR)</td>
<td>50</td>
<td>44</td>
</tr>
<tr>
<td>- Dynamic hip screw</td>
<td>14</td>
<td>20</td>
</tr>
<tr>
<td>- Total knee replacement (TKR)</td>
<td>20</td>
<td>18</td>
</tr>
<tr>
<td>- Bilateral TKR</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>- Hemiarthroplasty</td>
<td>8</td>
<td>14</td>
</tr>
<tr>
<td>- Revision THR</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>- Revision TKR</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Drugs taken at any time during study with the following potential side-effects (% number of patients):</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Fatigue</td>
<td>45</td>
<td>41</td>
</tr>
<tr>
<td>- Tachycardia</td>
<td>60</td>
<td>56</td>
</tr>
<tr>
<td>- Bradycardia</td>
<td>84</td>
<td>85</td>
</tr>
<tr>
<td>- Hypotension</td>
<td>85</td>
<td>85</td>
</tr>
<tr>
<td>- Hypertension</td>
<td>13</td>
<td>11</td>
</tr>
<tr>
<td>- Reduced mobility</td>
<td>95</td>
<td>94</td>
</tr>
<tr>
<td>- Constipation</td>
<td>94</td>
<td>95</td>
</tr>
<tr>
<td>- Diarrhoea</td>
<td>86</td>
<td>87</td>
</tr>
<tr>
<td>- Oedema</td>
<td>72</td>
<td>69</td>
</tr>
<tr>
<td>- Reduced resistance to infection</td>
<td>6</td>
<td>4</td>
</tr>
</tbody>
</table>
Table 6.3 Main health characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>LCG (SD)</th>
<th>UCG (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking (% number of subjects) One observation missing [4]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never smoked</td>
<td>32</td>
<td>34</td>
</tr>
<tr>
<td>Stopped smoking &gt;3 months</td>
<td>44</td>
<td>36</td>
</tr>
<tr>
<td>Stopped smoking &lt;3 months</td>
<td>12</td>
<td>11</td>
</tr>
<tr>
<td>Currently smoking</td>
<td>11</td>
<td>19</td>
</tr>
<tr>
<td>Mean pre-operative Hb (gm/l) 6 observations missing [5]</td>
<td>12.7(1.6)</td>
<td>12.2(2.0)</td>
</tr>
<tr>
<td>Mean post-operative Hb (gm/l) 10 observations missing [6]</td>
<td>10.8(1.4)</td>
<td>10.5(1.4)</td>
</tr>
<tr>
<td>Pre-operatively:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Missing data</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>% subjects Hb &lt;normal range</td>
<td>60</td>
<td>71</td>
</tr>
<tr>
<td>% subjects Hb within normal range</td>
<td>31</td>
<td>24</td>
</tr>
<tr>
<td>% subjects Hb &gt;normal range</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Median total Norton score (range)</td>
<td>20 (10-20)</td>
<td>19 (11-20)</td>
</tr>
<tr>
<td>Mean systolic blood pressure (mmHg)</td>
<td>133.6(16.3)</td>
<td>133.4(17.0)</td>
</tr>
<tr>
<td>Mean diastolic blood pressure (mmHg)</td>
<td>74.0(11.1)</td>
<td>74.1(11.3)</td>
</tr>
<tr>
<td>% subjects SBP &lt;normal range</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>% subjects SBP within normal range</td>
<td>70</td>
<td>69</td>
</tr>
<tr>
<td>% subjects SBP &gt;normal range</td>
<td>27</td>
<td>28</td>
</tr>
<tr>
<td>% subjects DBP &lt;normal range</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>% subjects DBP within normal range</td>
<td>85</td>
<td>87</td>
</tr>
<tr>
<td>% subjects DBP &gt;normal range</td>
<td>9</td>
<td>7</td>
</tr>
<tr>
<td>Mean pulse rate (beats/minute) (on admission)</td>
<td>80.9(13.3)</td>
<td>80.9(13.3)</td>
</tr>
<tr>
<td>% subjects PR &lt;normal range</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>% subjects PR within normal range</td>
<td>54</td>
<td>48</td>
</tr>
<tr>
<td>% subjects PR &gt;normal range</td>
<td>45</td>
<td>48</td>
</tr>
<tr>
<td>Mean fatigue score (VAS) (on admission)</td>
<td>4.1</td>
<td>4.0</td>
</tr>
<tr>
<td>Missing data [7]</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>% subjects score 1-3</td>
<td>32</td>
<td>37</td>
</tr>
<tr>
<td>% subjects score 4-6</td>
<td>56</td>
<td>58</td>
</tr>
<tr>
<td>% subjects score 7-9</td>
<td>9</td>
<td>4</td>
</tr>
<tr>
<td>Mobility prior to surgery/fracture (% number of subjects)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Full</td>
<td>70</td>
<td>60</td>
</tr>
<tr>
<td>Limited</td>
<td>28</td>
<td>40</td>
</tr>
<tr>
<td>House bound</td>
<td>2</td>
<td>-</td>
</tr>
<tr>
<td>Bed/chair bound</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

[4] Subject refused  
[5] No laboratory result available  
[6] No laboratory result available  
[7] Patient unable to complete (3) Missing data (1)
Table 6.4 Diagnoses/recent surgery

<table>
<thead>
<tr>
<th>Variable</th>
<th>LCG</th>
<th>UCG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osteo-arthritis</td>
<td>F</td>
<td>84</td>
</tr>
<tr>
<td>Cataracts</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Glaucoma</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Hypertension</td>
<td>P</td>
<td>30</td>
</tr>
<tr>
<td>Osteoporosis</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>F</td>
<td>3</td>
</tr>
<tr>
<td>Angina</td>
<td>F</td>
<td>9</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>P</td>
<td>2</td>
</tr>
<tr>
<td>Heart failure</td>
<td>PLF</td>
<td>9</td>
</tr>
<tr>
<td>Benign prostatic hyperplasia</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Alcohol abuse</td>
<td>F</td>
<td>2</td>
</tr>
<tr>
<td>Non-insulin dependent diabetes</td>
<td>F</td>
<td>4</td>
</tr>
<tr>
<td>Recent CABG</td>
<td>PLF</td>
<td>3</td>
</tr>
<tr>
<td>Urinary tract infection</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Constipation</td>
<td>-</td>
<td>4</td>
</tr>
<tr>
<td>Parkinson’s disease</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>Ulcerative colitis</td>
<td>F</td>
<td>1</td>
</tr>
<tr>
<td>Phlebitis of the leg</td>
<td>3</td>
<td>-</td>
</tr>
<tr>
<td>Reflux oesophagitis</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>Asthma</td>
<td>P</td>
<td>6</td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>F</td>
<td>3</td>
</tr>
<tr>
<td>Hypothyroidal alcohol</td>
<td>F</td>
<td>3</td>
</tr>
<tr>
<td>Hemiplegia</td>
<td>-</td>
<td>4</td>
</tr>
<tr>
<td>Meniere’s disease</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Galistones</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Emphysema</td>
<td>P</td>
<td>2</td>
</tr>
<tr>
<td>Clinical depression</td>
<td>F</td>
<td>4</td>
</tr>
<tr>
<td>Von Willebrandt’s disease</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Chest infection</td>
<td>P</td>
<td>2</td>
</tr>
<tr>
<td>Venous leg ulcer</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>COAD</td>
<td>F</td>
<td>1</td>
</tr>
<tr>
<td>Polymyalgia rheumatica</td>
<td>F</td>
<td>1</td>
</tr>
<tr>
<td>Hiatus hernia</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Sciatica</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Anterior sphincter detail</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Anaemia</td>
<td>PLF</td>
<td>2</td>
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<tr>
<td>Psoriatic arthropathy</td>
<td>F</td>
<td>1</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>PLF</td>
<td>1</td>
</tr>
<tr>
<td>Pansystoly</td>
<td>F</td>
<td>1</td>
</tr>
<tr>
<td>Alzheimer’s disease</td>
<td>-</td>
<td>2</td>
</tr>
<tr>
<td>diverticulitis</td>
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<td>4</td>
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<tr>
<td>Bronchitis</td>
<td>P</td>
<td>1</td>
</tr>
<tr>
<td>Insulin dependent diabetes</td>
<td>F</td>
<td>2</td>
</tr>
<tr>
<td>Back pain</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Eczema</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Transient ischaemic attack</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>Renal failure</td>
<td>LF</td>
<td>-</td>
</tr>
<tr>
<td>Hypothyroidal alcohol</td>
<td>1</td>
<td>-</td>
</tr>
</tbody>
</table>

Conditions which may affect the outcomes of interest are indicated by the following symbols:  
P = pulse or blood pressure  
L = leg volume  
F = fatigue

COAD = chronic obstructive airways disease  
Recent CABG = coronary artery bypass graft within 6 months
6.2 CHAIRNURSING TIME IN MOBILE AND IMMOBILE SUBJECTS

The distribution of bedrest and lengths of time sat in a chair at time of assessment for mobile and immobile patients (see 5.1 for definition) in the experimental and control groups is given in table 6.5. Overall patients in the UCG were much more likely to be assessed after being chairnursed for more than 2 hours (chi² = 326.44, p < 0.0001). Although mobile subjects in UCG were permitted to sit out longer than 2 hours, there was no significant difference in the time mobile subjects in either group spent in bed. However, immobile LCG subjects were more likely to be assessed in bed than immobile UCG subjects (z-test = 4.93, p < 0.001). Overall, mobile subjects were less likely to be sitting out for more than 2 hours than immobile ones (z-test = 5.04, p < 0.0001), regardless of group.

Table 6.5 Time sat up in chair at assessment for various groups of subjects (numbers of assessments)

<table>
<thead>
<tr>
<th></th>
<th>Time up in chair at assessment</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bed rest</td>
<td>0-2 hours</td>
<td>2-4 hours</td>
<td>4-6 hours</td>
<td>6-8 hours</td>
<td>&gt;8 hours</td>
</tr>
<tr>
<td>Mobile LCG</td>
<td>273</td>
<td>393</td>
<td>4</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>40.8</td>
<td>58.7</td>
<td>0.6</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Mobile UCG</td>
<td>198</td>
<td>213</td>
<td>54</td>
<td>29</td>
<td>16</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>38.2</td>
<td>41.0</td>
<td>10.4</td>
<td>5.6</td>
<td>3.1</td>
<td>1.7</td>
</tr>
<tr>
<td>Immobile LCG</td>
<td>478</td>
<td>173</td>
<td>6</td>
<td>1</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>72.6</td>
<td>26.3</td>
<td>0.9</td>
<td>0.2</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Immobile UCG</td>
<td>523</td>
<td>231</td>
<td>80</td>
<td>99</td>
<td>41</td>
<td>33</td>
</tr>
<tr>
<td></td>
<td>51.9</td>
<td>22.9</td>
<td>7.9</td>
<td>9.8</td>
<td>4.7</td>
<td>3.3</td>
</tr>
</tbody>
</table>

6.3 PART 1: INCIDENCE OF NEGATIVE OUTCOMES

6.3.1 Subjects

All subjects who were admitted to the study were assessed on an intention-to-treat basis - 100 in the LCG and 103 in the UCG. Table 6.4 lists those conditions which may affect the outcomes of interest. As noted in section 5.7.3, patients with a negative outcome present at baseline may be less likely to develop a 'new' occurrence of the negative outcome. To examine this effect, the data were re-analysed with all individuals with 'negative outcome' present at baseline excluded (appendix 7). There were no differences found using this method of analysis compared with analysis on an 'intention to treat' basis.

6.3.2 Severe self perceived fatigue (7-9 arbitrary units)

There was no significant difference in the mean fatigue score between the two groups on admission to the trial (4.1 and 4.0). There was also no significant difference in the percentage of subjects prescribed drugs which may cause fatigue (45% and 41%) as a side effect. The proportions of subjects developing severe fatigue were \( p_{LCG} = 41/100 \) and \( p_{UCG} = 69/103 \). The proportion of subjects developing severe fatigue was significantly higher in the UCG than in the LCG (\( p_{UCG} - p_{LCG} = 0.26; p < 0.001; 99\% CI = 0.09, 0.43 \)). Only chairnursing policy (LCG or UCG) was univariately associated with severe fatigue (\( p = 0.003 \)). The logistic regression model, containing chairnursing policy as the
only covariate, showed evidence that subjects in the UCG were 2.92 (99%CI = 1.38, 6.20) times more likely to have severe fatigue than subjects in the LCG.

6.3.3 Tachycardia (pulse rate > 100 beats per minute)
There was no difference in the mean pulse rate between the two groups on admission to the trial (81 beats per minute, SD 13.3). There was also no significant difference in the percentage of subjects prescribed drugs which may cause either tachycardia (60% and 56%) or bradycardia (84% and 85%) as a side effect at any time during the study. The proportions of subjects developing tachycardia were $p_{LCG} = 32/100$ and $p_{UCG} = 54/103$. The proportion of subjects developing tachycardia was significantly higher in the UCG than in the LCG ($p_{UCG} - p_{LCG} = 0.2$; $p = 0.0026$; 99%CI = 0.03, 0.38). Only chairnursing policy (LCG or UCG) was univariately associated with tachycardia ($p = 0.003$). The logistic regression model containing chairnursing policy as the only covariate showed evidence that subjects in the UCG were 2.34 (99%CI = 1.11, 4.96) times more likely to have tachycardia than subjects in the LCG.

6.3.4 Hypotension (systolic blood pressure <100 mmHg)
There was no significant difference in the mean systolic or diastolic blood pressure between the two groups on admission to the trial (74 and 81 mmHg respectively). There was also no significant difference in the percentage of subjects prescribed drugs which may cause either hypo (84% and 85%) or hypertension (85% and 85%) as a side effect. The proportions of subjects developing systolic hypotension were $p_{LCG} = 3/100$ and $p_{UCG} = 14/103$. The proportion of patients developing hypotension in the UCG was significantly higher than in the LCG ($p_{UCG} - p_{LCG} = 0.11$; $p = 0.0051$; 99%CI = 0.008, 0.20).

However, no factors were significant in the logistic regression model at the 1% significance level. Therefore, adequate evidence of an association between chairnursing policy and the incidence of hypotension was not found.

6.3.5 Lower limb oedema (increase of over 20% above baseline)
There was no significant difference in the mean volume of operated and unoperated limbs between the two groups on admission to the trial (1436 cm$^3$ and 1362 cm$^3$; 1404$^3$ and 1388 cm$^3$). There was also no significant difference in the percentage of subjects prescribed drugs which may cause oedema (72% and 69%) as a side effect. Two patients who had undergone bilateral surgery (one in each group) were excluded from the analysis.

The proportions of subjects developing lower leg oedema in the operated limb, calculated on an intention-to-treat basis (excluding subjects with bilateral surgery) were $p_{LCG} = 6/99$ and $p_{UCG} = 35/102$. The proportion of operated limbs that became oedematous in the UCG was significantly higher than in the LCG ($p_{UCG} - p_{LCG} = 0.28$; $p < 0.00001$; 99%CI = 0.15, 0.41). The logistic regression model, containing chairnursing policy as the only covariate, showed evidence that subjects in the UCG were 8.96 (99%CI = 2.57, 31.29) times more likely to develop operated limb oedema than subjects in the LCG.

The proportions of subjects developing lower leg oedema in the unoperated limb, also calculated on an intention-to-treat basis (excluding subjects with bilateral surgery) were $p_{LCG} = 11/99$ and $p_{UCG} = 37/102$. The proportion of unoperated limbs developing oedema was significantly higher in the UCG as compared with LCG ($p_{UCG} - p_{LCG} = 0.2$; $p < 0.00001$; 99%CI = 0.10, 0.40). Only chairnursing policy (LCG or UCG) was univariately associated with unoperated leg oedema. The logistic regression model, containing chairnursing policy as the only covariate, showed evidence that subjects in the UCG were 4.71 (99%CI = 1.72, 12.86) times more likely to develop unoperated limb oedema than subjects in the LCG.
6.3.6 Chest infection
There was no significant difference in the percentage of subjects prescribed drugs which may reduce resistance to infection as a side effect in the LCG and UCG (6% and 4% respectively). The proportions of subjects developing chest infection during the study were $p_{LCG} = 13/98$ and $p_{UCG} = 18/102$. These proportions were not significantly different ($p_{UCG} - p_{LCG} = 0.03; p = 0.4958; 99\%CI = -0.10, 0.17$). Not taking drugs which can cause oedema ($p = 0.004$) was univariately associated with increased risk of chest infection. The logistic regression model, containing oedema inducing drugs as the only covariate showed evidence that subjects not given these drugs were 2.89 times more likely to have a chest infection than those who were given them ($99\%CI = 1.04, 7.97$).

6.3.7 Pressure sores
There was no significant difference in the median total Norton score between the two groups on admission to the trial (19 and 20). No subjects had pressure sores on admission to the study and the proportions of subjects developing new pressure sores were $p_{LCG} = 0/100$ and $p_{UCG} = 9/103$. Since no patients in the LCG developed pressure sores during the study, the only valid test for this data was the Fisher's Exact Test which showed significantly greater incidence of pressure sores in the UCG as compared with LCG ($p = 0.003$). No other factors were significantly univariately associated with the development of pressure sores at the 1% level of significance.

6.3.8 Constipation
There was no significant difference in the percentage of subjects prescribed drugs which may cause either constipation (94% and 95%) or diarrhoea (86% and 87%) as a side effect. The proportions of subjects becoming constipated during the study were $p_{LCG} = 32/100$ and $p_{UCG} = 64/103$. The proportion of subjects developing constipation was therefore significantly higher in the UCG than in the LCG ($p_{UCG} - p_{LCG} = 0.31; p < 0.0001; 99\%CI = 0.14, 0.48$).

Factors univariately associated with the risk of developing constipation were chairnursing policy ($p < 0.0001$), ward ($p = 0.001$) and Austin-Moore’s hemiarthroplasty ($p = 0.006$). Chairnursing policy, ward and Austin-Moore’s hemiarthroplasty remained significant at the 1% level in the multivariate logistic regression model. UCG subjects were 3.66 times more likely to become constipated than LCG subjects ($99\%CI = 1.63, 8.24$). Subjects on one of the wards were 3.18 ($99\%CI = 1.40, 7.21$) times and subjects undergoing Austin Moore’s arthroplasty were 4.97 ($99\%CI = 1.16, 21.37$) times more likely to become constipated than subjects on the other ward and undergoing other types of surgery respectively.

6.3.9 Urinary tract infection (UTI)
There was no significant difference in the percentage of subjects prescribed drugs which may reduce resistance to infection (6% and 4%) as a side effect. The proportions of subjects developing UTI during the course of the study were $p_{LCG} = 7/100$ and $p_{UCG} = 11/103$. There was no significant difference in the incidence of UTI during the study between the UCG and LCG ($p_{UCG} - p_{LCG} = 0.04; p = 0.3542; 99\%CI = 0.065, 0.14$).

Factors univariately associated with increased risk of development of UTI were not being fully mobile on admission ($p = 0.004$) and the patient having emergency surgery as opposed to elective ($p = 0.001$). Only mobility on admission was significant in a multivariate model. Patients who were not fully mobile on admission were 4.27 times more likely to develop UTI than those who were ($99\%CI = 1.11, 16.48$).
6.4 PART 2: INDEPENDENT MOBILITY AND DISCHARGE

6.4.1 Subjects
98 subjects in both LCG and UCG remained in the trial until discharge. Pre-admission mobility and discharge destination is given in table 6.6. There were no significant differences in pre-admission mobility and discharge destination. No subjects died during the study.

Table 6.6 Pre-admission mobility and discharge destination

<table>
<thead>
<tr>
<th>Mobility prior to surgery/fracture (% number of subjects)</th>
<th>LCG</th>
<th>UCG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full</td>
<td>68 (69)</td>
<td>59 (60)</td>
</tr>
<tr>
<td>Limited</td>
<td>28 (29)</td>
<td>38 (39)</td>
</tr>
<tr>
<td>House bound</td>
<td>2 (2)</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Bed/chair bound</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Discharge destination (% number of subjects)</th>
<th>LCG</th>
<th>UCG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Home independently</td>
<td>70 (71)</td>
<td>63 (64)</td>
</tr>
<tr>
<td>Home with Early Discharge Scheme</td>
<td>25 (26)</td>
<td>30 (31)</td>
</tr>
<tr>
<td>Discharge to N/H or R/H</td>
<td>2 (21)</td>
<td>5 (5)</td>
</tr>
<tr>
<td>Discharge to acute hospital</td>
<td>1 (1)</td>
<td>-</td>
</tr>
</tbody>
</table>

NH = Nursing home      RH = Residential home

6.4.2 First day of independent mobilisation
Subjects in the LCG achieved independent mobility (as defined in section 5.1) at a mean of 5.8 days (SE=0.22, 95%CI=5.4, 6.2), more quickly than those in the UCG at 8.4 days (SE=0.46, 95%CI=7.4, 9.3). This difference was statistically significant (t-test, p<0.0001, 95%CI of the difference = 3.5, 1.5). Data were lost on one patient in LCG who was discharged before he attained independent mobility.

Because the distribution of the data was very skewed, a log transformation was carried out to bring the outcome measures into a normal distribution. Variables which were univariately significantly associated with time to mobilisation were:

- Chairnursing policy (LCG vs UCG) p<0.0001
- Age p=0.005
- Build (overweight vs normal vs underweight) p=0.004
- Volume of operated leg on admission p=0.011
- Volume of unoperated leg on admission p=0.002
- Norton score (20 vs <20) p<0.001
- Pulse rate on admission p=0.036
- Mobile on admission (fully mobile vs others) p=0.003
- Type of surgery (elective vs emergency) p<0.001
- Type of operation p<0.0001

The above variables were entered into a multivariate model. In this model chairnursing policy (p<0.001), build (p=0.006) and type of operation (p<0.0001) remained significant.
6.4.3 Length of hospital stay
Subjects in the LCG were discharged at a mean of 11.6 days (SE=0.51, 95%CI=10.6, 12.7), more quickly than those in the UCG at 14.0 days (SE=0.63, 95%CI=12.8, 15.3). This difference was statistically significant (t-test, p<0.0036, 95%CI of the difference = -4.0, -0.8).

Because the distribution of the data was very skewed, a log transformation was carried out to bring the outcome measures into a normal distribution. Variables which were univariately significantly associated with time to mobilisation were:

- Chair nursing policy (LCG vs UCG)  p=0.003
- Age  p=0.009
- Build (> normal vs normal vs < normal)  p=0.02
- Volume of operated leg on admission  p=0.05
- Norton score (20 vs <20)  p<0.001
- Pulse rate on admission  p=0.0008
- Mobile on admission (fully mobile vs others)  p=0.001
- Fatigue on admission  p=0.008
- Type of surgery (elective vs emergency)  p<0.001
- Type of operation  p<0.0001

The above variables were entered into a multivariate model. In this model chair nursing policy (p<0.004), SAF on admission (p=0.001) and type of operation (p<0.001) remained significant.

6.5 PART 3: PULSE RATE, BLOOD PRESSURE, LOWER LEG VOLUME AND FATIGUE

6.5.1 Assessments
2854 assessments were carried out during the course of the study, a mean of 14 assessments per subject. 1328 observations were carried out in the LCG and 1524 in UCG. The numbers of assessments carried out on various post-operative days are shown in table 6.7.

Table 6.7 Numbers of assessments on various post-operative days

<table>
<thead>
<tr>
<th>Group</th>
<th>Number of assessments</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Day 3</td>
</tr>
<tr>
<td>LCG</td>
<td>396</td>
</tr>
<tr>
<td>UCG</td>
<td>395</td>
</tr>
<tr>
<td>Total</td>
<td>791</td>
</tr>
</tbody>
</table>

There were significant differences between LCG and UCG groups in the numbers of assessments carried out on days 10 and 14 (z-test, day 10 p=0.011; day 14 p= 0.0002), which are attributable to the longer mean length of hospitalisation of the UCG group (see section 6.2.3).

6.5.2 Summary statistics
Two summary measures were chosen: firstly the mean overall assessments, independent of day for each patient and secondly, the mean of the means at each day of assessment per patient. However, since they produced similar results, only the results for the first summary statistic are presented. Table 6.8 shows the means and the standard error (SE) of these summary statistics for all outcomes.
Table 6.8 Means for pulse rate, blood pressure, lower leg volume and fatigue level during the study

<table>
<thead>
<tr>
<th>Outcome</th>
<th>LCG mean (SE)</th>
<th>UCG mean (SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean pulse rate</td>
<td>83.3 (0.85)</td>
<td>84.2 (0.82)</td>
</tr>
<tr>
<td>Mean systolic blood pressure</td>
<td>130.2 (1.15)</td>
<td>129.1 (1.02)</td>
</tr>
<tr>
<td>Mean diastolic blood pressure</td>
<td>71.1 (0.75)</td>
<td>69.5 (0.54)</td>
</tr>
<tr>
<td>Mean volume operated lower leg</td>
<td>1483.1 (32.63)</td>
<td>1390.0 (38.20)</td>
</tr>
<tr>
<td>Mean volume unoperated lower leg</td>
<td>1408.6 (30.55)</td>
<td>1354.4 (35.46)</td>
</tr>
<tr>
<td>Mean SAF</td>
<td>4.4 (0.11)</td>
<td>4.6 (0.11)</td>
</tr>
</tbody>
</table>

There was no significant difference between the means of the outcomes in the experimental and control groups. The following variables were univariately significantly associated with the outcomes:

- pulse rate: baseline level, type of surgery, total Norton score
- systolic blood pressure: baseline level
- diastolic blood pressure: baseline level
- operated lower limb volume: baseline level, age, type of surgery, total Norton score, build
- unoperated lower limb volume: baseline level, age, type of surgery, total Norton score, build
- Self assessed fatigue level: baseline level

When these factors were entered into a multivariate regression model in order to adjust for confounding, in all outcomes the only factor which remained highly significant (p value < 0.001) was the baseline value of the outcome. However, when the mean outcomes for each time of each post-surgical day were calculated and shown graphically (appendix 6), it appeared that there was variation between times and days and that the difference between the experimental and control group (ie the treatment effect) might vary depending on post-operative day and time of day.

Because of the hierarchical structure of the data, multi-level models were used (Goldstern 1995). These are models which regress the outcomes at a particular time, at a particular day, for a particular patient on the variables of interest, taking into account the variance structure between measurements within the same day and the same patient.
6.5.3 Multilevel models

According to the final models, mean outcomes (pulse rate, blood pressure, lower leg volume) differ between days and between times and the effect of experimental or control group (i.e., LCG and UCG) is not constant but differs between time points within days after adjusting for baseline rates. The coefficients and their standard errors for the group effect are shown in Table 6.9. The significance of the coefficients was tested using the Wald test (Armitage & Berry 1994). Fatigue was not analysed using multi-level models because of the additional requirements on software support which are needed to manage the categorical-like nature of the data for SAF.

Table 6.9 Coefficients (SEs) and their significance for the difference between UCH and LCH in pulse rate, blood pressure, lower leg volume and fatigue level

<table>
<thead>
<tr>
<th></th>
<th>1 9-10 am</th>
<th>2 12-1 pm</th>
<th>3 3-4 pm</th>
<th>4 6-7 pm</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pulse rate</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-2.89</td>
<td>1.45</td>
<td>1.45</td>
<td>1.45</td>
<td></td>
</tr>
<tr>
<td><strong>Systolic blood pressure</strong></td>
<td>1.42</td>
<td>0.88</td>
<td>0.88</td>
<td>0.89</td>
</tr>
<tr>
<td>-3.81</td>
<td>0.88</td>
<td>0.88</td>
<td>0.88</td>
<td></td>
</tr>
<tr>
<td><strong>Diastolic blood pressure</strong></td>
<td>0.90</td>
<td>0.68</td>
<td>0.68</td>
<td>0.68</td>
</tr>
<tr>
<td>0.04</td>
<td>0.96</td>
<td>0.96</td>
<td>0.96</td>
<td></td>
</tr>
<tr>
<td><strong>Volume of operated lower leg</strong></td>
<td>27.48</td>
<td>11.24</td>
<td>11.23</td>
<td>11.30</td>
</tr>
<tr>
<td>-62.11</td>
<td>25.59</td>
<td>25.59</td>
<td>25.59</td>
<td></td>
</tr>
<tr>
<td><strong>Volume of unoperated lower leg</strong></td>
<td>1.32</td>
<td>0.05</td>
<td>0.001</td>
<td>0.002</td>
</tr>
<tr>
<td>-1.60</td>
<td>1.32</td>
<td>1.32</td>
<td>1.32</td>
<td></td>
</tr>
<tr>
<td><strong>Systolic blood pressure</strong></td>
<td>1.42</td>
<td>0.88</td>
<td>0.88</td>
<td>0.89</td>
</tr>
<tr>
<td>-3.81</td>
<td>0.88</td>
<td>0.88</td>
<td>0.88</td>
<td></td>
</tr>
<tr>
<td><strong>Diastolic blood pressure</strong></td>
<td>0.90</td>
<td>0.68</td>
<td>0.68</td>
<td>0.68</td>
</tr>
<tr>
<td>0.04</td>
<td>0.96</td>
<td>0.96</td>
<td>0.96</td>
<td></td>
</tr>
<tr>
<td><strong>Volume of operated lower leg</strong></td>
<td>27.48</td>
<td>11.24</td>
<td>11.23</td>
<td>11.30</td>
</tr>
<tr>
<td>-62.11</td>
<td>25.59</td>
<td>25.59</td>
<td>25.59</td>
<td></td>
</tr>
<tr>
<td><strong>Volume of unoperated lower leg</strong></td>
<td>1.32</td>
<td>0.05</td>
<td>0.001</td>
<td>0.002</td>
</tr>
<tr>
<td>-1.60</td>
<td>1.32</td>
<td>1.32</td>
<td>1.32</td>
<td></td>
</tr>
</tbody>
</table>

The table is interpreted as follows. The difference between mean outcome in the UCG and LCG at time 9-10 am is given in column 1. The corresponding difference for 12-1 pm is given by summation of columns 1 and 2 and for 3-4 pm by the summation of columns 1 and 3 etc. Using diastolic blood pressure as an example, at 9-10 am mean diastolic blood pressure in UCG is 0.04 mmHg higher than in LCG. At 12-1 pm the difference is: 0.04 + (-2.64) = -2.6. This means that at 12-1 pm, the mean diastolic blood pressure in UCG is 2.6 mmHg lower than in the LCG. No significant differences between LCG and UCG were detected at 9-10 am but significant differences were detected for all outcomes in later time periods during the day.

6.5.4 Pulse rate, blood pressure, lower leg volume and fatigue after various lengths of chairnursing

6.5.4.1 Self-assessed fatigue (SAF)

Mean differences between pre-operative baseline SAF and at assessment during bed rest and after different lengths of time sat in a chair are given are given in Table 6.10. There were no significant differences between LCG and UCG which suggests that chairnursing had no cumulative effect on SAF and the data from the two groups were combined in figure 6.1.
Table 6.10 Mean difference between SAF level at baseline and after varying lengths of chairnursing

<table>
<thead>
<tr>
<th>Group</th>
<th>Time up in chair at assessment</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bed rest</td>
<td>0-2 hours</td>
<td>2-4 hours</td>
<td>4-6 hours</td>
<td>6-8 hours</td>
<td>&gt;8 hours</td>
<td></td>
</tr>
<tr>
<td>LCG: mean difference</td>
<td>-0.12</td>
<td>0.38</td>
<td>3.38</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td></td>
</tr>
<tr>
<td>SE</td>
<td>0.08</td>
<td>0.09</td>
<td>0.84</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td></td>
</tr>
<tr>
<td>Number of obs</td>
<td>711</td>
<td>542</td>
<td>8</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td></td>
</tr>
<tr>
<td>UCG: mean difference</td>
<td>0.17</td>
<td>0.33</td>
<td>1.25</td>
<td>2.41</td>
<td>2.63</td>
<td>3.42</td>
<td></td>
</tr>
<tr>
<td>SE</td>
<td>0.07</td>
<td>0.10</td>
<td>0.17</td>
<td>0.19</td>
<td>0.29</td>
<td>0.31</td>
<td></td>
</tr>
<tr>
<td>Number of obs</td>
<td>717</td>
<td>442</td>
<td>135</td>
<td>126</td>
<td>56</td>
<td>42</td>
<td></td>
</tr>
<tr>
<td>Tot: mean difference</td>
<td>0.25</td>
<td>0.36</td>
<td>1.37</td>
<td>2.41</td>
<td>2.63</td>
<td>3.42</td>
<td></td>
</tr>
<tr>
<td>SE</td>
<td>0.05</td>
<td>0.07</td>
<td>0.17</td>
<td>0.19</td>
<td>0.29</td>
<td>0.31</td>
<td></td>
</tr>
<tr>
<td>Number of obs</td>
<td>1428</td>
<td>984</td>
<td>143</td>
<td>126</td>
<td>56</td>
<td>42</td>
<td></td>
</tr>
</tbody>
</table>

obs = observations  SE = Standard error

Figure 6.1 Mean self assessed fatigue levels after bedrest and varying lengths of chairnursing (SE)
Severe fatigue (7-9 arbitrary units on VAS) was significantly associated with length of chairnursing ($\text{Chi}^2$ with 5 degrees of freedom = 423.33, $p < 0.001$). Observations of severe fatigue after various lengths of time sat in a chair are given in table 6.11.

### Table 6.11 Severe fatigue after varying lengths of chairnursing

<table>
<thead>
<tr>
<th>Time up in chair at assessment</th>
<th>Bed rest</th>
<th>0-2 hours</th>
<th>2-4 hours</th>
<th>4-6 hours</th>
<th>6-8 hours</th>
<th>&gt;8 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe fatigue (number of obs)</td>
<td>144</td>
<td>109</td>
<td>40</td>
<td>72</td>
<td>32</td>
<td>31</td>
</tr>
<tr>
<td>Total number of obs</td>
<td>1468</td>
<td>1007</td>
<td>144</td>
<td>128</td>
<td>56</td>
<td>42</td>
</tr>
<tr>
<td>% obs with severe fatigue</td>
<td>9.8</td>
<td>10.8</td>
<td>27.8</td>
<td>56.3</td>
<td>57.1</td>
<td>73.8</td>
</tr>
</tbody>
</table>

*obs = observations*

#### 6.5.4.2 Pulse rate

Mean differences between pre-operative baseline pulse rate and at assessment during bed rest and after different lengths of time sat in a chair are given in table 6.12. There were no significant differences between LCG and UCG which suggests that chairnursing had no cumulative effect on pulse rate and the data from the two groups was combined in figure 6.2.

### Table 6.12 Mean difference between pulse rate at baseline and after varying lengths of chairnursing

<table>
<thead>
<tr>
<th>Group</th>
<th>Time up in chair at assessment</th>
<th>Bed rest</th>
<th>0-2 hours</th>
<th>2-4 hours</th>
<th>4-6 hours</th>
<th>6-8 hours</th>
<th>&gt;8 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>LCG: mean difference</td>
<td>0.51</td>
<td>3.34</td>
<td>8.67</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td></td>
</tr>
<tr>
<td>SE</td>
<td>10.4</td>
<td>0.56</td>
<td>5.55</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td></td>
</tr>
<tr>
<td>Number of obs</td>
<td>742</td>
<td>561</td>
<td>9</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td></td>
</tr>
<tr>
<td>UCG: mean difference</td>
<td>0.89</td>
<td>0.81</td>
<td>6.61</td>
<td>13.23</td>
<td>16.61</td>
<td>16.71</td>
<td></td>
</tr>
<tr>
<td>SE</td>
<td>0.94</td>
<td>0.64</td>
<td>1.33</td>
<td>1.28</td>
<td>2.23</td>
<td>3.19</td>
<td></td>
</tr>
<tr>
<td>Number of obs</td>
<td>729</td>
<td>448</td>
<td>135</td>
<td>129</td>
<td>57</td>
<td>42</td>
<td></td>
</tr>
<tr>
<td>Tot: mean difference</td>
<td>0.70</td>
<td>2.22</td>
<td>6.74</td>
<td>13.23</td>
<td>16.61</td>
<td>16.71</td>
<td></td>
</tr>
<tr>
<td>SE</td>
<td>0.53</td>
<td>0.42</td>
<td>1.29</td>
<td>1.28</td>
<td>2.23</td>
<td>3.19</td>
<td></td>
</tr>
<tr>
<td>Number of obs</td>
<td>1471</td>
<td>1009</td>
<td>144</td>
<td>129</td>
<td>57</td>
<td>42</td>
<td></td>
</tr>
</tbody>
</table>

*obs = observations  SE = Standard error*
Tachycardia (a pulse rate of >100 beats per minute) was significantly associated with length of chairnursing (Chi² with 5 degrees of freedom = 245.39, p < 0.001). Observations of tachycardia after various lengths of time sat in a chair are given in table 6.13.

**Table 6.13 Tachycardia after varying lengths of chairnursing**

<table>
<thead>
<tr>
<th>Time up in chair at assessment</th>
<th>Bed rest</th>
<th>0-2 hrs</th>
<th>2-4 hrs</th>
<th>4-6 hrs</th>
<th>6-8 hrs</th>
<th>&gt;8 hrs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tachycardia (number of obs)</td>
<td>72</td>
<td>78</td>
<td>18</td>
<td>40</td>
<td>22</td>
<td>19</td>
</tr>
<tr>
<td>Total number of obs</td>
<td>1471</td>
<td>1009</td>
<td>144</td>
<td>129</td>
<td>57</td>
<td>42</td>
</tr>
<tr>
<td>% obs with tachycardia</td>
<td>4.9</td>
<td>7.7</td>
<td>12.5</td>
<td>31.0</td>
<td>38.6</td>
<td>45.2</td>
</tr>
</tbody>
</table>

*obs = observations*

### 6.5.4.3 Blood pressure

Mean differences between pre-operative baseline systolic and diastolic blood pressures and at assessment during bed rest and after different lengths of time sat in a chair are given in table 6.14. There were no significant differences between LCG and UCG which suggest that chairnursing had no cumulative effect on blood pressure and the data from the two groups was combined in figure 6.3.
Table 6.14 Mean difference between blood pressure at baseline and after varying lengths of chair nursing

<table>
<thead>
<tr>
<th>Group</th>
<th>Time up in chair at assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bed rest</td>
</tr>
<tr>
<td>LCG (systolic)</td>
<td>Mean difference</td>
</tr>
<tr>
<td></td>
<td>SE</td>
</tr>
<tr>
<td></td>
<td>Number of obs</td>
</tr>
<tr>
<td>UCG (systolic)</td>
<td>Mean difference</td>
</tr>
<tr>
<td></td>
<td>SE</td>
</tr>
<tr>
<td></td>
<td>Number of obs</td>
</tr>
<tr>
<td>Total (systolic)</td>
<td>Mean difference</td>
</tr>
<tr>
<td></td>
<td>SE</td>
</tr>
<tr>
<td></td>
<td>Number of obs</td>
</tr>
<tr>
<td>LCG (diastolic)</td>
<td>Mean difference</td>
</tr>
<tr>
<td></td>
<td>SE</td>
</tr>
<tr>
<td></td>
<td>Number of obs</td>
</tr>
<tr>
<td>UCG (diastolic)</td>
<td>Mean difference</td>
</tr>
<tr>
<td></td>
<td>SE</td>
</tr>
<tr>
<td></td>
<td>Number of obs</td>
</tr>
<tr>
<td>Total (diastolic)</td>
<td>Mean difference</td>
</tr>
<tr>
<td></td>
<td>SE</td>
</tr>
<tr>
<td></td>
<td>Number of obs</td>
</tr>
</tbody>
</table>

**Figure 6.3 Mean difference in blood pressure at baseline and after varying lengths of chair nursing (SE)**
Hypotension (a pulse rate of > 100 beats per minute) was significantly associated with lengthy chairnursing ($\chi^2$ with 5 degrees of freedom = 81.46, $p < 0.001$). Observations of hypotension after various lengths of time sat in a chair are given in Table 6.15.

**Table 6.15 Hypotension after varying lengths of chairnursing**

<table>
<thead>
<tr>
<th>Hypotension (number of obs)</th>
<th>Time up in chair at assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bed rest</td>
</tr>
<tr>
<td>Hypotension (number of obs)</td>
<td>7</td>
</tr>
<tr>
<td>% obs with hypotension</td>
<td>0.5</td>
</tr>
<tr>
<td>Total number of obs</td>
<td>1472</td>
</tr>
</tbody>
</table>

6.5.4.4 Lower leg volume

Mean differences between pre-operative baseline operated and unoperated lower leg volume and at assessment during bed rest and after different lengths of time sat in a chair are given in Table 6.16. There were no significant differences between LCG and UCG which suggest that chairnursing had no cumulative effect on lower leg volume and the data from the two groups were combined in Figure 6.4.

**Table 6.16 Mean difference between lower leg volume at baseline and after varying lengths of chairnursing**

<table>
<thead>
<tr>
<th>Group</th>
<th>Time up in chair at assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bed rest</td>
</tr>
<tr>
<td>LCG (unoperated leg)</td>
<td></td>
</tr>
<tr>
<td>Mean difference</td>
<td>-30.76</td>
</tr>
<tr>
<td>SE</td>
<td>5.63</td>
</tr>
<tr>
<td>Number of obs</td>
<td>656</td>
</tr>
<tr>
<td>UCG (unoperated leg)</td>
<td></td>
</tr>
<tr>
<td>Mean difference</td>
<td>-60.01</td>
</tr>
<tr>
<td>SE</td>
<td>9.04</td>
</tr>
<tr>
<td>Number of obs</td>
<td>636</td>
</tr>
<tr>
<td>Total (unop leg)</td>
<td></td>
</tr>
<tr>
<td>Mean difference</td>
<td>-45.16</td>
</tr>
<tr>
<td>SE</td>
<td>5.30</td>
</tr>
<tr>
<td>Number of obs</td>
<td>1292</td>
</tr>
<tr>
<td>LCG (operated leg)</td>
<td></td>
</tr>
<tr>
<td>Mean difference</td>
<td>34.77</td>
</tr>
<tr>
<td>SE</td>
<td>9.98</td>
</tr>
<tr>
<td>Number of obs</td>
<td>537</td>
</tr>
<tr>
<td>UCG (operated leg)</td>
<td></td>
</tr>
<tr>
<td>Mean difference</td>
<td>8.00</td>
</tr>
<tr>
<td>SE</td>
<td>7.43</td>
</tr>
<tr>
<td>Number of obs</td>
<td>507</td>
</tr>
<tr>
<td>Total (operated leg)</td>
<td></td>
</tr>
<tr>
<td>Mean difference</td>
<td>21.77</td>
</tr>
<tr>
<td>SE</td>
<td>6.29</td>
</tr>
<tr>
<td>Number of obs</td>
<td>1044</td>
</tr>
</tbody>
</table>

obs = observations  SE = Standard error
Oedema (defined as an increase of 20% over baseline) was significantly associated with length of chairnursing (Chi² with 5 degrees of freedom = 208.55, p < 0.001). Observations of oedema after various lengths of time sat in a chair are given in table 6.17 and 6.18.

Table 6.17 Oedema in operated limb after varying lengths of chairnursing

<table>
<thead>
<tr>
<th>Time up in chair at assessment</th>
<th>Bed rest</th>
<th>0-2 hours</th>
<th>2-4 hours</th>
<th>4-6 hours</th>
<th>6-8 hours</th>
<th>&gt;8 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oedema (number of obs)</td>
<td>56</td>
<td>89</td>
<td>15</td>
<td>26</td>
<td>21</td>
<td>17</td>
</tr>
<tr>
<td>Total number of obs</td>
<td>1044</td>
<td>759</td>
<td>101</td>
<td>85</td>
<td>39</td>
<td>30</td>
</tr>
<tr>
<td>% obs with oedema</td>
<td>5.4</td>
<td>11.7</td>
<td>14.9</td>
<td>30.6</td>
<td>53.9</td>
<td>56.7</td>
</tr>
</tbody>
</table>

Table 6.18 Oedema in un-operated limb after varying lengths of chairnursing

<table>
<thead>
<tr>
<th>Time up in chair at assessment</th>
<th>Bed rest</th>
<th>0-2 hours</th>
<th>2-4 hours</th>
<th>4-6 hours</th>
<th>6-8 hours</th>
<th>&gt;8 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oedema (number of obs)</td>
<td>36</td>
<td>74</td>
<td>20</td>
<td>25</td>
<td>15</td>
<td>21</td>
</tr>
<tr>
<td>Total number of obs</td>
<td>1292</td>
<td>908</td>
<td>127</td>
<td>111</td>
<td>51</td>
<td>20</td>
</tr>
<tr>
<td>% obs with oedema</td>
<td>2.8</td>
<td>8.2</td>
<td>15.8</td>
<td>22.5</td>
<td>29.4</td>
<td>51.2</td>
</tr>
</tbody>
</table>

obs = observations
7 DISCUSSION

7.1 MAIN FINDINGS

The main finding of this study was that implementing a policy of limiting chairnursing in patients who are unable to get out of a chair by themselves significantly reduced the incidence of chairnursing sessions lasting longer than 2 hours. This seemed to be associated with a beneficial effect on a range of outcomes which included a reduced incidence of constipation and pressure sores. There was a statistically significant reduction in the occurrence of tachycardia, lower leg oedema and severe fatigue while the reduction in hypotension approached significance. Subjects in the experimental, limited chairnursing group (LCG) achieved mobility (defined as being able to get out of their chair independently) more rapidly and were discharged sooner than those in the control, unlimited chairnursing group (UCG). No differences were found between the two groups in the incidence of UTI and chest infection. The LCG and UCG groups were evenly matched across a range of demographic, health and treatment characteristics (see section 6.1) on admission. This supports the conclusion that any difference in outcome found between the two groups is likely to be attributable to differences in chairnursing policy rather than baseline imbalances.

A secondary finding of the study was that mean pulse rate, blood pressure, lower leg volume and fatigue varied between LCG and UCG, but only when measured from 12 noon, onwards (table 6.9). A likely explanation for this phenomenon is that between 9-10am subjects in both groups would have been chairnursed for approximately the same amount of time, having been got out of bed at most 3 hours previously. By 12 noon, subjects who had been up at 9-10am in the LCG would have been returned to bed or been walking, while a proportion of those in UCG would have remained in their chairs. For the same reasons, increasingly longer periods of chairnursing would have been possible only as the day wore on. This explanation is supported by the observation that mean pulse rate and lower leg volume was higher while blood pressure was lower than baseline the longer subjects were chairnursed (figures 6.1, 6.2, 6.3). Thus subjects who were in the UCG were more likely to be sitting longer and therefore more likely to experience a higher pulse rate, higher leg volume and a lower blood pressure.

Subjects in the UCG who were not independently mobile appeared to spend more time in bed than those in the LCG. Despite this, there was no evidence of increased incidence of complications in this group. There was also no evidence that subjects in the LCG had become accustomed to being less mobile as a result of the limited chairnursing regime. When they became independently mobile, they appeared to spend no more time in bed than the UCG subjects. Since an approximately equal amount of time was spent out of bed by mobile LCG and UCG subjects and longer sessions of chairnursing were recorded in the mobile UCG subjects, it follows that LCG subjects were changing their posture more frequently. This is consistent with the findings of the preliminary study (Gebhardt & Bliss 1994, appendix 2).

The greater frequency of lengthy chairnursing (ie over 2 hours) in independently mobile UCG subjects as compared with LCG, suggests that patients were influenced by the regime and attitudes on the ward. This view is reinforced by some statements recorded by the researcher. For example one patient said: "I feel exhausted today, but I am pleased because I managed to sit out all day yesterday". Another said "They (the staff) are pleased with me because I am sitting out all day". The length of time they managed to sit seemed to be the yardstick by which they measured recovery. On the other hand, subjects nursed under the limited chairnursing regime were more likely to 'boast' of the number of times they had changed position or walked and this seemed to be their yardstick for recovery. "I've walked to the dayroom and back 6 times today", "I have been walking a lot" and "I walk about a bit, then lie down for half an hour, then walk some more" are some examples. This
suggests that the implementation of a particular chairnursing regime and the attitude of staff may be important influences on patient behaviour once they become independent. This may have implications for long-term rehabilitation although more research is need to confirm this and to clarify how best patient education should be managed.

However, the overall amount of chairnursing that was observed in the UCG was considerably less than that observed in other studies of orthopaedic wards where there was no restriction on chairnursing. The median length of time per session in the present study was 1-2 hours. In the Gebhardt & Bliss (1994) study, subjects in the UCG sat out for a mean of 6-7 hours and Bliss et al in other work (unpublished data) observed a mean of 6.5 hours. 83% of chairnursing sessions observed in the Bliss et al study (unpublished data) exceeded 2 hours. Only 34% UCG sessions did so in the present one. Although the Bliss study monitored chairnursing time continuously and the results of the present study represent 1524 assessments sampling at fixed times of day, it is unlikely that such a large difference was the result of sampling error. This difference requires some explanation.

Most likely, this lies in the ward staff being influenced by hospital policy and the known clinical views of the researcher who has a senior post within the organisation. The Pressure Sore Prevention and Wound Management Policy of the Trust which hosted the research (Gebhardt et al 1996) explicitly prohibits chairnursing for more than two hours per session of any patients deemed at risk of pressure sores. Arguably, all patients who had recently undergone hip surgery (Gebhardt & Bliss 1994) could be considered in this category as well as some patients who had undergone knee replacement. Education within the Trust on this subject (in which about 30% of the staff from the research wards had participated) stresses the importance of complying with the policy with regard to patients at risk of pressure sores and generally recommends frequent changes of posture, the encouragement of walking as opposed to sitting and compliance with patients' wishes regarding time spent in chairs. It is reasonable to expect that staff would not have made a distinction between the researcher as researcher and as senior manager, and would have made efforts to comply with policy and recommendations, when under the scrutiny of a research project. Additionally, it cannot be ruled out that despite an extended washout period there was some learning effect between periods. However if this was a significant factor, there should have been a difference in the number of lengthy chairnursing sessions between the first and second phases of the study. There was no evidence of such a difference.

The major implication of the small number of limited chairnursing sessions is that the limited and unlimited regimes were not very different. This means that any differences in outcomes may have been somewhat less than otherwise might have been the case. This has to be borne in mind when considering the outcomes individually. It is recommended that for any future confirmatory studies, sites should be chosen where prolonged chairnursing continues to be routinely practised and the research should not be implemented by an individual with known views regarding chairnursing.

7.1.1 Fatigue
Patients were more likely to have episodes of significant fatigue in the UCG as compared with the LCG (defined as a self assessed fatigue score of 7 or more on a VAS scale). There were no significant differences observed between LCG and UCG in factors which may influence fatigue such as pre-operative mobility, mean pre-operative self-assessed fatigue level, the incidence of diseases or conditions which can cause fatigue or the prescription of drugs which may cause fatigue as a side-effect. It is, therefore likely that any differences between LCG and UCG related to the difference in chairnursing regimes. Patients who were assessed after sitting for more than 2 hours were more likely to be severely fatigued than those who had not (table 6.18) and sessions of chairnursing lasting more than 2 hours were less common in LCG than UCG.
This finding supports the views of other authors (Bliss 1990, 1992, Mulley 1993) who believe that lengthy chairnursing is fatiguing and suggests that patient reports of fatigue following chairnursing (Dzielski 1999, Gebhardt & Bliss 1994) represent a common experience rather than isolated occurrences. Since fatigue is distressing to patients and may have negative impacts on general well being and possibly general health, the benefits of chairnursing will need to be demonstrated to justify the practice. The higher frequency of severe fatigue in the UCG may have contributed to the longer time that patients took to mobilise independently and to be discharged home.

Further research is needed to determine how mobilisation and rehabilitation regimes can be tailored to minimise fatigue, as it seems reasonable to assume that fatigued patients would have less ability and motivation to mobilise and exercise, important objectives of any rehabilitation regime. Even if fatigue were found to have no impact on other outcomes, reducing it seems worthwhile because of the distress it causes to many patients.

7.1.2 Tachycardia
Patients were observed to be tachycardic (for the purposes of this study defined as having a pulse rate > 100 beats per minute) more frequently in the UCG as compared with the LCG. There were no significant differences observed between LCG and UCG in factors which may influence pulse rate such as pre-operative mobility, baseline pulse rate, the occurrence of post-operative anaemia, diseases which produce tachycardic symptoms or the prescription of drugs which may cause tachycardia as a side-effect. It is therefore likely that the difference observed between LCG and UCG was related to the difference in chairnursing regimes. This is supported by the observations that patients who were assessed after sitting for more than 2 hours were more likely to be tachycardic than those who had not (table 6.11) and that sessions of chairnursing longer than 2 hours were significantly less common in LCG than UCG (table 6.5).

It is possible that other factors, not considered in the present study, may have had an impact on the incidence of tachycardia. Pain (eg Brand & Abraham-Inpijn 1996) and anxiety (eg Roth et al 1996) are known to increase pulse rate. Patients in the present study group were likely to be in pain pre-operatively due to either fracture or osteo-arthritis and post-operatively due to surgical wounds. Anxiety both before and after surgery is also common (Shafer et al 1996). It is recommended that the effect of these variables should be considered in future confirmatory studies of the effect of chairnursing on pulse rate.

The main dangers of tachycardia relate to an increased myocardial oxygen consumption coupled with reduced efficiency of the coronary circulation. It is reasonable to assume that a population which has a high incidence of cardiac morbidity such as the elderly would be particularly at risk. In this particular sample there was an overall incidence of cardiovascular disease of 21% (exclusive of hypertension). It also seems reasonable to assume that tachycardia of a long duration is more likely to cause complications than short bursts of adaptive tachycardia. The present, sampling study cannot provide information on whether the tachycardia was continuous or frequent. An observational study, monitoring patients continuously while they sat could elucidate this point. It is notable, perhaps, that two patients in the UCG dropped out of the study due to cardiac complications. It would probably be advantageous to monitor for heart failure, angina, cardiac arrhythmias and myocardial infarction in any future studies of the effects of prolonged chairnursing on heart rate, since these are outcomes of clinical interest.

The incidence of tachycardia in the study population was high regardless of which arm of the study the subjects belonged to (section 6.2.2). There are a number of possible explanations for this. The mean baseline pulse rate of 80 bpm in this study group was higher than the mean of 70 bpm that would be expected in healthy younger individuals though normal for this age group (Colbert 1993). Factors other than age related deconditioning may have been reduced mobility prior to admission and pre-operative anxiety. Pain, anxiety and anaemia which is caused by operative blood loss are likely
to play a part post-operatively. However, the *difference* in incidence of tachycardia between LCG and UCG may be due to failure of homeostatic mechanisms to compensate adequately for the translocation of blood volume from the thorax to the legs during prolonged sitting. This view is compatible with the observation that the likelihood of observing tachycardia increased, the longer the subject had been sitting prior to assessment (table 6.13).

Failure of homeostatic mechanisms for counteracting the effects of orthostasis may lead to tachycardia for a number of reasons. Firstly any reduction in circulating volume, means that the remaining volume has to be circulated more rapidly to fulfil the body’s requirements for the transportation of oxygen, carbon dioxide, nutrients and so on. Secondly, circulating blood volume is made up to some degree following pooling in the dependent parts of the body by a reduction in excretion (Guyton 1991). Because this volume is made up mainly with fluid, the carrying capacity of the blood is diluted. Thirdly, although in the sitting position there is an increased lung capacity this is mainly caused by a reduction in thoracic blood volume (Sjostrand 1951). While an increase in peripheral oxygen tension has been found during 10 minutes following sitting up into a semi-recumbent position from supine lying (Mynster et al 1996), the effects on peripheral perfusion of prolonged sitting with legs dependent are not known. It is possible that despite the increased air capacity, the reduced blood perfusion may actually lead to less efficient oxygenation and therefore require a more rapid circulation leading to tachycardia. In practice it is likely that the observed tachycardia is the result of the interplay of the above factors and possibly others as yet unknown.

### 7.1.3 Hypotension

Fewer episodes of hypotension were observed in the LCG subjects as compared with the UCG (hypotension was defined as a systolic blood pressure of $<100$ mmHg) and this difference was univariately statistically significant. However adequate evidence of association with chairnursing policy was not found in logistic regression analysis. As there were no significant differences observed between LCG and UCG in factors which may influence blood pressure such as pre-operative mobility, baseline blood pressure, cardiovascular disease or the prescription of drugs which may have hypotensive side-effects, it unlikely that the lack of difference is due to baseline imbalances between the two groups.

Bliss (1990) noted many elderly patients sat in chairs were "white, dizzy and confused". Mulley (1993) noted that "some ill patients feel faint or distressed because of postural hypotension". The findings of the present study support the above clinical observations to a degree. Blood pressure was found to be lower in patients who had been chaimursed for long periods of time (table 6.3). However, overall, the incidence of hypotension was low. One reason for this may be that, as in the general population (eg Nozawa et al 1996), subjects of this study were likely to vary in the efficiency of their orthostatic regulatory mechanisms. Thus, though overall mean blood pressure appeared to fall at times when patients were likely to have been chaimursed for prolonged periods (table 6.14), this could represent a minority who had a significant reduction in blood pressure and a majority who may have been able to maintain level or indeed slightly raised blood pressures. It would probably be clinically valuable to determine in future research whether such individual differences do in fact exist and if so, how individuals affected by hypotension can be identified so that their rehabilitation programmes can be modified accordingly.

Another reason is likely to be the small difference in the amount of chaimursing between the LCG and UCG and the somewhat low incidence of chairnursing prolonged beyond 2 hours as compared with other studies, which has been discussed in section 7.1. Since hypotension occurred 10 times more frequently in patients sitting for longer than 2 hours as compared to those who had not (table 6.15), it is tempting to believe that hypotension is likely to increase with more prolonged chairnursing. However, this study does not establish a causal link and it is possible that both outcomes (length of time up and incidence of hypotension) are the result of a common cause. For example staff may have selected patients to be chaimursed for longer periods who were also susceptible to
hypotension. Therefore, forced chairnursing may not necessarily increase the incidence of hypotension.

It is also quite likely that the definition of hypotension, arbitrarily set at a level of <100 mmHg based on accepted averages appropriate for young people, was too low for the target population - the elderly. Mean blood pressure tends to be higher in the over 60's (Landahl et al 1986, Powell & MacKnight 1998) and the incidence of hypertension is high. The study population was no exception, with an incidence of 29% of diagnosed hypertension on admission to the study. It is likely that as a consequence even when profound drops in blood pressure occur, they may not reach a threshold set for younger individuals with a lower baseline. However, this does not mean that unpleasant symptoms of acutely lowered blood pressure cannot occur in the elderly, rather hypotension in its usual definition of 'a blood pressure that is inadequate for the circulatory needs of the individual' (eg Anderson et al 1998) may occur at a higher threshold. What significant levels of hypotension are and their relationship to lengthy sitting should be researched, specifically by longitudinally observing patterns of blood pressure in elderly patients chairnursed for long periods of time. Such studies could suggest more clinically useful cut-offs for outcomes research.

Fundamentally, however, functional hypotension ie a blood pressure which is insufficient for the body's circulatory requirements is of greater clinical significance than a blood pressure defined by an arbitrarily chosen level, as has been measured in the present study. In other words, although the present study has shown that different chairnursing regimes can affect blood pressure, the clinical significance of these differences needs to be determined by further research. Therefore, in future studies of blood pressure during chairnursing, the incidence of symptoms of hypotension such as vertigo, dizziness, paleness, mental confusion and syncope should be considered as outcomes of interest as well as measurements of blood pressure itself.

The effects of chairnursing on functional hypotension require further research because inadequate blood pressure impacts negatively on varied aspects of health. Functional hypotension is likely to lead to tachycardia, the dangers of which have been discussed in section 7.1, inadequate perfusion of the organs of excretion, the bowel, the skin and eventually, when blood pressure drops to a sufficiently low level, the brain (Poli et al 1999). Underperfusion of the brain is likely to be a factor in mental confusion and hypotension syncope is a significant factor in falls (Dey et al 1997), which are a major cause of morbidity and mortality of the elderly (Hamdy et al 1997). It is notable, perhaps, that one patient in the UCG dropped out of the study because she developed acute dementia.

7.1.4 Lower limb oedema

Patients were more likely to have episodes of lower leg swelling >20% above baseline in the UCG as compared with the LCG. There were no significant differences observed between LCG and UCG in factors which may influence leg swelling such as pre-operative mobility, mean pre-operative leg volume, diseases which can cause leg oedema or the prescription of drugs which may cause oedema as a side-effect. It is therefore likely that this was related to the difference in chairnursing regimes since patients who were assessed after sitting for more than 2 hours were more likely to have swollen legs than those who had not and sessions of chairnursing of longer than 2 hours were more common in UCG than LCG.

These findings showed a considerably greater amount of swelling than would be expected from studies in younger adults (eg Waterfield 1931, Winkel & Jorgensen 1986, 1986a). This suggests that the anti-oedema mechanisms postulated by Winkel (1985) and which are thought to account for the slowing of leg swelling seen after 4 hours continuous sitting in healthy younger adults (Winkel & Jorgensen 1986) may be less effective in elderly patients, particularly if they have had surgery to the leg. Indeed, on more than half the occasions when subjects of the present study were assessed after sitting for 8 hours or more, a swelling of over 20% was noted. This is consistent with the theoretical
calculation by Winkel (1985) that without oedema-preventing mechanisms, foot swelling of the order of 20-25% could be expected after 8 hours sitting.

What these anti-oedema mechanisms are and why they fail in the elderly is not wholly understood. It is likely that there is a combination of age related changes which are both local and systemic. In the aged, acral skin is less elastic and shows no diurnal changes (Gniadecka et al 1994). The venoarteriolar vasoconstriction reflex becomes less effective even in healthy elders (Gniadecka et al 1994a). Systemic effects such as age-related deconditioning of mechanoreceptors and osmoreceptors involved in orthostasis (Blomqvist & Stone 1983) are also likely to play a part. Additionally, high temperatures appear to hasten leg swelling, possibly because of the dilatory effect on the circulation. The hospital wards were kept at a minimum of 21°C and the temperatures often exceeded this, especially in summer. This is likely to have made some contribution to leg swelling.

It is notable that although nearly all patients wore elasticated anti-embolic stockings, these could not prevent significant leg swelling. Also notable is that the view that postmenopausal women are particularly prone to leg swelling due to hormonal changes (Kroemer 1971) was not supported by the present study as no difference in leg swelling was found between men and women.

Leg swelling is unsightly which may have negative impacts on patient morale and is known to cause discomfort even in young individuals who experience less swelling than the subjects of the present study (Winkel 1985). The increased weight of the limbs may make mobilisation more arduous and this may have influenced the different rates of mobilisation and levels of fatigue in the LCG and UCG subjects. Extensive oedema of the lower limbs can make it impossible for the patient to wear their ordinary footwear which may further reduce their ability to mobilise (although no incidence of this were recorded in the present study). Gross oedema has also been linked to the development of leg ulcers (Bliss & Schofield 1995) and it is perhaps notable that one subject in the LCG developed a leg ulcer. For the above reasons, the effects of chairnursing on leg oedema need to be studied further and rehabilitation regimes need to be developed that avoid or minimise the formation of leg oedema.

7.1.5 Chest infections
There was no difference in the proportions of patients developing chest infections between the LCG and the UCG. There was close agreement between the two groups pre-operatively both generally and specifically in relation to variables which might directly affect the rate of chest infection such as smoking history, the taking of drugs which can reduce resistance to infection and pulmonary disease. It is, therefore, unlikely that the result could be attributed to baseline imbalances between the two groups rather than to a real lack of difference. The high p value suggests that the likelihood of type II error due to the reduction in power of the study to 73%, as a result of the smaller sample size, is low.

This study does not support the commonly held belief (appendix 1) that sitting in chairs rather than being in bed helps to prevent chest infections. Although immobile LCG patients were more frequently in bed than immobile UCG patients there was not a higher incidence in this group. It is surprising that in this study a significant correlation was not found with respect to smoking history as this has consistently been found to be of significance in previous studies (Dilworth & White 1992). The numbers developing chest infection was small (n = 32) and therefore it is likely that the sample size was too small to identify trends within the group. It is difficult to explain plausibly the increased risk of chest infection in patients not taking drugs which can cause oedema (p = 0.004). Bearing in mind the numbers of factors considered (tables 6.1, 6.2, 6.3 and 6.4) and therefore multiple significance testing, the potential exists that the association may be a chance one.
7.1.6 Pressure sores

There was a significantly higher incidence of pressure sores in the UCG as compared with LCG. This finding is consistent with that of the preliminary work (Gebhardt & Bliss 1994) and a previous study of pressure sores in orthopaedic patients (Bliss et al unpublished data) and with the observations of other workers who compared incidence of pressure sores in chairbound and bedbound patients (Nyquist & Hawthorne 1987, Barbenel et al 1977). However, the low incidence of pressure sores in both arms of the present study (0% and 9% in LCG and UCG respectively) requires some discussion, particularly in relation to the preliminary study where incidence was high (7% and 63% in LCG and UCG respectively).

Part of the explanation probably lies in the less prolonged chairnursing observed in this study and discussed in section 7.1. Development of pressure sores is a time-related event with necrosis occurring or not occurring depending on a complex interplay of tissue resistance, the intensity of the forces applied and the duration. Very long sessions (over 4 hours) were less common in this study as compared with the preliminary study and Bliss et al (unpublished) (8%, 33%[estimated] and 34% respectively). The subjects in this study appeared to be in better health overall than subjects in the preliminary study (eg median Norton scores 19 and 20 in the present and 11 in the preliminary studies) and efforts were made to minimise compressive and shearing forces applied to the buttocks during sitting by providing cushioning and appropriate seating. There was therefore a reduction to all the three critical factors described earlier (section 2.3.7). There may have also been other influences such as differences in surgical and anaesthetic technique resulting in lesser post-surgical sensory impairment and there may have been differences in the way pressure area care was managed.

Both the preliminary study and the present one have established a strong positive association between prolonged chairnursing and the incidence of pressure sores, which is supported by observations of other workers (St Clair 1992, Nyquist & Hawthorne 1987, Barbenel et al 1977). This is likely to pose some ethical dilemmas with regard to further research. Every effort will have to be made to prevent pressure ulceration if patients are subjected to lengthy chairnursing as part of any future study.

It is, perhaps, surprising that development of pressure sores was not positively associated with a low Norton score. However pressure sore prediction scores have been found to be poor predictors of sores in this patient group in other studies (Lewis & Robinson 1996). It is possible that in this group, pressure sores occur due to the process of surgical treatment rather than due to baseline poor health of the patient.

Often when an area of pressure sore risk is highlighted, it is suggested that installing pressure relieving equipment will resolve the problem (Phillips 1998). In this case there is little evidence that this is so. There is no evidence to suggest that cushions do effectively prevent pressure sores (Cullum et al 1995) and they are unlikely to redress any circulatory or postural disadvantages of sitting. On the other hand it has been shown that soft cushions make it more difficult for elderly people to get up from their chairs (Alexander et al 1996). Furthermore, pressure relieving or reducing cushions inherently reduce stability and instability has been linked with discomfort (Branton 1969).

7.1.7 Constipation

Subjects in the UCG were significantly more constipated as compared with the LCG. This finding is consistent with that of the preliminary study (Gebhardt & Bliss 1994, appendix 2) and with the general epidemiology of constipation which is more common among sedentary as opposed to active occupations (Gerhardsson et al 1986). The incidence of constipation was high in both arms of the study (32% and 65%) and this is likely to be a reflection of an elderly population which had been immobilised for at least one or two days completely, then had fairly limited mobility for a period of at least some days and in whom opioids had been extensively used to achieve post-operative analgesia. The constipatory effect of opioids is well known (Canty 1994). The taking of hypotensive drugs and
drugs with potential hypotensive side-effects was also significantly associated with the development of constipation. This may be related to the reduced gut motility associated with hypotension.

The difference in rates of constipation between the two groups may be related to the reduction in blood pressure found in patients who had been chairnursed for prolonged periods of time (table 6.13) and with the general reduction in the splanchnic circulation due orthostasis (section 2.2.1). Constipation in the elderly is not without complications (Clinch & Hilton 1998). It is generally distressing to patients and, as has been discussed previously (section 2.2.8) some complications are potentially fatal. Research is therefore indicated to help develop rehabilitation programmes which minimise constipation and promote good bowel function.

7.1.8 Urinary tract infection (UTI)
There was no difference in the proportions of patients developing UTI between the LCG and the UCG. There was close agreement between the two groups pre-operatively both generally and specifically in relation to variables which might directly affect the rate of UTI such as the taking of drugs which can reduce resistance to infection and general health. It is, therefore, unlikely that the result could be attributed to baseline imbalances between the two groups rather than to a real lack of difference. The high p value (p-value = 0.51) suggests that the likelihood of type II error due to the reduction in power of the study to 73%, as a result of the smaller sample size, is low.

This study does not support the hypothesis that sitting in chairs as opposed to bedrest is likely to prevent UTI since immobile LCG subjects were more frequently in bed than immobile UCG subjects. The only association found in this study was with pre-operative mobility and this may simply reflect the general health status of the patients. Alternatively it may reflect the effects of long term immobility on urinary function such as the formation of renal calculi which in turn predispose to UTI. Again, the chance effect of multiple testing of the sample should not be under-estimated. The finding may be a chance one.

7.1.9 Mobility
Patients in the LCG were able to move out of their chairs independently more quickly than those in the UCG. Since the two groups (LCG and UCG) did not vary significantly in factors likely to affect post-operative mobility it is unlikely that this finding was the result of baseline imbalances. Other factors found to be associated with length of time to independent mobility were the type of operation the patient had undergone and their body build. It is not surprising that type of operation would impact on mobility as different procedures place different limitations on post-operative movement (eg Maher et al 1998) for example in terms of what positions they can adopt to avoid dislocation of a joint prosthesis or how soon certain activities can be undertaken (Charnley 1979). Patients' build may have had varied influences on post-operative mobilisation which may have included the difficulty of assisting obese and overweight patients to rise and transfer from chair to bed and vise-versa in the early stages of post-surgical rehabilitation. Body weight may also have an impact on feelings of fatigue which may affect motivation and performance, however it should be noted that no association was found between body weight and fatigue, elsewhere in this study.

Possible reasons why limiting chairnursing may facilitate earlier attainment of independent mobility include fewer complications such as constipation, less fatigue, more stable (and possibly more effective) circulation (less tachycardia and hypotension). Measurement of mobility should be included in future research as it is an important measure of general progress, to the patient as well as to healthcare professionals. It may be beneficial in future studies to investigate goals other than, or as well as independent rising, such as walking a set distance or being able to perform other activities. Ideally these goals should be representative of goals used in day-to-day clinical practice for each group of patients to enable an assessment of the clinical implications of chairnursing on this aspect of patient recovery.
7.1.10 Discharge

The day on which a patient is discharged home depends on a complex combination of factors. Of these, the medical and physical state of the patient and their fitness for discharge may not be the most critical (Falcone et al 1991). Indeed the pressure on beds in a particular ward or department may also be important (Dawson 1993) and it is notable that length of stay decreased in the research wards during the second half of the study, as the hospital came under great pressure to deal with large numbers of emergencies with fewer beds than had been the case in previous years.

As the complex interactions between social and organisational factors involved in patient discharge had not been accounted for, apart from destination on discharge (which showed no significant difference), the mean earlier discharge of LCG as compared with UCG should be viewed with considerable caution. However, it is not unreasonable to expect patients who suffer fewer complications and mobilise sooner to be medically and physically fit for discharge more quickly.

Again type of operation was found to be a determinant of how soon patients are discharged and again this is not surprising since different surgical procedures are thought to require different post-operative regimes to ensure good prosthetic and tissue union. Mean pre-operative fatigue was the most significant determinant of mean post-operative fatigue. Since post-operative fatigue level is likely to influence the ability of patients to mobilise and progress with rehabilitation this may well explain the significance of pre-operative fatigue level.

7.2 LIMITATIONS OF THE PRESENT STUDY

7.2.1 Multiple outcomes

The findings should be viewed with some caution as the multiple outcomes increase the probability of 'fishing error'. That is, because multiple analyses are carried out on the same data set, the probability of some of these outcomes being significantly different through pure chance is increased. Frequently significance levels are adjusted to compensate for this (Grove & Andreasen 1982). Whether this is appropriate for the testing of multiple 'a priori' hypotheses as opposed to unrelated data is being debated in the medical and epidemiological press (Perneger 1999, Savitz & Olsham 1995, Rothman 1990). One argument is that the data 'says what it says' and needs to be analysed as such. This approach has been taken with regard to the present study. However, over 20 variables were considered when assessing group comparability and when univariately selecting variables for inclusion in the multivariate models used to determine which factors were independently predictive of the development of negative outcomes. Since these outcomes were identified by multiple significance testing without 'a priori' hypotheses, the possibility of any factors thus identified being significant by chance must be considered.

7.2.2 Historical threats to validity

The present study, is of a type (randomisation by ward with crossover of wards mid-way through the study) that is particularly prone to historical threats to validity. These are events which are not related to the planned study but occur during its course. The main threat is to the comparability of the two experimental groups. As has been discussed above (section 7.1), attitudes and clinical opinions of staff are likely to have an effect on patient behaviour and management.

Studies, such as the present one, which compare an experimental regime with an 'ad lib' component (patients were allowed to have unlimited chairnursing in LCG once they became independently mobile) with a wholly ad libitum regime are particularly prone to this. For example, if a ward has the experimental regime and staff who support lengthy chairnursing before crossover and the ad
libitum regime and staff who believe in limited chairnursing after crossover there is likely to be little
difference in the treatment the two groups receive. During the course of this study there were
numerous changes in ward staff on the two wards including one ward manager, 15 staff nurses and
12 other nursing staff and regular coming and going of student nurses. Junior medical staff changed
four times during this time and there were a number of changes in physiotherapy staff. Attempts were
made to minimise these effects by regular updating of staff during the course of the study and
indoctrinating any new staff, particularly on the experimental wards.

Another historical influence on validity is the potential for changes in admission and treatment policy.
For example, to reduce orthopaedic waiting lists, a number of beds were ring-fenced for elective
arthroplasty. This meant that for a period of the study, one arm of the study had a higher proportion
of elective surgical patients than the other, although overall, this did not lead to an significant
imbalance between the two groups. The impact of such changes can be minimised by close liaison
with hospital managers, so that (for example) dedicated beds can be spread equally between the two
study wards rather than be placed on one ward only.

The likelihood of historical threats to validity increases with the length of the study. In a long study
historical threats may also be magnified by the process of maturation - that is the changes that occur
over time due to the researcher and ward staff becoming more familiar and experienced in the process
of research but also potentially more bored, tired and de-motivated. The present study took
significantly longer than originally expected and this should be considered when assessing the clinical
significance of the results. However, analysis did not reveal any significant time-related trends in the
data.

Other potential threats to validity were hypothesis guessing by patients who may have guessed the
research hypotheses and were trying to 'help along' by exaggerating, for example, fatigue when they
had been sitting for long periods. This effect can be minimised by not intimating to the patients the
researcher's expectations of the data as far as possible. Believing that they were expected not to sit
for more than two hours at a time or vice-versa, independently mobile patients may have been
untruthful when asked how long they had been up. To minimise this error, the importance of accuracy
was reiterated to patients at each data collection stage. The expectations of the researcher are also a
biasing factor. The researcher who has a hypothesis is likely to be biased in favour of those
measurements that favour his hypothesis and against those which disprove it. These biases are
minimised by selecting end points which are objective and unambiguous - for example pressure sores
were only counted if they were actual skin breaks greater than 0.5 cm across while 'persistent
erythema' which is open to interpretation was not counted.

7.2.3 Generalisability

The selection of patients to minimise variation between the experimental and control groups inherently
limits generalisability. Thus, the findings of this study can only be applied to orthopaedic patients of
reasonably good health pre-operatively, who are sound of mind and do not suffer from cancer.
Patients with cancer are known to have different patterns of fatigue as compared with those who do
not (Schroeder & Hill 1993). However, only two patients presented with a diagnosis of cancer and
were excluded from this study. Few eligible patients refused admission to the study (n = 7, 3%) and
they did not appear to be demographically or medically different to the experimental population. It
is possible that they were particularly fatigued or feeling unwell, which may have had a bearing on
the outcomes. However small numbers and even distribution between the two groups (3 and 4 in LCG
and UCG respectively) suggests these are unlikely to have influenced the overall findings or their
generalisability.

This study, however, has a generalisability problem common to much research into the care of elderly
patients. Patients were selected who were able to give informed consent and who were alert enough
to participate in the study. This effectively means that demented and confused patients (who are likely
to be the sickest; Norton et al 1962) were excluded. They formed a significant proportion of the overall population that was being studied (patients over 65 undergoing major orthopaedic surgery) \( n = 74, 27\% \). It is possible that these subjects were significantly different in their cardiovascular parameters and behaviour than the subjects who were included.

Demented and confused patients pose a difficult ethical dilemma as they are unable to give informed consent to participate in research. However if they are wholly excluded from research, they are denied evidence-based care and treatment which is considered to be the goal of modern healthcare (NHS Executive 1999). This too can be deemed unethical. In the case of this study, confused/demented patients were excluded because they may have been unable to appropriately self-report fatigue and comply with a limited chair-nursing regime in addition to their inability to give informed consent. However, in confirmatory studies not reliant on self-reporting, attempts should be made to enable the inclusion of this important sub-group.

Another factor limiting generalisability of the results of this study is that it was carried out in a teaching hospital which welcomes research, was willing to part-finance its employee to carry the present study and where staff are used to research being conducted. Other organisations are resistant to the conduct of research. It is recognised that these two types of organisation may vary in important ways (Burns & Grove 1987). This may be particularly true of interventions such as a policy for care. It is possible that institutions which are resistant to the conduct of research may also be resistant to its implementation. The introduction of an effective policy requires the co-operation of both staff and administrators for it to be successful. If one or both are lacking, the policy is not likely to be adhered to and consequently will probably be unsuccessful. This may therefore limit the generalisability of the present findings made in an environment which is committed to examining new strategies, to one that is not.

7.3 FURTHER RESEARCH

One of the aims of this study was to stimulate further research into what should be considered an important area of nursing and medical practice, but which has hitherto received scant attention. The importance stems from the possibility that patients are needlessly subjected to management which may cause discomfort and distress or even dangerous complications and that better knowledge may lead to better health outcomes for patients and savings in healthcare resources.

Factors to be considered when conducting confirmatory studies have already been discussed individually for each of the main outcomes considered in this study. In general it is recommended that such studies seek one main outcome or possibly combined outcomes (to enable appropriate sample selection and detailed analysis of associated variables) and are carried out on sites where prolonged chairnursing is regularly practised. Data should be collected by individuals who have no managerial or clinical authority over the staff of the research institution to limit bias. Sites with a high throughput of suitable subjects should be used where possible, in order that historical and maturation threats to validity are minimised.

A wide range of types of patient should be studied to increase generalisability of findings. In particular patients who are immobile for long periods of time should be studied as compared with those with fairly short loss of mobility such as the majority of patients who were subjects in the present study. Individuals who spend much time sitting such as wheelchair-bound paraplegics or some nursing home residents may develop adaptive mechanisms for coping with prolonged orthostatic stress and immobility and thus be significantly different. Such adaptations may also shed light on the processes that occur in individuals without such coping mechanisms. There are also other outcomes which have not been addressed in this study but which deserve attention.
The incidence of deep vein thrombosis (DVT) was discussed in the literature review. The particular significance of this complication is that it can lead to fatal pulmonary embolism. Its post-operative incidence is likely to have been reduced in recent years due to extensive implementation of preventative therapy such as heparinisation and the use of anti-embolic stockings (Planes et al 1998). However, because of its life-threatening nature, even small further reductions in incidence of DVT are worth pursuing. Early mobilisation or ambulation has long been advocated as an effective preventative intervention (Newburger 1943). Despite this, there are very little data available on the effectiveness of this strategy or how it should be implemented (Pauschert et al 1998). Lengthy chairnursing appears unlikely to be very helpful and it is suggested may even promote DVT rather than prevent it (Kakkar, cited in Bliss 1992, Wells 1979).

It was not possible to study the incidence of DVT as part of the present study because funding was not available for the laboratory blood tests necessary to screen subjects for the presence of DVT indicators. Because of the significance of this complication, it seems important that studies of mobilisation regimes be carried out with DVT as the main outcome.

It has been suggested that urinary problems associated with bedrest such as bladder and urethral stones and urinary tract infection (UTI) may be partially due to reduced effectiveness of kidney and bladder drainage due to horizontal posture (Olson & Schroeder 1967). Should this assumption prove correct, lengthy chairnursing may be beneficial since it would maintain the kidneys and bladder in a more advantageous vertical position. On the other hand chairnursing tends to reduce urinary output (Guite et al 1988) due to orthostasis (section 2.3.9). There is no evidence from the present study that chairnursing does prevent UTI, even though patients in the LCG spent more time in bed prior to achieving independent mobility than UCG patients. To create a fuller understanding of the processes which take place under different mobilisation regimes studies could be carried out to examine diurnal variations in urine output, blood chemistry and the deposition of renal calculi using various imaging techniques.

Bone demineralisation occurs during bedrest and this is thought to occur because of reduced stresses and strains put on the bones. It is highly likely that different mobilisation regimes may influence this process by putting different types of stresses and strains on the skeleton. As many patients, especially elderly females, are often already suffering from osteoporosis to some degree, it is important to design mobilisation protocols which minimise further loss. Traditionally bone demineralisation has been measured by analysing mineral content of urine, which is a convenient, non-invasive technique.

The design of the present study did not allow the establishment of a causal relationship between time chairnursed and pulse rate, blood pressure, lower leg swelling and fatigue. For an understanding of these factors, there needs to be a development of models of physiological response to chairnursing, to guide practice and further outcomes research. Such models could be developed from investigations of changes in parameters of interest (such as oxygen tension, pulse, blood pressure, fatigue) over time, probably along the lines proposed by Mynster et al (1996). However, such studies need to observe patients over long periods of continuous sitting representative of usual chairnursing sessions, rather than the 10 minute observation periods of Mynster et al's actual study.

With respect to other research, Lassen & Borris (1991) pointed out on the basis of their findings of significant effect of early as compared to late mobilisation on the development of DVT, that mobilisation regimes are likely to be a confounding factor in studies of DVT prevention, if they are not properly controlled for. The findings of the present study suggest that this may also be true of studies which investigate many other parameters such as post-operative fatigue, bowel function, the circulatory system, pressure sore development, length of hospital stay and others. It is recommended that chairnursing be carefully controlled in any clinical studies where the outcome is likely to be affected by cardiovascular events.
Finally, there appears to be a strong association between fatigue and low mood in patients subjected to lengthy chairnursing. For example many patients recall being tearful "I was reduced to tears" (Dzielski 1999) and "I was so utterly exhausted I wanted to cry" (Gebhardt & Bliss 1994) while Dzielski (1999) says she thinks of chairnursing "with great unhappiness". Because of the possible influence on fatigue and mood of central control mechanisms (section 2.3.1) this area may also repay careful psychological as well as physiological research. It is possible that this research has the potential to lead to better management of post-operative rehabilitation which may improve patients' mood as well as physical well-being, thereby increasing motivation for engaging in the rehabilitative process and possibly increasing the rate of progress and achieving better final outcomes.

7.4 CONCLUSIONS

The null hypotheses (chapter 4) with respect to the incidence of tachycardia, lower limb oedema, severe fatigue, pressure sores and constipation, the time to independent mobilisation, time to discharge and mean pulse rate, blood pressure and lower leg volume were refuted. The null hypotheses regarding hypotension, chest infection and UTI were upheld.

In simple terms, if chairnursing was limited, patients appeared to 'do better' in a number of aspects and to go home sooner than patients allowed ad libitum chairnursing. Nor did patients in the LCG do worse in any other aspect studied. However, as is probably often the case when entering new avenues of research, more questions are raised than answered. Primarily, as little research of this type has been attempted in the past, it is difficult to determine if these findings are representative of the study population or indicative of some extreme of the spectrum. This limits the current clinical applicability of the results. Other questions relate to the generalisability of the findings to patient groups other than elderly orthopaedic, the impact of variables which have not been considered in the present study, the precise mechanisms involved and whether the findings represent general trends among the majority of patients or the mean results for different sub-groups which respond differently to the challenge of prolonged chairnursing.

Further research should be aimed at confirming or refuting the present findings, determining physiological models on which to base further clinical research and conducting investigations of the effects of chairnursing on other important outcomes such as deep vein thrombosis, uro-lithiasis, bone demineralisation and tissue oxygenation. These investigations should be extended to groups of patients other than the post-operative elderly to determine generalisability of the findings. Mobilisation regimes should also be considered as potential confounding factors in the study of other aspects of post-operative recovery.

The results of this study do not support the practice of lengthy chairnursing. It is recommended that, until the issues raised by this study and by the literature review are resolved, elderly post-operative orthopaedic patients be nursed for no more than two hours per session if they are unable to mobilise independently. Otherwise, it is probably best to allow the patients to choose for themselves how long they sit. After all that is what we would do for ourselves, given the option.
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APPENDIX 1

Nurses’ views on mobilisation and chairnursing: a questionnaire

Sample
In 1993, a convenience sample of 21 nurses on two orthopaedic wards was asked to complete a questionnaire on their views of mobilisation and chairnursing. The sample consisted of 21 subjects (2 ward sisters, 12 staff nurses, 4 student nurses and 3 healthcare assistants). 5 subjects had under 3 years’ nursing experience, 9 had 3-5 years’ and 7 had over 5 years’ nursing experience. However, only one had worked more than 2 years on their current ward. Only 5 had attended post-graduate training on pressure sore prevention and of those only three within the last year.

Questionnaire
The subjects were asked whether they agreed, disagreed or neither agreed nor disagreed with the following statements:

1. early mobilisation prevents the following complications: deep vein thrombosis (DVT), pulmonary embolism (PE), chest infection, pressure sores, constipation;
2. chairnursing prevents the following complications: DVT, PE, chest infection, pressure sores, constipation;
3. chairnursing is equivalent to mobilisation;
4. it is safe for a patient at risk of pressure sores to sit in a chair for the following lengths of time: half an hour, up to an hour, 1-2 hours, 2-3 hours etc up to 8 hours or more;
5. patients at risk of pressure sores on this ward sit out longer than is ‘safe’.

Subjects who agreed with 5 were asked to explain what the reason was in their view.

All subjects were asked to grade a series of 12 tasks according to priority with 1 having the highest priority and 12 the lowest.

Results
18, 15 and 19 subjects agreed that ‘early mobilisation’ prevents DVT, PE, and chest infection respectively. 3, 6 and 2 respectively had no view. All subjects agreed that early mobilisation prevented pressure sores and constipation.

The numbers agreeing/disagreeing neither agreeing nor disagreeing for question 2 were as follows: DVT 7/7/7, PE 5/5/8, chest infection 12/3/6, pressure sores 7/9/5, constipation 4/10/7.

The majority (15) believed that chairnursing was not equivalent to mobilisation, with 3 agreeing that it was and three not expressing a view.

None of the sample indicated that they believed that it was safe for patients at risk of pressure sores to sit in a chair for more than 2 hours. 1 subject did not complete this part of the questionnaire.

The majority (14) agreed that patients at risk of pressure sores did sit out longer than it was safe, only one disagreeing and 6 not expressing a view. The most common reason given for this was shortage of staff (13). Other reasons were patients’ request (2) and varying priorities, forgetfulness and lack of knowledge among nurses (1). 7 subjects did not offer a view on this matter.
The 12 tasks were prioritised overall as follows:

1. resuscitate a patient who has had a cardiac arrest;
2. carry out post-operative observation on a patient who has just arrived from surgical theatre;
3. give commode to patient requesting it;
4. put patient at risk of pressure sores back to bed after 4 hours in a chair;
5. carry out post-operative observation on a patient 2 hours after arrival from surgical theatre;
6. drug round;
7. put patient at risk of pressure sores back to bed after 2 hours sitting in a chair;
8. answer telephone;
9. attend consultant ward round;
10. carry out wound dressing;
11. bedbath a patient;
12. make bed ready for an admission.

Discussion

The questionnaire confirmed the view that most nursing staff believed that early mobilisation of patients prevented the whole range complications presented here. It was curious to note that 3 staff believed early mobilisation prevented DVT but not PE. The views on chairnursing were more mixed with the majority either not believing that chairnursing was effective or not expressing an opinion. Only in the case of chest infection, did a narrow majority believe that chairnursing prevented this complication. Few believed that chairnursing was equivalent to mobilisation and all believed patients who are at risk of pressure sores should sit in chairs for no more than 2 hours and generally less.

However, the majority believed that patients did sit out longer than was 'safe' for their pressure areas and they felt this was due mainly to insufficient staff to help the patients walk or get back into bed. In terms of priorities most agreed that the priorities given in the list were perhaps idealised, and in practice if in fact the telephone was ringing it would be answered and the consultant would take priority if he or she turned up. Therefore it was easy to be distracted from moving a patient from his or her chair at the allotted time, and very easy for time to pass by quickly so that the patients often spend many more hours in chairs than intended.

Conclusions

There was evidence that nurses believed early mobilisation was beneficial for the prevention of post-operative complications. Few believed that chairnursing was equivalent to mobilisation and there was some uncertainty about its relative merits. The majority view seemed to be that it occurs primarily due to inadequate resources to carry out active mobilisation which would be preferable.
APPENDIX 2

chairnursing detrimental? Journal of Tissue Viability; 4: 51-54
Preventing Pressure Sores in Orthopaedic Patients - is Prolonged Chair Nursing Detrimental?

Krzystof Gebhard1, Mary R Bliss2

1Research Nurse, 2Consultant Geriatrician, Department of Medicine for the Elderly, Homerton Hospital, London

Many patients spend a long time each day in chairs. The results of the study show that the development of pressure sores is strongly correlated with the length of individual periods of time spent sitting. The study recommends that patients should spend less than two hours sitting at each chair nursing session.

Summary

A randomised, controlled study to compare two types of pressure relieving support for preventing pressure sores - alternating pressure and constant low pressure - showed less benefit from alternating pressure mattresses in orthopaedic compared with intensive care patients. Discriminant function analysis suggested that this might be due to the greater amount of time which the orthopaedic patients were being nursed in chairs, 68% days compared with the intensive care patients' 14% days. A crossover study was therefore carried out between two orthopaedic wards to see if limiting chair nursing in elderly patients >65 years for the first two weeks postoperatively was associated with a reduction in the incidence of pressure sores. All the subjects had large celled alternating pressure mattresses on their beds. In one ward chair nursing was limited to 2 hours per session (with more than 1 session per day if desired) and in the other, the amount of time the patients spent up was decided by the ward staff.

The median hours of chair nursing per session in the limited and unlimited groups was 2 (range 0.25-12.0) and 6 (range 0.25-15.25) with the incidence of sores being 7% and 63% respectively (p=0.001, 95% CI of the difference -77 to -36%). Apart from one patient in the limited chair nursing group who developed a pulmonary embolus whilst still on bedrest postoperatively, no patient showed clinical signs of venous thrombosis. Patients in the limited group had fewer chest infections (0:3), a lower incidence of constipation (10:16, p=0.05) and more were independently mobile before the end of the trial than in the unlimited group (17:9).

Chair nursing for more than two hours per session seems likely to cause pressure sores, and perhaps to exacerbate other post operative complications and delay mobility in elderly orthopaedic patients.

Introduction

In 1990-92, a randomised controlled trial was carried out to compare the efficacy, practicability and cost of the two basic types of pressure relieving support - alternating pressure (AP) and constant low pressure (CLP) - for preventing pressure sores in a district general hospital14. Mattresses and overlays were assigned to one or other of the two groups according to their principal mode of action and placed in three cost bands according to purchase price. Newly admitted patients aged over 18 years with a Norton risk assessment score1 of <14 and without existing sores were randomly allocated to AP or CLP supports according to the last digit of their case notes number and given a mattress from the cheapest cost band suitable for their weight chosen by a table of random numbers. They were then assessed twice weekly until: their condition improved so that they were no longer at risk of developing sores; they were discharged/ transferred to another hospital; they died. Apart from the support, all other aspects of the patient's care - including time spent out of bed - were decided by the ward staff. If during the course of the trial the pressure areas showed persistent erythema indicating tissue damage, or a break in the skin, the mattress was changed for a more sophisticated type in a higher cost band in the same group.

Each mattress change therefore indicated a failure of the support group. However it was noted that results varied in different types of patient. In the orthopaedic wards 15.9% of patients developed sores on AP and 26.7% on CLP supports, whilst in the intensive care unit only 4.3% developed sores on AP compared with 55% on CLP supports. This was particularly surprising as the orthopaedic patients were less ill than the intensive care patients as shown by their median Norton Scores, 11 and 5, and the death rate, 13.5% and 25.6% respectively. Eighty four percent of the intensive care patients were unconscious on entry to the trial and almost all were receiving inotropes likely to increase peripheral vasoconstriction and susceptibility to pressure necrosis4.

The principal difference in management observed between the two groups of patients was the time spent out of bed. Eighty seven percent of the orthopaedic patients spent a mean 7.6 hours/day in chairs, compared with 40% of intensive care patients, mean 3.4 hours/day (the rest remained in bed). Discriminant function analysis in the orthopaedic patients showed that those who developed sores tended to spend longer periods sitting in chairs during the first 14 days of the trial than those who did not (coefficient 1.03).
As a result of these observations the following study was
carried out to see if limiting chair nursing reduced the incidence
of pressure sores.

Method
The study was conducted in two orthopaedic wards, 1 and 2,
which were identical in design and shared the same consultants.
The only difference in policy was that ward 2 had the benefit of
a consultant geriatrician to assist with medical management
and discharge planning of elderly patients; however, since even
daily discharge took place more than 2 weeks postoperatively,
this was unlikely to seriously affect the trial. All patients >65
years who had had a fracture or major orthopaedic surgery of the
pelvis, hip or lower limb within the previous four days were
recruited to the trial and given a large celled AP mattress.

In the experimental ward, chair nursing was restricted to two
hours per session. There was no restriction on the number of
sessions per day but patients had to have at least two hours in
or on the bed between sessions. Patients in the control ward
were nursed according to the normal routine of that ward with
the amount of time they spent in chairs decided by the ward
staff. All patients were allowed physiotherapy and encouraged
to walk with assistance if possible. The times that the patients
were sat out in chairs and put back to bed were recorded by the
staff with the assistance of the researcher.

The patients' Norton scores, mental and physical condition,
drug therapy and pressure areas were assessed on admission
and then every 3-4 days. Pressure sores were defined as actual
skin breaks. Patients remained in the trial for two weeks or until
they could walk independently; they were discharged/transferred
to another ward; they died. No patient entered the study more
than once.

A power calculation (80% power, 5% significance level) based
on the results of the previous study and allowing for 10% drop out,
suggested 114 subjects would be needed in each group to
detect a difference of 20% from a base of 5%. Records of the
patients admitted to the orthopaedic wards in 1992 showed that
a total of 8 months would be necessary to achieve this sample
size. To obviate differences in ward management, a crossover
between the experimental and control wards was planned after
4 months with an intervening month's adjustment period.

Analysis
The results were analysed to assess the comparability of the two
patient groups and any association between the number of chair
hours and the development and progress of pressure sores and
other complications. Demographic, health characteristics and
treatment variables were compared using Chi square test for
categorical variables; t-tests for normally distributed and the
Mann Whitney test for non-normally distributed continuous
data. Significant differences were accepted at the p=0.05 level.

Results
Analysis of the data after three months showed that the difference
in the incidence of pressure sores between the limited and
unlimited groups was almost three times greater than expected,
so on ethical grounds it was decided to reduce the study period
to 6 months, i.e. 3 months followed by crossover, a month's
adjustment period, and another 3 months' data collection (7
months in total).

Fifty seven patients were recruited to the trial, 30 in the limited
and 27 in the unlimited group with more patients being admitted
to ward 2 in both groups (Table 1), possibly due to the
consultant geriatrician input.

The number of days spent on full bedrest was similar in the
limited and unlimited groups, mean 2.9 (SD±2.3) and 3.2 (SD±
3.0) days respectively. However, on the days when patients sat
out, 83% of sessions in the limited group were restricted to two
hours or less and only 8% lasted longer than 3 hours. In contrast,
in the unlimited group, only 18% of chair nursing sessions were
for under 2 hours and 73% were for more than 3 hours, including
21% for over 9 hours. By far the majority of the unlimited
patients were got up for long periods once daily, compared with
patients in the limited group who on 16% of days got up twice
and on 14% between 3 and 8 times. The median length of chair
nursing session in the limited and unlimited groups was 2
(range 0.25-12.0) and 6 (range 0.25-15.25) hours respectively.

Table 2 shows the characteristics of the patients in the two
groups. There was no significant difference in the patients'
mean ages, sex or physical or mental state. However, 7%
patients in the limited chair nursing group developed pressure
sores compared with 63% in the unlimited group (p <<0.001.
95% CI of the difference: -77 to -36%) (Table 3).

Discussion
When a regime departs from usual clinical practice it is inevitable
that despite the best efforts of the researcher and ward staff, it
will not be adhered to at all times. Some patients in the
experimental wards sat out for longer than two hours, eg. two
demented patients who refused to comply with the limited
regime and accounted for 31% of sessions longer than 2 hours.
52% longer than 3 hours and all sessions longer than 6 hours.
No patients in either group who sat out for <3 hours per session
developed sores.

The results of this study accord with those of numerous surveys
which have shown that patients being nursed in chairs are
more likely to have sores than patients in bed. Although the
sores here were small (mean maximum diameter 1.4cm), had it
not been for the presence of the researcher and the raised
awareness of pressure area care generated by the study, it is
likely that at least some would have deteriorated further. Even
the smallest sores were often very painful - 'My arse is sore as
hell!'.

Chair nursing is thought to prevent other complications in
fracture/postoperative patients besides pressure sores,
particularly venous thrombosis, chest and urinary tract infections
and constipation. Our observations did not support this. No
patient in either group showed clinical evidence of a deep vein
thrombosis, and although one patient in the limited chair nursing group developed a pulmonary embolus, this was whilst she was still on complete bedrest postoperatively. About half of the patients in both groups were receiving anticoagulants. No patient in the limited chair nursing group developed a chest infection compared with 3 in the unlimited group. Incidences of urinary tract infection were similar, 10% and 11% respectively, but patients in the limited group were significantly less constipated than those in the unlimited group (33 and 59%; p=0.05). In addition, despite the fact that the limited group had more patients with cardiovascular disease, ankle and foot oema occurred almost exclusively in the unlimited chair nursing patients (3 and 26%; p=0.02). One patient in the unlimited group had swelling to the knees and two patients were unable to wear their slippers which may have delayed mobilisation. Sixteen (53%) patients in the limited group became independently mobile before the end of the trial compared with only 9 (33%) in the unlimited group although this was not statistically significant (p=0.07).

### Table 1. Patients and chair nursing

<table>
<thead>
<tr>
<th>Patients</th>
<th>Limited</th>
<th>Unlimited</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>total</td>
<td>30</td>
<td>27</td>
<td></td>
</tr>
<tr>
<td>Ward 1</td>
<td>11</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Ward 2</td>
<td>19</td>
<td>17</td>
<td></td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Days in trial</th>
<th>Limited</th>
<th>Unlimited</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>total</td>
<td>294</td>
<td>320</td>
<td></td>
</tr>
<tr>
<td>in chair (%)</td>
<td>206 (70)</td>
<td>248 (77)</td>
<td></td>
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</table>

<table>
<thead>
<tr>
<th>Chair sessions</th>
<th>Limited</th>
<th>Unlimited</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>total</td>
<td>327</td>
<td>260</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>no per day (%)</td>
<td>153 (74)</td>
<td>236 (95)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>28 (14)</td>
<td>12 (5)</td>
<td></td>
</tr>
<tr>
<td>3-8</td>
<td>25 (12)</td>
<td>0 (0)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>hours per session (%)</th>
<th>Limited</th>
<th>Unlimited</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤2</td>
<td>272 (83)</td>
<td>47 (18)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>&gt;2-3</td>
<td>28 (9)</td>
<td>22 (9)</td>
<td></td>
</tr>
<tr>
<td>&gt;3-6</td>
<td>14 (4)</td>
<td>71 (27)</td>
<td></td>
</tr>
<tr>
<td>&gt;6-9</td>
<td>4 (1)</td>
<td>66 (25)</td>
<td></td>
</tr>
<tr>
<td>&gt;9-12</td>
<td>9 (3)</td>
<td>51 (20)</td>
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<tr>
<td>&gt;12</td>
<td>3 (1)</td>
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</tr>
</tbody>
</table>

| median hours per patient per session (range) | 2 (0.25-12.0) | 6 (0.25-15.25) |

### Table 2. Patients' characteristics

<table>
<thead>
<tr>
<th></th>
<th>Limited</th>
<th>Unlimited</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total patients</td>
<td>30</td>
<td>27</td>
</tr>
<tr>
<td>Age in years mean (range)</td>
<td>78 (65-94)</td>
<td>79 (65-92)</td>
</tr>
<tr>
<td>Sex: male/female</td>
<td>5/25</td>
<td>4/23</td>
</tr>
<tr>
<td>Build: no (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>emaciated</td>
<td>4 (13)</td>
<td>5 (19)</td>
</tr>
<tr>
<td>average</td>
<td>11 (37)</td>
<td>10 (37)</td>
</tr>
<tr>
<td>obese</td>
<td>15 (50)</td>
<td>12 (44)</td>
</tr>
<tr>
<td>Grip strength in Kg mean (range)</td>
<td>13.1 (0-30)</td>
<td>13.0 (5-28)</td>
</tr>
<tr>
<td>Smokers: no (%)</td>
<td>4 (13)</td>
<td>3 (11)</td>
</tr>
<tr>
<td>Mental Test Score ≤8: no (%)</td>
<td>9 (30)</td>
<td>8 (30)</td>
</tr>
<tr>
<td>Norton Score ≤ median (range)</td>
<td>11 (7-16)</td>
<td>11 (7-16)</td>
</tr>
<tr>
<td>Associated diagnoses: no (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arthritis</td>
<td>13 (43)</td>
<td>11 (41)</td>
</tr>
<tr>
<td>Circulatory disease</td>
<td>7 (23)</td>
<td>2 (7)</td>
</tr>
<tr>
<td>Bowel disorders</td>
<td>2 (7)</td>
<td>2 (7)</td>
</tr>
<tr>
<td>Dementia/confusion</td>
<td>4 (13)</td>
<td>3 (11)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1 (3)</td>
<td>3 (11)</td>
</tr>
<tr>
<td>Infections</td>
<td>1 (3)</td>
<td>3 (11)</td>
</tr>
<tr>
<td>Breast cancer</td>
<td>1 (3)</td>
<td>1 (4)</td>
</tr>
<tr>
<td>Surgery - acute</td>
<td>18 (60)</td>
<td>18 (67)</td>
</tr>
<tr>
<td>planned</td>
<td>12 (40)</td>
<td>9 (33)</td>
</tr>
<tr>
<td>Catheterised</td>
<td>16 (53)</td>
<td>14 (52)</td>
</tr>
<tr>
<td>Anticoagulated during trial</td>
<td>14 (47)</td>
<td>14 (52)</td>
</tr>
</tbody>
</table>

Table 2. Patients' characteristics
Patients nursed for long periods in chairs frequently complained of exhaustion, eg.

'I was out too long. I couldn't get back into bed quick enough. I was so utterly exhausted I wanted to cry.'

'I was up all day yesterday - it wore me out.'

In contrast, some of the patients on limited chair nursing became very active. One got in and out of bed 8 times a day. They did not feel that this was difficult, some seeing it as 'breaking up the monotony of the day' and 'good exercise'.

**Conclusion**

Elderly orthopaedic patients who were supplied with large celled alternating pressure mattresses on their beds and in whom chairnursing in the early postoperative period was restricted as far as possible to <2 hours per session, rarely developed pressure sores compared with similar patients who sat out for longer periods. They also suffered from fewer chest infections, were less constipated and appeared less fatigued and more active and motivated. They mobilised earlier than patients treated with prolonged chair nursing. We therefore recommend that chairnursing should be limited to two hours/session in the first week after fracture/surgery in elderly patients. They also suffered from fewer chest infections, were less constipated and appeared less fatigued and more active and motivated. They mobilised earlier than patients treated with prolonged chair nursing. We therefore recommend that chairnursing should be limited to two hours/session in the first week after fracture/surgery in elderly patients. Patients who are fit should be encouraged to get in and out of bed as frequently as they wish. Ideally sitting out should involve other activities, eg, a meal, watching television, seeing visitors etc. Patients who are not ambulant should be put back to bed if they request it, and in any case after no more than three hours. Nursing patients for long periods out of bed against their wishes is inhumane and likely to increase the risk of pressure sores.

**Acknowledgements**

We would like to thank the nursing staff of the orthopaedic wards, Homerton Hospital, London, for their kind cooperation in carrying out this study, Talley Group Ltd. for the loan of alternating pressure mattresses, and Miss Janice Thomas, Computer Services Department, St. Bartholomews Hospital Medical College, London and Dr Michael Clark for help with the analysis. This and related studies have been made possible by means of generous grants from the Locally Organised Research Scheme of the North East Thames Regional Health Authority.

**Address for correspondence.**

Mr K Gebhardt, Department of Medicine for the Elderly, Homerton Hospital, Homerton Row, London E9 6SR

**References**

APPENDIX 3

Subjects’ consent form, for agreement to participation in the trial
ST GEORGE'S HEALTHCARE

NURSING RESEARCH/ETHICS COMMITTEE

SUBJECT CONSENT FORM
Form of consent to participation in nursing research
for use by patients/clients/volunteers

1. Brief Title of Project

Chair nursing outcomes project

2. Patient Information (must be in lay language)

As part of your rehabilitation following surgery you will be asked to sit in a chair. We are trying to find out if sitting for short periods at a time (up to 2 hours) or for longer periods is better. I would therefore like you to sit in your chair for no longer than 2 hours at a time until you can walk by yourself. You may sit out as often as you wish, so long as you spend an hour in between lying down or walking. I would also like to ask your permission to record some details about yourself, your medical condition and how long you spend in your chair. I would also like to measure your pulse, blood pressure and the diameter of your lower legs 4 times on some days during your stay and to ask you to record how fatigued you are on special charts on those days. Once all the data about you has been collected your data set will be made anonymous. No one apart from the research team will know that it has come from you. Your treatment will not be affected in any way as a result of this study.

Signed by the person in charge of the project:

Signature: [Signature] Date: 2/7/96

The District Nursing Research/Ethics Committee has approved the above statement. Written/Verbal-consent is required for this project.

Signed by the Chairperson of that committee:

Signature: [Signature] Date: 3/July 96
Form of consent to participation in nursing research for patients/clients/volunteers: (*) Delete as appropriate).

I ________________________________ of ________________________________

______________________________________________________________

agree to participate in the nursing research described overleaf. The nature, purpose and possible consequences of the procedures involved, summarised overleaf, have been explained to me by ______

____________________ and are acceptable to me.

I understand that I am entering this project of my own free will and am free to withdraw at any time, without necessarily giving any reasons. In addition, my participation or non-participation in this project will in no way affect the care that I will receive.

Signed: (* Patient/client/volunteer) ________________________________

Witnessed by: ________________________________

Date: ________________________________

Hard Disk: Mins/Ethics
Ethics96
APPENDIX 4

Visual Analogue Scale (not to scale)

FATIGUE ASSESSMENT

Subject number _________  Day ____  Assessment ____

Directions:

Please place a horizontal mark through the vertical line. This horizontal mark should be placed at the exact spot on the line which best indicates the degree to which you are experiencing the feeling.

How much fatigue are you experiencing at the moment?

A GREAT DEAL OF FATIGUE

MODERATE FATIGUE

SLIGHT FATIGUE

NO FATIGUE
APPENDIX 5

Data collection proforma
CHAIR NURSING OUTCOMES STUDY

SUBJECT PROTOCOL

Case number: 

Regime: Limited chairnursing/Unlimited chairnursing

Pre-study screen:

Patient over 65
Major surgery of lower limb
Is not para/tetraplegic
Mental test score >8
Consent obtained
PR mattress supplied
Chair cushion available if UCN
Nurse information sheet complete
Bed labels attached

Protocol

Pre-operative assessment
Operation date
day 1
Post operative assessment 1
day 3
Post operative assessment 2
day 5
Post operative assessment 3
day 7
Post operative assessment 4
day 10
Post operative assessment 5
day 14

Post-study checklist
Bed labels removed
Cushion retrieved
Follow up data complete

Comments:

Name: ____________________________
Ward: ____________________________
PRE-OPERATIVE ASSESSMENT - GENERAL

Subject number

Regime (1 limited 2 unlimited)

Ward (1 Holdsworth 2 Gunning 3 Keate)

Surgery (1 elective 2 emergency)

Type of surgery (c/list 1)

Age

Sex (1 male 2 female)

Other pathology (c/list 2)

Pre-operative mobility (1 fully ambulant 2 limited 3 chair/wheelchair bound 4 bedbound)

Smoking (1 never 2 no >two months 3 no <two months 4 yes)

Weight (kg)

Height (cm)

Build (1 cachectic 2 underweight 3 average 4 overweight 5 obese)

Haemoglobin (Hb)

Initial Norton score - general condition
  mental state
  activity
  mobility
  incontinence
  TOTAL

TED stockings (1 yes 2 no) 16†
PRE-OPERATIVE ASSESSMENT - DAILY

Chest infection (1 yes 2 no) [definition c/list 3]

Pressure sores: present (1 yes 2 no)
   widest diameter (cm)
   grade [definition c/list 4]

Independently mobile (1 yes 2 no) [definition c/list 5]

Drug therapy

Drugs taken which might affect fatigue
(1 yes 2 no) [see c/list 6]

Drugs taken which might affect mobility
(1 yes 2 no) [see c/list 7]

Drugs taken which might affect BP and pulse
(1 yes 2 no) [see c/list 8]

Fatigue

2.7 What was most tiring today? [see c/list 9]

2.8 What would relieve tiredness [see c/list 10]
9-10 am

<table>
<thead>
<tr>
<th>Time up before assessment</th>
<th>1 in bed</th>
<th>1-2 hrs</th>
<th>2</th>
<th>1-2 hrs</th>
<th>3</th>
<th>2-4 hrs</th>
<th>4</th>
<th>4-6 hrs</th>
<th>5</th>
<th>6-8 hrs</th>
<th>6</th>
<th>8hrs +</th>
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<tr>
<td>Diastolic blood pressure</td>
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<td></td>
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</tr>
</tbody>
</table>

| Operated limb: h (cm)     |         |        |   |         |   |         |   |         |   |         |   |        |
| Diameter at ankle (cm)    | ___     |        |   |         |   |         |   |         |   |         |   |        |
| Diameter at widest point of calf (cm) | ___ |        |   |         |   |         |   |         |   |         |   |        |
| Calf volume (cc)          |         |        |   |         |   |         |   |         |   |         |   |        |

| Unoperated limb: h (cm)   |         |        |   |         |   |         |   |         |   |         |   |        |
| Diameter at ankle (cm)    | ___     |        |   |         |   |         |   |         |   |         |   |        |
| Diameter at widest point of calf (cm) | ___ |        |   |         |   |         |   |         |   |         |   |        |
| Calf volume (cc)          |         |        |   |         |   |         |   |         |   |         |   |        |

Fatigue self assessment
Description of fatigue (own words)

12-1 pm

<table>
<thead>
<tr>
<th>Time up before assessment</th>
<th>1 in bed</th>
<th>1-2 hrs</th>
<th>2</th>
<th>1-2 hrs</th>
<th>3</th>
<th>2-4 hrs</th>
<th>4</th>
<th>4-6 hrs</th>
<th>5</th>
<th>6-8 hrs</th>
<th>6</th>
<th>8hrs +</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulse rate</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Systolic blood pressure</td>
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<td></td>
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<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
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<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

| Operated limb: h (cm)     |         |        |   |         |   |         |   |         |   |         |   |        |
| Diameter at ankle (cm)    | ___     |        |   |         |   |         |   |         |   |         |   |        |
| Diameter at widest point of calf (cm) | ___ |        |   |         |   |         |   |         |   |         |   |        |
| Calf volume (cc)          |         |        |   |         |   |         |   |         |   |         |   |        |

| Unoperated limb: h (cm)   |         |        |   |         |   |         |   |         |   |         |   |        |
| Diameter at ankle (cm)    | ___     |        |   |         |   |         |   |         |   |         |   |        |
| Diameter at widest point of calf (cm) | ___ |        |   |         |   |         |   |         |   |         |   |        |
| Calf volume (cc)          |         |        |   |         |   |         |   |         |   |         |   |        |

Fatigue self assessment
Description of fatigue (own words)
3-4 pm

Time up before assessment
(1 in bed 2 1-2 hrs 3 2-4 hrs 4 4-6 hrs 5 6-8 hrs 6 8 hrs+

Pulse rate

Systolic blood pressure

Diastolic blood pressure

Operated limb: h (cm)
Diameter at ankle (cm)
Diameter at widest point of calf (cm)

Calf volume (cc)

Unoperated limb: h (cm)
Diameter at ankle (cm)
Diameter at widest point of calf (cm)

Calf volume (cc)

Fatigue self assessment
Description of fatigue (own words)

6-7 pm

Time up before assessment
(1 in bed 2 1-2 hrs 3 2-4 hrs 4 4-6 hrs 5 6-8 hrs 6 8 hrs+

Pulse rate

Systolic blood pressure

Diastolic blood pressure

Operated limb: h (cm)
Diameter at ankle (cm)
Diameter at widest point of calf (cm)

Calf volume (cc)

Unoperated limb: h (cm)
Diameter at ankle (cm)
Diameter at widest point of calf (cm)

Calf volume (cc)

Fatigue self assessment
Description of fatigue (own words)
POST-OPERATIVE ASSESSMENT 1

Chest infection (1 yes 2 no) [definition c/list 3]

Pressure sores: present (1 yes 2 no)
  widest diameter (cm)
  grade [definition c/list 4]

Independently mobile (1 yes 2 no) [definition c/list 5]

Drug therapy

Drugs taken which might affect fatigue
(1 yes 2 no) [see c/list 6]

Drugs taken which might affect mobility
(1 yes 2 no) [see c/list 7]

Drugs taken which might affect BP and pulse
(1 yes 2 no) [see c/list 8]

Haemoglobin level (Hb)

Fatigue
  2.7 What was most tiring today? [see c/list 9]
  2.8 What would relieve tiredness [see c/list 10]
### 9-10 am

**Time up before assessment**: (1 in bed 2 1-2 hrs 3 2-4 hrs 4 4-6 hrs 5 6-8 hrs 6 8hrs+)

<table>
<thead>
<tr>
<th>Pulse rate</th>
<th>Systolic blood pressure</th>
<th>Diastolic blood pressure</th>
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</thead>
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<table>
<thead>
<tr>
<th>Operated limb: h (cm)</th>
<th></th>
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<tbody>
<tr>
<td></td>
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</table>

<table>
<thead>
<tr>
<th>Diameter at ankle (cm)</th>
<th></th>
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<tbody>
<tr>
<td></td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Diameter at widest point of calf (cm)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Calf volume (cc)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Unoperated limb: h (cm)</th>
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</thead>
<tbody>
<tr>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Diameter at ankle (cm)</th>
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</tr>
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<tbody>
<tr>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Diameter at widest point of calf (cm)</th>
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<tbody>
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</table>

<table>
<thead>
<tr>
<th>Calf volume (cc)</th>
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<tr>
<td></td>
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</tbody>
</table>

**Fatigue self assessment**

**Description of fatigue (own words)**

---

### 12-1 pm

**Time up before assessment**: (1 in bed 2 1-2 hrs 3 2-4 hrs 4 4-6 hrs 5 6-8 hrs 6 8hrs+)

<table>
<thead>
<tr>
<th>Pulse rate</th>
<th>Systolic blood pressure</th>
<th>Diastolic blood pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<table>
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<tr>
<th>Operated limb: h (cm)</th>
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<table>
<thead>
<tr>
<th>Diameter at ankle (cm)</th>
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<table>
<thead>
<tr>
<th>Diameter at widest point of calf (cm)</th>
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<table>
<thead>
<tr>
<th>Calf volume (cc)</th>
<th></th>
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<table>
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<tr>
<th>Unoperated limb: h (cm)</th>
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<td></td>
<td></td>
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<tr>
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**Fatigue self assessment**

**Description of fatigue (own words)**

---
<table>
<thead>
<tr>
<th>Time</th>
<th>3-4 pm</th>
<th>6-7 pm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time up before assessment</td>
<td>1 in bed 2 1-2 hrs 3 2-4 hrs 4 4-6 hrs 5 6-8 hrs 6 8hrs +</td>
<td>1 in bed 2 1-2 hrs 3 2-4 hrs 4 4-6 hrs 5 6-8 hrs 6 8hrs +</td>
</tr>
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<td>Pulse rate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
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<td></td>
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<td></td>
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</tbody>
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POST-OPERATIVE ASSESSMENT 2

Chest infection (1 yes 2 no) [definition c/list 3]

Pressure sores: present (1 yes 2 no)
  widest diameter (cm)
  grade [definition c/list 4]

Independently mobile (1 yes 2 no) [definition c/list 5].

Drug therapy

Drugs taken which might affect fatigue
  (1 yes 2 no) [see c/list 6]

Drugs taken which might affect mobility
  (1 yes 2 no) [see c/list 7]

Drugs taken which might affect BP and pulse
  (1 yes 2 no) [see c/list 8]

Fatigue
  2.7 What was most tiring today? [see c/list 9]
  2.8 What would relieve tiredness [see c/list 10]
9-10 am

Time up before assessment (1 in bed 2 1-2 hrs 3 2-4 hrs 4 4-6 hrs 5 6-8 hrs 6 8 hrs +)

Pulse rate

Systolic blood pressure

Diastolic blood pressure

Operated limb: h (cm) — — —
Diameter at ankle (cm) — — —
Diameter at widest point of calf (cm) — — —

Calf volume (cc)

Unoperated limb: h (cm) — — —
Diameter at ankle (cm) — — —
Diameter at widest point of calf (cm) — — —

Calf volume (cc)

Fatigue self assessment
Description of fatigue (own words)

12-1 pm

Time up before assessment (1 in bed 2 1-2 hrs 3 2-4 hrs 4 4-6 hrs 5 6-8 hrs 6 8 hrs +)

Pulse rate

Systolic blood pressure

Diastolic blood pressure

Operated limb: h (cm) — — —
Diameter at ankle (cm) — — —
Diameter at widest point of calf (cm) — — —

Calf volume (cc)

Unoperated limb: h (cm) — — —
Diameter at ankle (cm) — — —
Diameter at widest point of calf (cm) — — —

Calf volume (cc)

Fatigue self assessment
Description of fatigue (own words)
3-4 pm

Time up before assessment (1 in bed 2 1-2 hrs 3 2-4 hrs 4 4-6 hrs 5 6-8 hrs 6 8hrs +

Pulse rate

Systolic blood pressure

Diastolic blood pressure

Operated limb: h (cm) ___ ___
Diameter at ankle (cm) ___ ___
Diameter at widest point of calf (cm) ___ ___

Calf volume (cc)

Unoperated limb: h (cm) ___ ___
Diameter at ankle (cm) ___ ___
Diameter at widest point of calf (cm) ___ ___

Fatigue self assessment

Description of fatigue (own words)

6-7 pm

Time up before assessment (1 in bed 2 1-2 hrs 3 2-4 hrs 4 4-6 hrs 5 6-8 hrs 6 8hrs +

Pulse rate

Systolic blood pressure

Diastolic blood pressure

Operated limb: h (cm) ___ ___
Diameter at ankle (cm) ___ ___
Diameter at widest point of calf (cm) ___ ___

Calf volume (cc)

Unoperated limb: h (cm) ___ ___
Diameter at ankle (cm) ___ ___
Diameter at widest point of calf (cm) ___ ___

Fatigue self assessment

Description of fatigue (own words)
POST-OPERATIVE ASSESSMENT 3

Chest infection (1 yes 2 no) [definition c/list 3]

Pressure sores: present (1 yes 2 no)

widest diameter (cm)

grade [definition c/list 4]

Independently mobile (1 yes 2 no) [definition c/list 5]

Drug therapy

Drugs taken which might affect fatigue
(1 yes 2 no) [see c/list 6]

Drugs taken which might affect mobility
(1 yes 2 no) [see c/list 7]

Drugs taken which might affect BP and pulse
(1 yes 2 no) [see c/list 8]

Fatigue

2.7 What was most tiring today? [see c/list 9]

2.8 What would relieve tiredness [see c/list 10]
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<tr>
<th>Time</th>
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<th>Diastolic blood pressure</th>
<th>Operated limb: h (cm)</th>
<th>Diameter at ankle (cm)</th>
<th>Diameter at widest point of calf (cm)</th>
<th>Calf volume (cc)</th>
<th>Unoperated limb: h (cm)</th>
<th>Diameter at ankle (cm)</th>
<th>Diameter at widest point of calf (cm)</th>
<th>Calf volume (cc)</th>
<th>Fatigue self assessment</th>
<th>Description of fatigue (own words)</th>
</tr>
</thead>
<tbody>
<tr>
<td>9-10 am</td>
<td></td>
<td></td>
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<tr>
<td>12-1 pm</td>
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</tbody>
</table>
### 3-4 pm

<table>
<thead>
<tr>
<th>Time up before assessment</th>
<th>Pulse rate</th>
<th>Systolic blood pressure</th>
<th>Diastolic blood pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1 in bed 2 1-2 hrs</td>
<td>3 2-4 hrs</td>
<td>4 4-6 hrs 5 6-8 hrs 6 8hrs +</td>
<td></td>
</tr>
<tr>
<td>Description</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| Operated limb: h (cm)     | ___ ___    |                         |                          |
| Diameter at ankle (cm)    | ___ ___    |                         |                          |
| Diameter at widest point of calf (cm) | ___ ___ |                          |

| Calf volume (cc)          | ___ ___    |                         |                          |

| Unoperated limb: h (cm)   | ___ ___    |                         |                          |
| Diameter at ankle (cm)    | ___ ___    |                         |                          |
| Diameter at widest point of calf (cm) | ___ ___ |                          |

| Calf volume (cc)          | ___ ___    |                         |                          |

| Fatigue self assessment   |           |                         |                          |
| Description of fatigue (own words) |           |                          |

### 6-7 pm

<table>
<thead>
<tr>
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| Operated limb: h (cm)     | ___ ___    |                         |                          |
| Diameter at ankle (cm)    | ___ ___    |                         |                          |
| Diameter at widest point of calf (cm) | ___ ___ |                          |

| Calf volume (cc)          | ___ ___    |                         |                          |

| Unoperated limb: h (cm)   | ___ ___    |                         |                          |
| Diameter at ankle (cm)    | ___ ___    |                         |                          |
| Diameter at widest point of calf (cm) | ___ ___ |                          |

| Calf volume (cc)          | ___ ___    |                         |                          |

| Fatigue self assessment   |           |                         |                          |
| Description of fatigue (own words) |           |                          |
POST-OPERATIVE ASSESSMENT 4

Chest infection (1 yes 2 no) [definition c/list 3]

Pressure sores: present (1 yes 2 no)
widest diameter (cm)
grade [definition c/list 4]

Independently mobile (1 yes 2 no) [definition c/list 5]

Drug therapy

Drugs taken which might affect fatigue
(1 yes 2 no) [see c/list 6]

Drugs taken which might affect mobility
(1 yes 2 no) [see c/list 7]

Drugs taken which might affect BP and pulse
(1 yes 2 no) [see c/list 8]

Fatigue
2.7 What was most tiring today? [see c/list 9]

2.8 What would relieve tiredness [see c/list 10]
### 9-10 am

<table>
<thead>
<tr>
<th>Time up before assessment</th>
<th>Pulse rate</th>
<th>Systolic blood pressure</th>
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</tr>
</thead>
<tbody>
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<td>1 in bed</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>2 1-2 hrs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 2-4 hrs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 4-6 hrs</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>5 6-8 hrs</td>
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<td></td>
</tr>
<tr>
<td>6 8 hrs +</td>
<td></td>
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<table>
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<th>Operated limb: h (cm)</th>
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<table>
<thead>
<tr>
<th>Fatigue self assessment</th>
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<tbody>
<tr>
<td>Description of fatigue (own words)</td>
</tr>
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<td>-----------------------------</td>
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### 12-1 pm

<table>
<thead>
<tr>
<th>Time up before assessment</th>
<th>Pulse rate</th>
<th>Systolic blood pressure</th>
<th>Diastolic blood pressure</th>
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<tr>
<td>1 in bed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 1-2 hrs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 4-6 hrs</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>5 6-8 hrs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 8 hrs +</td>
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<td></td>
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### 3-4 pm

**Time up before assessment**: (1 in bed 2 1-2 hrs 3 2-4 hrs 4 4-6 hrs 5 6-8 hrs 6 8hrs +)

<table>
<thead>
<tr>
<th>Pulse rate</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>Systolic blood pressure</td>
<td></td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
<td></td>
</tr>
</tbody>
</table>

**Operated limb**: h (cm) 
**Diameter at ankle (cm)**
**Diameter at widest point of calf (cm)**

**Calf volume (cc)**

**Unoperated limb**: h (cm) 
**Diameter at ankle (cm)**
**Diameter at widest point of calf (cm)**

**Calf volume (cc)**

**Fatigue self assessment**

**Description of fatigue (own words)**

---

### 6-7 pm

**Time up before assessment**: (1 in bed 2 1-2 hrs 3 2-4 hrs 4 4-6 hrs 5 6-8 hrs 6 8hrs +)

<table>
<thead>
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<th>Pulse rate</th>
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<tbody>
<tr>
<td>Systolic blood pressure</td>
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<td>Diastolic blood pressure</td>
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**Operated limb**: h (cm) 
**Diameter at ankle (cm)**
**Diameter at widest point of calf (cm)**

**Calf volume (cc)**

**Unoperated limb**: h (cm) 
**Diameter at ankle (cm)**
**Diameter at widest point of calf (cm)**

**Calf volume (cc)**

**Fatigue self assessment**

**Description of fatigue (own words)**

---
POST-OPERATIVE ASSESSMENT 5

Chest infection (1 yes  2 no) [definition c/list 3]

Pressure sores: present (1 yes 2 no)

widest diameter (cm)

grade [definition c/list 4]

Independently mobile (1 yes  2 no) [definition c/list 5]

Drug therapy

Drugs taken which might affect fatigue
(1 yes  2 no) [see c/list 6]

Drugs taken which might affect mobility
(1 yes  2 no) [see c/list 7]

Drugs taken which might affect BP and pulse
(1 yes  2 no) [see c/list 8]

Fatigue

2.7 What was most tiring today? [see c/list 9]

2.8 What would relieve tiredness [see c/list 10]
### 9-10 am

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<tr>
<th>Time up before assessment</th>
<th>1 in bed</th>
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<th>5 6-8 hrs</th>
<th>6+ 8 hrs</th>
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</table>

<table>
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<tr>
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<table>
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### 12-1 pm

<table>
<thead>
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<th>Time up before assessment</th>
<th>1 in bed</th>
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3-4 pm

Time up before assessment (1 in bed 2 1-2 hrs 3 2-4 hrs 4 4-6 hrs 5 6-8 hrs 6 8hrs +

Pulse rate
Systolic blood pressure
Diastolic blood pressure

Operated limb: h (cm)
Diameter at ankle (cm) ______
Diameter at widest point of calf (cm) ______

Calf volume (cc)

Unoperated limb: h (cm)
Diameter at ankle (cm) ______
Diameter at widest point of calf (cm) ______

Calf volume (cc)

Fatigue self assessment
Description of fatigue (own words)

6-7 pm

Time up before assessment (1 in bed 2 1-2 hrs 3 2-4 hrs 4 4-6 hrs 5 6-8 hrs 6 8hrs +

Pulse rate
Systolic blood pressure
Diastolic blood pressure

Operated limb: h (cm)
Diameter at ankle (cm) ______
Diameter at widest point of calf (cm) ______

Calf volume (cc)

Unoperated limb: h (cm)
Diameter at ankle (cm) ______
Diameter at widest point of calf (cm) ______

Calf volume (cc)

Fatigue self assessment
Description of fatigue (own words)
SITTING OUT
(to nearest quarter-hour)

Day 1 post-op

1st time up

Last time to bed

Day 2 post-op

1st time up

Last time to bed

Day 3 post-op

1st time up

Last time to bed

Day 4 post-op

1st time up

Last time to bed

Day 5 post-op

1st time up

Last time to bed
Day 10 post-op

Day 12 post-op

Day 11 post-op

Day 13 post-op

1st time up

Last time to bed

1st time up

Last time to bed

1st time up

last time to bed
Day 14 post-op

- 1st time up
- Last time to bed

COMPLICATIONS

Constipation (1 yes 2 no)
UTI (1 yes 2 no)

FOLLOW UP

Post-operative day independently mobile
Post-operative day discharged from ward

Fate: (1 discharged home
2 discharged to long term care
3 t/ferred to other acute ward/hospital
4 died
5 discontinued due to complications
6 early Discharge Scheme
APPENDIX 6

Graphical presentation of mean pulse, blood pressure and leg oedema by day and time of day.

In the following graphical representations the day refers to day after surgery (with day of surgery being counted as day one). The times of day are as follows:

1 = 9am - 10am
2 = 12 noon - 1pm
3 = 3pm - 4pm
4 = 6pm - 7pm
Mean self-assessed fatigue levels

Arbitrary units

Day

Time of day

LCG

UCG
Mean heart rate

Beats per minute

○ LCG
△ UCG

Day

Time of day

1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4

0 LC
94
84
78

0
1
2
3
4
5
7
10
14
Mean systolic blood pressure

![Graph of mean systolic blood pressure over time for two groups, LCG and UCG, with data points showing variation across days and times.](image)
Mean diastolic blood pressure

![Graph showing diastolic blood pressure over time](image)
Mean volume of operated leg

- LCG
- UCG

Day

Time of day

1 2 3 4

Volume in mm³

1500
1200

3 5 7 10 14

1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4
Mean volume of unoperated leg

Graph showing the mean volume of unoperated leg over time, with markers for LCH and UCH.
APPENDIX 7

Comparison of incidence of negative outcomes between LCG and UCG when patients with outcome at baseline were removed from the analysis.

Severe self perceived fatigue (7-9 arbitrary units)
There was no significant difference in the mean fatigue score between the two groups on admission to the trial (4.1 and 4.0). There was also no significant difference in the percentage of subjects prescribed drugs which may cause fatigue (45% and 41%) as a side effect. After excluding 17 subjects who were severely fatigued on admission to the study, the proportions of subjects developing severe fatigue were \( p_{LCG} = \frac{36}{88} \) and \( p_{UCG} = \frac{65}{98} \). The proportion of subjects developing severe fatigue was significantly higher in the UCG than in the LCG (\( p_{UCG} - p_{LCG} = 0.25; p < 0.001; 99\% CI = 0.07, 0.44 \)).

The following factors were univariately associated with severe fatigue: chairnursing policy (LCG or UCG) (\( p = 0.001 \)) and total Norton score (20 vs < 20) approached significance (\( p = 0.03 \)). Only chairnursing policy remained significant in the multivariate model. Subjects were 2.85 times more likely to report severe fatigue in the UCG as compared with the LCG (99% CI = 1.3, 6.23).

Tachycardia (pulse rate > 100 beats per minute)
There was no difference in the mean pulse rate between the two groups on admission to the trial (81 beats per minute, SD 13.3). There was also no significant difference in the percentage of subjects prescribed drugs which may cause either tachycardia (60% and 56%) or bradycardia (84% and 85%) as a side effect at any time during the study. After excluding 13 subjects who were tachycardic on admission to the study, the proportions of subjects developing tachycardia were \( p_{LCG} = \frac{28}{88} \) and \( p_{UCG} = \frac{54}{102} \). The proportion of subjects developing tachycardia was significantly higher in the UCG than in the LCG (\( p_{UCG} - p_{LCG} = 0.21; p = 0.0003; 99\% CI = 0.03, 0.39 \)). The following factors were univariately associated with tachycardia: chairnursing policy (LCG or UCG) (\( p = 0.004 \)) and not taking potentially fatigue inducing drugs (\( p = 0.026 \)). Only chairnursing policy was significant at the 1% level and remained significant in the multivariate logistic regression model. Subjects in the UCG were 2.41 (99% CI = 1.1, 5.26) times more likely to have tachycardia than subjects in the LCG.

Hypotension (systolic blood pressure < 100 mmHg)
There was no significant difference in the mean systolic or diastolic blood pressure between the two groups on admission to the trial (74 and 81 mmHg respectively). There was also no significant difference in the percentage of subjects prescribed drugs which may cause either hypo (84% and 85%) or hypertension (85% and 85%) as a side effect. After excluding 11 subjects who were hypotensive on admission to the study, the proportions of subjects developing systolic hypotension were \( p_{LCG} = \frac{3}{96} \) and \( p_{UCG} = \frac{13}{96} \). The proportion of patients developing hypotension in the UCG was significantly higher than in the LCG (\( p_{UCG} - p_{LCG} = 0.10; p = 0.008; 99\% CI = 0.003, 0.21 \)).

The following factors were univariately associated with hypotension: chairnursing policy (LCG or UCG) (\( p = 0.016 \)), not taking potentially fatigue inducing drugs (\( p = 0.03 \)) and having Austin Moore’s hemiarthroplasty as opposed to any other type of surgery (\( p = 0.02 \)). None of these were significant at the 1% level. The best multivariate logistic regression model includes only chairnursing policy. Patients in the UCG were 2.41 (1.1, 5.26) times more likely to have hypotension than patients in the LCG. However adequate evidence of an association between chairnursing policy and the incidence of hypotension was not found.
Lower limb oedema (increase of over 20% above baseline)
There was no significant difference in the mean volume of operated and unoperated limbs between the two groups on admission to the trial (1436 cm³ and 1362 cm³; 1404³ and 1388 cm³). There was also no significant difference in the percentage of subjects prescribed drugs which may cause oedema (72% and 69%) as a side effect.

The proportions of subjects developing lower leg oedema in the operated limb, calculated on an intention-to-treat basis (excluding subjects with bilateral surgery) were p_LCG = 6/99 and p_UCG = 35/102. The proportion of operated limbs that became oedematous in the UCG was significantly higher than in LCG (p_UCG - p_LCG = 0.29; p < 0.00001; 99%CI = 0.14, 0.44). Factors which were univariately associated with operated lower leg oedema were chairnursing policy (p < 0.0001), type of operation (dynamic hip screw vs all others) (p = 0.001), Norton score (< 20 vs 20) (p = 0.004), smoking history (p = 0.03) and the pre-operative volume of the operated (p = 0.03) and the unoperated leg (p = 0.018).

The proportions of subjects developing lower leg oedema in the unoperated limb, also calculated on an intention-to-treat basis (excluding subjects with bilateral surgery) were p_LCG = 11/99 and p_UCG = 37/102. The proportion of unoperated limbs developing oedema was significantly higher in the UCG as compared with LCG (p_UCG - p_LCG = 0.26; p = < 0.00001; 99%CI = 0.11, 0.42). Factors associated with operated lower leg oedema were chairnursing policy (p < 0.0001), type of operation (dynamic hip screw vs all other types) (p = 0.03), type of surgery (emergency vs elective) (p = 0.007) and mobility on admission (fully mobile vs others).

Multivariately, only chairnursing policy remained significant at the 1% level for both limbs, while having a dynamic hip screw as opposed to any other surgery approached significance. Subjects in the UCG were 8.96 times more likely to develop oedema in the operated leg (99%CI = 2.57, 31.29) and 4.71 times more likely to develop oedema in the unoperated leg (99%CI = 1.3, 6.23) than LCG subjects.

Chest infection
There was no significant difference in the percentage of subjects prescribed drugs which may reduce resistance to infection as a side effect in the LCG and UCG (6% and 4% respectively). After excluding 3 subjects who had chest infections on admission to the study, the proportions of subjects developing chest infection during the study were p_LCG = 13/98 and p_UCG = 18/102. These proportions were not significantly different (p_UCG - p_LCG = 0.04; p = 0.39; 99%CI = -0.09, 0.18).

Not taking drugs which can cause oedema (p = 0.004) was univariately associated with increased risk of chest infection. Factors associated with a reduced risk of developing chest infection were having a total Norton score of 20 (as opposed to < 20) and quitting smoking > 3 months previously (p = 0.01). The best multivariate logistic regression model contained only the drugs causing oedema variable. Patients not given these drugs were 3.13 times more likely to have a chest infection than those who were given them (99%CI = 1.11, 8.77).

Pressure sores
There was no difference in the median Norton score between the two groups on admission to the trial (18). No subjects had pressure sores on admission to the study and the proportions of subjects developing new pressure sores were p_LCG = 0/100 and p_UCG = 9/103. Since no patients in the LCG developed pressure sores during the study, the only valid test for this data was the Fisher’s Exact Test which showed significantly greater incidence of pressure sores in the UCG as compared with LCG (p = 0.003).
Other factors univariately associated with the development of pressure sores were the type of surgery \((p=0.0078)\) and total Norton score \((20 \text{ vs } <20)\). Only type of surgery was significant in the multivariate model. Subjects undergoing emergency surgery were 5.6 times more likely to develop pressure sores than those having surgery electively.

**Constipation**

There was no significant difference in the percentage of subjects prescribed drugs which may cause either constipation \((94\% \text{ and } 95\%)\) or diarrhoea \((86\% \text{ and } 87\%)\) as a side effect. After excluding 4 subjects who were constipated on admission to the study, the proportions of subjects becoming constipated during the study were \(p_{\text{LCG}} = 32/99\) and \(p_{\text{UCG}} = 64/100\). The proportion of subjects developing constipation was significantly higher in the UCG than in the LCG \((p_{\text{UC}} - p_{\text{LC}} = 0.32; \ p < 0.0001; \ 99\% \text{CI} = 0.14, 0.49)\).

Factors univariately associated with the risk of developing constipation were chairnursing policy \((p < 0.0001)\) and Austin-Moore’s hemiarthroplasty \((p=0.007)\). Taking drugs which may cause hypotension or drugs which may cause bradycardia approached significance \((p = 0.03 \text{ in each case})\). Chairnursing policy and Austin-Moore’s operation remained significant at the 1\% level in the multivariate logistic regression model. UCG subjects were 3.68 times more likely to become constipated than LCG subjects \((99\% \text{CI} = 1.63, 8.31)\). Subjects undergoing Austin Moore’s arthroplasty were 4.78 times more likely to become constipated \((99\% \text{CI} = 1.11, 20.52)\) all other things being equal.

**Urinary tract infection (UTI)**

There was no significant difference in the percentage of subjects prescribed drugs which may reduce resistance to infection \((6\% \text{ and } 4\%)\) as a side effect. After excluding 5 subjects who had UTI on admission, the proportions of subjects developing UTI during the course of the study were \(p_{\text{LCG}} = 7/97\) and \(p_{\text{UCG}} = 11/101\). There was no significant difference in the incidence of UTI during the study between the UCG and LCG \((p_{\text{UCG}} - p_{\text{LCG}} = 0.04; \ p = 0.37; \ 99\% \text{CI} = -0.07, 0.14)\).

Factors univariately associated with increased risk of development of UTI were not being fully mobile on admission \((p=0.0039)\) and the patient having emergency surgery as opposed to elective \((p = 0.009)\). Only mobility on admission was significant in a multivariate model. Patients who were not fully mobile on admission were 4.32 times more likely to develop UTI than those who were \((99\% \text{CI} = 1.12, 16.69)\).
APPENDIX 8

Illustrations

Figure 1 Dependent oedema of the legs, slippers cut to accommodate feet

Figure 2 Superficial ischial pressure sore in orthopaedic patient
Figure 3 Full-thickness ischial and superficial sacral pressure sores in elderly patient
APPENDIX 9

Studies of postoperative fatigue

A literature search using MEDLINE, CINAHL, ASSIA and PSYCLIT with CD-ROM for the period 1966-1999 was used to extract a sample of studies which assessed post-operative fatigue. The abstracts of all papers which have the word 'fatigue' anywhere in the text of the entry (title, keyword or abstract) were examined. Those which described measurement of fatigue in postoperative patients were retained. Further literature was gathered through references in the papers already obtained and reviews (Kehlet 1988, Christensen & Kehlet 1993). This procedure is designed to produce a convenience sample rather than an exhaustive search (Chalmers et al 1989) which was beyond the scope of this thesis.

It was not possible to determine the method of measurement in three foreign language studies (Bauwens et al 1998, Lopez-Vidaur et al 1995, Alonso Chico et al 1998). Also excluded were studies which gave inadequate information to determine method of assessment used (eg Cimino & Kogan 1989, Yano et al 1991, Dickson & Mintz 1996, Iannotti et al 1996, Oliveira et al 1997, Hatasaka et al 1997) and if the study was of adjuvant therapy rather than the surgery itself (Lindley et al 1998). Papers which report unsolicited self reporting of fatigue as a symptom by patients (eg Fremes et al 1990) were excluded. Only final papers were included, with reports on subsets of the same total group being excluded (eg Jenkins et al 1983). Finally it was not possible to obtain some papers prior to completion (Wu et al 1998).

The search yielded 72 studies which attempted to measure self perceived fatigue in postoperative patients. Two studies used both a questionnaire and VAS, hence the total number of references quoted in sections 1-5 is 74 rather than 72. There were five basic approaches.

1. Fatigue expressed as present or absent (table 1): the patient is simply asked if he was fatigued or not during a specified period of time as part of a symptoms checklist (13 references).

2. Questionnaire (table 2): the patient is asked to complete a battery of questions. These may be a specialised fatigue inventory (2 references) or part of a larger questionnaire measuring mood or health status (13 references).

3. Visual analogue scale (VAS): the patient is presented with a linear analogue (eg figure 1) and asked to place a mark on the scale to indicate the level of fatigue they feel (42 references).

4. Numerical scale (NS): the patient is presented with a numerical ascale (eg 1 = no fatigue, 2 = slight fatigue, 3 = moderate fatigue) and asked to indicate the level of fatigue they feel by circling the appropriate number (4 references).

5. Semistructured interview: patient is asked if they feel fatigued and if so how much. Patients response recorded (1 study, Fitzpatrick et al 1998)
Figure 1 Constructed visual analogue scale after Christensen et al (1982)

<table>
<thead>
<tr>
<th>10</th>
<th>FATIGUED</th>
<th>(Cannot cope with daily chores or short walks, pronounced need of sleep)</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>TIRED</td>
<td>(Particularly doing house work, gardening or walking stairs, increased need of sleep)</td>
</tr>
<tr>
<td>7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>SLIGHTLY TIRED</td>
<td>(Can manage daily chores, occasionally more strenuous tasks)</td>
</tr>
<tr>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>FIT</td>
<td>(Tired only by violent exertion, normal need of sleep)</td>
</tr>
<tr>
<td>1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 1.1 Studies recording fatigue as present or absent

<table>
<thead>
<tr>
<th>Author</th>
<th>Instrument</th>
<th>Timing of measurement</th>
<th>Study population</th>
<th>Subject numbers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lanz et al (1982)</td>
<td>Symptom checklist</td>
<td>Afternoon of day 1 post-op</td>
<td>Orthopaedics</td>
<td>174</td>
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<td>Takabayashi et al (1990)</td>
<td>Symptom checklist</td>
<td>12 weeks post-op</td>
<td>Gynaecological surgery</td>
<td>50</td>
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<tr>
<td>Uden et al (1992)</td>
<td>Symptom checklist</td>
<td>2-3 weeks preop 1-3 weeks and 6 months postop</td>
<td>Initial neck surgery for Hyperparathyroidism</td>
<td>250</td>
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<tr>
<td>Redeker (1993)</td>
<td>Symptom checklist</td>
<td>during hospitalisation</td>
<td>Coronary artery bypass surgery</td>
<td>129</td>
</tr>
<tr>
<td>Tezelman et al (1993)</td>
<td>Symptom checklist</td>
<td>pre-op and 1-4 weeks, 6 months and at 1-yearly intervals post-op</td>
<td>Parathyroidectomy</td>
<td>416</td>
</tr>
<tr>
<td>Forsberg et al (1996)</td>
<td>Symptom checklist</td>
<td>pre-op and 6 weeks post-op</td>
<td>Major surgery for colorectal or gastric cancer</td>
<td>79</td>
</tr>
<tr>
<td>Shinoda et al (1997)</td>
<td>Symptom checklist</td>
<td>pre-op and 1-4 weeks, 6 months and at 1-yearly intervals post-op</td>
<td>Parathyroidectomy</td>
<td>416</td>
</tr>
<tr>
<td>Hariz &amp; de Salles (1997)</td>
<td>Symptom checklist</td>
<td>up to 36 months post-op (mean 12 months post-op)</td>
<td>Posteroventral pallidotomy</td>
<td>138</td>
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<tr>
<td>Wyatt &amp; Friedman (1998)</td>
<td>Symptom checklist</td>
<td>1 week, 6 weeks, 3 and 6 months post-op</td>
<td>Breast surgery for cancer</td>
<td>46</td>
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Table 1.2 Studies measuring fatigue with questionnaire(s)

<table>
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<th>Timing of measurement</th>
<th>Study population</th>
<th>Subject numbers</th>
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<tbody>
<tr>
<td>Meikle et al (1977)</td>
<td>POMS</td>
<td>pre-op and 6 wks, 3 months post-op</td>
<td>Hyterctomy (55) Cholecystectomy (38) Tubal ligation (60)</td>
<td>153</td>
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<tr>
<td>Jenkins et al (1983)</td>
<td>POMS</td>
<td>pre-op and 6 months post-op</td>
<td>CABG (372) CVS (91)</td>
<td>463</td>
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<tr>
<td>Kiyak et al (1985)</td>
<td>POMS</td>
<td>pre-op and 1 day, 4-6 weeks, 6 months post-op</td>
<td>Orthogonathic surgery</td>
<td>90</td>
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<tr>
<td>Oberst &amp; James (1985)</td>
<td>STAI BSI</td>
<td>1-2 days prior to discharge; 10, 30, 60, 90, 180 days post-op</td>
<td>Surgery for cancer bowel/genitourinary</td>
<td>40</td>
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<tr>
<td>Vogele &amp; Steptoe (1986)</td>
<td>POMS</td>
<td>2, 1 days pre-op; 1, 2, 3, 4, 5, 7, 9, 11 days post-op</td>
<td>Hip replacement (8) Arthroscopy (7)</td>
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<tr>
<td>Oberst &amp; Scott (1988)</td>
<td>STAI BSI</td>
<td>1-2 days prior to discharge; 10, 30, 60, 90, 180 days post-op</td>
<td>Surgery for cancer stoma (20) nonstoma (20)</td>
<td>40</td>
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<tr>
<td>Krouse et al (1988)</td>
<td>STAI BSI</td>
<td>Pre-op and 3, 12 months post-op</td>
<td>Head and neck surgery for cancer</td>
<td>33</td>
</tr>
<tr>
<td>Cleary et al (1991)</td>
<td>Own questionnaire</td>
<td>pre-op and postop; 3 months (TURP, cholecystectomy) 6 months (CABG) 12 months (THR)</td>
<td>Cholecystectomy (476) TURP (425) CABG (361) THR (335)</td>
<td>1597</td>
</tr>
<tr>
<td>Suxton et al (1992)</td>
<td>5 dimension 7 point scale</td>
<td>pre-op; 2, 6 days postop (23) pre-op; 2, 8, 15, 46 days post-op (13)</td>
<td>Abdominal surgery (22) Mastectomy (1)</td>
<td>23</td>
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<tr>
<td>Jones et al (1992)</td>
<td>EORTC + head &amp; neck module</td>
<td>4-26 months postoperatively</td>
<td>Head and neck surgery for cancer</td>
<td>48</td>
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<tr>
<td>Fasschieter et al (1993)</td>
<td>Short POMS</td>
<td>Pre-op; every 8 hrs for 48 hrs postop</td>
<td>Elective upper abdominal surgery</td>
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<td>Schroeder &amp; Hill (1993)</td>
<td>POMS</td>
<td>Pre-op; 7, 14, 28, 90 days post-op</td>
<td>Abdominal surgery</td>
<td>84</td>
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<tr>
<td>Mangione et al (1993)</td>
<td>SF-36</td>
<td>Pre-op</td>
<td>Major elective non-cardiac surgery</td>
<td>745</td>
</tr>
<tr>
<td>Yamadera (1994)</td>
<td>SAM</td>
<td>Pre-op; 2-22 days, 3-6 months post-op every 2 hrs, from 0800 to 2000 incl</td>
<td>Surgery for obstructive sleep apnoea syndromes</td>
<td>17</td>
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<tr>
<td>Croog et al (1994)</td>
<td>STAI POMS RSF-36</td>
<td>1 week pre-op I; 1 week pre-op II; 10 days post-op II</td>
<td>Periodontal surgery</td>
<td>42</td>
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<tr>
<td>Kyerulff &amp; Lagenberg (1995)</td>
<td>POMS</td>
<td>Unable to determine</td>
<td>Hysterectomy</td>
<td>1205</td>
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<tr>
<td>Pick et al (1994)</td>
<td>STAI</td>
<td>Pre-op; 1, 4, 21 days post-op</td>
<td>Elective CABG</td>
<td>74</td>
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<tr>
<td>Ritter et al (1995)</td>
<td>RSF-36</td>
<td>Pre-op; 6 months, 1 and 2 years post-op</td>
<td>THR (85) TCR (93) SSBTKR (65)</td>
<td>243</td>
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<td>Aarons et al (1996)</td>
<td>POMS</td>
<td>pre-op and weeks 1 and 7 post-op</td>
<td>THR and TCR</td>
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<td>Belle et al (1996)</td>
<td>LTD QOL</td>
<td>pre-op and 1 year post-op</td>
<td>Liver transplant</td>
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</table>

POMS: Profile of Mood States  
BDI: Beck’s Depression Inventory  
EORTC: European Organisation for Research into the Treatment of Cancer Questionnaire  
SAM: Spacemedicine Fatigue Checklist  
RSF-36: Read SF-36 Health Status Questionnaire (TPE Specification, Quality Quest)  
SF-36: Medical Outcomes Study Short Form Health Survey  
LTD QOL: Liver Transplant Database Quality of Life questionnaire  
STAI: State-Trait Anxiety Inventory  
BSI: Brief Symptom Inventory  
TURP: Trans-urethral prostatectomy  
CABG: Coronary artery bypass graft  
THR: Total hip replacement  
SSBTKR: Single stage bilateral total knee replacement
Table 1.3 (part 1) Studies measuring fatigue with visual analogue scale (VAS)

<table>
<thead>
<tr>
<th>Author</th>
<th>Instrument</th>
<th>Timing of measurement</th>
<th>Study population</th>
<th>Subject numbers</th>
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<tbody>
<tr>
<td>Christensen et al (1982)</td>
<td>Constructed VAS</td>
<td>pre-op, 10, 20, 30 days postop</td>
<td>Abdominal surgery</td>
<td>16</td>
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<td>Christensen &amp; Kehlet (1984)</td>
<td>VAS after Christensen</td>
<td>pre-op and 10, 20, 30 days postop</td>
<td>Elective abdominal surgery</td>
<td>36</td>
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<td>Christensen et al (1985)</td>
<td>VAS after Christensen</td>
<td>pre-op and 10, 20 days postop</td>
<td>Elective abdominal surgery</td>
<td>20</td>
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<td>Hyortso et al (1985)</td>
<td>VAS after Christensen</td>
<td>pre-op and 10, 30, 60 days postop</td>
<td>Elective abdominal surgery</td>
<td>100</td>
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<td>Christensen et al (1985a)</td>
<td>VAS after Christensen</td>
<td>pre-op and 10, 20, 30 days postop</td>
<td>Elective abdominal surgery</td>
<td>67</td>
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<td>Christensen et al (1986)</td>
<td>VAS after Christensen</td>
<td>pre-op and 10, 30 days postop</td>
<td>Elective abdominal surgery</td>
<td>15</td>
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<td>Shulze et al (1988)</td>
<td>VAS after Christensen</td>
<td>pre-op, 3, 6 hrs and days 1, 2, 4, 6 post-op</td>
<td>Elective cholecystectomy</td>
<td>24</td>
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<td>Christensen et al (1988)</td>
<td>VAS after Christensen</td>
<td>pre-op and 20 days postop</td>
<td>Elective abdominal surgery</td>
<td>12</td>
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<td>Christensen et al (1989)</td>
<td>VAS after Christensen</td>
<td>pre-op and 20 days postop</td>
<td>Elective abdominal surgery</td>
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</tr>
<tr>
<td>Christensen et al (1990)</td>
<td>VAS after Christensen</td>
<td>pre-op, 20 days postop</td>
<td>Elective abdominal surgery</td>
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<tr>
<td>Zeideman et al (1990)</td>
<td>VAS after Christensen</td>
<td>pre-op and 3rd day post-op</td>
<td>Elective abdominal surgery</td>
<td>14</td>
</tr>
<tr>
<td>Shulze et al (1990)</td>
<td>VAS after Christensen</td>
<td>pre-op and days 1, 2, 4, 6 post-op</td>
<td>Elective cholecystectomy</td>
<td>12</td>
</tr>
<tr>
<td>Shroeder &amp; Hill (1991)</td>
<td>VAS after Christensen</td>
<td>pre-op, 7, 14, 28, 90 days postop</td>
<td>Elective major abdominal surgery</td>
<td>38</td>
</tr>
<tr>
<td>Ghouri et al (1991)</td>
<td>VAS modified from Christensen</td>
<td>pre-op, 30, 60, 90 minutes post-op</td>
<td>Arthroscopy (40%); General surgery (24%); Plastic surgery (23%); Laparoscopy (23%)</td>
<td>38</td>
</tr>
<tr>
<td>Stage et al (1991)</td>
<td>VAS after Christensen</td>
<td></td>
<td>Minicholecystectomy</td>
<td>24</td>
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<tr>
<td>Wadstrom et al (1991)</td>
<td>Constructed VAS</td>
<td>pre-op and after 10% and 18% weight loss</td>
<td>Gastroplasty in obese patients</td>
<td>16</td>
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<tr>
<td>Kho et al (1991)</td>
<td>VAS after Christensen</td>
<td>pre-op and 1, 2, 3, 5, 10, 15, 20 days post-op</td>
<td>Retroperitoneal lymph node dissection</td>
<td>29</td>
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<tr>
<td>Cimprich (1992)</td>
<td>VAS</td>
<td>Day before discharge</td>
<td>Surgery for breast cancer</td>
<td>32</td>
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<tr>
<td>Buxton et al (1992)</td>
<td>VAS after Christensen</td>
<td>pre-op, 2, 8, days post-op (23); pre-op, 2, 8, 15, 46 days post-op (13)</td>
<td>Abdominal surgery (22); Mastectomy (1)</td>
<td>23</td>
</tr>
<tr>
<td>Parker et al (1992)</td>
<td>VAS after Christensen</td>
<td>Every 8 hrs for 24 hrs post-op</td>
<td>Caesarian sections</td>
<td>170</td>
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<tr>
<td>Troidl et al (1992)</td>
<td>VAS (1-100mm)</td>
<td>pre-op and days 2 and 14 post-op</td>
<td>Laparoscopic cholecystectomy</td>
<td>300</td>
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<tr>
<td>Cordova Martinez &amp; Marcon (1992)</td>
<td>VAS after Christensen</td>
<td>3, 9, 27, 45 days post-op</td>
<td>Abdominal surgery</td>
<td>60</td>
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</table>
Table 2.3 (part 2) Studies measuring fatigue with visual analogue scale (VAS)

<table>
<thead>
<tr>
<th>Author</th>
<th>Instrument</th>
<th>Timing of measurement</th>
<th>Study population</th>
<th>Subject numbers</th>
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<tbody>
<tr>
<td>Shulze &amp; Thorup (1993)</td>
<td>VAS after Christensen</td>
<td>pre-op and 1,2,3,4,5,6,7,8 days post-op</td>
<td>Laparoscopic cholecystectomy</td>
<td>50</td>
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<tr>
<td>Schroeder &amp; Hill (1993)</td>
<td>VAS after Christensen</td>
<td>pre-op, 7, 14, 28, 90 days post-op</td>
<td>Major uncomplicated gastrointestinal surgery</td>
<td>84</td>
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<tr>
<td>Hill et al (1993)</td>
<td>VAS after Christensen</td>
<td>pre-op, 1, 2, 3, 14, 28, 90 days post-op</td>
<td>Open cholecystectomy <em>(16)</em> Laparoscopic <em>(15)</em></td>
<td>31</td>
</tr>
<tr>
<td>Jakeways (1994)</td>
<td>VAS after Christensen</td>
<td>Pre-op, 24 hrs and 48 hrs postop</td>
<td>Elective cholecystectomy</td>
<td>24</td>
</tr>
<tr>
<td>Moiniche et al (1993)</td>
<td>VAS (1-100mm)</td>
<td>pre-op, days 1-7 post-op</td>
<td>TEA <em>(20)</em> TNA <em>(22)</em></td>
<td>42</td>
</tr>
<tr>
<td>Moiniche et al (1995)</td>
<td>VAS (1-100mm)</td>
<td>pre-op, 6 hrs, days 1-6 postop</td>
<td>Colonic resection</td>
<td>17</td>
</tr>
<tr>
<td>Delauney et al (1995)</td>
<td>VAS after Christensen</td>
<td>pre-op, 1, 10 days postop</td>
<td>Laparoscopic cholecystectomy</td>
<td>9</td>
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<tr>
<td>Vasa-Thorbeck et al (1996a)</td>
<td>VAS after Christensen</td>
<td>pre-op and days 5, 30 post-op</td>
<td>Elective cholecystectomy</td>
<td>48</td>
</tr>
<tr>
<td>Galloway &amp; Graydon (1996)</td>
<td>VAS</td>
<td>72 hours or less prior to discharge</td>
<td>Colonic cancer surgery</td>
<td>40</td>
</tr>
<tr>
<td>Kum et al (1996)</td>
<td>VAS after Christensen</td>
<td>Days 1-3 post-op</td>
<td>Elective laparoscopic cholecystectomy <em>(42)</em> Emergency laparoscopic cholecystectomy <em>(54)</em></td>
<td>478</td>
</tr>
<tr>
<td>Kum et al (1996a)</td>
<td>VAS 0-100</td>
<td>6, 24, 48 hours post-op</td>
<td>Elective laparoscopic cholecystectomy</td>
<td>45</td>
</tr>
<tr>
<td>Jensen &amp; Heesov (1997)</td>
<td>VAS after Christensen</td>
<td>pre-op (elective only), at discharge, 20, 50, 80, 110, 180 (elective only) days post discharge</td>
<td>Elective colorectal <em>(32)</em> Acute obstruction and severe peritonitis <em>(21)</em></td>
<td>53</td>
</tr>
<tr>
<td>Watters et al (1997)</td>
<td>VAS after Christensen</td>
<td>pre-op and day 6 post-op</td>
<td>Esophagectomy Pancreatoduodenectomy</td>
<td>28</td>
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<tr>
<td>Keele et al (1997)</td>
<td>VAS after Christensen</td>
<td>pre-op, day 3 post-op and on discharge</td>
<td>Major and minor abdominal surgery</td>
<td>86</td>
</tr>
<tr>
<td>Galloway et al (1997)</td>
<td>VAS (0-100mm)</td>
<td>48 hours or less prior to discharge 4 weeks after discharge</td>
<td>Abdominal aortic surgery</td>
<td>43</td>
</tr>
<tr>
<td>Stage et al (1997)</td>
<td>VAS after Christensen</td>
<td>pre-op, days 1-10 and 30 post-op</td>
<td>Laparoscopic <em>(18)</em> and open <em>(14)</em> resection for adenocarcinoma</td>
<td>29</td>
</tr>
<tr>
<td>Chen et al (1998)</td>
<td>VAS 100mm</td>
<td>pre-op, 24, 48, 72 hrs postop</td>
<td>Total hysterectomy Myomectomy</td>
<td>100</td>
</tr>
<tr>
<td>Schwenk et al (1998)</td>
<td>VAS-F</td>
<td>pre-op; 1-7 days post-op</td>
<td>Colonic resection open <em>(30)</em> laparoscopic <em>(30)</em></td>
<td>60</td>
</tr>
<tr>
<td>Kissmeyer-Nielsen et al (1999)</td>
<td>VAS after Christensen</td>
<td>2 days pre-op, 10, 30, 90 days post-op</td>
<td>Ileocat J-pouch surgery</td>
<td>24</td>
</tr>
<tr>
<td>Author</td>
<td>Instrument</td>
<td>Timing of measurement</td>
<td>Study population</td>
<td>Subject numbers</td>
</tr>
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<td>-------------------</td>
<td>------------</td>
<td>------------------------------------------------------------</td>
<td>----------------------------------------</td>
<td>-----------------</td>
</tr>
<tr>
<td>Baidram et al (1995)</td>
<td>NS 0-2</td>
<td>Once per day for first 10 postop days</td>
<td>Laparoscopic colonic surgery</td>
<td>8</td>
</tr>
<tr>
<td>Cason et al (1996)</td>
<td>Likert-type 0-5</td>
<td>Pre-op, day of surgery and days 1, 2, 3, 4, 7 post-op</td>
<td>Laparoscopic cholecystectomy</td>
<td>53</td>
</tr>
<tr>
<td>Wyatt &amp; Friedman (1998)</td>
<td>NS 0-2</td>
<td>1 week, 6 weeks, 3 and 6 months post-op</td>
<td>Breast surgery for cancer</td>
<td>46</td>
</tr>
<tr>
<td>Chen et al (1998)</td>
<td>NS 0-5</td>
<td>pre-op and at least 6 months post-op</td>
<td>Parathyroidectomy</td>
<td>26</td>
</tr>
</tbody>
</table>
APPENDIX 10

The Norton pressure sore risk assessment score (Norton et al 1962)

<table>
<thead>
<tr>
<th>Physical condition</th>
<th>Mental state</th>
<th>Activity</th>
<th>Mobility</th>
<th>Incontinence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Good</td>
<td>4 Alert</td>
<td>4 Ambulant</td>
<td>4 Full</td>
<td>4 Net</td>
</tr>
<tr>
<td>Fair</td>
<td>3 Apathetic</td>
<td>3 Walks with help</td>
<td>3 Slightly limited</td>
<td>3 Occasionally</td>
</tr>
<tr>
<td>Poor</td>
<td>2 Confused</td>
<td>2 Chairbound</td>
<td>2 Very limited</td>
<td>2 Usually urine</td>
</tr>
<tr>
<td>Very bad</td>
<td>1 Stuporous</td>
<td>1 Bedfast</td>
<td>1 Immobile</td>
<td>1 Double</td>
</tr>
</tbody>
</table>

**Assessment of risk**

Under 14 = at risk

*Fig 1. The Norton score*¹²

The Norton score was designed to indicate elderly patients at risk of developing pressure sores. It is a numerical scoring system with five criteria: physical condition, mental state, activity, mobility and incontinence. One level is selected for each criterion and the numbers for all criteria are summed. A score of 14 or less is considered indicative of susceptibility to pressure sores with patients with a score of 12 or less being considered especially at risk.
## APPENDIX 11

List of abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>5-HT</td>
<td>5-Hydroxy tryptamine</td>
</tr>
<tr>
<td>APAM</td>
<td>Alternating pressure air mattress</td>
</tr>
<tr>
<td>BCAA</td>
<td>Branched chain amino acid</td>
</tr>
<tr>
<td>BP</td>
<td>Blood pressure</td>
</tr>
<tr>
<td>CI</td>
<td>Confidence interval</td>
</tr>
<tr>
<td>CUS</td>
<td>Compression ultrasonography</td>
</tr>
<tr>
<td>CV</td>
<td>Closing volume</td>
</tr>
<tr>
<td>DBP</td>
<td>Diastolic blood pressure</td>
</tr>
<tr>
<td>DD</td>
<td>D-dimer</td>
</tr>
<tr>
<td>DVT</td>
<td>Deep vein thrombosis</td>
</tr>
<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
</tr>
<tr>
<td>FRC</td>
<td>Fundamental respiratory capacity</td>
</tr>
<tr>
<td>Hb</td>
<td>Haemoglobin</td>
</tr>
<tr>
<td>IPG</td>
<td>Impedence plethysmography</td>
</tr>
<tr>
<td>LCCT</td>
<td>Liquid crystal contact thermography</td>
</tr>
<tr>
<td>LCG</td>
<td>Limited chairnursing group</td>
</tr>
<tr>
<td>LCP</td>
<td>Limited chairnursing policy</td>
</tr>
<tr>
<td>LRTI</td>
<td>Lower respiratory tract infection</td>
</tr>
<tr>
<td>mmHg</td>
<td>Millimetres of mercury</td>
</tr>
<tr>
<td>NS</td>
<td>Numerical scale</td>
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<tr>
<td>p</td>
<td>Potenz (power)</td>
</tr>
<tr>
<td>PCA</td>
<td>Patient controlled analgesia</td>
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<td>PE</td>
<td>Pulmonary embolus</td>
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<tr>
<td>POF</td>
<td>Post-operative fatigue</td>
</tr>
<tr>
<td>POMS</td>
<td>Profile of mood states</td>
</tr>
<tr>
<td>PR</td>
<td>Pulse rate</td>
</tr>
<tr>
<td>SAF</td>
<td>Self-assessed fatigue</td>
</tr>
<tr>
<td>SBP</td>
<td>Systolic blood pressure</td>
</tr>
<tr>
<td>SD</td>
<td>Standard deviation</td>
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<tr>
<td>SE</td>
<td>Standard error</td>
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<tr>
<td>Tc</td>
<td>Technicum</td>
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<tr>
<td>THR</td>
<td>Total hip replacement</td>
</tr>
<tr>
<td>TKR</td>
<td>Total knee replacement</td>
</tr>
<tr>
<td>UCG</td>
<td>Unlimited chairnursing group</td>
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<tr>
<td>UCP</td>
<td>Unlimited chairnursing policy</td>
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<td>UK</td>
<td>United Kingdom</td>
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<tr>
<td>US</td>
<td>Ultrasound</td>
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<td>USA</td>
<td>United States of America</td>
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<tr>
<td>UTI</td>
<td>Urinary tract infection</td>
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<tr>
<td>VAS</td>
<td>Visual analogue scale</td>
</tr>
<tr>
<td>VC</td>
<td>Vital capacity</td>
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<tr>
<td>ZCG</td>
<td>Impedence cardiography</td>
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