2011

Scald burn admissions to Princess Margaret Hospital in 1998 and 2008: A comparative analysis.

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Scald burn admissions to Western Australia's Princess Margaret Hospital in 1998 and 2008: a comparative analysis

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Date of submission: June 2011

USE OF THESIS

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Abstract

Scald is the most common form of burn in young children, accounting for over 50% of all paediatric burn admissions in Western Australia (WA). Interventions implemented over the past few years focussed on prevention, first aid, and improved services post-injury (Department of Health Western Australia, 2009). The population of children in WA is rising and an epidemiological study was undertaken to assess changes in the profile of scald injury among WA children and to assess priorities for future action.

Data were collected from all acute inpatient admissions presenting with scald injury for 1998 and 2008. These data were compared with state-wide admission data, local presentation data, population growth and shift within WA. Comparisons of types and severity of injury, treatments, healing, infection, remote and rural patients, first aid administered and by whom, and scar management were analysed.

The incidence of scald injury was not reduced, but numbers of admissions were, possibly due to changes in patient management. The admitted wounds were larger, but did not require more surgery in 2008. An important group to target for preventive strategies were the 1-2 year olds who were scalded by hot drinks. The amount of first aid given by carers had not improved, and inappropriate substances were applied to wounds by carers as first aid treatments in both years, often influenced by culture. Alterations in analgesic drugs and administration routes meant less intramuscular injections for all children. Dressing type changed with the adoption of Acticoat™ dressings. Preadmission infection rates seem to be influenced by delayed presentation, non-metropolitan living and Aboriginality. Post discharge infection rates have increased.

Several recommendations emerge from the data. Preventive strategies should target the most affected suburbs, injury in toddlers from hot drinks and community education promoting burns first aid. In hospital measures include the development of information packages for parents about pain and coping after a burn injury, and the provision of new distraction techniques, as well as the development of novel wound depth assessment tools. Further research is needed to investigate socio-economic risk factors and of how dressing use is associated with bacterial colonisation and infection. Data collection: The implementation of an outpatient data collection program and routine collection of scar data is necessary for on-going evaluation.

DECLARATION

I certify that this thesis does not, to the best of my knowledge and belief:

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ACKNOWLEDGEMENTS

I'd like to thank my supervisors, Associate Professor Jacques Oosthuizen and Mr Kim Clark of Edith Cowan University, and Professor Suzanne Rea of the Burns Service of Western Australia for their support and guidance whilst undertaking this work. I'd also like to thank Professor Fiona Wood for allowing me the opportunity to conduct this study, for the encouragement given and the means to present the findings.

CONTENTS

CHAPTER 1

INTRODUCTION

Epidemiological studies are crucial for determining priorities for burn prevention and treatment. A number of interventions introduced during the last ten years may have already changed the profile of burn injury admissions to Western Australia's only paediatric burn unit. However, the population is constantly growing as a result of migration, and the sub population of very young children will continue to expand over the coming years. The most common cause of burn injury in children is a scald burn; these are burns that arise from hot liquids specifically. It is these injuries that are considered in this study.

This study used hospital data to explore the profile of serious childhood scald injuries that occurred in Western Australia in both 1998 and 2008. The study examined the characteristics of admitted patients, the circumstances of injuries, the actions of carers' and the care received from non-specialist and specialist practitioners for these two 12-month periods. In doing so, the research revealed indicators of progress, areas where further change and research may be required, and targets that might be useful in shaping preventive strategies. The study was primarily a quantitative analysis of patient medical records, complemented by the interpretation of qualitative data.

Background

Worldwide, injuries result in the deaths of five million people each year, accounting for 9% of deaths (World Health Organisation, 2010). In 2004, among those aged less than 18 years, there were approximately 950,000 deaths due to injury, 60% of which were attributed to five causes; road traffic accidents, drowning, burns, falls, and poisonings. Thus, while the profile of injury deaths differs somewhat by age and country, burns are a leading overall cause of mortality (Yarrow, Moiemen, & Gulhane, 2009) in many countries, and accounted for approximately one-in-ten global childhood deaths in 2004 (World Health Organisation, 2008b).

While the extent of burn-related mortality among children is significant, for every death there are many more non-fatal cases, which collectively result in an extensive burden of pain and suffering and use of health-care resources. While the causes of non-fatal injury differ from their fatal counterparts, burn injuries are common in both outcome categories. Thus, in 2004, among children aged less than 15 years, burn injury accounted for 5.9% of all unintentional injuries and 152.7 per 100,000 hospital

admissions. While the exact cost incurred by unintentional childhood injuries is unknown, it has been estimated that in the United States alone, medical costs and losses in productivity as a result of injuries to children under 14 years old are approximately US$ 50 billion (World Health Organisation, 2008b). As 95% of burn injuries occur in low to middle income countries, the scale of the problem is clearly substantial. In light of this, the World Health Organisation has developed a plan to reduce the global burden of burn injury (World Health Organisation, 2008a).

A core problem in accurately interpreting the scope of the problem of burn injuries is the quality and completeness of data. This has led to a dearth of comparable studies and calls for standardised data to be collected on the issue (Burd & Yuen, 2005). Data problems have also resulted in some ambiguity about trends in burn injuries. There are some data from developing countries (Fadeyibi et al., 2011; Forjuoh, 2006) but data may not be comparable (Tourtier, Raynaud, Murat, & Gall, 2010). Data from the United States obtained between 1993 and 2004 showed a decline in emergency department presentations for burn injury (Fagenholz, Sheridan, Harris, & Pelletier, 2007). However, another recent epidemiological study presents a variable picture, with a decline between 1995 and 2000, then a plateau between 2000 and 2005 (Taira, Singer, Thode Jr, & Lee, 2010). Peck, Brigham & Patterson (2007) claim that this change is not due, as is widely attributed, to injury prevention campaigns, but are linked to societal changes that have reduced risk exposure. Several studies report current incidences in varying countries (Chipp, Walton, Gorman, & Moiemen, 2007; Farooq et al., 2011) some of which focus on children (Fadeyibi et al., 2011; Rawlins, Khan, Shenton, & Sharpe, 2007) however, among these analysis of trends has not been common.

In part, the problem of interpreting trends arises because burns are treated in many different settings which often depend upon place of residence (Australian Institue of Health and Welfare, 2005). For example, it is possible that the same category of burn injury might result in any level of care on the continuum from home based treatment without any medical attention, presentation for treatment at a primary care service, attendance at a hospital emergency service, or hospital admission. It is also possible that patterns of treatment seeking behaviour might change over time as a consequence of influences such as service availability, economic factors, and broader social norms with respect to treatment (Australian Institue of Health and Welfare, 2005; National Rural Health Alliance, 2002). Each of these alternatives has implications for the collection of injury data, and therefore quantification of the incidence of burn injuries is challenging. Thus, while data on deaths from burn injury appear likely to be relatively

complete, data on non-fatal burn injury do not. A Canadian study conducted injury surveillance followed a cohort of children for 5 years. They found that 9556 of 96 359 children sustained a burn injury in this time (9.9%) equating to one child in 10 sustaining a burn injury before the age of ten years (Spady, Saunders, Schopflocher, & Svenson, 2004).

The lack of adequate burn injury trend data applies to Australia. Streeton and Nolan (1997) reported a reduction in Victorian hospital admissions resulting from burns among children over a 25 year period; however this study is now dated and its scope was limited. A more recent Victorian burn injury study was undertaken by Wasiak et al. (2009), covering 2000-2007 admitted and non-admitted episodes of hospital treatment, and this indicated that there had been little change in burn-injury incidence over these seven years.

Despite the overall weaknesses in burn injury data and the gap in research on trends, Australian records on hospital admissions offer a useful descriptive picture of the causes of more serious events occurring across the nation. These data indicate that between 1999 and 2004, the Australia-wide admission rate for burn injury was 47.9 per 100,000 people (personal communication, Duke, 2010). Records on these injuries show that a third of these injuries are the result of scalds (34%) resulting from contact with fluids such as hot water or cooking oils. For children aged four years or younger, the hospital admission rate for burns over the same period was more than twice as high at 109.6 per 100,000 people, 63% of which were due to scalds (Australian Institute of Health and Welfare, 2006). Thus, children under five are at greater risk of hospitalisation as a result of scald injuries than older children.

Problem statement

Scald is a common cause of injury, especially for children less than five years old. These injuries result in short-term health problems but can also leave sufferers with scarring that may have lifelong physical and psychological effects (Falder et al., 2009; Phillips, Fussell, & Rumsey, 2007; Stubbs et al., 2011; ter Smitten, deGraaf, & Van Loey, 2011; Williams & Griffiths, 1991). While the incidence of hospital admissions due to scalds appears to have declined in WA over the last few years, the change seems likely to relate to a change in patient management rather than a change in incidence (Department of Health Western Australia, 2009). Consequently, the true picture regarding trends in serious scalds is unclear, leaving open the possibility that with population growth the WA public health system will be unprepared to deal with future demand for both short- and long-term childhood scald injury treatment services.

Professional significance.

This study attempts to clarify trends in scald injury among Western Australian children; a significant and costly category of morbidity and mortality among this segment of the community. Interpretation of the underlying trends in serious scald injuries among Western Australian children is necessary to clarify priorities for treatment and prevention services that are appropriate to current and emerging levels of need over the coming years. If incidence is unchanged then greater resources are required to manage these injuries, and effective prevention programs are required to target this group.

Overview of methodology

The methods used are described in a subsequent chapter of the thesis. Data were collected by scrutinising the hospital records of every patient (aged 0-15 years) admitted to PMH with scald injury for the calendar years of 1998 and 2008. The analysis was primarily quantitative and entailed use of both Predictive Analytic Software (PASW) version17 for windows, formerly Statistical Package for the Social Sciences (SPSS) and STATA MP 10. Where appropriate, qualitative case review data were used to supplement the quantitative analysis. The data collected were broadly grouped as follows:

1. The pre-hospital situation of scald injuries; the circumstances of the injury, causative agent, people present, severity, and initial treatment measures;
2. Management of the scald injury during the early period of medical intervention (usually by non-specialist medical staff or other health care practitioners) and up to admission to the specialist paediatric burns unit.
3. Specialist management of the burn injury, including the amount and nature of scar management required.

These categories were then used to guide the analysis of data

Objectives and research questions

The primary objective of the study was to assess changes to the profile of hospital admissions for children with scald injury in Western Australia between 1998 and 2008. Comparison data on scalds for this period from the only specialist paediatric burns unit in WA was used to interpret incidence and changes in patterns of treatment of children who have suffered this form injury. Changes that occurred across the time frame were also used to ascertain possible needs for further preventive or treatment interventions.

A secondary objective of the study was to assess whether there were specific sub-populations that were at higher risk for scald injury.

These objectives have not previously been addressed in Western Australian studies of scalds.

Question 1:

Did the epidemiology, treatment and outcomes of Western Australian paediatric scald injuries admitted to the Total Care Burns Unit, PMH in 1998 and 2008 differ?

Sub-questions:

During the study period was there: :

a reduction in the incidence of scald injuries among this cohort?

a change in the severity of scald injury?

a change in the type of scald injury presenting to hospital?

an improvement in first aid given to children with scalds during the pre-hospital period?

changes in the actions of the carers, present at the time of injury?

changes in medical treatment and nursing care of paediatric patients with scald injury?

Question 2:

Which target groups or focus areas do the data suggest might be appropriate for further resourcing and effort to reduce the incidence and severity of burns and associated scar outcomes from scald injuries among Western Australian children?

Limitations

The limitations of this study are that these are data from Western Australia, and may not necessarily be able to be generalised to other locations. The data was collected for PMH admissions only, and therefore represents a limited set of scald injuries. The latest data collected is from 2008 and therefore does not account for subsequent trends.

CHAPTER 2

LITERATURE REVIEW

This chapter explores the current state of knowledge about children and scald injuries, which has predominantly been derived from research into other populations. It reveals the norms and patterns of injury and treatment, whether Western Australia conforms to these, and where local data are lacking. It examines who gets scalded, how and why it happens, and the subsequent course of events. Chronologically, it explores the journey, from data such as ethnicity, age and gender, to modes, patterns and severity of scald injury, the type and duration of first aid received and the treatment received from both generalist and specialist health care professionals.

Scald injury in childhood: an overview of epidemiology and risk

The social, economic and geographic situation of Western Australia (WA) is unique. Seventy percent of its current population of 2.2 million people live in the capital city, Perth. The land area of WA spans over 2.5 million square kilometres, and occupies a third of Australia (About Australia Pty Ltd, 2011). Perth is the world's most isolated city of over 1 million people and is physically closer to Jakarta and Singapore than to the Australian capital, Canberra. Two thirds of the indigenous population of WA live in remote or rural areas (Australian Bureau of Statistics, 2006b). They are a population who not only have the disadvantage of greater distance and time delays to access specialist medical care, but also contend with substantial socio-economic disadvantage (Australian Institue of Health and Welfare, 2005). Distances from Perth to the State's regional centres and the subsequent distances of these centres to local communities creates a remoteness that is unusual, and that brings challenges to the provision of equitable care (National Rural Health Alliance, 2002). In 2001 (AIHW & ABS, 2005, p. 5) 45.1% of the Western Australian indigenous community lived in remote areas, and a further 22.8% lived in regional areas.

Low socioeconomic status is widely accepted to be a major risk factor for burn injury (Poulos, Hayen, Finch, & Zwi, 2007; Wood, La Hei et al., 2006). Health disparities between indigenous and non-indigenous people are well documented (National Health and Medical Research Council, 2010), and the socioeconomic disadvantage of the indigenous people increases their risk of exposure to behavioural and environmental risk factors, many of which increase the risk of burn injury, and in turn those factors can also be a risk factor for developing complications such as burn wound infection. For

example, in 2001 (AIHW & ABS, 2005) 10% of indigenous households in Australia were overcrowded, mothers are younger,have more children than non-indigenous families and housing lacks basic facilities. Overcrowding, poverty, not being fisrt-born, or not being the child of the head of the house-hold all increase burn risk (Delgado et al., 2002; Van Niekerk, Rode, & Laflamme, 2004). Weedon & Potterton (2011) report that burn risk increases with single parent homes, and this risk increases when working mothers leave their children in the care of elderly relatives.

It is widely reported that boys have a slightly higher incidence of scald injury than girls (Delgado et al., 2002; Van Niekerk et al., 2004). The age related pattern of scald injury is linked to the normal stages of child development (Flavin, Dostaler, Simpson, Brison, & Pickett, 2006). Scald injuries are most common in the under 5's, either by spill in the kitchen, or immersion/hot tap water injury in the bathroom (Lowell, Quinlan, & Gottlieb, 2008; Tse et al., 2006a). These often present as shoulder, trunk and upper limb injury in the case of spillage, and lower limb, buttock and genitalia burns injury in the bath (Drago, 2005).

Infants who have not yet learned to walk are at risk due to a variety of factors. Bottles warmed in jugs of hot water are a common mechanism of injury, as babies are often being held by the parent whilst the bottle is warming and might kick or grab the jug. Hot drinks spilt by carers whilst holding a baby are also a risk, which increases if the baby is unwell and needs constant comforting. As a baby becomes more mobile, and starts crawling, grabbing, pulling himself up onto his feet and toddling the dangers of grabbing a hot drink that previously would not have posed a risk increases. Low coffee tables provide an ideal height for accessibility to hot drinks, and an excellent surface for a young child to gain leverage to pull up to a standing position. (Burlinson, Wood, & Rea, 2009). Cognitive limitations can fail to distinguish the relationship between cause and effect. Concepts, such as temperature or extreme heat, are beyond the understanding of children less than two years old, who will try to categorise information within their limited realm of experience (Davies, 2011, p. 204). Thus the first encounter with very hot substances might be the catastrophic event. These toddlers like to imitate others, which increases the chances of grabbing a cup to copy adult behaviour.

Reports about temporal differences in scald injury vary. In South Africa, Van Niekerk, Rode & Laflamme (2004) reported an increase in burn incidence in the winter. This is supported by some other studies carried out in Sub-Saharan Africa (Albertyn, Bickler, & Rode, 2006). However, a study undertaken in Hong Kong saw no marked change

except for an increase in childhood burn admissions in the month of August, which corresponds to the school summer holiday period (Tse et al., 2006b). This was supported by a study in Taiwan (Tung et al., 2005). Han et al (2005) found no seasonal variation of note in South Korea. The times of day that burns most often occur correlate mostly to meal preparation time (Lin, Wang, Lai, & Lin, 2005). There is little information about variations in burn incidence across the week.

Scalds and burns: first aid recommendations

Appropriate first aid is aimed at cooling the burn to limit cell damage in the skin. Immediate cooling of the burnt area is recommended, and whilst the temperatures in the tissues remain higher than 44 degrees Celsius, the injury continues (Hartford & Kealey, 2007). The Australian and New Zealand Burn Association (ANZBA) recommends that the appropriate first aid following burn injury is twenty minutes of cool running water within three hours of injury (2010). This has been their recommendation since 1996. Moist compresses are a practical method of cooling with water in the ambulatory setting (Hartford & Kealey, 2007). It is important to maintain core body temperatures, and the care giver should be mindful of the fact that hypothermia can occur, particularly in children because of their high surface area to body mass ratio (Australia and New Zealand Burns Association, 2010, p. 66; Taira, Singer, Cassara, Salama, & Sandoval, 2010). The American Medical Association recommends that cooling should not extend beyond 30 minutes in duration for this reason (Pham, Gibran, & Heimback, 2007).

Extensive and recent work by Cuttle, Kempf, Lui, Kravchuk, & Kimble (2010) demonstrate that twenty minutes is the optimal duration for cool water application, that, if applied immediately, speeds healing in the first two weeks after injury. It is known that if healing occurs in the first two weeks after burn injury then scarring is less (Cubison, Pape, & Parkhouse, 2006; Deitch, Wheelahan, Rose, Clothier, & Cotter, 1983). Cuttle et al (2010) found that after this initial two week period, the rate of healing was unchanged. She deduced that the more superficial the burn, the greater the benefits of immediate and adequate cool water application and subsequent healing. Furthermore, the investigators reported that delayed application of cool water (3 hours following injury) has less effect during this time but improved healing rates at three weeks post injury compared to controls. In these cases there might have been a confounding effect of general anaesthetic on inflammatory mediators. Her work supports the recommendations of ANZBA, but emphasises the importance of immediate first aid for burn injury, and the importance of promoting these recommendations.

However, Cuttle et al (2010) were of the opinion that different durations and delays in the application of cold water did not influence scarring significantly. Scar assessment was undertaken at six weeks post-injury. However, scars can continue to mature for up to 15 months following injury (Gangemi et al., 2008), so assessment at six weeks would be inconclusive. . Improving scar outcome is a long-term treatment aim in the management of burn injuries and any difference in the cosmetic appearance of an injury is of key importance. Deitch (1983) states that the longer the wound takes to heal, the greater the probability of developing hypertrophic scarring. This theory is supported by more recent studies (Cubison et al., 2006) and a retrospective review of 127 scars indicate that this might be linked to bacterial colonisation (Baker, Townley, Mckeon, Linge, & Vijh, 2007).

There are many different substances other than water that are applied to burn wounds in the first few minutes following injury and the choice of agent is often cultural in origin (Skinner, Brown, Peat, & Muller, 2004; Tse et al., 2006a). Aloe Vera, saliva (Cuttle, Kempf, Kravchuk, George et al., 2008; Seoighe, Baker, & Conroy, 2011), ink, toothpaste, and a selection of handy items from the kitchen cupboard such as oil, soy sauce, honey, eggs and butter are common choices (Cuttle, Pearn, McMillan, & Kimble, 2009). Over a century ago it was suggested by Martin that the elimination of air from the wound surface is beneficial, and that any available agent to achieve this will benefit healing (cited by Cuttle, Pearn et al., 2009) and whilst the absence of air on a wound surface has an analgesic effect, so does cooling the wound. Pain following burn injury often motivates cooling. Immediate first aid often ceases when the pain is more manageable (Cuttle, Pearn et al., 2009; Taira, Singer, Cassara et al., 2010). The analgesic effect can give the impression that the application of a substance is beneficial when it can actually be deleterious. Some substances applied to an acute burn wound, such as ice, might deepen the injury.

The use of ice to cool wounds is not recommended (The Australian and New Zealand Burn Association Limited, 2010). It is very effective in reducing pain, and therefore intuitively thought to be appropriate. The use of ice is purported to reduce oedema formation and early studies from the 1960s showed that a benefit of ice is reduced need for narcotic and sedative medications. Cuttle (2009) also cites Fay (1959) who claimed that use of 'local refrigeration' was bacteriostatic. Now, it is considered that ice will constrict the microcirculation and extend the zone of stasis, therefore increasing the size and depth of the burn (Venter, Karpelowsky, & Rode, 2007). One recent study showed that ice application did not hinder healing, but neither did it help (Cuttle, Kempf, Kravchuk, Phillips et al., 2008). In addition, the effects of ice application might be

compounded by the pressure with which the ice is applied. The associated risk of causing hypothermia is an important issue, particularly in children because of their high body surface area relative to mass, or in larger burn injuries where the area to be cooled is more extensive (Cuttle, Kravchuk, Wallis, & Kimble, 2009).

Skinner and Peat (2002) assessed the first aid received by 165 adult and paediatric patients who were admitted with a burn injury in New Zealand. Approximately 40% had received adequate first aid, and the impact of this was particularly notable within the group that had sustained a scald burn. There was a statistically significant reduction in the rates of split skin graft procedures between those who had adequate burns first aid and those who did not. The adequacy of first aid in this paper was defined as ten minutes of cool running water (as from a 1994 education campaign), not twenty, so a greater proportion of patients would have been categorised to the 'adequate' group than is appropriate by current recommendations.

Early first aid is important to minimising the effects and scarring of burns. This emphasises the need for correct and adequate burns first aid to be common knowledge in the community, especially for parents of young children. The internet is a common source of public knowledge. Tiller, Rea, Silla & Wood (2006) assessed the quality of burns aid information available on the internet. Forty-seven websites were evaluated for the accuracy and quality of their content, and ranked on a five point scale from poor, through fair, good and very good, to excellent. None of the websites achieved a ranking in the 'excellent' category and 77% (36/47) ranked as 'poor'. Thus first aid advice retrieved from the internet is often incorrect.

Although internet access is available across Australia, the quicker broadband access is not. While the Australian Government have plans to increase availability, this major undertaking starts with five selected sites in Australia, none of which are in Western Australia (NBN Co Ltd, 2010). At the time of writing, the roll out of broadband access in WA is predicted to be eight years away at best, and then there will still be areas not served. So, in some areas, gaining information in an emergency situation would be slow and cumbersome and not a viable option.

For those who have access; the type of information sought on the Internet varies according to socioeconomic status (SES) (Zillien & Hargittai, 2009). A recent German study showed that the percentage of German users that accessed health information was only 28.5% in the lowest SES class and 45% in the highest class. It is likely that this European population does not compare to the population of Western Australia. As lower socio economic status is associated with a higher rate of burn injury, the

population most likely to sustain burn injuries are least likely to use the internet as an advice tool. Thus, burns first aid information given on the Internet, is poor in quality, and unlikely to reach to the population in need of education.

Burns first aid knowledge needs to be known in advance of injury, as it requires immediate attention. Thus, carers whose children have sustained a scald injury will act as they think best at the time. Seeking this information before an injury occurs would show an awareness of risk, and this awareness would lessen the risk itself.

Awareness of correct burns first aid does not ensure compliance. Knowing what to do does not ensure people do what they know. Cooling is often stopped for transfer to medical attention. The application of wet dressings for transfer can be ineffective as the swabs soon warm and the effects of cooling are negated (Taira, Singer, Cassara et al., 2010). The actions of the carers in the provision of first aid are important, and equally, so is their choice of subsequent action. Speed to medical attention, and the actions of the first medical personnel to attend the child will impact on the initial experience of a traumatic event and the outcome (Tiller et al., 2006).

Scalds and burns: physiological effects of injury and initial care

It is essential that wound size is accurately assessed by medical personnel. The estimated total body surface area burned (TBSAB) underpins fluid resuscitation calculations. If wrongly estimated, under or over hydration occurs, and both these situations can be detrimental to the child. Commonly small burns are over estimated and large burns are underestimated. The variability between practitioners' estimation of burn size increases with burn size initially, plateaus in large burns and the decreases slightly in extensive burns (Wachtel, Berry, Wachtel, & Frank, 2000). Paediatric burn size is estimated using the Lund and Browder chart (Appendix 1), and for small burns the patient's palm size is regarded as being equivalent to 1% body surface area. Erythema, which is a superficial reddening of the skin caused by dilation of the blood vessels, is not included in the TBSAB. These areas can develop into deeper burns or can resolve. This is due to an inflammatory response on the microcirculation that is unique to burn injury (Kramer, Lund, & Beckum, 2007, p. 93). Appropriate wound cooling and acute management of the injury can limit this process by stabilising the mast cells in the skin, thereby reducing histamine release, and oedema (Hartford & Kealey, 2007).

When tissue is damaged by burn, there are three zones of injury apparent. Jackson first described the three zones in 1951, and this seminal work has remained the model of burn injury (cited in Shupp et al., 2010). Centrally, there is a zone of necrosis, which

is not salvageable. Surrounding this is the zone of stasis, which is the area of tissue that is potentially salvageable. The goal of treatment is to salvage this zone, which can either progress to areas of deep burn injury or improve and heal. The progression of a burn injury in this way is known as 'conversion' and is due to cell death (Shupp et al., 2010). The third area, between this and the unburnt skin is the zone of hyperaemia. This third zone is the tissue that will recover unless complicated by infection or severe hypoperfusion (Pham et al., 2007, p. 120), In a burn of approximately 25%, the zone of hyperaemia can extend throughout the whole body (Australian and New Zealand Burns Association, 2010b).

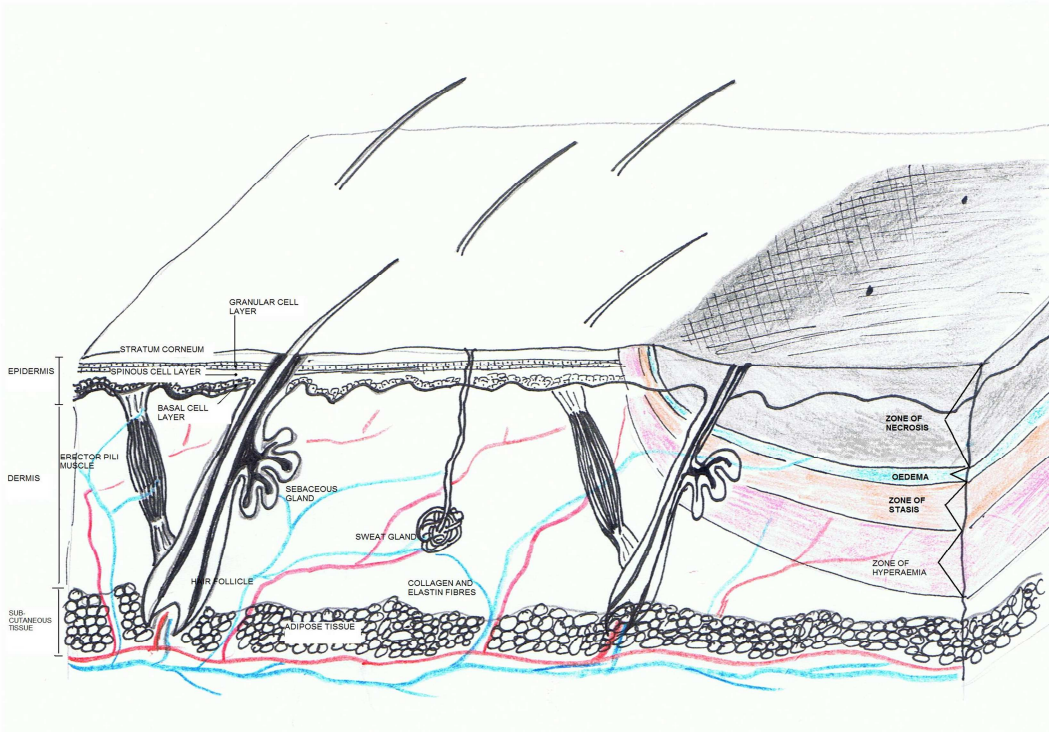
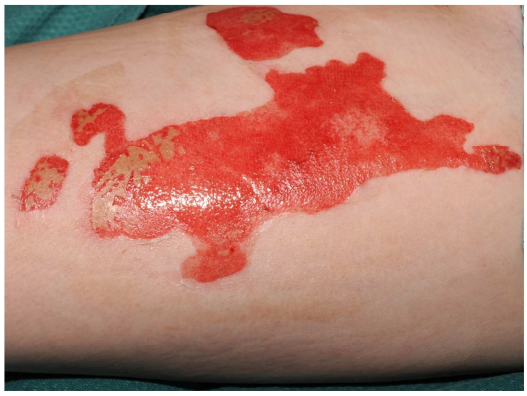


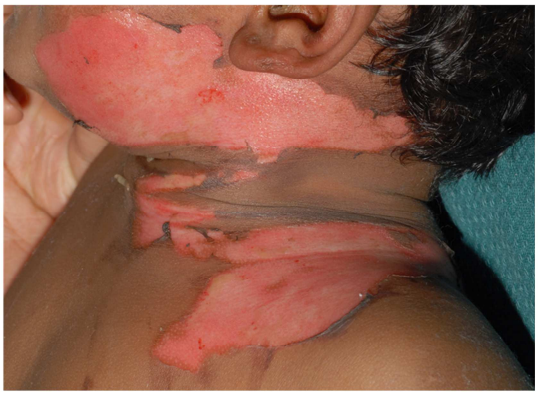
Figure 1: Skin anatomy showing Jackson's burn model (Shupp et al., 2010).

Table 1: Clinical diagnosis of burn depth (EMSB Course Manual, 14th edition)

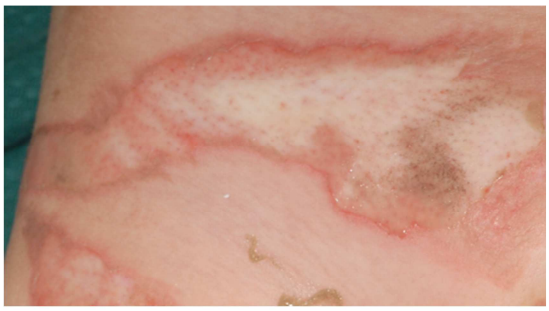
Depth	Colour	Blisters	Capillary refill	Sensation	Healing
Epidermal	Red	No	Present	Present	Yes
Superficial	Pale pink	Small	Present	Painful	Yes
Mid-dermal	Dark pink	Present	Sluggish	+/-	Usual
Deep dermal	Blotchy red	+/-	Absent	Absent	No
Full thickness	White	No	Absent	Absent	No



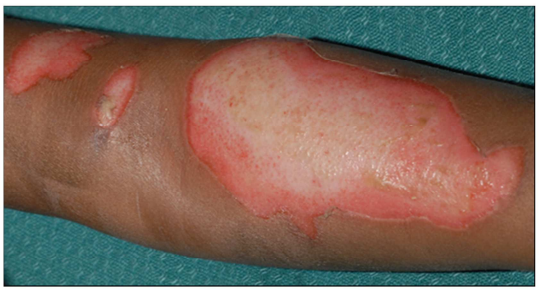
Case 1: Hot water*



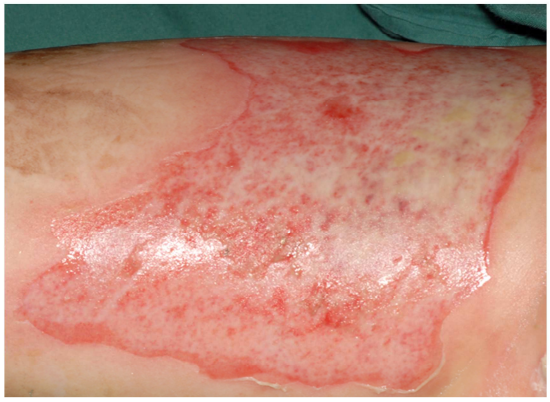
Case 2: Scald from hot tea*



Case 3: Spill from hot noodles*



Case 4: Forearm scald from coffee*



Case 5: Spill from soup*

*(PMH Medical Illustrations, 2011) All photographs used with parental informed consent

The examples of paediatric scald shown above demonstrate how scald wounds vary in depth. The whiter areas show greater burn depth, and case 5 demonstrates the blotchiness of a deep dermal burn. Apart from case 1, which healed without the need for surgery, all had surgical intervention to close the wound. The optimal time for burn assessment is at 48 hours post-injury because of the potential improvements or worsening of the areas of erythema. It is clear that the assessment of burn depth is

closely linked to TBSAB. Traditionally depth has been assessed by clinical methods. A recent study by Bloemen, van Zuijlen & Middelkoop et al (2011) indicates that experienced practitioners make accurate assessments of burn wound healing potential and the potential success of skin graft take, and that correct assessment correlates to practitioner experience.

The type of scald agent and how the injury occurs are relevant to the depth and pattern of injury to the child. The more viscous the agent is, the more it adheres to the skin and the longer the removal time. Water will run off the skin readily, whereas soup or noodles will have a longer contact time. The longer the contact time the deeper the burn, depending on temperature of the agent. The amount of heat energy in the agent depends on the source of the agent and time elapsed since removal from source. A burn sustained from a hot liquid is described as a scald injury, a burn from a solid is described as a contact burn injury. Debate will continue about whether a burn is classified as a scald or contact burn depending on the viscosity of the agent, which can run on a continuum from liquid to solid, and may be dependent on heat to be a liquid. Some substances might be liquid when hot, such as toffee or wax, but solid when cool; cooling occurs following impact to the skin, when heat is transferred.

Laser doppler imaging has provided a validated and accurate method of depth assessment, but still should be done at 48 hours post burn (Holland, Ward, & Farrell, 2007; Merz, Pfau, Blumenstock, Tenenhaus, & Rennekampff, 2010; Nguyen, Ward, Lam, & Holland, 2010) although some argue that 24 hours post burn will also give an accurate prediction of burn depth (Mill, Cuttle, Harkin, Kravchuk, & Kimble, 2009). This scan detects vascular flow, and works on the doppler principle that frequency shift occurs when light waves are reflected by moving objects, in this case red blood cells (Chatterjee, 2006). In a superficial burn, where the skin integrity is just breached but the microvasculature is largely intact, the vascular flow is high. As burn depth increases, the vessels are destroyed, and the vascular flow progressively decreases. The laser doppler image displays these changes via a continuum through the spectrum from red, indicating superficial injury, to blue, indicating full thickness injury. This is particularly useful for scald injury, which can be of varying depth. The advantage of laser doppler scanning is that this is a non-invasive, non-contact method of assessment, that is able to assess large areas to determine surgical need (McGill, Sorensen, MacKay, Taggart, & Watson, 2007). The disadvantage of using this method is that the child needs to be stationary with a debrided, uncovered and painful wound for the duration of the scan for clear and accurate imaging.

Appropriate and adequate fluid resuscitation is essential after a burn injury to prevent burn shock. Globally, current recommended guidelines use the Parkland formula to calculate fluid requirements (Alvarado, Chung, Cancio, & Wolf, 2009; Baker, Akhavani, & Jallali, 2007) and in Australia and New Zealand, a modified version of this is used (Australian and New Zealand Burns Association, 2010b) (Appendix 2). This uses a crystalloid, Hartmann's solution, and volumes are based on the patient's weight and the percentage TBSAB burned. A calculated volume is given in the 24 hours from the time of the injury, with 50% administered in the first eight hours and the next 50% in the remaining 16 hours. The calculations are made from the time of injury, so greater volumes might be infused for the first 8 hour period in order to 'catch up' the fluid lost.

In children, the comparatively high body surface area to mass ratio lowers the threshold for fluid resuscitation requirement. Fluid resuscitation is necessary in adults if the burn is greater than 15%, but in children the threshold is much lower. A child over 18 months old requires fluid resuscitation for a burn injury greater than 10% TBSAB and for those younger than 18 months old, the threshold is 8% TBSAB. Maintenance fluids are also given concurrently. Problems can occur from over-hydration or under-hydration if the TBSAB is inaccurately calculated. Commonly burns under 20% are over estimated and those under 20% are underestimated (Collis, Smith, & Fenton, 1999). Benicke et al (2009) maintain that fluid requirements are more complex and individual than is recognised by the Parkland formula. Concurrent systemic decompensation or acidosis may result in the need for an immediate response in fluid therapy (Alvarado et al., 2009).

Concurrent physiological mechanisms occur in severe burn injury. Multiple mediators of inflammation affect the microcirculation and combine with hypovolaemia and other systemic factors that depress the myocardium. This increases both pulmonary and systemic vascular resistance and exacerbates the systemic inflammatory response, which escalates into a vicious circle leading to organ death (Kramer et al., 2007, p. 93).

Urine output measurements are the best evaluation of adequate fluid resuscitation. Monitoring is best achieved with hourly urine output measurements via an indwelling urinary catheter. Early insertion of a urinary catheter is recommended if fluid resuscitation criteria are met. Monitoring fluid status via the vital signs is unreliable in burn injury for several reasons, especially in children. Children and young adults have good compensatory mechanisms and intravascular fluid volumes must deplete vastly before hypotension is evident. Therefore, hypotension is not a warning sign of imminent hypovolaemia, but an indicator that significant and damaging hypovolaemia

has already occurred. Catecholamines are released by the stress response; maintaining normotensive blood pressures despite reduced blood volume. Catecholamines cause peripheral vasoconstriction which results in radial intra-arterial blood pressure monitoring misrepresenting the central vascular pressures by producing reassuringly high readings in the face of emergent hypovolaemia (Wheeler, Wong, & Shanley, 2009a, p. 91). Tachycardia, another sign of reduced circulating volume will be present due to pain. Thus urine output is currently the best measure of adequacy of circulating volume, and low urine output, or oliguria, indicates inadequate fluid resuscitation. The aim is to achieve a urine output of 0.5-1ml/kg/hour (Australia and New Zealand Burns Association, 2010).

Children require close monitoring of blood sugar levels. Hypoglycaemia is a risk; children have high energy needs, compounded by limited glycogen stores leading to reduced reserve for gluconeogenesis. Dilutional hyponatraemia can also occur, and therefore intake of free water should be limited and carbohydrate given. This is why glucose is added to intravenous fluids, but it is beneficial to begin enteral feeding early (Australian and New Zealand Burns Association, 2010b). Overestimation of burn size leads to over-hydration and can be a problem in children if the TBSAB is not accurately reassessed in a timely manner. Routine addition of glucose to infusions to meet the additional energy requirement in children can lead to cerebral oedema if volumes are given excess to requirement.

Hypovolaemia occurs because burn injury triggers the release of inflammatory mediators such as histamine, serotonin, prostaglandins, thromboxane, and nitric oxide (Wheeler, Wong, & Shanley, 2009b, p. 125). These cause an increase in vascular permeability, resulting in an almost immediate shift of fluid into the interstitium. As proteins shift into the interstitial space, a reverse osmotic gradient causes more fluid shift to correct the pressure difference, and further oedema occurs. The burn injury also denatures collagen fibres, causing expansion of the third space and resulting in greater negative pressure gradients(Santos et al., 2000). These processes peak at 6-12 hours post burn, which is when the capillary barrier starts to recover. The initial increased vascular permeability precludes the use of colloids in the initial resuscitation period; as large protein molecules would filter into the interstitium, increasing osmotic pressure and drawing more fluid from the circulation into the tissues, worsening oedema and intravascular hypovolaemia (Baker, Akhavani et al., 2007). Lymph vessels become blocked with platelets, leucocytes and erythrocytes, hindering drainage of the tissues back into the venous circulation. Oedema volumes maximise at about 24 hours post injury, and begin to resolve after 1-2 days (Tricklebank, 2009).

In addition, oedematous burnt limbs can develop compartment syndrome, requiring close observation for early intervention. Limbs must be elevated and assessed for circulatory compromise, particularly if the injury is circumferential or near-circumferential. Therefore loss of pulses in a limb can be due to the development of compartment syndrome secondary to over-hydration or peripheral vascular shutdown due to under-hydration.

Scalds and burns: understanding and controlling pain

Adequate assessment of pain following burn is multi-faceted. Deeper wounds are less painful due to the destroyed nerve endings, and conversely, the more superficial the scald injury, the more painful, due to exposure of raw nerve endings to the air or dressing. So pain is often inversely related to depth, but it also is not always directly related to burn size (Difede, Jaffe, Musngi, Perry, & Yurt, 1997). Acute burn pain is burning, smarting or pricking in nature, and can be agonising in intensity (Tengvall, Wickman, & Wengstrom, 2010). Hyperalgesia is an increase in sensitivity to pain, and can be primary, directly involving the traumatised tissue, or secondary, involving tissue that has not had direct damage. Both types occur following burn injury, but in the acute stage it is primary hyperalgesia that requires attention (Summer, Puntillo, Miaskowski, Green, & Levine, 2007).

Feelings of helplessness increase pain severity in children and adolescents (Tremblay & Sullivan, 2010). Catastrophising and seeking emotional support results in lower pain tolerance, and feelings of helplessness are an element of catastrophising pain (Lu, Tsao, Myers, Kim, & Zeltzer, 2007). Patterson et al (2007) claim that in adults pain experience in acute burn injury was a better indicator of prolonged adjustment times following injury than size of the burn or length of stay in hospital. This has been demonstrated in other types of painful experience in different situations and other age groups. For example, inadequate analgesia in procedures on neonates have been shown to have had long term effects alterations in their pain coping and perception. A second is example is that post traumatic stress disorder is more common in those who have experienced pain (Zempsky, Cravero, & Committee on Paediatric Emergency Medicine and Section on Anaesthesiology and Pain Medicine, 2004).

Convincing health care professionals that good pain management in the acute stages assists recovery and long term management is difficult; acute pain is seen as a temporary problem and the reluctance of health care professionals to use opioid analgesia has long been noted (Patterson et al., 2007). Accurate pain assessment at triage, and regular reassessment, with appropriate analgesic intervention should be

intrinsic to emergency department care, and is a key factor in patient and carer satisfaction (Joint Commission Resources & The American Academy of Pediatrics, 2010).

Various interventions improve the pain experienced. Cooling the wound reduces pain whilst cooling continues, although will not improve ongoing pain (Werner, Lassen, Pedersen, & Kehlet, 2002) and covering the wound reduces acute pain by occluding contact with air, which contributes to mechanical primary hyperalgesia. Analgesia is imperative. It needs to be fast-acting, administered without needles, and preferably have anxiolytic effects (Finn et al., 2004).

Pain is categorised into three types, each of which occurs at three different time points. Background pain is the pain felt at rest, breakthrough pain is the pain felt on movement, and procedural pain is the pain felt at dressing change. These occur and differ at the acute phase, the healing phase and the rehabilitation phase (Summer et al., 2007; Tengvall et al., 2010). Ongoing pain is frequent and neuropathic in nature. Burning, stabbing, shooting pains and 'pins and needles' are triggered or worsened by temperature, touch, position and weight bearing activities for up to 15 months after injury (Schneider et al., 2006).

Coping mechanisms for pain tolerance in children vary. Lu et al (2007) identify positive self statements and behavioural distraction as pain-resisting coping mechanisms. Poor pain control during oncological procedures leads to higher pain scores in further procedures (Zempsky et al., 2004) and this situation occurs in acute burn care (De Jong, Middelkoop, Faber, & van Loey, 2007). Distraction therapies for pain control has recently expanded, with successful use of virtual reality distraction (Hoffman et al., 2006; Rutter, Dahlquist, & Weiss, 2009; Schmitt et al., 2011). This has been explored for control of procedural pain in paediatric burn injury with a multi-modal distraction device (Miller, Rodger, Bucolo, Greer, & Kimble, 2010) and in physiotherapy for paediatric burn injury (Schmitt et al., 2011). Hypnosis has been used as supplementary as a successful adjunct to traditional analgesia in adult burn dressing changes (Berger et al., 2010). A systematic review of non-pharmacological interventions for pain relief in adult burns concurs with this; active hypnosis together with rapid induction analgesia, and distraction techniques were found to be the most effective (De Jong et al., 2007).

Gender differences have been noticed with procedural pain. In adults, females report higher levels of pain, but in children differences are minimal and seem to emerge during adolescence. The cause of these differences is not established but may have

both hormonal and neurobiological origins (Fillingim, King, Ribeiro-Dasilva, Rahim-Williams, & Riley III, 2009).

Pain assessment tools vary depending on the age and capabilities of the child. For children aged 2-7 the Face, Legs, Activity, Cry and Consolability Pain Scale is appropriate (FLACC) (Merkel, Voepel-Lewis, & Malviya, 2002), and for children aged 0-23 months the Children and Infant's Postoperative Pain Scale (CHIPPS) is suitable (Buttner & Finke, 2000). The Revised Faces Pain Scale is the correct pain assessment tool for older children (Stinson, Kavanagh, Yamada, Gill, & Stevens, 2006). It has been recognised that the perception of pain by others is influenced by how the pain is expressed. Pain can be expressed in two main ways; by involuntary, reflexive actions and by intentional, purposive reactions, The former elicits an immediate emotional response, whereas the latter creates thoughts about the nature and origins of the pain (Craig, Versloot, Goubert, Vervoot, & Crombert, 2010).

It is important that parents and carers understand the ongoing nature of pain after burn injury, and advice given. Rest, massage, affected limb elevation and pressure garments may help (Schneider et al., 2006). The problem is often associated with itch. Itch occurs in the acute stage of injury and as a chronic problem post- burn (LaSalle, Rachelska, & Nedelec, 2008). Itch is caused by a range of pruritogenic molecules (Brooks, Malic, & Judkins, 2008) and the mechanisms are not fully understood (Falder et al., 2009). It is associated with inflammation and scarring (Brooks et al., 2008) and adds the risk of external damage due to scratching (Casaer, Kums, Wouters, Van den Kerckhove, & Van den Berghe, 2008). Advice needs to be given on the pharmacological and non-pharmacological management of both pain and itch.

Scalds and burns: infection risk and patterns

The breach of intact skin by burn injury reveals an excellent microenvironment for bacterial growth; a moist, warm wound bed, possibly with necrotic tissue or eschar, that is open to opportunistic pathogens. Wound infection is a risk for all burn patients and strategies to prevent infection are a foundation of treatment. Peck et al (1998) describe the diagnosis of burn wound infection as a "subjective art". They propose definitions for the purpose of surveillance rather than clinical use, which are designed to eliminate the inclusion of false-positives (Appendix 3). The authors emphasise the need for clinical judgement in the diagnosis of burn wound infection.

Much of the published data on infection in burns patients has been analysed in an adult population with severe burn injury who have had invasive treatment. These factors

mean that systemic infections have predominated. The risk of burn wound infection increases with severity of burn. The immunosuppressive effects of large burn injury are well documented (Edwards-Jones & Greenwood, 2003; Pruitt, McManus, Him, & Goodwin, 1998) and increase the burns patients' vulnerability to infection.

Initial colonisation of gram positive organisms such as *Staphylococcus aureus*, *coagulase-negative Staphylococcus*, and *Enterococci* is replaced by predominantly gram-negative organisms after approximately a week post injury. This transition is well documented. (Lari & Alaghehbandan, 2000); (Edwards-Jones & Greenwood, 2003) . Tropical climates favour the growth of *Acinetobacter baumanii* (Chim, Tan, & Song, 2007). *Escherichia coli* was the second most common bacteria associated with burn wound infection in Switzerland (Guggenheim et al., 2009). Increasing resistance of staphylococcus continues to emerge (Taneja, Emmanuel, Chari, & Sharma, 2004). If clinical infection occurs, wound closure is delayed and the use of antibiotics may lead to colonisation by antibiotic resistant flora, yeasts and fungi (Weber & McManus, 2004). Advances in topical wound treatment, with the use of antimicrobial dressings, have helped suppress the multiplication of organisms isolated in burn wounds (Neely et al., 2009).

While much has been written about burn injury infection in adults, there is far less literature about the incidence of infection in children, particularly in relation to the diverse population characteristics and distribution in Australia. However, age is a known risk factor in burn wound infection. Pruitt (1998) attributes the greatest risk to children, and the elderly at intermediate risk; although Edward-Jones et al (2003) claims that the reverse is true. Appelgren, Bjornhagen, Bragderyd, Jonsson & Ransjo (2002) also attributed old age as a risk factor for burn wound infection. Which population is at greater risk is an unnecessary argument; vigilance is required with all.

Severity of burn injury is the biggest risk factor for the development of wound infection (Appelgren et al., 2002; Wong, Tan, Ling, & Song, 2002). Burn injury greater than 30% TBSAB (Pruitt et al., 1998) and the presence of full-thickness burns (Wong et al., 2002) are significant factors. The longer the wound remains open, the greater the chance of colonisation, and once colonised, the greater the chance of overt infection. The larger wounds are often deeper, and so remain open for longer. Other factors intrinsically linked, that are also risk factors in themselves are that more severe burns require more surgery, longer hospital stays, more invasive monitoring, and increased likelihood of the patient requiring mechanical ventilation. These are all associated with the development of infection (Easterlow, Hoddinott, & Harrison, 2011; Weber & McManus,

2004; Wong et al., 2002), especially when coupled with physiological changes such as immunosuppression. Weber and McManus (2004) state that urinary catheterisation is a risk factor for UTI, which may progress to a systemic infection and that in paediatric patients nosocomial UTI is probably only found in patients with urinary catheters (Wong et al., 2002).

A variety of issues involving the wound microenvironment and the characteristics of the colonising bacteria are key to whether colonisation progresses to infection. Wound temperature and dryness, secondary impairment of blood flow and acidosis can combine to provide an ideal medium for the proliferation of bacteria. Polymicrobial colonisation, virulence and motility of the infecting organism, antibiotic resistance and the extracellular products produced by the organism are key factors for the development of burn wound infection (Edwards-Jones & Greenwood, 2003: Appelgren et al., 2002).

Pseudomonas aeruginosa produces endo-toxins and exo- enzymes that break down the wound as they degrade a path to the blood supply and systemic sepsis can follow (King, Croft, Ward, Williams, & Sockett, 2003). *S. Aureus* can produce exotoxins that can progress to cause toxic shock syndrome (Edwards-Jones & Greenwood, 2003). *Streptococcus pyogenes* can cause graft failure, necrosis and is the causative agent behind necrotising fasciitis (Edwards-Jones & Greenwood, 2003; King et al., 2003). *Bacillus cereus* can also cause wound degradation, systemic sepsis and markedly increased wound pain (Attwood & Evans, 1983) although an Italian study by Damgaard et al (1997) found that some of the bacteria thought to be bacillus cereus turned out to be *Bacillus thuringiensis*, which is known to produce endo-toxins. Bacillus cereus is a type of bacteria responsible for food poisoning, whereas *B. thuringiensis* is used as an agent for agricultural pest control. The latter was found in the water used to treat burn injury in the hospital.

Prior use of antibiotics is a known risk factor for the development of wound infection, particularly with multi-resistant organisms (Weber & McManus, 2004; Wong et al., 2002). Vancomycin or third generation cephalosporin use is a risk factor for infection with *vancomycin-resistant enterococci* (VRE) (Weber & McManus, 2004); this was also a significant factor for the development of *A. baumannii* (Wisplinghoff, 1999). Previous skin infection in the past year (OR 7.4), skin infection in a household member (OR 3.6), nasal colonisation (OR 3.7), and nail biting (OR 3.0) were identified as significant risk factors for the development of a skin or soft tissue infection with *methicillin-resistant Staphylococcus aureus* (MRSA) in children in the United States (Fritz, Epplin, Garbutt,

& Storch, 2009). A UK study that investigated toxin producing strains of *S. aureus* also found that nasal colonisation of the same strain was found to cause wound infection (Khojasteh, Edwards-Jones, Childs, & Foster, 2007).

Pre-existing disease is a further factor for the development of infection (Edwards-Jones & Greenwood, 2003; Pruitt et al., 1998). Systemic physiological changes integral to conditions such as diabetes, circulatory and neurological disorders can impact on susceptibility to infection for a variety of reasons. Lower socioeconomic status has been associated as a significant risk factor for both burn injury and the risk of infection (Forjuoh, 2006).

Rapid wound closure is the key to optimal burn care, and as well as reducing infection risk, it helps to minimise scar outcome (Deitch et al., 1983). Before the achievement of wound closure it is essential to protect against infection. Firstly, protection of the moist wound bed from contamination by opportunistic pathogens is achieved by adequate wound covering and the use of aseptic techniques, and secondly it is necessary to eliminate any pathogens that have accessed the wound. A wound dressing that enables the wound bed to stay moist is necessary to the healing process (Hartford & Kealey, 2007). Moisture allows the delivery of oxygen and nutrients to the wound surface, and allows the migration and spread of epithelial cells. It increases re-epithelialisation by 30-50% and collagen synthesis by about 50% and reduces surface inflammation. Devitalised tissue is removed by surface proteases. In dry wound healing, where a scab (containing fibrin, dead neutrophils and other debris) is allowed to form, the keratinocytes use proteases to cut through viable tissue beneath the scab to allow healing.

Antimicrobial dressings are fundamental in maintaining an infection free wound. These need to have a broad-spectrum of activity against pathogens, penetrate eschar with little systemic absorption and must not inhibit healing. Silver is toxic to all bacteria and fungi, including MRSA and VRE (Atiyeh, Costagliola M, Hayek, & Dibo, 2007), and has a broad spectrum of activity which is effective against gram negative and gram positive bacteria (Fong, Wood, & Fowler, 2005). Preparations can be metallic, nanocrystalline and ionic. There are many different wound dressings that contain silver (Patel, Vasquez, Granick, & Rhee, 2008).

Silver sulfadiazine (SSD), a cream formulation introduced in 1968 is a combination of silver nitrate and the antibiotic, sodium sulfadiazine. Silver nitrate had previously been used as a topical agent in wound care. The maximum concentration of this substance is a 1% solution or cream, because nitrate is cytotoxic and can slow healing. (Atiyeh et

al., 2007). Silver sulfadiazine transformed the outcomes of paediatric burn patients at risk of *Pseudomonas* septicaemia at 2 weeks post burn injury (Burd, Chiu, & Huang, 2008). This preparation was combined with chlorhexidine in Australia in response to an outbreak of *Staphylococcus*. The application of this cream causes the formation of a pseudo-eschar. There are two schools of thought on this. Some believe that the pseudo-eschar needs removal, thereby creating longer, more painful dressing changes while others question the need to remove the pseudo-eschar, claiming that leaving it intact has the benefit of providing a barrier to colonising bacteria (Burd et al., 2008). However, the pseudo-eschar is produced by the removal of surface moisture and therefore does not provide a moist wound healing environment. This means that the rationale given by Burd et al, is similar to the out-dated rationale for the use of wound exposure as the method of choice for wound healing. Before the advent of antibiotics, both systemic and topical, the formation of a 'scab' was beneficial for infection control, in a time when mortality from sepsis secondary to burn wound infection was high.

The nanocrystalline form delivers silver more rapidly to the wound. There are a variety of new silver dressings that contain the silver within the dressing, some of which work by delivering the silver onto the wound so that the bactericidal properties occur at patient level and others lift the bacteria into the dressing, so the silver kills bacteria at dressing level. Acticoat™ (Smith and Nephew, UK) is a nanocrystalline silver dressing that delivers silver with an initial bolus followed by a sustained release over three days. This offers the advantage of reducing painful dressing changes to every third day, not daily, as is necessary with SSD dressings. There are reports that nanocrystalline silver prolongs inflammation and healing, and therefore should not be used in non-infected wounds (Widgerow, 2010). Acticoat™ stains the unburnt skin, and is not suitable for areas, such as the face.

In Western Australia, several risk factors for the development of wound infection are present, particularly in rural areas. High ambient temperatures, high ground water temperatures, long transfer times, unusual native bacteria and social circumstances conspire to produce circumstances that increase the likelihood of an acquired wound infection (Kienzle, Mullera, & Pegg, 2000). Colonisation is a risk before arrival at PMH, and therefore the application of silver at the earliest opportunity is preferred.

A variety of non-silver dressings are available for burn wounds. Paraffin gauze dressings, low-adherent silicone coated dressings (e.g. Mepitel® [Molnlycke Health Care, Sweden), biosynthetic collagen dressings (e.g. Biobrane® [Smith & Nephew, UK]), and alginate dressings are commonly used worldwide (Al-benna, Collin,

Spalding, & Jeffery, 2007). Biobrane® is a synthetic dual layer dressing, which uses a nylon mesh coated in porcine collagen, with an outer layer of silicone. It is useful for clean, debrided partial thickness wounds, to which it adheres until healing has occurred below and has been used for many years in the UK and the US (Bishop, 1995; Whitaker, Worthington, Jivan, & Phipps, 2007). Hydrocolloid dressings (such as Duoderm® [ConvaTec, UK]) provide a moist wound healing environment, resulting in quicker healing times (Edwards, 2010; Hartford & Kealey, 2007, p. 75). Duoderm® is associated with a high protease wound environment that resulted in good healing with an architecturally normal epidermis following a partial thickness burn (Caulfield, Tyler, Austyn, Dziewulski, & McGrouther, 2008). A study comparing the use of Biobrane® and Duoderm® in children did not show a clinical benefit in healing time or pain scores between the two dressings (Cassidy et al., 2005). A retrospective study claims that the use of Duoderm® might reduce the incidence of surgery to heal compared to Jelonet (a paraffin based gauze), but this retrospective audit was subject to bias due to non-randomisation (Martin, O'Sullivan, Regan, McCann, & Kelly, 2010). The practice at Princess Margaret Hospital (PMH) for partial thickness burn injury is to use Acticoat™ for antibacterial control, covered by Duoderm® for moist wound healing and activation of the nanocrystalline silver. Special areas such as the face should be treated with emollient or chlorhexidine wash. If eyes are affected an ophthalmology review is required and Chloramphenicol eye ointment might be required for infection prophylaxis.

Scalds and burns: what affects the resultant scar?

Hypertrophic scarring (HTS) was defined by Peacock (1970) as a scar raised above the skin level that stays within the confines of the original lesion (cited in Bloemen et al., 2009). This is distinct from keloid scarring, which extends beyond the margin of the original lesion. Hypertrophic scars usually decrease over time, whereas keloid scars often reactivate and proliferate over time (Berman, Viera, Amini, Huo, & Jones, 2008).

The time taken to heal a burn wound is directly related to the degree of the resultant scar. Deitch et al (1983) found that if a wound took between 14 and 21 days to heal, then one-third of the wound scarred, and if the wound took more than 21 days to heal, 78% scarred. The investigators did not differentiate between keloid and hypertrophic scar (HTS) formation, and 73 of the 100 patients studied were black. A non-significant increase in scar formation occurred in the black population at shorter healing times. This study included adult and paediatric patients and maintains that the children had a higher rate of scar formation than the adults. However, the analysis revealed that the biggest predictor of scar formation was healing time. McDonald and Deitch (1997)

followed this study with a prospective study of hypertrophic scarring of skin-grafted burn areas. They found that incidence of scarring in the skin-grafted wounds were highest in black people, in children, in patients grafted after 14 days post-burn and in those whose wounds did not contain dermal elements.

Therefore, our population of scald injury, all of whom are children and a disproportionately high percentage of whom are Aboriginal are at of risk from scarring after traditional skin graft surgery. This assumes that the results can be extrapolated from the American non-white population to the Australian non white population, which may not be true. However, although the factors of age and race are constant, it is important to account for these when assessing the potential for scar formation. Importantly, they identify that prudent dermabrasion or wound excision with the goal of maintaining dermal elements before grafting is an important factor in scar prevention. Therefore timely and judicious surgical techniques are a key factor in scar prevention.

A study of 89 patients, conducted in the US, which included adult and paediatric patients, found that overall the prevalence of hypertrophic scarring was 67%, and the non-white population had a slightly higher prevalence (75%) than the white population (60%). This was a retrospective study that did not account for healing time or other factors (Bombaro et al., 2003).

The relationship between healing time and hypertrophic scar formation *per se* was investigated in a British paediatric burn population by Cubison et al (2006). Again, this study was retrospective. Time to healing was estimated by annotations in patients' notes recorded at outpatient review stating that the wound was healed or that dressings had been discontinued. This method is subject to inaccuracy as the status is dependent on appointment date not the true healing time. For example, a wound might have healed by day 13 but review at outpatients was on day 15. Inaccuracy would have been reduced by categorising the variable, but still remains. In addition, the diagnosis of HTS was reasonably established by the subsequent use of steroid injections or silicon treatment, but also by the use of pressure garments, unless the notes clarified that the use of pressure garments were prophylactic. The latter method of HTS diagnosis is also subject to inaccuracy in a retrospective study. The investigators conclude that healing is preferred within 25 days for a low risk of HTS.

The location of the injury on the body has been identified as a risk factor, with hands, face and neck healing better than chest, upper extremities and feet, which were more likely to develop HTS (Deitch et al., 1983). Skin that overlies bony prominences are more likely to develop hypertrophic or keloid scarring (Berman et al., 2008). So, many

factors that are considered to affect scar formation, either directly as an independent factor, or indirectly by affecting healing time. These include the wound severity and location, wound colonisation and infection, and the race, age, genetics, hormone levels and immunological response of the individual (Bloemen et al., 2009; Gangemi et al., 2008).

The prevalence of HTS after burn injury differ vastly between studies, with reports of between 7% and 68% in the Caucasian population, and 30% to 75% in the non-Caucasian population (Corica, Wigger, Edgar, Wood, & Carroll, 2006). The most likely causes of these conflicting reports is poor, inconsistent scar evaluation (Bloemen et al., 2009), and the dearth of studies that have assessed this using small samples and inconsistent methods (Bombaro et al., 2003; Gangemi et al., 2008), many of which do not differentiate between keloid and hypertrophic scarring.

Every intervention from the time of injury will have an effect on the scar. Good scar outcome is the ultimate goal of all treatments given, and is orientated towards good function and cosmetic appearance (Gangemi et al., 2008). The scar can take up to two years to fully mature, although it will be apparent at 1-3 months following injury (Bloemen et al., 2009). It goes through three phases; the inflammatory phase, the proliferative phase and the matrix remodelling phase (Berman et al., 2008). Identification of risk factors as described above; gender, age, wound site and severity, prolonged inflammation and past medical or family history of excess scarring will reveal those at highest risk of scarring. However, interventions need to aim for the optimal result for scarring in all individuals.

Early surgery to facilitate wound closure is advocated in wounds assessed not to heal within a specified time period. The length of time to healing recognised as acceptable is often 3 weeks, but varies between centres. In Western Australia it is preferred that a wound will heal within 10 days. If a child is admitted with deep or full thickness burn injury, then surgery is performed within a few days of injury or presentation. If a burn is considered likely to heal within 10 days then a conservative approach is taken; involving dressings every one to three days depending on treatment choice and as clinical judgement dictates. Typically when a child who has a burn wound of indeterminate depth is admitted, and therefore healing time is unclear, they will have initial conservative treatment and frequent review with a view to surgery at the 10-14 day period post-burn.

Surgical dermabrasion or debridement of the wound needs to preserve the dermal elements associated with re-epithelialisation, and needs to be adequate, but no

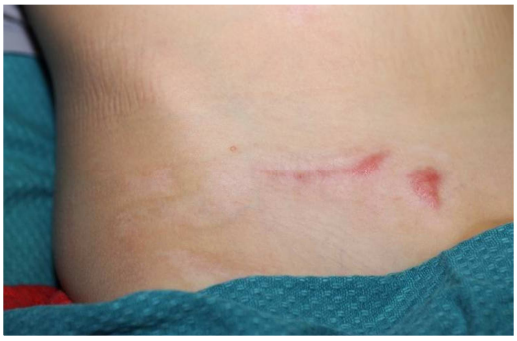
deeper, in order to preserve as many viable cells as possible. This can be achieved through established methods by dermabrasive tools or tangential excision, or newer techniques such as Versajet (Gravante, Delogu, Esposito, & Montone, 2007). Versajet uses pressurised jets of fluid to cut or debride tissue. Therefore, the development of accurate novel methods to guide and perform dermabrasion may prove to be invaluable (Dorafshar, Gitman, Henry, Agarwal, & Gottlieb, 2010; Singer, Taira, Anderson, McClain, & Rosenberg, 2010).

Traditional skin graft techniques can be used for dermal reconstruction. Autologous cell based sprays can be used, and are harvested from the dermal/epidermal junction. These have been used in Western Australia to enhance wound healing since the early 1990s. (Wood, Kolybaba, & Allen, 2006). Initially, cell sprays were cultured from biopsies taken a few days before surgery, and in recent years cells are harvested for immediate use during theatre time from a small skin graft site (1cm^2 graft site for each 80cm^2 area of burn grafted) for application in one procedure (ReCell). The spray can be used in isolation in mid dermal injury or in combination with meshed split thickness grafts in deep dermal injury. The advantage of the ReCell technique is the small donor site area as compared to the larger areas required for traditional grafting, which themselves can be painful and require time to heal (Wood, 2003). The disadvantage being slightly prolonged theatre and anaesthetic time (Gravante, Di Fede et al., 2007).

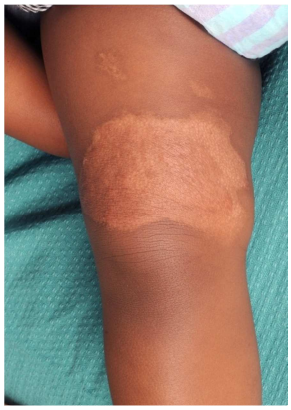
In Western Australia, in 1998 cell application was achieved using cultured epithelial autograft (CEA). This requires a skin biopsy to be taken before surgery, which then needs to be cultured in the laboratory for five days before it is ready for use. By 2008, this technique had been replaced by ReCell, which is a cell suspension that does not require the cells to be cultured and is prepared whilst the patient is in theatre in 20 minutes. Recently, a case study reported a similar technique piloted in the adult outpatient setting by Gerlach et al (2011) on non-healing deep dermal flame burns. A 1:20 spray (i.e. 1cm^2 graft site for each 20cm^2 area of burn grafted) was applied at day 10, following mechanical debridement and cell harvesting under oral fentanyl analgesic cover. Healing occurred 4 days after treatment. This would not be an option in the paediatric setting because of the induction of pain, anxiety and post-burn psychological sequelae. However, this study demonstrates that tissue engineering solutions for burn wound closure remain a priority for future research (Klein, Donelan, & Spence, 2007).

Scar assessment is currently via the ranking of the scar using an assessment tool. The original and most commonly used scale is the Vancouver Scar Scale which assesses the pigmentation, pliability, height and vascularity, scoring higher numbers the further

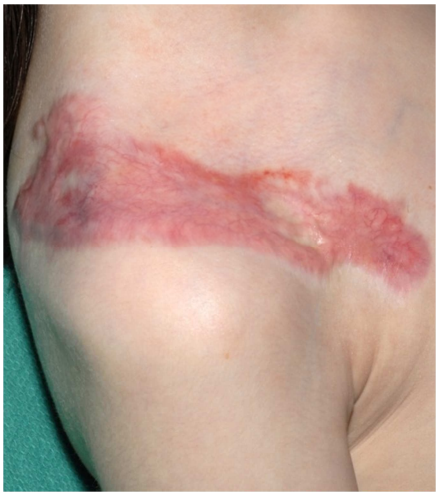
each factor deviates from normal. Various modifications have been made to this original scale to account for itch, pain and mixed pigmentation, or for remote assessment by visual means only; therefore not reliant on tactile assessment of pliability (Oliveira et al., 2005). The version currently in use at PMH accounts for mixed pigmentation (Baryza & Baryza, 1995). It remains an imperfect evaluation; reports of inter-rater reliability vary (Nedelec, Correa, Armour, & LaSalle, 2008), improves with practice amongst groups (Wang et al., 2009) but may differ between groups and is not culturally sensitive to colour (Forbes-Duchart, Marshall, Strock, & Cooper, 2007).



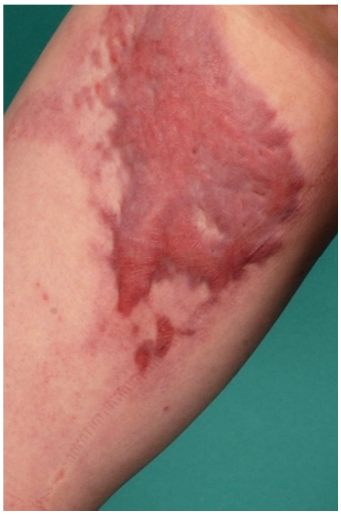
Case 6: Small scar areas (corresponding to areas of hypergranulation)*



Case 7: Hypopigmentation in dark skin*



Case 8: Poor scar outcome following graft failure*



Case 9: Deep dermal burns have resulted in poor scar outcome in this child*

*(PMH Medical Illustrations, 2011) All photographs used with parental informed consent

After healing has occurred, the emphasis shifts definitively from preventive strategies to treatment strategies, however because the scar continues to mature the goal is still to minimise the scarring process, so management combines preventive and curative elements.

Silicone, used as a rubber, gel or liquid, improves scar appearance and is used both preventively and curatively. The exact mechanism is unknown, but it is thought to act upon the collagen remodelling phase of healing. Increased local hydration causes reduced capillary activity and reduced collagen production, and increased local temperature causes additional collagenase activity thereby reducing collagen levels by collagen breakdown. It is also postulated that a static electrical field might be created by a negative charge generated by friction between the skin and the silicone layer which might cause collagen realignment and the involution of scars (Bloemen et al., 2009; Mustoe, 2008).

Pressure therapies are also used as preventive and curative measures. Preventively, it is thought to act by accelerating the remodelling phase of healing, and curatively by thinning the scar and improving pliability. The possible mechanisms are due to decreases in hydration levels and blood flow, stabilising the mast cells and reducing the formation of new blood vessels. It is not clear why the alterations in hydration levels, which are in contrast to the actions of silicone seem to be effective in both circumstances. Compression triggers the release of Prostaglandin E_2; which increases collagenase levels, so reducing collagen synthesis (Reno, Grazianetti, & Cannas, 2001). Scar tissue is known to have microvasculature changes; large vessels with few interconnections and pressure to these reduces scar activity (Leung, Clark, Cheng, & Leung, 1989). Garments are custom made, expensive, hot and uncomfortable. There is a high rate of non-compliance due to physical discomfort, embarrassment and stigma. Rashes and pruritis can occur and occasionally skin breakdown (Chang et al., 1995; Macintyre & Baird, 2006). Areas of high movement or flexion are difficult to treat with pressure. Pressure is recommended to be maintained at 20-30 mmHg above capillary pressure (Bloemen et al., 2009) but lower pressures of 15 mmHg have been shown to benefit (Van den Kerckhove et al., 2005) . Garments need to be worn 18-24 hours a day until the scar matures, and early release can cause rebound hypertrophy (Berman et al., 2008; Bloemen et al., 2009).

Massage is a widely used and accepted method for improving vascularity and pliability of scars (Davoodi, Fernandez, & Seung-Jun, 2008; Gangemi et al., 2008; Mustoe et al., 2002) but there are few studies that have proven the effects (Silverberg, Johnson, &

Moffat, 1996). It is used as an adjunct to other therapies such as silicone and pressure (Cubison et al., 2006) and is not effective alone (Li-Tsang et al., 2010) possibly due to fibroblast stimulation, so needs to be used with anti-collagen modalities (Davoodi et al., 2008). Massage effects are mostly mechanical; improving venous return and lymphatic drainage, and stimulating movement between the muscle fibres. It also may improve pruritis (Patino, Novick, Merlo, & Benaim, 1998). There is even less evidence in the literature for the importance of sun protection on both hypertrophic scars and well healed burns that are at risk from permanent pigment changes from sun exposure in the post burn period. Davoodi et al (2008) assert that the healed burn loses its ability to retain moisture and its protective ability against ultraviolet light. This is very relevant in the climate of WA and the local practice is to advocate both massage and sun protection for two years post burn injury, longer if scarring is ongoing. Judicious exposure to the sun can begin on the advice of the burns specialist with a view to normalising pigment if the burnt area remains pale, particularly in darker skins. Massage can be undertaken with sun protection creams to combine techniques.

Physiotherapy to prevent and treat burn scar contractures is important to optimise range of motion. Assessment is often subjective and methods lack consistency, both in clinical practice and in research which makes comparison between studies difficult. Methods include specific tailored exercises and splinting of joints (Parry, Walker, Niszczak, Palmieri, & Greenhalgh, 2010).

This review details the current body of knowledge that revolve around burn injury and childhood scald in particular. The nature of these injuries means that the evidence is based on observation and clinical experience, together with some supportive evidence from animal studies. Evidence is available in the literature that demonstrates common threads of all issues discussed that vary dependent upon location and culture. The evidence pertaining to Western Australian children is currently unknown and is explored further in this study.

Scald injury: health promotion, injury prevention and public health

Globally, burn prevention needs differ in high income countries (HIC) and low-middle income countries (LMIC). The latter have a high incidence of burn injury leading to an excessive burden of death and disability as the health care systems are unable to cope. The World Health Organisation (WHO) have joined forces with the International Society for the Prevention of Burn Injuries (ISBI) to support governments, especially of LMICs to develop burn prevention and care strategies. In consultation with many burn care experts, health care practitioners and burn victims, from 14 countries, the WHO

plan for burn prevention and care was devised (World Health Organisation, 2008). This provides a framework for the advancement of effective and sustainable burn prevention and care.

High income countries have used a combination of methods to lower rates of injury. In Australia, for burn injury overall, legislation about the use of smoke detectors and flame retardant nightwear for children has done much towards lowering injury rates. For scald injury *per se* legislation about the temperature of hot water systems has been introduced (Mock, 2009).

Alongside these enforcements, community education is important, and to be effective must target specific high risk groups. The program must be relevant and meaningful to the population at risk. In Australia, a round table forum on burns prevention was conducted in 2010 in Canberra. Twelve recommendations were made for the advancement of burn prevention in Australia. These include, and are based on the collation of accurate data (Commonwealth of Australia, 2010).

Burn injury data collection is coordinated in Australia by the Australian and New Zealand Burns Association (ANZBA) in collaboration with Monash University. This data repository collates data from 17 burns centres across Australia and New Zealand. This information can be used both nationally and locally to identify needs for burn prevention (Australian and New Zealand Burns Association, 2010a). A burn prevention committee has been in operation by ANZBA since 2009. This committee comprises of representatives from states and territories of Australia, and New Zealand. It is developing a national first aid campaign seeking to educate all members of the population on correct and timely first aid for all people sustaining burn injuries. This will be delivered by multiple means including Community Service Announcements (CSA) and an internet-based education module.

A scald prevention that targets childhood injury was run by Kidsafe WA in 1999 and 2002. This campaign, “Hot water burns like fire”, distributed posters and brochures which gave practical advice to parents and carers on the dangers of kettle cords, saucepans, hot drinks and hot tap water (Leeds & Wicks, 2007)

Health professionals are educated across the state with monthly videoconferencing from Princess Margaret Hospital. These sessions simultaneously link all sites to receive expert burn care and first aid information. This multidisciplinary program repeats every six months and is widely advertised and available to all interested health care professionals (McWilliams, 2011). In addition, the burn care consultants liaise

with the ED consultants for the appropriate regular review of burns first aid and care guidelines, which are then disseminated to ED staff at PMH.

Summary

Western Australia is vast and the delivery of equitable care across the State is challenging. The rural population, and indigenous families especially, have risk factors for burns and scalds that increase risk of burn injury and complications such as wound infection. Childhood scald injury is most common in those less than 5 years old, often from spill in the kitchen or bathroom. Cooling the burn limits damage. The recommended method of cooling the burn in Australia is to apply 20 minutes of cool running water. Many other substances have been applied to burn wounds to ease pain and the choice of first aid is often cultural.

Burn wounds continue to develop for 48 hours after injury. Accurate assessment of size and depth of burn wounds can be difficult, but is important for the correct calculation of fluid resuscitation. Over or under hydration can be detrimental, so careful monitoring of fluid status needs to occur. Oedema is common, and careful attention must be paid to this, especially in limbs. Loss of circulation due to swelling needs to be observed for early surgical intervention, especially if the burn is circumferential.

Pain mechanisms are complex and need appropriate management. Adequate pain control is essential at acute and chronic stages of injury. Wound infection is a risk. Early application of antimicrobial dressings are important for control. Rapid wound closure is important to minimise scarring, and might need to be achieved by skin grafting. Scar management after injury can involve massage, pressure garments, silicon, steroid injections and surgery.

Prevention is better than cure. A combination of prevention strategies have helped reduce the incidence of burns and scalds. Continued education of health care professionals occurs across the State.

CHAPTER 3

METHODS

Study design

This was an observational, retrospective cohort study. The primary inclusion criterion consisted of every burn injury admission to PMH, where the mode of burn injury was classified as scald, from January 1, 1998 to December 31, 1998 and from January 1, 2008 to December 31, 2008. These years were chosen to assess the changes that had occurred in a ten year period. Patients scalded in 2008 were the latest group of patients to allow the collection of complete scar assessment and management. It was essential not to exclude any patient that met this criterion from the study to prevent bias and assure study validity. For this reason, and also because the data collection was observational and retrospective, patient consent was not required by any of the approving ethics committees.

Study Objectives

The primary objective of the study was to assess changes to the profile of hospital admissions for children with scald injury in Western Australia between 1998 and 2008. Comparison data on scalds for this period from the only specialist paediatric burns unit in WA was used to interpret incidence and changes in patterns of treatment of children who have suffered this form injury. Changes that occurred across the time frame were also used to ascertain possible needs for further preventive or treatment interventions.

A secondary objective of the study was to assess whether there were specific sub-populations that were at higher risk for scald injury.

Data Collection

A list of burn injury admissions for the years 1998 and 2008 was compiled from hospital data base systems and provided by administrative staff at PMH. Relevant patient notes were scrutinised and data collected as per the Burn Minimum Data Set (Appendix 5). These notes were originally compiled by medical staff, nursing staff and all allied health professionals involved in the care of each patient. The Burn Minimum Data Set is a data collection form adopted for use in research studies in the burns unit at PMH and is derived from these notes. Data for the patients who sustained burn

injury due to a scald mechanism was de-identified during the process of data entry into the statistical software database.

Sample size

The sample size was determined by the number of admissions in each year. It was important to include every admission and assess the whole cohort to avoid bias. A preliminary assessment of numbers of admissions revealed that there were approximately 100 patients in each year. Formal sample size calculations were not required because this was an observational study assessing many parameters and compared changes to generate new hypotheses and recommendations for future work.

Outcome measures

A comparative analysis was undertaken to assess changes in gender, age, scald agent, time and date of injury, ethnicity, area of residence, whether Australian born, severity of burn (extent of total body surface area burned and depth of deepest burn area), location of body site affected, whether adequate first aid was received by the patient, numbers of admissions, length of stay, follow up appointments for each episode, whether on-going scar management is required, the incidence of surgery and mortality rate.

Area of residence was assessed by using the Accessibility/Remoteness Index of Australia (ARIA). This is the current Australian Bureau of Statistics (ABS) standard geographic classification of remoteness. The ARIA provides an index of remoteness scale based on geographic location and access to a range of services. The ARIA provides classifications for five different levels of remoteness (Commonwealth of Australia, 2004). This classification has been chosen as in Western Australia it is broadly indicative of distance from Perth, and therefore representative of the use of the Telehealth service. The ARIA map for Australia is shown in Figure 2. Regional data was also assessed using the Regional Development Commissions Act (RCDA) classifications to ascertain any marked difference in injury incidence or service provision in the regions. This was included for the benefit of the burns Telehealth service to identify specific needs of health provision in these areas .

Where numbers were too small for meaningful analysis by ARIA, or when it was useful to perform logistic regression analysis, then residential area was classified into metro and non-metro areas. For analysis specific to Aboriginality and where numbers allowed meaningful analysis, residency classification by local government area (LGA) was completed, as requested by the West Australian Aboriginal Health Information and

Ethics Committee (WAAHIEC). Postcode was used to rank the areas of residence for socioeconomic status via The Socio-Economic Indexes for Areas (SEIFA). This is a method developed by the ABS to rank areas according to their level of social and economic well-being. It is derived from census data and uses 4 indices that assess income, education, unemployment, dwellings without motor vehicles, further economic evaluation, and occupation (Australian Bureau of Statistics, 2006a).

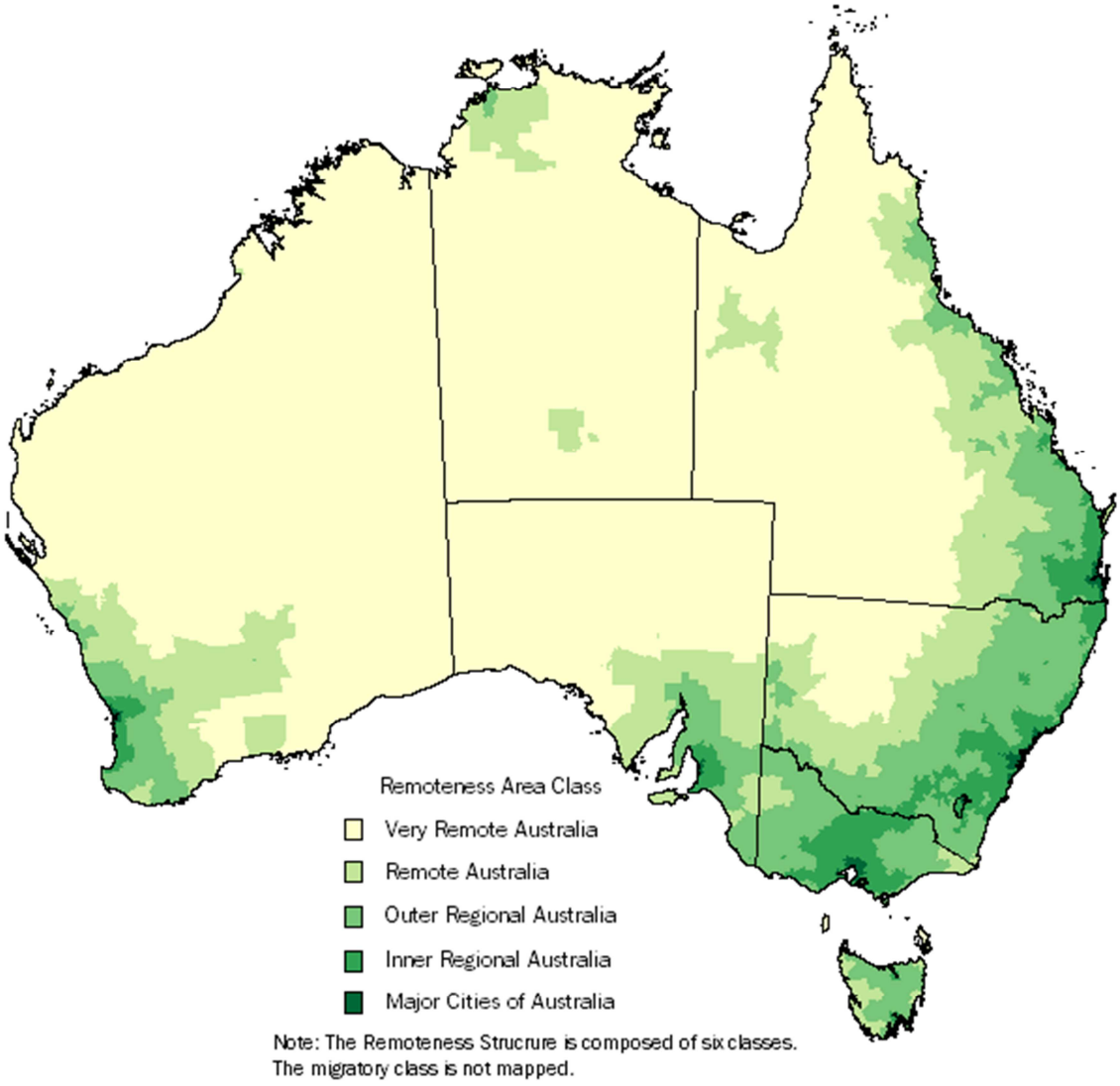


Figure 2: ARIA classification (Commonwealth of Australia, 2004)

The following variables were classified dichotomously: gender (male/female); ethnicity (Aboriginal or Torres Strait Islander/non-Aboriginal); Australian born (yes/no); incidence to surgery for index admission (yes/no); ongoing scar management, defined by use of

pressure garments or reconstructive surgery (yes/no). The need for surgery was used as a measure of the depth of the wound.

The following variables were classified categorically: area of residence (as above); first aid (nil/not recorded, inadequate or inappropriate, adequate); scald agent (hot beverage no milk, hot beverage with cold milk, soup, noodles, other food, hot oil, other substance), age (in yearly increments i.e. 0-1, 1-2, 2-3 etc), and the day of the week, and the month of the year that the injury occurred.

The following variables were classified initially by continuous data: Total body surface area burned (%); length of stay (days), distances (km) and the number of readmissions and clinic appointments resulting from the index admission.

Table 2: Outcome measures

Variable	Original measurement	Categorisation for the study	Type	Analysis
Age at time of injury	Determined as a decimal value (to 2 decimal places) between date of birth and date of injury	Age	Continuous	Independent samples T-test analysis
		Year of age	Categorical (0-1; 1-2; 2-3 etc)	Pearson's Chi^2 test
		Age group	Categorical (0-2; 2-5; 5-10; 10-15)	Pearson's Chi^2 test
Sex	Male / female	Male/female	Dichotomous	Fishers exact test
Country of birth	Classified as Standard Australian Classification of Countries 2nd Ed.	By country	Categorical	Fishers exact test
		Australian born	Dichotomous	
Indigenous status	Aboriginal or Torres Strait Islander/Other	Aboriginal or Torres Strait Islander/Other	Dichotomous	Fishers exact test
Residential area	Postcode of injury or residence if unknown	Metro	Categorical	Fishers exact test Pearson's Chi^2 test
		ARIA	Categorical	
		RDCA	Categorical	
		LGA	Categorical	
Scald agent	Specific agent as documented in notes (descriptive eg tea with milk)	Hot water, hot drinks, soup, noodles, other food, oil, milk	Categorical	Pearson's Chi^2 test
Total body surface area burned (TBSA)	As documented by the most senior member of the burns team	Percentage	Continuous data	Independent samples T-test analysis

Time of injury	mm:hh	hours and minutes	Continuous data	Independent samples T-test analysis
Date of injury	dd:mm:yyyy	Day of injury	Categorical	Pearson's Chi^2 test
		Month of injury	Categorical	Pearson's Chi^2 test
		Season of injury	Categorical	Pearson's Chi^2 test
First aid given	Descriptive data from notes	By carer: duration of water; ice; other	Categorical	Pearson's Chi^2 test
		By first attender: duration of water; ice; other		
		By PMH ED: duration of water; ice; other		
Delay to treatment	More than 3 hours from injury to first presentation for treatment	Over 3 hours: Yes/no	Dichotomous	Fishers exact test
Dressing use	Specific type of dressing recorded	Silver dressing	Dichotomous	Fishers exact test
		Acticoat /SSD	Dichotomous	
Analgesia use	Drug, dose and route, place:	First attender: drug type and administration	Categorical	Pearson's Chi^2 test
		PMH ED: drug type and type of administration		
		Ward: drug type and type of administration		
Antibiotic use	Drug, dose and route, indication	Antibiotic for wound	Dichotomous	Pearson's Chi^2 test
		Wound infection preadmission, during admission, after discharge	Categorical	
Surgery	Was surgery given to close the wound?	Surgery yes/no	Dichotomous	Fishers exact test
		No of days to surgery	Categorical	Mann-Whitney
Time to healing	Estimate no of days post healing that wound closure occurred	Number of days	Continuous data	Independent samples T-test analysis Mann-Whitney tests

Scar management	Use of scar management techniques	Tubigrip, pressure garments, silicon, steroid injections, surgical scar management, physiotherapy	Categorical	Pearson's Chi^2 test
Length of stay			Continuous data	Independent samples T-test analysis

Statistical methods

Analysis was conducted using PASW (formerly SPSS) version 17 for windows and STATA MP 10. Firstly, descriptive data analyses were performed on all variables. Statistical tests that were used included T-test analyses, Pearson's chi square analyses, Fisher's exact tests, correlations, and logistical and linear regression analyses, as appropriate. Appropriate transformation techniques were used to ensure normality. Categorisation of continuous variables was used where necessary for meaningful analysis. If transformations were inadequate, unequal variance t-tests were used, or Mann-Whitney rank-sum tests for equality of medians. Binary categorisation was used when possible to allow Fisher's exact testing in preference of Chi^2 tests. Level of significance was taken as 0.05 on a two sided test. Two-sided tests were always used, even if the hypothesis could be estimated to be one-way. Some qualitative analysis was undertaken to assess specific areas of interest. All these measures were used to err on the side of caution and ensure good quality statistical analysis. Firstly, all variables between 1998 and 2008 were compared to assess changes within this time. Secondly, some whole cohort analyses were competed to assess risk factors for infection.

Ethics approval

Ethics approval was gained from the PMH Ethics Committee, the West Australian Aboriginal Health Information and Ethics Committee, and Edith Cowan University before commencement of the study.

CHAPTER 4

RESULTS

As stated in Chapter 1, this study attempts to clarify the differences in scald injury in children between 1998 and 2008. The change in numbers of children who present and are admitted for scald injury are quantified. This chapter contains the results of the analysis and is organised chronologically, as per the patient journey, starting with the circumstances of injury and the patient socio-economic characteristics, through first aid received and the care given by medical personnel, both non-specialist and specialist and follows the framework of elements identified in question 1. The section that describes the characteristics of the cohort introduces the children at risk of scald injury, and together with the results that follow reveal the target groups for intervention. This is integrated in the discussion. Data display guidelines taken from Freeman, Walters, & Campbell (2008). For a reminder of the research questions posed:

Question 1:

Did the epidemiology, treatment and outcomes of Western Australian paediatric scald injuries admitted to the Total Care Burns Unit, PMH in 1998 and 2008 differ?

Sub-questions:

During the study period was there: :

a reduction in the incidence of scald injuries among this cohort?

a change in the severity of scald injury?

a change in the type of scald injury presenting to hospital?

an improvement in first aid given to children with scalds during the pre-hospital period?

changes in the actions of the carers, present at the time of injury?

changes in medical treatment and nursing care of paediatric patients with scald injury?

Question 2:

Which target groups or focus areas do the data suggest might be appropriate for further resourcing and effort to reduce the incidence and severity of burns and associated scar outcomes from scald injuries among Western Australian children?

Patient characteristics

Which target groups or focus areas do the data suggest might be appropriate for further resourcing and effort to reduce the incidence and severity of burns in Western Australian children? There was a slightly higher proportion of boys than girls who sustained a scald injury in both years. The 2008 male:female ratio in Western Australia was 1:0.97, in 1998, the admitted male:female ratio was 1:0.91 and in 2008 it was 1:086. The number of Aboriginal children admitted with scald injury was higher than the proportion in the population of WA in both years, and has displayed a non-significant reduction. There were no Torres Strait Islanders in either cohort.

Table 3: Cross-tabulation of year of admission (in columns) and patient characteristics (in rows) for scald injury (n=210)

	Year		p value[a]
	1998 n=113	2008 n=97	
Male	52% (59/113)	54% (52/97)	p=0.890
Aboriginal	15% (17/113)	12% (12/97)	p=0.689
Australian born	94% (106/113)	91% (88/97)	p=0.443

[a] p-values from Fishers Exact Test

In 1998, 6% (7/113) children were not Australian born in WA compared with 9% (9/97) in 2008. The countries of birth for these children did not show any specific trend (Table 4).

Table 4: Countries of birth if not Australian born

Year	
1998 n=7	2008 n=9
New Zealand (2/7)	New Zealand (1/9)
England (1/7)	India (1/9)
El Slavador (1/7)	Malaysia (2/9)
Indonesia (1/7)	Japan (1/9)
Sri Lanka (1/7)	South Africa (3/9)
At sea (1/7)	Saudi Arabia (1/9)

The change in areas of residence was statistically different between 1998 and 2008, showing a drop in admissions from the outer metro areas and an increase from the non metro areas between 1998 and 2008 (Table 5). These changes remained significant when stratifying area by the accessibility/remoteness index of Australia (Table 6). The patients who did not reside in the Perth metropolitan area as defined by the Regional Development Commissions Act (RDCA) were distributed across the regions and did not show significant differences either between regions or between years. Statistical tests have not been completed on these figures as the numbers of children admitted from each region were not large enough for meaningful analysis. A table has been included for descriptive information (Table 7).

Table 5: Cross-tabulation of year of admission (in columns) and area of residence by inner, outer and non-metro areas (in rows) (n=210)

Metropolitan area	Year		p value [a]
	1998 n=113	2008 n=97	
Inner metro	36% (40)	41% (40)	
Outer metro	49% (55)	31% (30)	p=0.019[b]
Non metro	16% (18)	28% (27)	

[a] P-value from Chi^2, χ^2=7.9802 on 2 degrees of freedom

[b] Statistically significant for p≤0.05

Table 6: Cross-tabulation of year of admission (in columns) and area of residence by Accessibility/Remoteness *Index* of Australia (ARIA) (in rows) (n=210)

ARIA	Year		p value [a]
	1998 n=113	2008 n=97	
Major city	83% (94)	70% (68)	
Regional	6% (7)	12% (12)	
Outer regional	3% (3) [b]	8% (8)	p=0.014[b]
Remote	5% (6)	1% (1) [b]	
Very remote	3% (3) [b]	8% (8)	

[a] P-value from Chi^2, χ^2=12.4588 on 4 degrees of freedom

[b] Statistically significant for p≤0.05

Table 7: Year of admission (in columns) and area of residence by region as established by the Regional Development Commissions Act (RDCA) (in rows)

RDCA Region	Year	
	1998	2008
Southwest	2	6
Pilbara	2	4
Kimberley	3	3
Peel	3	3
Midwest	2	3
Great Southern	0	3
Goldfields-Esperance	1	2
Wheatbelt	3	1
Gascoyne	0	1

Other local trend data of burn admissions from 1998 to 2008 visually show changes in the 0-5 year old age group when stratified by metro or non-metro area of residence (Figure 3). The study data also displays a downward trend in the number of admissions in the metropolitan area in this age group (Figure 4) although this is not apparent in the non-metropolitan patients (Table 8).

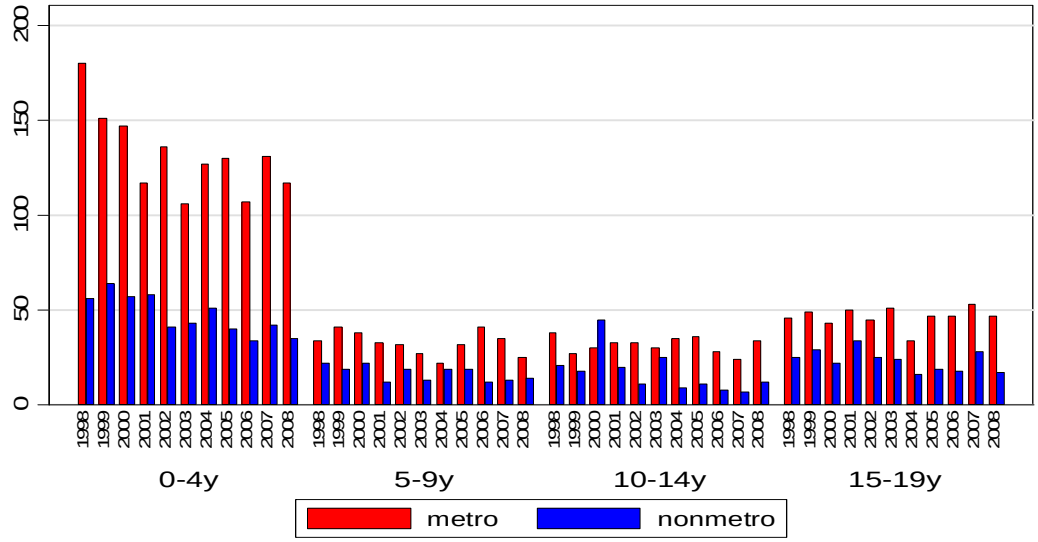


Figure 3: Bar chart showing number of burn admissions by age group and metropolitan area for 1998 to 2008

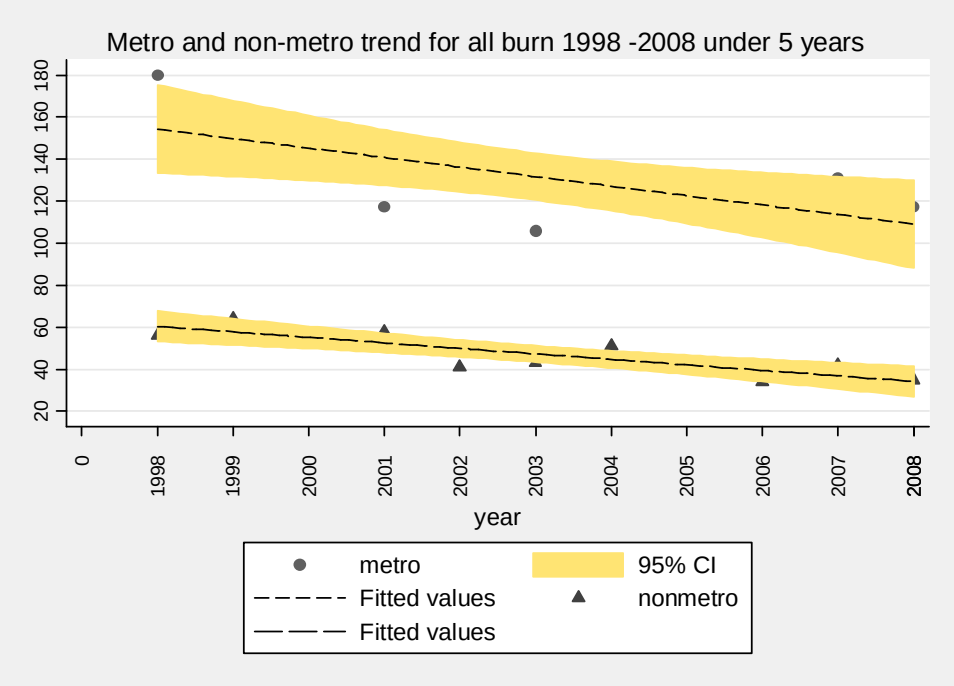


Figure 4: Time series chart showing West Australian state-wide admission trends for children less than 5 years old from 1998 to 2008

Table 8: Cross-tabulation of scald injury of year of admission (in columns) and area of residence by metro area (in rows) for those less than 5 years (n=155)

Residence by metropolitan area	Year		p value[a]
	1998 n=86	2008 n=69	
Metropolitan area	83% (71/86)	65% (45/69)	p=0.016[b]
Non –metropolitan area	17% (15/86)	35% (24/69)	

[a] p-values from Fishers Exact Test
[b] Statistically significant for p≤0.05

Distributional population change data (Australian Bureau of Statistics, 2009) was used to predict 2008 hospital admission, using baseline 1998 admission data for inner, outer and non-metro areas. The data from the Australian Bureau of Statistics shows population growth of 10.2% in the inner metropolitan area, 26.1% growth in the outer metropolitan area, and 15.6% growth in the non-metropolitan area between 1998 and 2008. The following predictions are based on the assumptions that the incidence of scald injury has not reduced, and that the population changes are consistent across age groups. Predictions are shown and compared to actual admission numbers in 2008, in Figure 5. The population declined between 1998 and 2008 in the non-metro areas, and numbers of admissions increased, while the reverse was true in the inner and outer metropolitan areas.

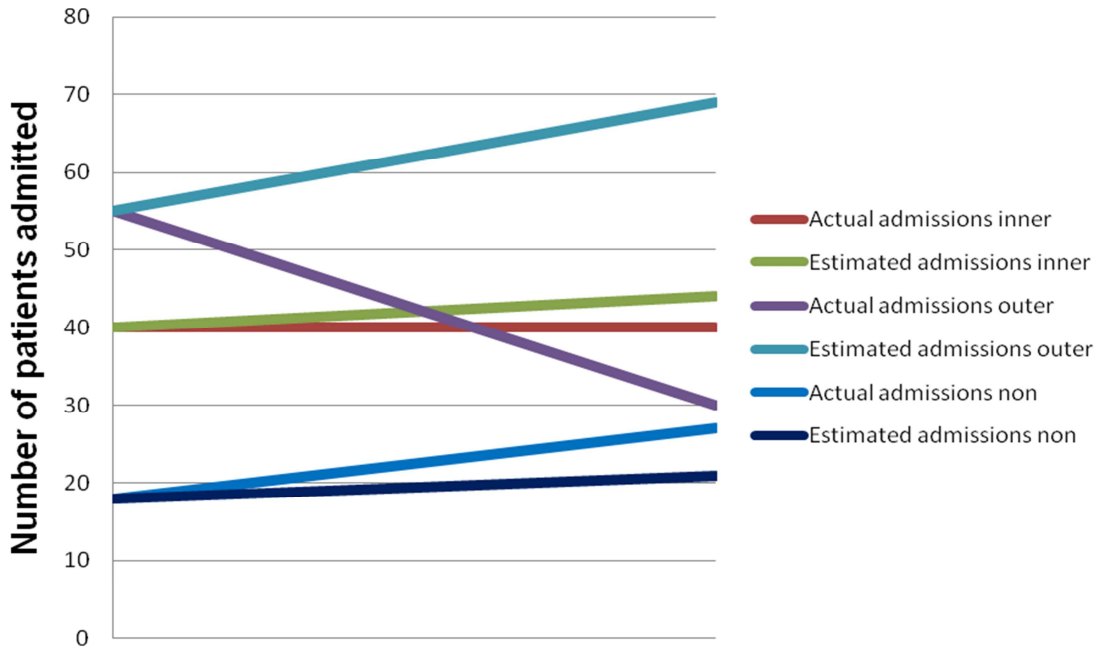


Figure 5: Line graph showing actual and predicted admission trends by inner, outer and non metro areas from 1998 to 2008

There are 142 local government areas in Western Australia. In 1998 and 2008 collectively all admissions came from 49 of these. Admissions from the local government areas of Stirling, Swan and Wanneroo were most likely to be admitted with a scald injury in both 1998 and 2008, and these suburbs remained the top three suburbs for admission numbers for both years. Data from the Australian Bureau of Statistics (ABS) reveal the Stirling, Swan and Wanneroo have the biggest populations and are also the top three areas for largest growth (Australian Bureau of Statistics, 2009). Taking population size into account, the incidence of injury is not highest for these areas. The top 15 LGAs are displayed in order of incidence in Figure 6; incidence is represented by the height of the blue bars. The Socio-Economic Indexes for Areas (SEIFA) is the ABS measure of socioeconomic status, and is indicated by the green line in the graph (Australian Bureau of Statistics, 2006a). The average SEIFA is 1000, and is indicated by the black line. Suburbs that have a SEIFA, as calculated by the Australian Bureau of Statistics (2006a), below 1000 indicate relative disadvantage, those above, relative advantage. The graph below shows the green line falls below the black line for 3 of the 15 top suburbs; Bassendean, Armadale and Belmont. In addition to this, for the 49 LGAs from which the admitted patients came, the mean SEIFA score was 984.20 (SD 94.43), and the LGAs from which no admissions came had a mean SEIFA score of 974.90 (SD 85.55). This suggests that the areas from which the scald injuries came were slightly more affluent, although this difference was not statistically different (t [140] =-0.5940, p=0.5535).

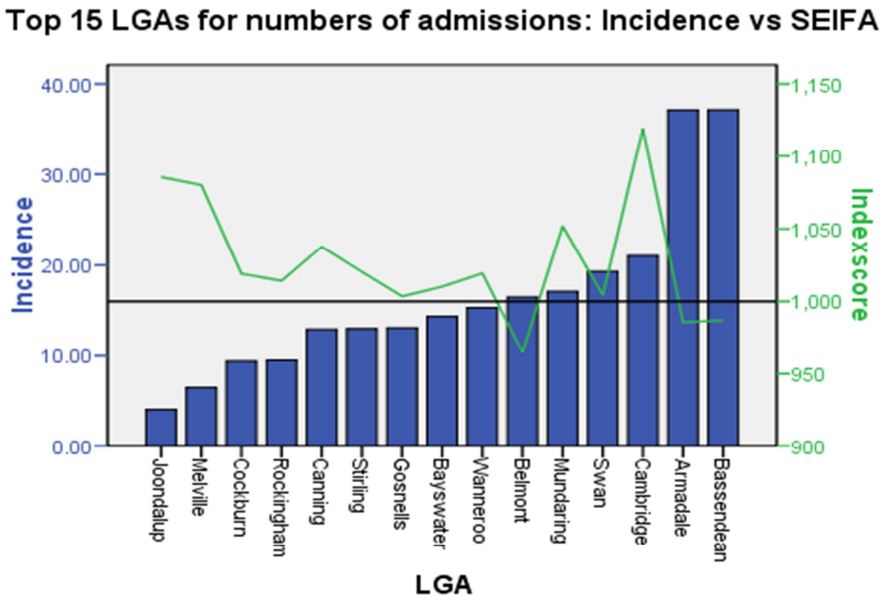


Figure 6: Bar chart showing highest incidences of scald admission from local government areas (LGA). The green line shows the Socio-economic indices for area (SEIFA) value for each LGA. The black line illustrates the SEIFA midpoint of 1000.

Age at time of injury showed a positively skewed distribution for both 1998 and 2008. Log transformation normalised this, and all t-test analyses were performed on the log transformed data. The slight increase in age between 1998 and 2008 did not reach significance. The analysis of categorised data demonstrated that only children aged between one and two years showed a reduction in admission numbers (p=0.015).

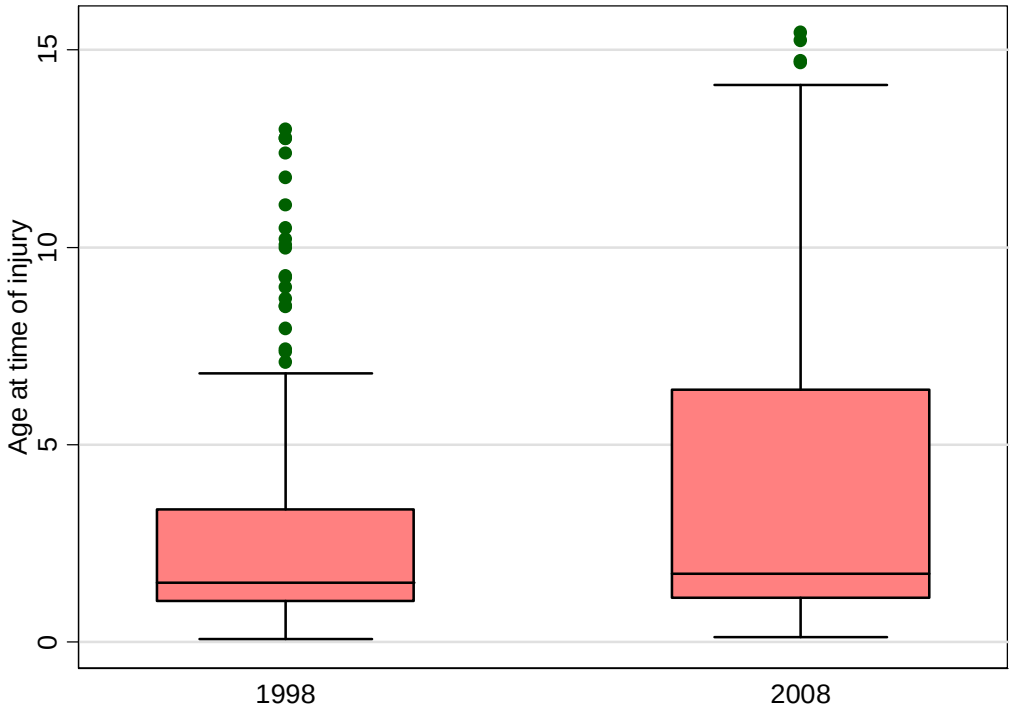


Figure 7: Box and whisker plot to show distribution of age at time of injury

H_o: mean age (1998) = mean age (2008)

H_A: mean age (1998) ≠ mean age (2008)

The box plot (figure 7) shows the distribution is positively skewed, and log transformation was conducted for the comparison of means.

Independent sample t-test were performed on log transformed age data

t= -1.3874 (208) p=0.1668

P value > 0.05 therefore, the null hypothesis is not rejected.

Table 9: Summary statistics for age at time of injury

Summary statistic	Year		p value[a]
	1998 n=113	2008 n=97	
Mean (SD)	3.09 (3.41)	3.88 (4.12)	0.1668
Median (IQR)	1.48 (1.05 to 1.48)	1.73 (1.12 to 6.40)	

[a]P value from Independent samples t test on log transformed data

During the study period was there a reduction in the incidence of scald injuries between 1998 and 2008?

The numbers of children presenting with a burn injury to PMH increased between 1998 and 2008 at the same rate as the population in WA, therefore displaying no change in incidence. Scald injury presentations increased at a rate greater than the population change. Numbers of children admitted were significantly reduced for all burn injury, but the reduction was not significant for scald injury *per se* (Table 10).

Table 10: Calculated incidence rates for burn and scald presentations and admissions to PMH in 1998 and 2008

	1998	2008	percentage change	p value[a]
Population of WA for persons aged 0-15y	424,524	457,659	+8%	
All burn presentations to PMH (incidence)	388 (91/100,000)	418 (91/100,000)	No change	1.000
All scald presentations to PMH (incidence)	176 (41/100,000)	228 (49/100,000)	+19%	0.211
All burn admissions to PMH TCBU (incidence)	240 (56/100,000)	185 (40/100,000)	-29%	0.033[b]
All scald admissions to PMH TCBU (incidence)	113 (26/100,000)	97 (21/100,000)	-19%	0.327

[a] P-values from Chi^2 test

[b] Statistically significant for $p \leq 0.05$

During the study period was there a change in the type of scald injury presenting to hospital?

Mode of injury

Hot water accounted for 53% (60/113) of the admitted scald injuries in 1998 and 41% (40/97) in 2008. The source of hot water was often not stated, and documentation was no better in 2008 than in 1998. Of the 60 scalds caused by hot water in 1998, 42% (25/60) of the medical records did not state whether the source was from a tap or from another source such as a kettle or pan. In 2008; of the 40 admitted scalds from hot water, 37% (n=15) did not state a source. This reduction was not significant (Pearson's chi^2 (2)=0.1008, p=0.951), and statistical test revealed no change. Scald injuries incurred through the spillage of hot drinks remained constant; 35% (40/113) in 1998, 35% (34/113) in 2008. The addition of milk to hot drink scalds was not documented for 43% (17/40) of these patients in 1998, and for 56% (19/34) of patients in 2008. Other agents such as soup, noodles, oil, milk and sauces increased from 12% of total scald injuries in 1998 to 24% in 2008. An increase that was statistically significant p=0.048 (χ^2=6.0805 [df=2, n=210]). Scald injury from noodles showed an increase from 0.9% (1/113) in 1998 to 8.2% (8/97) in 2008. Simplified mode of injury data are illustrated in Figure 8, and an examination of scald injury due to other agents in Figure 9.

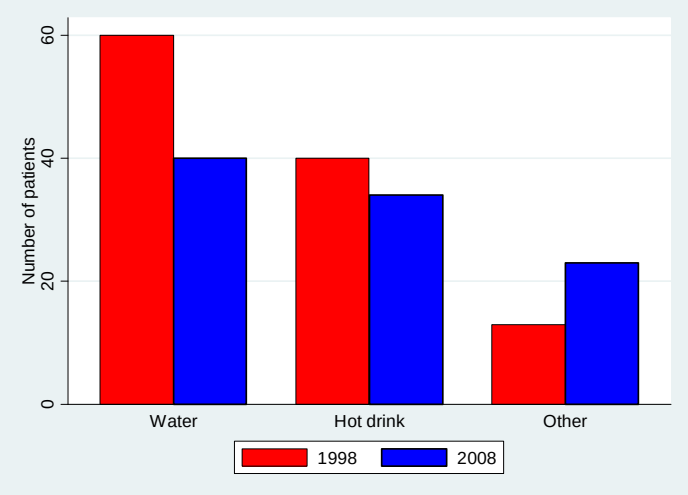


Figure 8: Bar chart showing mode of scald injury by year

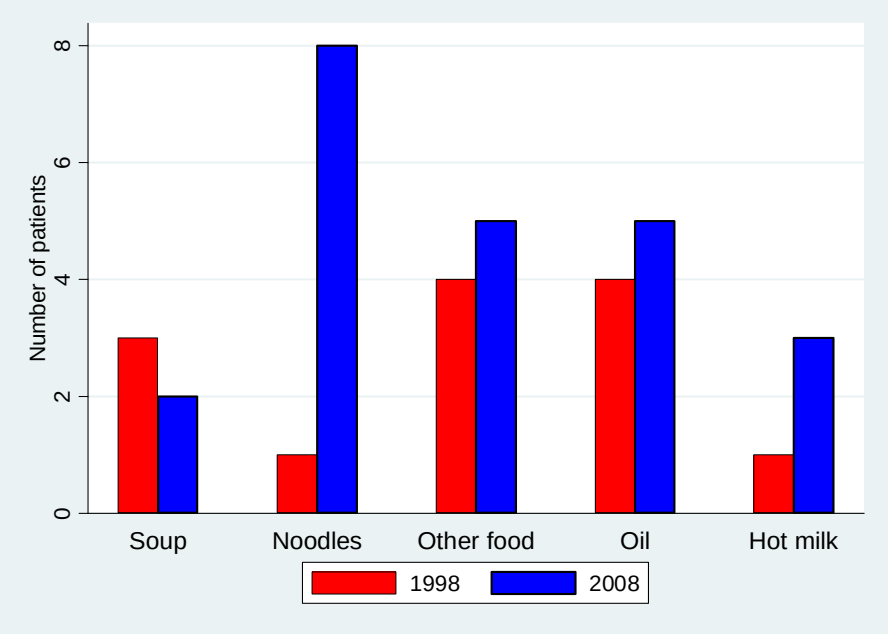


Figure 9: Bar chart showing other types of scald injury by year

The mean age for a scald by hot water was 3 years 6 months in 1998, rising to 4 years 3 months in 2008 (t(97) =-0.4660 p=0.6423). The mean age of injury due to hot drinks rose from 1 year 9 months to 2 years 9 months, (t(70) =-1.6872 p=0.0960). For those scalded by foods and other agents the mean age showed a drop from 5 years 6 months to 4 years 9 months (t=0.5709 (33) p=0.5720). All p values were calculated by independent t-tests on log transformed age data and were non-significant.

The changes in mode of injury between 1998 and 2008 are statistically significantly different in the children aged 1-2 years only ($chi^2(2) = 8.4528$ p = 0.015). Injuries due to hot beverages were unchanged, those due to water were decreased, and there was an increase in injuries from food and other agents.

Table 11 Cross-tabulation of year of admission (in columns) and mode of injury for those aged 1-2 years (in rows) (n=96)

Mode of injury	Year		p value [a]
	1998 n=53	2008 n=38	
Water	41% (22)	29% (11)	p=0.015[b]
Hot drink	55% (29)	47% (18)	
Other	4% (2)	24% (9)	

[a] P-value from Chi^2, χ^2=8.4528 on 2 degrees of freedom

[b] Statistically significant for p≤0.05

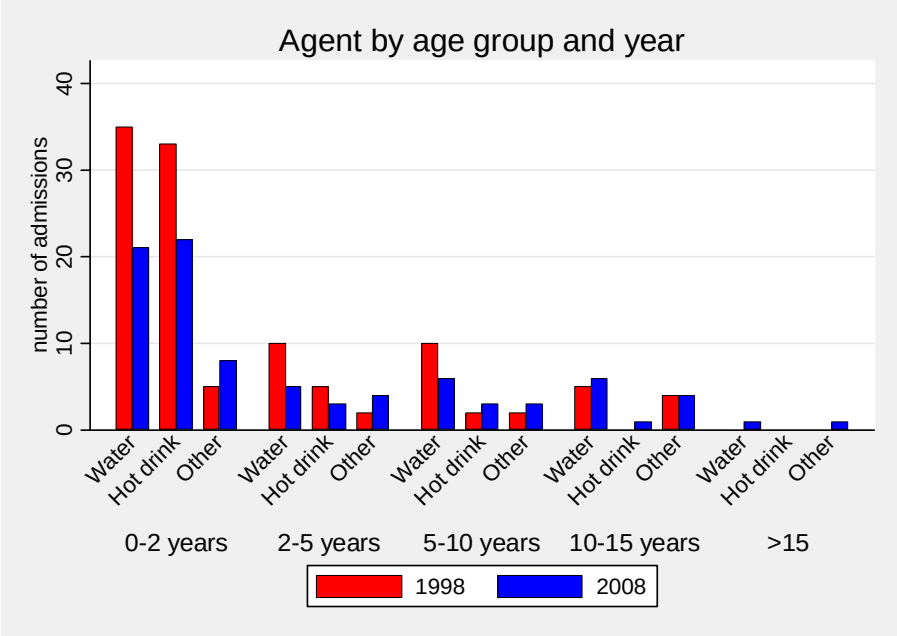


Figure 10: Bar chart showing mode of injury by age group for both years collectively

Temporal patterns

Study data did not show an increase in admissions in any particular season or month of the year, and the figures do not appear to relate to school holidays. When the admissions are assessed by day of the week, a change in pattern between 1998 and 2008 can be detected. The most common time for a scald injury in 1998 was Monday and Tuesday. In 2008 there was a tendency for less scald injuries mid week, with a spike in injury occurrence on a Friday. These changes were not statistically significant.

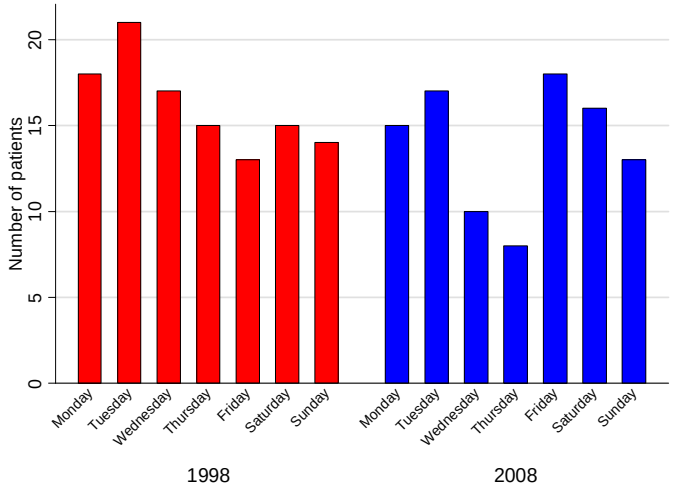


Figure 11: Bar chart showing number of admissions for each day of the week (by year) P= 0.592 calculated by Chi2 =4.6323 on 6 degrees of freedom

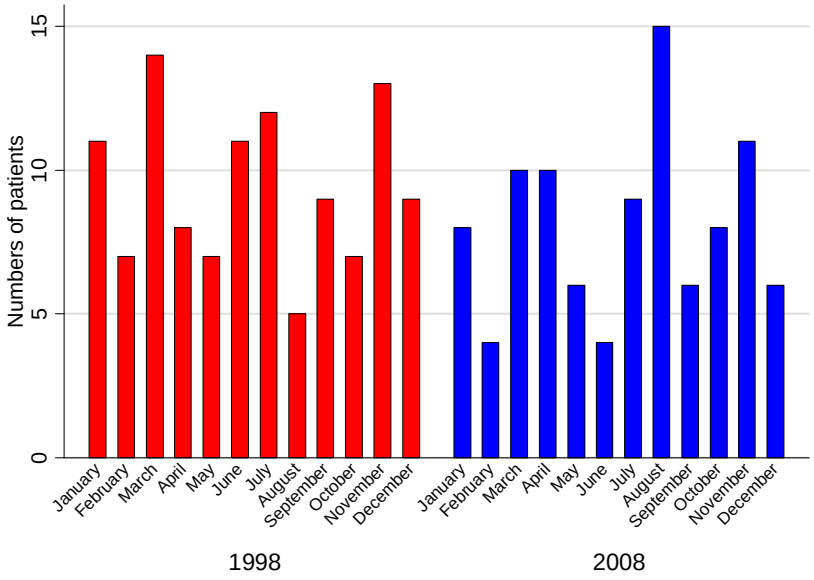


Figure 12: Bar chart showing numbers of admissions by month of year (by year) P= 0.424 calculated by Chi2 =11.2324 on 11 degrees of freedom

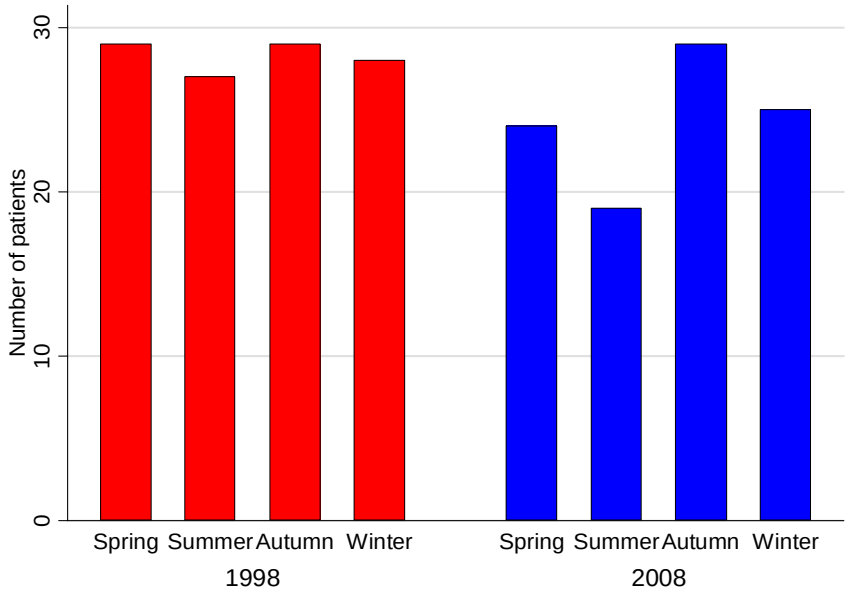


Figure 13: Bar chart showing season of year (by year) p=0.845 calculated by Chi2 =0.8185 on 3 degrees of freedom

Diurnally, the injuries showed a bimodal distribution with peaks at breakfast time and in the early evening. The evening peak had increased in 2008 compared to 1998. Injuries from hot drinks were more common at breakfast but steady through the day. Injuries from other agents tended to happen in the afternoon and evenings, especially those from soup, which only occurred at this time. For the injuries that occurred late at night, between 10 and 11pm: three children between 1 and 2 years were scalded from their carers' hot drinks, one 3 year old girl was scalded to her chest and abdomen by boiling water from a saucepan, a 20 month old child had patchy full thickness burns to both feet from tap water, and an older child spilt hot chocolate drink to the lap. Between 11pm and 6 am there were only two injuries sustained and these were both in older children. One ten year old girl sustained a 1.5% tap water scald to her left hand, and a 13 year old boy was using a steam inhaler for an upper respiratory tract infection, which he spilt in his lap.

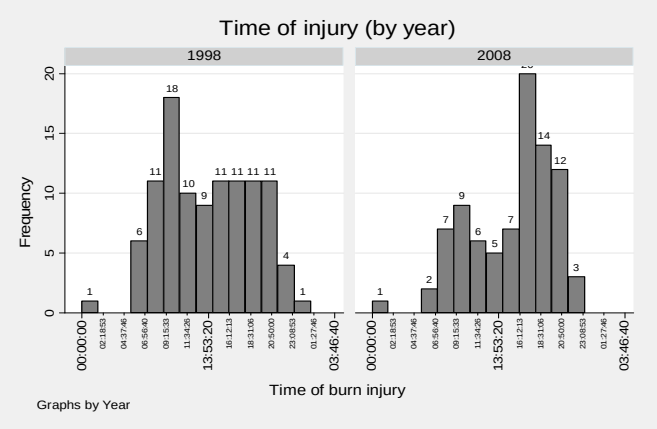


Figure 14: Histogram displaying time of injury by year

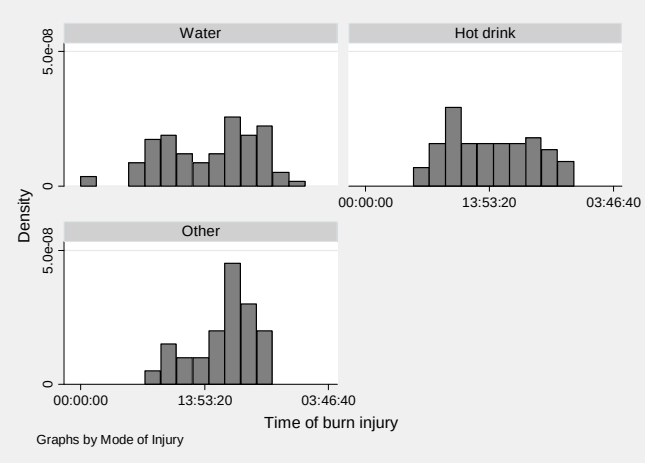


Figure 15: Histogram showing time of injury, stratified by mode of injury

During the study period was there a change in the severity of scald injury between 1998 and 2008?

Size of injury

Total body surface area burnt is recorded as a percentage, and has been treated as continuous data. The distribution of the raw data is skewed to the right, and all the following t-test mean comparisons have been performed on log transformed data to create a normal distribution. The observations are independent and consist of the whole population of admitted scalds in the two years studied.

For all patients, the mean TBSAB in 1998 was 4.6% compared to 5.4% in 2008. This rise was not statistically significant (t=-1.8491 (208), P=0.0659). Further T-test analysis did not show statistical differences in mean TBSAB between 1998 and 2008 for gender, Aboriginality, mode of injury and area of residence by metropolitan area. When analysed by age, a statistically significant increase in wound size was revealed in those aged one to two years old only. In this age group the mean TBSAB was 5.15% in 1998 and 6.49% in 2008 (t(89)=-2.3547, p=0.0207).

For all TBSAB comparisons:

H_0: mean TBSAB 1998=mean TBSAB 2008

H_A: mean TBSAB 1998 ≠ mean TBSAB 2008

Independent samples t-test (2-sided test completed) on log transformed data

Level of significance: α≤0.05

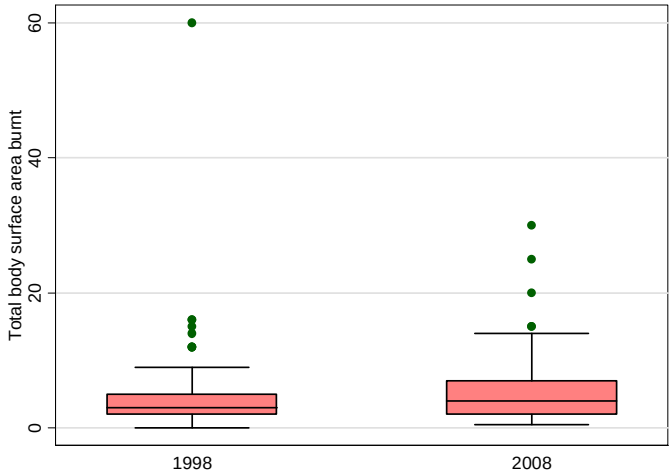


Figure 16: Box and whisker plot comparing distribution of TBSAB in 1998 and 2008

Depth of injury

As previously described, depth of injury is hard to assess, and the study has used the need for surgery as a method of quantifying severity of injury for depth. Overall, the surgery rates, although lower in 2008 compared to 1998, were not statistically different. Further analysis of the need for surgical intervention to heal was repeated for age, gender, indigenous status, area of residence and mode of injury and did not reveal a statistically significant difference for any specific group.

Table 12: Cross-tabulation of year of admission (in columns) and wound closure surgery (in rows)

Surgery	Year		p value[a]
	1998 n=113	2008 n=97	
No	65% (74)	69% (67)	0.659
Yes	35% (39)	31% (30)	

[a] p-values from Fishers Exact Test

When TBSAB is analysed by rates of surgery; the mean TBSAB is statistically bigger for the surgical patients in both 1998 and 2008.

Table 13: Comparison of mean TBSAB between nonsurgical and non-surgical patients for 1998 and 2008

Surgery	Year 1998 n=113	p value[a]	Year 2008 n=97	p value[a]
No	3.16 (2.22)	p=0.0006	4.31 (4.37)	p=0.0001[b]
Yes	6.94 (9.67)		7.77 (5.46)	

[a] P value from Independent samples t test on log transformed data showing mean (SD)

[b] Statistically significant for p≤0.05

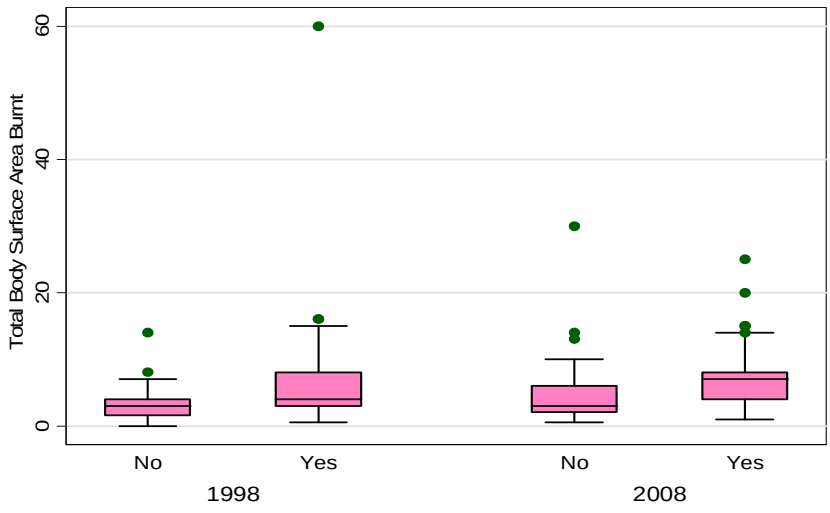


Figure 17: Box and whisker plot comparing TBSAB by surgery by year

During the study period was there an improvement in first aid given to children with scalds during the pre-hospital period?

What first aid was given at the scene by the carer of the scalded child?

Analysis of the data shows that documentation of burns first aid was recorded for 73% (83/113) of patients in 1998 and 87% (85/97) in 2008. In 1998, 7% (8/113) of all patients were known to have received adequate first aid from their carer, and in 2008 this increased to 12% (12/97). However, 27% (n= 30) of patients in 1998 and 12% (n=12) in 2008 did not have first aid documented. The slight improvement in first aid given by the carer was not statistically different (Pearsons Chi^2 (5) =8.9483, p=0.111). Further analysis was completed on the known data.

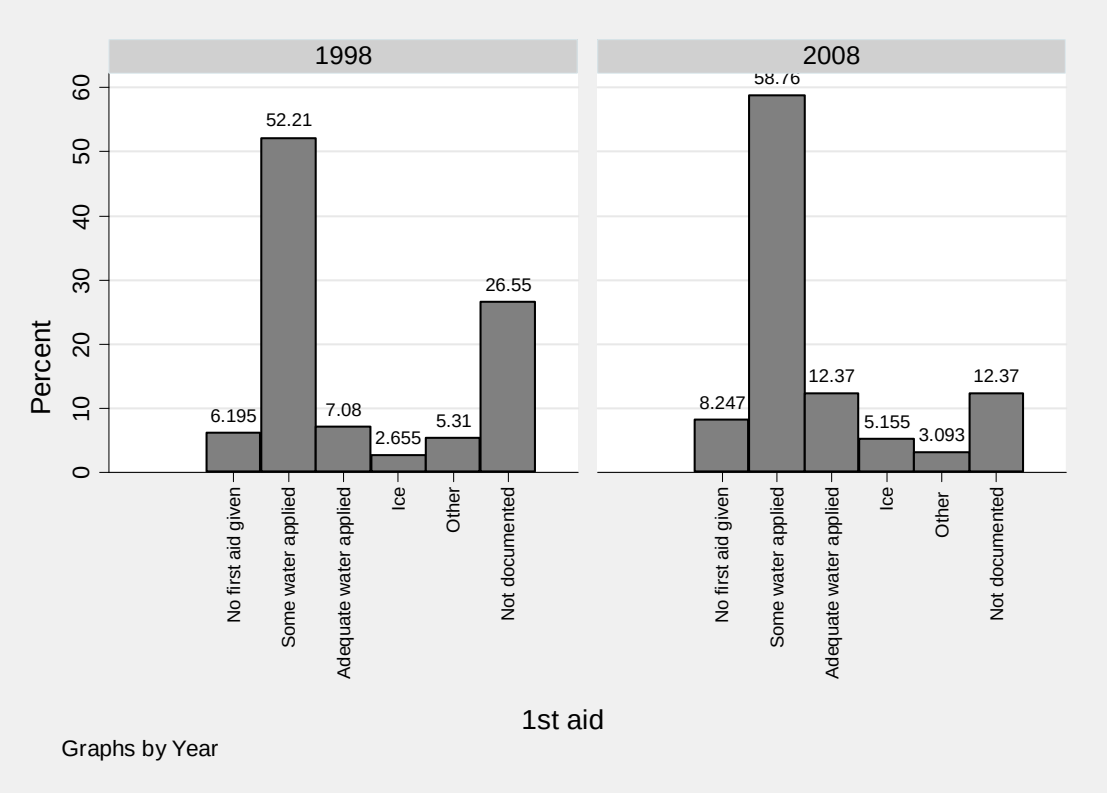


Figure 18: Bar chart comparing the first aid given by the carer in 1998 and 2008

Considering the cases in which first aid was documented, analysis of the first aid given by the carer shows a small increase between 1998 and 2008 in the number of carers who applied twenty minutes or more of cool water to the wound, which increased from

10% (8/83) to 14% (12/85) however this wasnot statistically significant (Pearsons Chi^2 (4) =2.3777 p=0.667).

A further 68% (56/83) received some cool water to their wound in 1998, although the administration time was not recorded in 48% (27/56) of these cases. In 2008, a further 57% (48/85) received some cool water to their wound, administration time not recorded in 17% (8/48) of these cases. In 1998, 76% and 71% in 2008 of those admissions where first aid was documented received cool water to their wound. Of these patients, 11% (9/83) in 1998, and 12% (10/85) in 2008 received inadequate first aid, of less than five minutes of cool water. The differences in known cool water application were not statistically significant (p=0.931, Pearson chi^2).

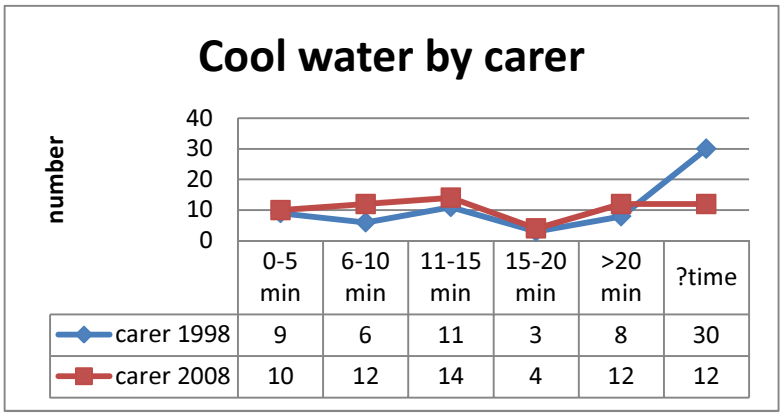


Figure 19: Line graph comparing the duration of cool water given by carer in 1998 (blue line) and 2008 (red line)

In 1998, 7% (6/83) of patients were documented to have received no first aid by their carer, compared to 9% (8/85) in 2008. Overall, the patients who did not receive first aid were slightly older than average with a mean age of 4y 3m (SD 4.806), median 1y 11m (IQR 1-5.6 years) compared to a mean of 3y3m and a median of 1y 7m although this was not statistically significant on log transformed data one sample t-test t=1.7386 (14) p=0.4726), and more were boys (60% compared to 52% of the whole sample) but this was non-significant (z=0.6202 p=0.5351). Their wounds were also larger with a mean TBSAB of 9% (SD 15.1292), but this was not statistically significant on log transformed one sample t-test t=0.6943 (14) p=0.4989, and they had a higher incidence of surgery at 53.33% (non-significant; z=1.7342 p=0.0829). There was no significant difference in ethnicity between this group and the whole sample, with 20% being of Aboriginal origin (z=0.6325 p=0.5271), and 93.33% being Australian born.

In 1998, 4% (3/82) of patients were documented to have received ice to their wound by their carer, this increased non-significantly to 6% (5/85) in 2008. The group that received ice to their burn wound were older (mean age 5y 2m) but not statistically significantly so (p=0.3641), had smaller injuries (mean TBSAB 2.1%) which was statistically significant (p=0.0210), healed in a similar time to average (18 days) and were less likely to require surgery to heal. There is a cultural difference displayed here. None of these patients were of Aboriginal origin, compared to 14% of the entire sample (p=0.2663), and only 50% were Australian, compared to 92% of the sample. The data shows that three of the eight patients were near the median age of 19 months, and the remaining five patients were all over 7y 8m. More boys had ice applied than girls; 62% of the patients who received ice were boys, a higher percentage when compared to whereas the whole sample which comprised of 52% boys but this was not statistically significant (p=0.5522).

Table 14: Patient characteristics for those who received ice to their burn wound by their carer

Year	*Sex*	*Age*	*Ethnicity*	*Scald agent*	*TBSAB*	*Depth of injury*	*Location of injury*	*Surgery*
1998	M	8y	Sri Lankan	Kettle water	4%	Deep partial	Left shoulder	Yes
1998	F	1y 3m	Australian	Soup	1.5%	Partial	Face and left hand	No
1998	M	14m	Swiss	Tap water	3%	Deep partial	Soles both feet	No
2008	F	9m	Saudi Arabia	Black tea	1%	Partial	Right arm/hand	No
2008	F	10y	India	Noodle	1.5%	Partial	Genitalia	No
2008	M	17m	Australian	Tea	3.5%	Deep partial	Left thigh	Yes
2008	M	7y 8m	Australian	Noodle	1%	Superficial partial	Left thigh and genitalia	No
2008	M	10y	Australian	Oil	2%	Superficial partial	Right arm and hand	No

In 1998, 6% (5/83) of patients and 4% (3/85) in 2008 received inappropriate substances to their burn wounds. These eight patients that received non recommended agents to their wound were slightly younger (p=0.3332, t-test), had smaller injuries (p=0.184, t-test) and healed more quickly (p=0.0837, t-test) than the whole sample, although none of these differences were significant. There was a clear cultural element with a variety of ethnic backgrounds evident.

The bimodal distribution of time of injury, was divided into morning and afternoon groups, and transformed for normality. Linear regression analysis was performed on these data to assess the first aid given at the scene in relation to time of day. This revealed a significant difference; there was a tendency to give inappropriate and inadequate first aid if the injury occurred later in the day when compared to the patients who received 20 minutes or more cool running water (Chi^2 (5)=662.08), p=0.000). The mean burn time for poor first aid occurred on average 2hrs and 45 minutes later than those who received good first aid.

Chi^2 testing was completed to compare the differences in first aid given for each day of the week for 1998 and 2008. None of these showed a significant difference. In both years, 7% (8/113 in 1998 and 7/97 in 2008) did not seek medical attentionwithin three hours of injury.

Table 15: Patient characteristics for those who received other types of non-recommended first aid

Year	*Sex*	*Age*	*Ethnicity*	*Scald agent*	*TBS AB*	*Location of injury*	*Surgery*	*First aid*
1998	M	20m	Australian	Hot coffee, no milk	7%	Left arm	Yes	Butter
1998	F	21m	Australian Aboriginal	Tap water	2.5 %	Both feet Right lat thigh	Yes	Butter
1998	M	12m	Somalian	Tea	3%	Face, shoulder	No	Sugar
1998	M	17m	Australian	Hot water from cup	3%	Chest, left neck & trunk	No	Aloe gel
1998	M	9y	Australian	Boiling water	7%	Genitalia Bilateral thighs	No	Sodium bicarbonat e solution
1998	F	12m	English	Coffee	4%	Neck, anterior chest right arm	No	Antiseptic cream
2008	M	21m	Ethiopian	Hot milk	5%	Chest, left axilla/arm chin & neck	No	Vegetable oil
2008	F	6m	Viet-namese	Hot water	2.5 %	Right thigh	Refused by parents	Microwave lemon then vinegar
2008	M	2y	Arabic	Hot tea	3%	Right arm	Yes	Egg white

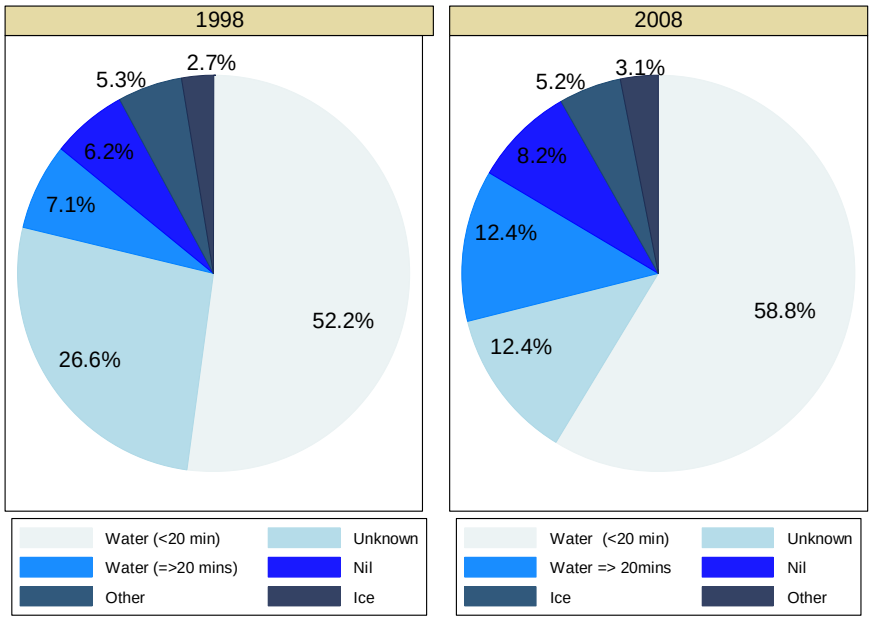


Figure 20: Pie chart comparing first aid given by carer in 1998 and 2008

The amount and type of first aid received was not statistically different in the Aboriginal population in either 1998 or 2008 (1998: Pearsons Chi^2 (5) =4.8549, p=0.434; 2008: Pearsons Chi^2 (5) =5.4329, p=0.365). The first aid received by boys and girls was similar in both years, and had not changed between the years. There was no association between area of living and the first aid given to the child (Chi^2(16)=23.4595; p=0.102).

What first aid was given by others?

Of the 12 patients who were seen by either St John's Ambulance service (SJA) or the Royal Flying Doctor Service (RFDS) in 1998, only two are documented to have received cool running water administered by them, and only one of these were documented to have received it for 20 minutes or more. In 2008, 22 patients had been attended at the scene by SJA or RFDS and of these 5 had received 20 minutes or more of cool water given by emergency personnel. Other actions taken included wet wrap (2 cases in each year) and in 2008, other techniques were emerging; the use of burn aid dressings (2/22), saline irrigation (1/22) and one patient who is not documented to have received cool water to their wound but had it covered in glad wrap. Unfortunately, records were not available in all cases (5/12 in 1998 and 7/22 in 2008), and the small numbers make these figures hard to interpret.

For those who presented initially to GPs, no one received 20 minutes of cool water to their wound in either year, and only 2 in 1998 and 1 in 2008 received any cool running

water at all. Three patients received a wet wrap before transfer to hospital in 1998. In 1998, one patient received ice from the GP.

There were 40 and 44 patients who directly presented to the emergency department of the local hospital in 1998 and 2008 respectively. In 1998, any first aid given in the department was not recorded in 62.5% (25/40) of cases and the most common type of first aid given was a wet wrap to the wound 22.5% (9/40). All except one of remaining patients received cold water (12.5% or 5/40), but no one was documented to have received 20 minutes of cold water. The final patient had ice applied. In 2008, of the 44 patients, 45.5% (20/44) were not documented to have received first aid in the local ED. For the all patients 31.8% (14/44) received cool water, 4 of which were of the required duration of 20 minutes or more, 13.6% (6/44) received wet wrap, and 9.1% (4/44) were documented to have had no first aid.

Did the distance to the place of first attendance affect the first aid given at the scene?

In 1998 the mean distance to first medical attention was 13.7km and in 2008 it was 10.0km. However, the distribution was skewed to the right and the medians in both years were 7km. A non-parametric equality of medians test was performed on data split by year of admission. Patients who had been attended by ambulance personnel or medical professionals from the RFDS were removed from the analysis, as were those for whom documentation of first aid at the scene was not completed. In 1998, there was no difference in the median distance to first medical intervention in each group (Pearson chi^2 =5.05615; p=0.783). Linear regression analysis was completed comparing the distance to PMH to the place of first attendance, and whether this had an impact on whether carers used emergency services, their GP, or an emergency department. This analysis did not show that distance from PMH impacted the type of first medical attention sought by carers .

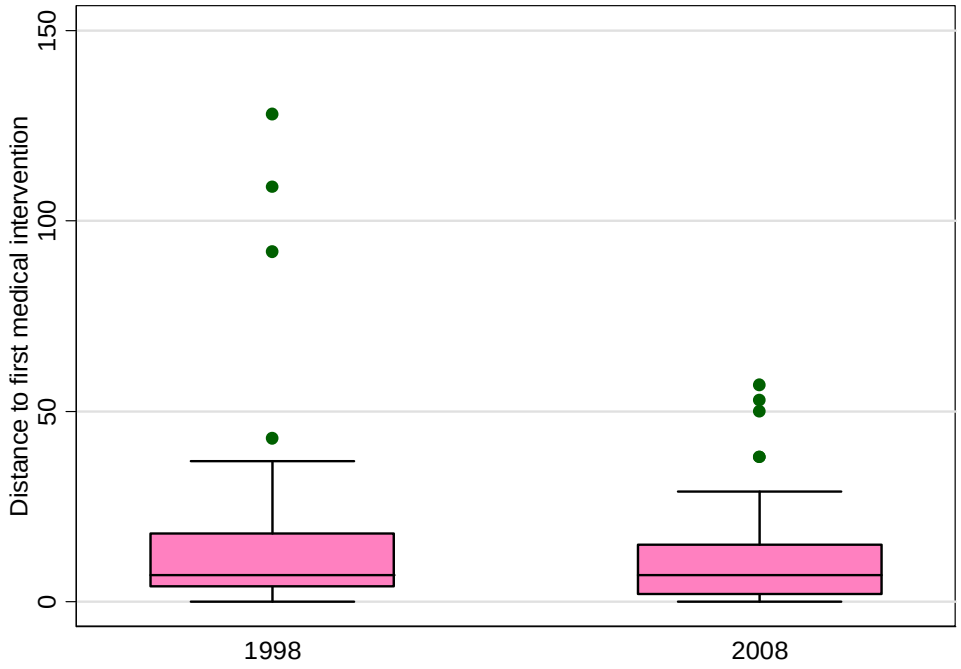


Figure 21: Box and whisker plot comparing distance to first medical intervention

Table 16: Table comparing distance to first medical intervention in 1998 and 2008

Summary statistic	Year		p value[a]
	1998 n=113	2008 n=97	
Mean (SD)	13.6 km (19.01)	10.0km (11.37)	0.578
Median (IQR)	7 km (4 to 18km)	7km (2 to 15km)	

[a] Wilcoxon (Mann-Whitney) rank sum test z=1.911

During the study period were there changes in the actions of the carers present at the time of injury?

Not accounting for the cases in which times were inadequately documented; in 1998 63.37% (64/101) of carers either self presented to their local emergency department or called the emergency services, either the state ambulance service or the Royal Flying Doctor Service (RFDS). By 2008, this had increased to 77.65% (66/85). For those who called emergency services, there was an increase in the numbers who received 20 minutes of cool water between the years. For those who received adequate first aid in 1998, 25% (n=2) were attended at the scene by emergency services, and by 2008 this had increased to 50% (n=6). See figure 22. The small figures do not allow meaningful statistical analysis for these data. There was a reduction in the number of parents or carers who sought first medical attention for their child from their General Practitioner. In 1998, 18.25% (21/112) of children attended their GP first, and this had dropped to 7.22% (7/97) in 2008. These differences were statistically significant on Pearson's Chi2 tests (Chi2(4)=18.5987, p=0.001). The data was also analysed to assess whether the type of medical help sought caused delays of over 3 hours. Seeking advice from a GP did not prolong the time to first medical intervention.

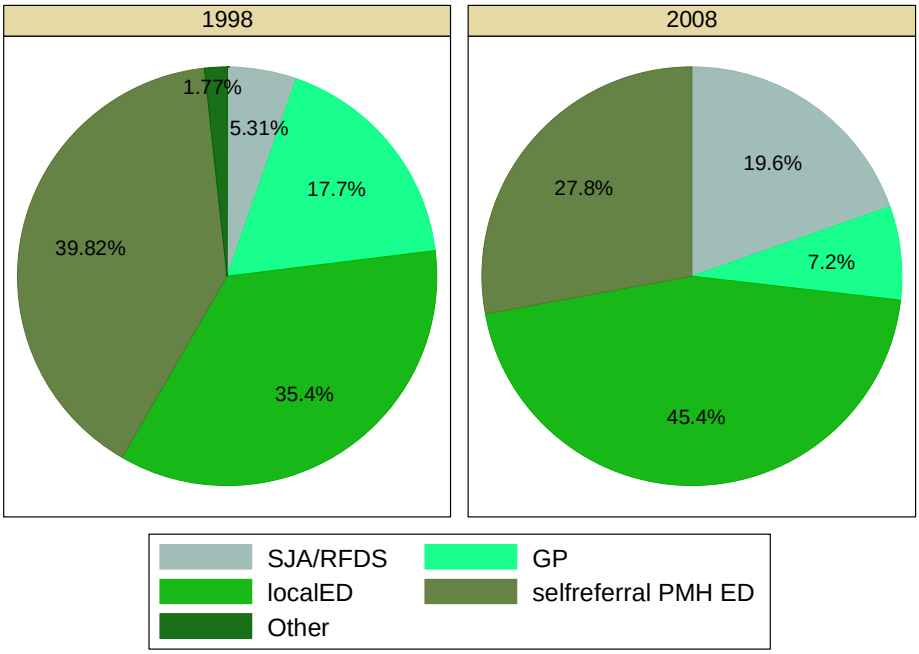


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Figure 22: Pie chart comparing the initial action taken by the carer in 1998 and 2008

Those with a higher total body surface area burnt were more likely to call emergency services. The mean TBSAB for those patients who did in 1998 (mean=6.4%) was not significantly different to the overall mean in 1998 (4.46%) on t-test analysis (t[4]=0.7685, p=0.1517). However, in 2008 the mean TBSAB was statistically larger for the patients that called emergency services having risen from 5.38% to 11.14% (t[17]=4.5844, p=0.003).

Time in PMH ED

The distribution of the time spent in PMH ED was positively skewed and able to be normalised by log transformation. For all patients who attended the emergency department in both years (n=193) the mean time spent in 1998 was 1 hour (SD 35mins), and in 2008 had increased to 1 hour 44 minutes (SD 1 hour 18). This finding was statistically significant (t=-4.8221; p=0.000). These data were further assessed by two separate criteria. Firstly, their referral source; whether the patient came directly from home, was referred from another ED, the GP or SJA/RFDS and secondly, their care pathway from PMH ED whether the patient was destined for ward admission, or was to return home for later review in OPC.

Table 17: Table comparing mean time spent in PMH ED by year stratified by referral source

Source	Year		p value[a]
	1998 Mean(SD)	2008 Mean(SD)	
SJA/RFDS	00:56 (00:27) n=6	01:12 (00:38) n=19	0.8015 (t(22)= -0.2545)
GP	01:06 (00:42) n=20	02:11 (01:18) n=7	0.0246 (t(25=-2.3924)
other ED	00:51 (00:29) n=33	01:33 (01:04) n=39	0.0013[b] (t(67)= -3.3532)
Self-presentation	01:03 (00:36) n=45	02:15 (01:45) n=27	0.0002[b] (t(70)= -3.8917)

[a] P value calculated from independent sample t-test on log transformed data (hh:mm)
[b] Statistically significant for p≤0.05

Table 18: Table comparing mean time spent in PMH ED by year stratified by destination

Source	n	Mean(SD)	t	df	p
OPC	3	01:4 (00:44)	-0.3794	12	0.7110
	11	00:59 (00:34)			
Ward	100	02:03 (01:07)	-4.2992	178	0.000[b]
	80	01:40 (01:19)			

[a] P value calculated from independent sample t-test on log transformed data (hh:mm)
[b] Statistically significant for p≤0.05

Wound severity was found to be a factor that influenced the speed of ward admission. The two variables were correlated in both years: in 1998: $r(78) = -0.2188$, $p = 0.0287$; in 2008: $r(98) = -0.2667$, $p = 0.0168$. This means that the more severe wounds are a predictor of faster admission times, and shorter lengths of stay in PMH ED.

During the study period were there changes in medical treatment and nursing care of paediatric patients with scald injury?

Analgesia

Prior to presentation at PMH

Forty percent of patients in 1998 and 50% in 2008 received pain relief before reaching PMH ED. There was a statistically significant reduction in the use of IM analgesia and a statistically significant increase in the use of IN fentanyl for pain relief. Of the 5 and 6 patients respectively that received an IV cannula and analgesia, 4 patients in 1998 and 3 in 2008 received an IV cannula for hydration. The groups in the tables below are not exclusive, 30% (14/46) in 1998 and 19% (19/49) in 2008 received more than one type of analgesia.

Table 19: Comparison of analgesia given 1998 and 2008 for those patients who received analgesia before presenting to PMH ED

Analgesic (route)	Year		p value[a]
	1998 n=46	2008 n=49	
Pethidine IM	44% (20)	2% (1)	0.000[b]
Pethidine IV	7% (3)	0%	0.110
Morphine IM	20% (9)	29% (14)	0.345
Morphine IV	4% (2)	14% (7)	0.160
Morphine PO	0%	16% (8)	0.009[b]
Fentanyl IN	0%	33% 16	0.000[b]
Paracetamol PO	9% (4)	20% (10)	0.149
Painstop PO	41% (19)	16% (8)	0.012[b]
Ibuprofen PO	0%	14% (7)	0.012[b]
Ketamine IM	2% (1)	4% (2)	1.000
Paracetamol PR	2% (1)	4% (2)	1.000

[a] pvalue calculated from Fishers exact test
[b] Statistically significant for p≤0.05

Table 20: Cross-tabulation comparing route of analgesia for 1998 and 2008 for those patients who received analgesia before presenting to PMH ED

Route of analgesia	Year		p value[a]
	1998 n=46	2008 n=49	
IM	67% (30)	39% (19)	0.008[b]
IV	11% (5)	12% (6)	1.000
PO	53% (24)	55% (27)	1.000
IN	0%	33% (16)	0.000[b]

[a] pvalue calculated from Fishers exact test
[b] Statistically significant for p≤0.05

In PMH ED

Overall, 40% of patients in 1998 and 50% in 2008 received pain relief in PMH ED. IM analgesia was not used in 2008 and therefore showed a statistically significant decrease from 1998. There was a statistically significant increase in the use of intranasal (IN) fentanyl and oral analgesics. The groups in Table 21 are not exclusive, however, in 1998 no patient had more than one type of analgesia but in 2008 44% (21/48) of those who received analgesia had more than one type.

Table 21: Comparison of analgesia given 1998 and 2008 for those patients who received analgesia in PMH ED

Analgesic (route)	Year		p value[a]
	1998 n=61	2008 n=48	
Pethidine IV	1.6% (1)	0%	1.000
Morphine IM	57% (35)	2% (1/48)	0.000[b]
Morphine IV	5% (3)	27% (13/61)	0.002[b]
Morphine PO	28% (17)	6% (3/48)	0.005[b]
Fentanyl IN	0%	44% (21)	0.000[b]
Paracetamol PO	0%	12% (6)	0.006[b]
Painstop PO	10% (6)	25% (12)	0.041[b]
Ibuprofen PO	0%	33% (16)	0.000[b]
Codeine PO	0%	2% (1)	0.440

[a] p-value calculated from Fishers exact test
[b] Statistically significant for p≤0.05

Table 22: Cross-tabulation comparing route of analgesia for 1998 and 2008 for those patients who received analgesia in PMH ED

Route of analgesia	Year		p value[a]
	1998 n=61	2008 n=48	
IM	56% (34)	0%	0.000[b]
IV	8% (5)	21% (10)	0.091
PO	36% (22)	73% (35)	0.000[b]
IN	0%	50% (24)	0.000[b]

[a] p-value calculated from Fishers exact test
[b] Statistically significant for p≤0.05

On ward

Table 23: Comparison of analgesia given on ward (in rows) by year (in columns)

Analgesic (route)	Year		p value[a]
	1998 n=113	2008 n=97	
Morphine (PO)	86% (97)	79% (77)	0.271
Paracetamol (PO)	77% (87)	72% (70)	0.431
Painstop/ Liquegesic (PO)	69% (79)	61% (56)	0.083
Ibuprofen (PO)	0%	79% (77)	0.000[b]
Entonox (inh)	7% (9)	1% (1)	0.022[b]
Morphine (IV)	3% (3)	1% (1)	0.370
Pethidine (IV)	1% (1)	0%	1.000
Pethidine (IM)	1% (1)	0%	1.000
Fentanyl (IV)	0%	1% (1)	1.000
Ketamine (IV)	0%	1% (1)	1.000

[a] p value calculated by Fishers Exact Test
[b] Statistically significant for p≤0.05

Table 23 shows the most common types of analgesics used on the ward. The most used agents were given orally, with the occasional use of IV agents. Pethidine was

used in 1998, not in 2008, and given IM on one occasion. Ibuprofen is an agent used frequently in 2008 that was not used in 1998.

Table 24: Comparison of analgesia combinations for both years (groups not exclusive) n=210

Analgesic (route)	Paracetamol (PO)	Painstop/ Liquegesic (PO)	Ibuprofen (PO)	Entonox (inh)	Morphine (IV)
Morphine (PO)	143	120	69	8	2
Paracetamol (PO)		111	63	9	2
Painstop/ Liquegesic (PO)			54	5	3
Ibuprofen (PO)				1	0
Entonox (inh)					0
Morphine (IV)					

Table 24 shows that most patients received a combination of analgesic agents during their admission, the core choices were oral morphine, paracetamol, painstop or liquegesic and ibuprofen.

Intravenous access and hydration

There was no change in the numbers of children who had IV cannulae inserted between 1998 and 2008 before reaching PMH ED (p=0.411) and a non-significant increase in those who received a cannula in PMH ED between the two years (p=0.150) and no difference for those who received IV hydration (p=0.409).

Twelve patients in 1998 and thirteen patients in 2008 met the criteria for fluid resuscitation. Two patients in 1998 who did not receive hydration were just under 18 months old both with a TBSAB of 8%, and one patient did not receive fluids at 1y 7m with a TBSAB of 10%.

For the remaining patients; in 1998 4 patients had fluids started before PMH presentation, 4 patients had fluids started in PMH ED, and 2 had fluids started on the ward (one of whom was admitted directly to the ward.). In 2008, 3 patients had fluids

started before PMH presentation, 6 were cannulated in ED, 2 of whom had fluids started there, the other 4 had fluids started on the ward, and 2 had an IV cannula inserted and fluids on the ward. One 13 month old patient with an 8% scald to bilateral thighs was managed at a regional centre and referred to PMH as an outpatient, with no notes of care given. Thus, the early management of this patient is unknown.

Table 25: Numbers of patients assessed to meet the resuscitation fluid criteria

Scalds assessed to require resus fluids	1998 n=113	2008 n=97
8% TBSAB & <=18m	9	6
10% TBSAB & >18m	3	7

Table 26: Cross-tabulation of IV access and hydration status before ward admission

IV access or hydration status	Year		p value
	1998	2008	
IV access pre PMH			
No	75% 47/62	80% 48/60	p=0.411[a] $Chi^2(2)=1.7782$
Yes	18% 11/62	18% 11/60	
Unsuccessful	6% 4/62	2% 1/60	
IV hydration pre PMH (for those given IV access)			
No	33% 3/11	33% 3/11	p=1.000[b]
Yes	88% 8/11	88% 8/11	
IV access PMH ED			
No	83% 88/106	72% 63/88	p=0.150[a] $Chi^2(2)=3.7885$
Yes	8% 9/106	8% 14/88	
In situ	8% 9/106	12% 11/88	
IV hydration PMH ED			
No	88% 93/106	82% 71/87	p=0.409[a] $Chi^2(2)=1.709$
Yes	5% 5/106	9% 8/87	
Continuing	8% 8/106	9% 8/87	

[a] p value calculated by Chi^2 test
[b] p value calculated by Fishers Exact Test

Dressings

Table 27 demonstrates who applied silver dressings, and at which stage of the patient journey. Table 28 shows the growing use of Acticoat™ in 2008 as a preferred dressing by specialist staff, but not fully adopted by non-specialist medical staff.

Table 27: Comparison of the place of application of silver dressings (in rows) by year (in columns)

Silver dressings	Year		p value[a]
	1998	2008	
Pre PMH silver dsg	20% (7/63)	46% (21/61)	0.002[b]
PMH ED silver dsg	4% (4/94)	18% (12/65)	0.004[b]
Ward silver dsg	94% (96/102)	91% (58/64)	0.540
Total silver	95% (107/113)	94% (91/97)	1.000

[a] p-value calculated from Fishers exact test
[b] Statistically significant for p≤0.05

Table 28: Comparison of the use of SSD vs Acticoat™ by place of application (in rows) and year (in columns)

Place	Dressing type	Year		p value[a]
		1998	2008	
Pre PMH	SSD	100% (7/7)	90% (19/21)	p=0.397 $Chi^2(1)=0.7179$
	Acticoat™	0%	10% 2/21	
PMH ED	SSD	100% (3/3)	67% (8/12)	p=0.243 $Chi^2(1)=1.3636$
	Acticoat™	0%	33% (4/12)	
Ward	SSD	100% 96/96	36% (21/58)	p=0.000[c] $Chi^2(2)=80.6083$
	Acticoat™	0%	48% (28/58)	
	Combined[b]	0%	16% (9/58)	

[a] p-value calculated from Chi^2 test
[b] SSD used on wound sites inappropriate for Acticoat™
[c] Statistically significant for p≤0.05

Microbiology

Infection rates were compared between years for wound, upper respiratory tract, systemic sepsis, and other types. A number of patients had pyrexia of unknown origin, for which antibiotics were given, so these have been accounted separately. Those with wound infections were stratified by when the infection occurred; pre admission, during admission or after discharge. The types of colonising bacteria are also recorded. Factors affecting the causes of wound infections are explored, as are the effects of wound infection on scar outcome.

There was no change in the incidence of upper respiratory tract infection between 1998 and 2008, with the incidence remaining at 7% and 8% in respective years (FET; 0.798). There was one case of sepsis in 2008, and none in 1998, indicating no significant change (FET; p=0.462). For other infections, which included otitis media (3 cases), urinary tract infections (1 case), rotavirus (1 case), and lower respiratory tract infections (2 cases) there was no statistical change between the years (FET; p=0.706). For wound infections Table 29 shows an increase in infection rate, which showed significance in the post-discharge group only.

Table 29: Comparison of the types of wound infection (in rows) by year (in columns) stratified by time of occurrence

	Year		p value[a]
	1998 113	2008 97	
Pre-admission	6% (7)	10% (10)	p=0.317
During admission	3% (3)	3% (3)	p=1.000
Post discharge	1% (1)	6% (6)	p=0.050[b]
All wound infection	10% (11)	20% (19)	p=0.042[b]

[a] p-value calculated from Fishers exact test
[b] Statistically significant for p≤0.05

In 1998, 10% of patients got a wound infection, with infection rate increasing significantly to 20% in 2008. These data were stratified according to time when infection was acquired.

Preadmission wound infections

In 1998, 63% (7/11) and in 2008, 53% (10/19) of wound infections were acquired before admission. For both years; independent factors for preadmission infections included delay to medical attention (Fishers exact test; p=0.010), the Aboriginal population (Fishers exact test; p=0.003), patients admitted from the non-metropolitan areas neared significance (Fishers exact test; p=0.059). Being older or younger than

three years of age (Fishers exact test; p=0.783) and gender (Fishers exact test; p=0.624) did not affect the tendency for preadmission infections. The application of silver dressings before PMH presentation (Fishers exact test; p=1.000), or at any time (Fishers exact test; p=0.062), made no difference to the rate of infection. Analysis of these data with logistical regression analysis would ascertain the odds ratio for these factors, but sample size was not large enough to calculate this. Sample size analysis for three covariates, with a preadmission infection rate of 7%, shows that 395 patients are needed for such an analysis.

Between 1998 and 2008 there was no difference to preadmission infection rate in relation to any of these factors. For non metropolitan patients (Fishers exact test; p=0.745) for delayed patients (Fishers exact test; p=0.569), and for Aboriginal patients (Fishers exact test; p=1.000).

S. Aureus, *S. Pyogenes,* and *B. Cereus* were found to be highly significant colonisers of these wounds. *P. aeruginosa* was a prominent coloniser, but did not reach statistical significance (Fishers exact test; p=0.078), and there were no wounds with preadmission wound infections that were colonised with *Acinetobacter, Enterobacter, coagulase-negative Staphlococcus* or *Aeromonas.* (Table 30).

Table 30: Comparison of preadmission wound infection (in columns) by causative bacteria (in rows)

Bacteria	Preadmission wound infection		p value[a]
	No	Yes	
S. aureus	11% (19/180)	71% (12/17)	0.000[b]
S. pyogenes	1% (2/180)	35% (6/17)	0.000[b]
B. cereus	0.5% (1/180)	12% (2/17)	0.018[b]
S. pneumoniae	0.5% (1/180)	12% (2/17)	0.018[b]
P. aeruginosa	2% (4/180)	12% (2/170	0.078
Group G streptococci	0.5% (1/180)	6% (1/17)	0.157
Acinetobacter	4% (8/180)	0%	1.000
Enterobacter	3% (6/180)	0%	1.000
coagulase-negative Staphlococcus	9% (16/180)	0%	0.370
Aeromonas	1.5% (3/180)	0%	1.000

[a] p-value calculated from Fishers exact test
[b] Statistically significant for p≤0.05

Wound infection during admission

In both 1998 and 2008 3% of patients acquired a wound infection while in hospital (3/113 and 3/97). This accounted for 27% (3/11) of all wound infections in 1998, and 16% (3/19) in 2008. For all patients; wound infections were not more common among those who delayed seeking medical attention (Fishers exact test; p=0.379) but were more common in patients admitted from the non-metropolitan areas (Fishers exact test; p=0.020). The Aboriginal population were more likely to acquire a wound infection whilst an inpatient (Fishers exact test; p=0.036). Being older or younger than three years of age (Fishers exact test; p=0.363) and gender (Fishers exact test; p=0.424) did not affect the tendency for in-hospital wound infections. The application of silver dressings before PMH presentation (Fishers exact test; p=1.000), made no difference to the rate of infection, and nor did silver dressings overall (Fishers exact test; p=0.301).

S. aureus, *S. pyogenes,* and *P. aeruginosa* all showed highly significant colonisers of these wounds. *Coagulase-negative Staphylococcus* was found in two of the infected wounds but its presence made no difference to the likelihood of wound infection (Fishers exact test; p=0.420), and there were no wounds with infections that were colonised with *B. cereus, S. pneumoniae, Group G streptococcus.. Acinetobacter, Enterobacter,* or *Aeromonas. Providencia stuartii* was late coloniser of the severe 60% TBSAB scald in 1998.

Table 31: Comparison of during admission wound infection (in columns) by causative bacteria (in rows)

Bacteria	Wound infection during admission		p value[a]
	No	Yes	
	180	6	
S. aureus	11% (19)	100% (6)	0.000[b]
S. pyogenes	1% (2)	33% (2)	0.028[b]
B. cereus	0.5% (1)	0%	1.000
S. pneumoniae	0.5% (1)	0%	1.000
P. aeruginosa	2% (4)	33% (2)	0.018[b]
Group G streptococcus	0.5% (1)	0%	1.000
Acinetobacter	4% (6)	0%	1.000
Enterobacter	3% (6)	0%	1.000
Coagulase-negative Staphlococcus	9% (16)	33% (2)	0.420
Aeromonas	2%(4)	0%	1.000
Providencia	0%	16% (1)	0.041[b]

[a] p-value calculated from Fishers exact test
[b] Statistically significant for p≤0.05

Between 1998 and 2008 there was no difference to wound infection rate during admission in relation to any of these factors. There was no change for non-metropolitan patients (Fishers exact test; p=1.000), for delayed admission patients (Fishers exact test; p=1.000), and for Aboriginal patients (Fishers exact test; p=0.553).

Post-discharge wound infection

For wound infections after discharge; 1% (1/113) in 1998, and 6% (6/97) required antibiotic treatment. The increase in the post-discharge wound infection over the study period was significant (p=0.050). There was no increased incidence for aboriginal patients (Fishers Exact Test; p=0.597), children under or over 3 years (Fishers Exact Test; p=1.000), gender (Fishers Exact Test; p=0.451), non-metropolitan residence (Fishers Exact Test; p=0.350), delay to presentation (Fishers Exact Test; p=0.489), or type of silver dressing applied to the wound (Fishers Exact Test; p=1.000). Length of hospital stay was compared for these patients, but independent t-test analysis showed no difference in mean stay on square root transformed data (t=0.1308, p=0.9212). The only coloniser for these wounds was *Staphylococcus* (FET; 0.000). All six post-discharge wound infections occurred in an upper limb or shoulder wound.

Risk factors for wound infection

Overall, for both years, length of stay comparison for all patients classified as having a wound infection showed a statistically significant increase (t=-2.9184(df33.5543); p=0.0062). However, when this was stratified by pre, during admission or post discharge wound infection, the difference was only significant for those who sustained a wound infection as an inpatient (t=-3.6717(df5.1367); p= 0.0137). Mean length of stay increased from 7.5 days for those with no infection to 28.2 days for patients acquiring an infection during admission.

Overall, for both years, TBSAB comparison for all patients classified as having a wound infection was not different from those who did not have wound infection (t=-1.6300(df32.454); p=0.1128). However, when this was stratified by pre, during admission or post discharge wound infection, those who acquired a wound infection as an inpatient had larger wounds (t=-4.3553[df5.2687]; p= 0.0065). Mean TBSAB for those who did not acquire a wound infection in hospital infection was 4.4% compared to 20.3% for patients who acquired an infection during admission. The mean TBSAB for those who got a wound infection after discharge was 10.3% compared to 4.6% TBSAB for no post-discharge infection (t=-2.4425(df6.3809); p=0.0479).

Time to healing was not able to be estimated for all patients. The data did not follow a normal distribution and was not able to be normalised successfully by transformation. Non-parametric comparison of medians Mann-Whitney testing was completed on this variable. The presence of a wound infection increased median time to healing 15 days (IQR 10-21) to 20.5 days (IQR 15-38). This was significant (z=-2.953; p=0.0031). On stratification, this was only true for the post-discharge wound infection group (z=-2.979; p=0.0029). Incidence of surgery was greater for those who acquired a wound infection whilst in hospital (Table 32).

Table 32: Comparison of incidence of surgery (in columns) with wound infection (in rows)

Wound infection type	Surgery		p value[a]
	No n=142	Yes n=68	
Pre-admission	6% (9)	12% (8)	0.186
During admission	<1% (1)	7% (5)	0.014[b]
Post discharge	3% (4)	3% (3)	0.684
All wound infection	10% (14)	23% (16)	0.011[b]

[a] p-value calculated from Fishers exact test
[b] Statistically significant for p≤0.05

The warm summer conditions might have increased the incidence of wound infection but on Chi^2 tests no infection type was affected by season; preadmission infection (Pearson $chi^2(3) = 2.5493$; p=0.466), wound infection during admission (Pearson $chi^2(3)=2.0260$; p= 0.567) or after discharge (Pearson $chi^2(3) = 4.9425$; p=0.176).

Effects of wound colonisation

An analysis of all colonising bacteria, with or without clinical wound infection, showed an association with increased time for healing for *S. aureus* (z=-2.927; p=0.0034) and *B. cereus* (z=-1.967; p= 0.0492) only. TBSAB was larger in the *S. aureus* group only (t=-2.399; p=0.0199), and this was true for incidence to surgery (FET; p=0.002). For all other colonising bacteria, there was no statistical difference found for these variables.

Surgery

In 1998, 34.5% (39/113) of admissions required wound closure surgery, this remained constant in 2008, when 30.9% (30/97) had surgery (Fisher Exact Test, p=0.769). One of these patients also received Integra™ (Integra Lifesciences, New Jersey) to their wound in 1998. This is a porous collagen matrix that allows cellular and capillary growth in deep wounds to scaffold and remodel the skin. One patient, not included in the table below, went to surgery for dermabrasion only, and others re-attended theatre for dressing changes. As this was not surgery designed for wound closure, these cases were not accounted for in these initial figures. All surgical types were not significantly changed between the years, and this was unchanged when stratified by type of surgery.

Table 33: Comparison of type of surgery (in rows) by year (in columns)

Surgery type	Year		p value[a]
	1998 n=39	2008 n=30	
SSG only	21% (8)	10% (3)	0.229
SSG and CEA/ReCell	36% (14)	43% (13)	0.839
CEA/ReCell only	44% (17)	47% (14)	1.000

[a] p-value calculated from Fishers exact test

Independent groups T-test analysis of age showed no difference for each surgical type between 1998 and 2008. Independent groups T-test analysis of TBSAB showed an increase for the application of cells only in 2008 compared to 1998. There was no difference in TBSAB between 1998 and 2008 for the other two surgical types. One child had Integra™ used in surgery as an additional procedure due to severe full thickness burns with 60% TBSAB. Median time to surgery following injury was unchanged in both years for all surgical types.

Table 34: Comparison of median time to surgery by surgical type (in rows) between years (in columns

Surgery type	Year		p value[a]
	1998 Day 1st surgery[b]	2008 Day 1st surgery[b]	
SSG only	8 (7 to 11)	9 (6.5 to 10)	0.7993
SSG and CEA/ReCell	7 (6 to 9)	9 (6 to 9)	0.7114
CEA/ReCell only	8 (7 to 10)	10.5 (8 to 13)	0.0891

[a] p-value calculated from Mann-Whitney Wilcoxon rank sum test
[b] median (IQR)

Table 35: Comparison of TBSAB by year, stratified by type of surgery

Year	Surgery type	TBSAB–mean (SD)	p value
1998	SSG	3.25 (3.71)	p=0.6992
2008	SSG	4.33% (6 .93)	[t(9)= -0.3989]
1998	SSG+ CEA	11.79%(14.86)	p=0.8120
2008	SSG + ReCell	7.96% (4.89)	[t(25)= 0.2404]
1998	CEA/ rapid cells only	4.71% (2.02)	t(29)= -2.4442
2008	ReCell only	8.32% (6.10)	p=0.0208[b]

[a] p-value calculated independent samples t-test
[b] Statistically significant for p≤0.05

In both years, length of stay was statistically significantly longer for those patients who had surgery. When stratified for residence by metropolitan areas, those who lived in outer metro areas in 2008 and who had surgery did not have longer stays.

Table 36: Comparison of length of stay (in columns) by surgery and areas of residence by year (in rows)

Length of stay	Area of residence	Length of stay[b]				p value[a]
		Surgery		No surgery		
		N		n		
1998	All areas	39/113	14.97 (9.99)	74/113	5.36 (3.56)	0.000[c]
	Inner metro	11/113	12.4 (7.24))	29/113	5.21 (4.02)	0.0039[c]
	Outer metro	20/113	12.5 (4.49)	35/113	5.37 (3.54)	0.00001[c]
	Non-metro	8/113	24.9 (16.5)	10/113	5.8 (2.15)	0.0001[c]
2008	All areas	29/97	12.9 (9.72)	68/97	4.62 (3.74)	0.005[c]
	Inner metro	9/97	12 (6.24)	31/97	4.03 (3.92)	0.0000[c]
	Outer metro	8/97	6.5 (6.55)	22/97	5.96 (4.05)	0.5347
	Non-metro	12/97	19.1 (10.5)	15/97	4.2 (2.96)	0.0007[c]

[a] p-value calculated from independent group ttest
[b] mean (SD)
[c] Statistically significant for p≤0.05

Scar management

Scar management techniques were unchanged between the years overall or when stratified by surgery. When assessed by need for surgery; there was a greater use of pressure garments and silicone in both years for the surgical patients, and greater use of tubigrip only in 1998 for these. Steroid injections were only required in the patients who had received surgery. Scar reconstruction was required in one 1998 non-surgical patient, and no patients in 2008. Physiotherapy need was unrelated to surgery in either year, but was related to the need for scar reconstruction overall (FET; p=0.000).

Table 37: Cross-tabulation showing scar management intervention (in rows) by year (in columns); all analysis and stratified analysis by surgery

	Year	1998 % (n)	2008 % (n)	p value[a]
Tubigrip	All	51% (58/113)	46% (45/97)	0.492
	Surgery	67% 26/39	55% 16/29	0.450
	No surgery	43% 32/74	43% 29/68	1.000
Pressure garments	All	27% (30/113)	28% (27/97)	0.877
	Surgery	56% 22/39	79% 23/29	0.070
	No surgery	11% 8/74	6% 4/68	0.372
Silicone	All	15% (17/113)	21% (20/97)	0.364
	Surgery	36% 14/39	41% 12/29	0.801
	No surgery	4% 3/74	12% 8/68	0.118
Scar reconstruction	All	5% (6/113)	2% (2/97)	0.291
	Surgery	36% 5/39	7% 2/29	0.690
	No surgery	1% 1/74	0% 0/68	1.000
Steroid injections	All	4% (4/113)	4% (4/97)	1.000
	Surgery	10% 4/39	14% 4/29	0.715
	No surgery	0% 0/74	0% 0/67	-
Physio	All	5% (6/113)	3% (3/97)	0.510
	Surgery	10% 4/39	7% 2/29	1.000
	No surgery	3% 2/74	1% 1/68	1.000

[a] p-value calculated independent samples t-test

Table 38: Cross-tabulation showing scar management intervention (in rows) by surgery (in columns) stratified by year

Scar treatment	Surgery			
	Year	Yes % (n)	No % (n)	p (by surg)
Tubigrip	1998	67% 26/39	43% 32/74	0.029[b]
	2008	55% 16/29	43% 29/68	0.275
Pressure garments	1998	56% 22/39	11% 8/74	0.000[b]
	2008	79% 23/29	6% 4/68	0.000[b]
Silicone	1998	36% 14/39	4% 3/74	0.000[b]
	2008	41% 12/29	12% 8/68	0.002[b]
Scar reconstruction	1998	13% 5/39	1.4% 1/74	0.018[b]
	2008	7% 2/29	0% 0/68	0.087
Steroid injections	1998	10% 4/39	0% 0/74	0.013[b]
	2008	14% 4/29	0% 0/68	0.007[b]
Physio	1998	10% 4/39	3% 2/74	0.179
	2008	7% 2/29	1% 1/69	0.212

[a] p-value calculated independent samples t-test
[b] Statistically significant for p≤0.05

CHAPTER 5

DISCUSSION

This chapter discusses the main findings and their clinical significance. Each section is discussed, then summarised. The chapter concludes by answering the research questions:

Did the epidemiology, treatment and outcomes of Western Australian paediatric scald injuries admitted to the Total Care Burns Unit, PMH in 1998 and 2008 differ?
and
What target groups or focus areas do the data suggest might be appropriate areas for further resourcing and effort to reduce the incidence, severity and scar outcomes from scald injuries to Western Australian children?

Following this, a number of recommendations are listed.

Which target groups or focus areas do the data suggest might be appropriate for further resourcing and effort to reduce the incidence and severity of burns and associated scar outcomes from scald injuries among Western Australian children? The identification of who is at risk, and why, is paramount to the design of effective prevention programs. This section discusses the characteristics of the children admitted with a scald injury, exploring gender, ethnicity, age and area of residence to compare to the expected patterns discussed previously and to identify those children that are most at risk from scald injury. Once the most "at risk" groups of children have been identified, these groups can be investigated further to establish why they are sustaining more scald injuries than others. The aim of this section is two-fold. Firstly, to discover if the injuries correspond to physical characteristics and socio-economic patterns described in the literature, or whether the particular characteristics of Western Australia and its people have specific concerns. Secondly, to explore how these patterns have changed between1998 and 2008.

Slightly more boys than girls were admitted in both years, following the pattern described in the literature (Delgado et al., 2002; Van Niekerk et al., 2004). The proportion of Aboriginal patients admitted has remained constant between 1998 and 2008 but at approximately 14% this figure is much higher than the proportion of Aboriginal children in the community, which was approximately 4% in 2008. This is despite the fact that the rural and remote areas of WA have a higher proportion of

Aboriginal people compared to metro areas, who may be more likely to be managed at other hospitals if criteria are not met for transfer to PMH. The proportion of young children in the Aboriginal communities are higher than average, introducing the risk factors of larger families and more crowded households. This increased risk is confirmed by other studies (Delgado et al., 2002; Van Niekerk et al., 2004).

In 1998, 94% of admitted patients were Australian born compared with 91% in 2008. In 1998 other countries of birth were New Zealand, England, Indonesia, Sri Lanka and El Salvador. In 2008 other countries of birth comprised of New Zealand, India, Malaysia, Japan, South Africa and Saudi Arabia. These changes were not statistically significant, but differences emerged in the subsequent actions taken by the carer, discussed later. Therefore, if these changes are part of a trend, this could indicate a growing need for ongoing community education. Most of these children are very young, so if they were not born in Australia, then their families are likely to be new immigrants, possibly in a state of flux with accommodation and jobs with reduced social support networks, which could increase risk. The multicultural diversity observed here, means that a plethora of new cultural beliefs and lifestyles could introduce new risks for injury and responses to injury so far unseen.

There was a statistically significant shift of residential area between 1998 and 2008 ($p=0.019$). Admissions of patients from the inner metro area remained static at 40, the number of admissions from the outer metro area decreased, and those in non-metro areas increased. When assessed by ARIA the difference remains significant ($p=0.014$), but not by region. For children under 5 years, the number of admissions due to scald injury from the metropolitan area reduced and the number of admissions in non-metropolitan areas increased. This was in contrast to the data that is also available for burn admissions from all causes in this age group which shows a downward trend in admissions. However, the latter information incorporates all types of burn injury. Flame burns in older children, and contact burns, especially to soles of feet from hot ashes, are another common burn risk in rural and remote WA. Therefore, this discrepancy might be due to, and indicative of, a reduction in other types of burn injury, which is outside the scope of this study.

A prediction of 2008 admission numbers from 1998 data in conjunction with population shift data shows that actual admissions from inner metro areas were slightly reduced, while admissions from outer metro areas were reduced and admissions from non metro areas were increased; these changes were significant ($p=0.016$). This would assume

all other influences on numbers are unchanged. However, the discrepancy between predicted admissions and actual numbers of admissions fit in with the philosophy of the West Australian Burn Injury Model of Care. This strives towards inclusive, cohesive and equitable care for all, acknowledging the constraints associated with geographical area.

The concept that the provision of burn care should be a state-wide service in Western Australia is integral to the underlying values and beliefs of the key service providers, and these predictions illustrate how the drive towards this goal has progressed before formal documentation in the Model of Care document. Those residing in both inner and outer metropolitan areas now have the benefit of attending the PMH outpatient clinic for treatment in the acute stage of injury, so preventing the need for admission in certain cases. The increasing provision of peripheral hospitals during the time frame studied has enabled initial local assessment for patients in the outer metropolitan areas. Medical practitioners at these sites can collaborate with the specialists at PMH to provide appropriate care before referring to PMH outpatient clinic for expert management resulting in a large drop in admissions from these areas. These patients have benefitted from the drive towards having the right resources in the right place at the right time.

Care revolves around the needs of the patient and is evolving with strong partnerships between care givers. The patients who reside in the non-metropolitan areas have gained from the advances in communication and the increased awareness by rural and regional health care practitioners of the need for specialist care; this has resulted in the identification of scald-injured children who require expert assessment and guided treatment that would account for the small increase in admissions. The care received by patients across the state is progressing against the obstacles of distance, workforce education and specialist services distribution resulting in more equitable care state-wide. However, a limitation of the predicted data is that they are based on the 1998 figures; if these numbers were spurious, the 2008 projection would be inaccurate.

The introduction of the PMH Burns Telehealth service, which started with multidisciplinary tele-consultations in 2005, and Telehealth clinics in 2006 for rural and remote paediatric burn patients, has enabled a change in management and admission for all rural and remote patients (McWilliams, Gilroy, & Wood, 2007). As a result, improved communication between rural centres and PMH burns specialists has increased appropriate identification of burns patients who require admission. Further to

this, it has allowed local care for children with burn injuries that can be managed in other centres. Over 350 patients have been cared for under the PMH Burns Telehealth Service, providing wound management advice via digital photography and multidisciplinary scar management via videoconference.

The Socio-Economic Indexes for Areas (SEIFA) is a method developed by the ABS to rank areas according to their level of social and economic well-being. It is derived from census data and uses 4 indices that assess income, education, unemployment, dwellings without motor vehicles, further economic evaluation, and occupation. Each local government areas (LGA) is ranked and the index has a midpoint of 1000; scores higher than 1000 signify comparative advantage and those below 1000 signify comparative disadvantage. In addition to this, LGAs are ranked by their index score from 1 to 1,111 were 1 indicates the most disadvantaged and 1,111 indicates the most advantaged areas across Australia.(Australian Bureau of Statistics, 2006a).

Of the 142 LGAs in Western Australia all admissions came from 49 of these in both years. Patients from the biggest LGAs of Stirling, Swan and Wanneroo were most likely to be admitted with a scald injury in both years. Taking population size into account, the incidence of injury was not higher for these areas. When compared to population size, the top 15 LGAs for admission numbers are shown in Figure 6. Only 3 of these 15 LGAs, Bassendean, Armadale and Belmont, fall below the average SEIFA score of 1000. This is an interesting finding, as burn injury has been shown to be associated with lower socioeconomic status (Delgado et al., 2002). This study suggests that in Western Australia scald injury *per se* is a universal injury and is not bound by the same rules about socioeconomic status as other burn injuries, such as flame or contact burns.

The LGAs from which the patients came had a higher SEIFA score than the other LGAs, although this difference was not statistically different. These results need cautious interpretation. The SEIFA rating is taken from a complex analysis of census data and can be distorted by skewed data (Australian Bureau of Statistics, 2001). Therefore, because the estimation of socioeconomic status is taken from the residing suburb of the patient and not the circumstances of the patient themselves sampling error can occur.

Scald injury typically occurs among younger children, and the distribution of age found in this study for scald admissions is typical (Hankins, Tang, & Phipps, 2006; Rimmer et al., 2008; Tse et al., 2006a). The children admitted in 2008 (median age 20m) were

slightly older than those admitted in 1998 (median age 18m), but not significantly so. In 1998, 75% of the admitted scald population was under 2 years and in 2008 this had dropped to 65%. The most likely age for a child to be admitted with a scald burn was between the ages of one and two years; 46% of 1998 admissions and 39% of 2008 admissions fell into this age group. The reduction in admissions in this age group was the only group to show a significant change in admission numbers between 1998 and 2008 ($p=0.015$). The most likely reason for these changes is that this patient group, by far the largest, has benefitted most through changes in patient management that allow them to be managed more frequently as outpatients. An analysis of outpatient data would confirm whether treatment pathways have caused this by maximising patient management. If so, it is necessary to define the characteristics that have enabled success in order to extrapolate these to younger or older scald injury patients.

In summary, the pattern of scald injury in West Australian children is slightly over represented by boys, and well over represented by indigenous children. These findings are important because they emphasise the need for culturally sensitive education in prevention and the provision of appropriate safety resources for the Aboriginal community to help 'close the gap'.

The biggest group of patients remain the 12-24 month old children, and concurs with the literature (Flavin et al., 2006; Lowell et al., 2008). This age group that has had the greatest reduction in admission rates probably due to changes in nursing and medical management, which have recently been formalised in the Burn Model of Care (Department of Health Western Australia, 2009). The findings are significant because they highlight the need to assess why the changes in treatment have impacted this group of patients more than the other patients, and how of the Burn Model of Care can be improved to benefit all patients in the same way.

The literature claims that low socioeconomic status is a risk factor in burn injury (Poulos et al., 2007; Wood, La Hei et al., 2006), because risk is inherently linked with the cultures and practices of low-middle income countries where burn injury risk is very high. However, the findings here are contrary to this, and socioeconomic status does not appear to be a factor in paediatric scald injury in Western Australia. It might be that burn injury in children has other prevailing risk factors compared to adults, or that scald injury risk has different major risks for other types of burn, or that factors that affect risk are atypical in WA. Using SEIFA to measure socioeconomic status for individual families might be not sensitive enough for accuracy. Further investigation of this finding is required.

These findings help to identify those at greatest risk from scald injury as any child between the ages of one and two years old. These children are particularly at risk from scalds due to the spillage of hot drinks. Campaigns such as the Kidsafe WA 'Hot water burns like fire' in 1999 and 2002, and therefore both falling between the years studied, do not appear to have had any lasting impact in injuries from hot drinks

During the study period was there a reduction in the incidence of scald injuries between 1998 and 2008?

Accurate figures about those who were admitted with a scald injury were obtained from hospital records; the number of admissions for a scald injury can be retrieved directly from the hospital coding system, currently ICD-10 (World Health Assembly, 1990). This allows the collation of data for all hospital separations and is a standardised process across all member states of the World Health Organisation.

Scald injury presentations to the Emergency Department who are subsequently managed as outpatients under specialist burn consultant care, are not represented in the ICD coding data. Obtaining these figures requires a separate method of collection, which is hospital specific. These figures were obtained from PMH for this study.

An attempt to gain data from the community for scald injuries that do not seek medical attention at the time of injury is unachievable for many reasons; the children involved are usually pre-schoolers and are unable to report for themselves, parents and carers may not be aware of the significance of an injury, or may be unwilling to state that their child has had an injury, particularly if they had failed to act at the time. A representative sample of the population may be difficult to obtain. These are often young families who come from lower income backgrounds, therefore accounting for a specific part of the population.

Data obtained from the Department of Health (2010) and used in association with data obtained from the Australian Bureau of Statistics reveal that the state-wide incidence of hospital separations for all burn injury has reduced by 34% over the ten year study period. As some of these patients will have been admitted to more than one hospital for the same injury, these data are difficult to interpret. For example, a child sustaining a scald injury near a regional hospital may be admitted locally and then transferred to Perth and admitted to PMH. Additionally, the pattern of two hospital admissions per patient might have changed between 1998 and 2008 making the data from both years

incomparable. These patients would be in a small minority as the usual pathway would be to attend the patient in the local emergency department (ED), not admit them and then transfer directly to PMH from ED. However, there are some cases that might be admitted before transfer depending on the clinical decision making process of both the on-site doctor and the team at PMH. The recent advent of the PMH burns Telehealth service has allowed regional and remote practitioners to manage some of these patients at their local site. This service commenced in 2005, and uses clinical photos accompanied by telephone advice to enable appropriate patient management.

At PMH, between 1998 and 2008 the number of admissions for all burn injury declined by 23%, and for scald injury by 14%. It is these admissions that have been closely reviewed in this study. Conversely, the number of presentations has increased for all burn injury by 8% and for scalds by 29%.

If these figures are compared to the population growth of 8% between 1998 and 2008, it is clear that the 8% rise in burn presentation demonstrates no change in incidence. The incidence of scald presentations has risen non-significantly. The decline in admission rate, when analysed in relation to population change was significant for all burn ($p=0.033$), but not scald.

Consequently, it can be concluded that the number of reported burn injuries occurring in the Western Australian population increased between 1998 and 2008. When compared to population growth, incidence is unchanged. This finding supports the study in Victoria, Australia by Wasiak (2009). The number of reported scald injuries also increased, however when compared to population growth, incidence is higher in 2008 compared to 1998. Despite this, admissions of both burn injury overall, and scald injury *per* se have decreased. Therefore, the reduction in admissions is not due to a reduction in rates of injury. If injury severity is unchanged, then the reduction in admissions must reflect a change in clinical management. Globally, there has been a trend towards outpatient management and ambulant care in many areas of health care, especially for children. To assess this, the characteristics of the admission population have been assessed, as well as the type, size and severity of their wounds.

During the study period was there a change in the type of scald injury presenting to hospital?

Having identified the children most at risk, it is then important to establish how they are at risk. This relates to the causative scald agent, and age of the child, as well as the time of injury in relation to time of day, week, month and season. The aim of this section is to establish which circumstances contribute to the highest incidence of injury, which will in turn lead to the establishment of targets for prevention programs.

The most common cause of scald injury was hot water, either from kettles and pans in the kitchen, or tap water. This accounted for more than half of the admitted scald injuries in 1998 and approximately 40% in 2008, and this finding is supported by Lowell et al (Lowell et al., 2008). The source of hot water was often not stated. Injuries due to the spillage of hot drinks remained constant at 35%, and other scald agents such as soup, noodles, oil, milk and sauces increased significantly from 12% in 1998 to 24% in 2008 ($p=0.048$).

The temperature of the liquid is relevant to the depth of the scald injury, therefore impacting on healing time and surgery rates. Thus, the addition of cold milk to a hot beverage is relevant information, unfortunately these data were not recorded for 47% of patients admitted for hot drink scalds in 1998 and in 2008 were not documented for 53% of patients. This change was not statistically significant but certainly there has been no improvement in data collection related to milk in hot drinks. There appears to be the tendency to document if milk is not used, more than if it is used, reflecting some awareness that this is relevant to the injury, and possibly reflecting the common use of milk with tea and coffee in Australian culture.

For all ages, scald injury from noodles showed an increase from <1% (1/113) in 1998 to 8.25% (8/97) in 2008. Scald injuries from noodles might be considered to be a risk for Asian children ; however this was not evident from our data. One child scalded with hot noodles was born in India, and the rest were Australian born, one of whom was Australian Aboriginal. Five of these children were under three years old, all with a similar injury pattern involving face, neck, chest, abdomen and unilateral (usually right) upper arm. This concurs with the literature (Drago, 2005). Three of the remaining four patients with noodle scalds spilt the noodles into their lap whilst eating sustaining burns to thighs and genitalia, none of who required surgery to heal, and one child, aged 7

years spilt noodles whilst preparing them, sustaining deep burns to his right arm and hand, that did require surgery to heal.

The social circumstances surrounding the increased incidence of childhood noodle scalds are outside the scope of this analysis. Lifestyle differences between 1998 and 2008 could contribute to this; there were more single parents and more double income families in 2008 compared to 1998 (Commonwealth of Australia, 2011). A possible reason for the rise in scald burns from noodles or other foods might be a result of greater independence for children at a younger age with more children managing their own meals at home, or an elderly relative caring for children whilst mothers are at work; these are both risk factors and so this theory would fit with current evidence (Weedon & Potterton, 2011). Further investigation would be required to assess this.

Age rose in 2008 for hot water and hot drink scalds and fell for scalds from other agents, although these changes were not statistically significant. However, the pattern of mode of injury by age remains consistent. In 2008, the youngest children were scalded by hot drinks at a median age of 1y 6m (mean 2y 9m), the next most frequently recorded agent causing injury was hot water with a median age of 1y 11m (mean 4y 3m). Other agents were the main cause of scald in the older child, a median age of 2y 1m (mean 4y 9m). Further analysis of the pattern of mechanism of injury within the age groups reveals that for those less than two years old there was an equal chance of being scalded by water and hot drinks. However, for children over 2 years old the likelihood of hot drink scalds rapidly and progressively reduced, and the most common scald agent was water, from tap, pan or kettle (Figure 10).

The changes in mode of injury between 1998 and 2008 were statistically significantly different in the children aged 1-2 years only ($p=0.015$). These differences comprised of a reduction in admitted scalds from water and hot drinks and an increase in scalds from other agents. There were three new scald agents in this age category in 2008; noodles, oil and hot milk, accounting for 3, 2 and 2 admissions respectively. It is interesting to note that the reduction in admitted scalds in this age group was the only change between the years that showed statistical significance and poses two questions: Has there been a real change in incidence of scald injuries in this age group? Or are there successful management changes that have been implemented for these patients? If it is assumed that the incidence has not changed, based on the previous discussion, then it is possible that this group is being managed in an optimal

manner, and the biggest impact would be to concentrate future management strategies on other age categories.

Changes in legislation regulating the temperature of hot water systems were introduced in 1997. This meant that new domestic systems had to limit their maximum water temperature to 50^{o}C. As this regulation applies only to new housing, the effects of the intervention may take many years to filter through for many young families, however, it could have lead to decreased numbers of hot water admissions by 2008. KidsafeWA introduced an anti-scald campaign, 'Hot water burns like fire', in 1999 and 2002. This targeted all scald injuries, from both hot water and hot drinks. There was no corresponding reduction in hot drink scalds. Such a campaign would need to be evaluated through the assessment of all scald presentations across the State, and include ED presentations, outpatients and inpatients for the period in which the campaign ran, compared with corresponding baseline data from the preceding period. Therefore, evaluation of the impact of these interventions is not possible from our data but these health promotion campaigns do not appear to have had any lasting impact on the incidence of scald injury.

These findings help to identify those at greatest risk from scald injury as any child between the ages of one and two years old. These children are particularly at risk from scalds due to the spillage of hot drinks. Campaigns such as the Kidsafe WA 'Hot water burns like fire' in 1999 and 2002, and therefore both occurring between the years studied, do not appear to have had any lasting impact in injuries from hot drinks. It is possible that legislative changes in hot water systems might be having an impact.

There was no increase in admissions in any particular season or month of the year, and the figures do not appear to relate to school holidays. This finding is supported by Han et al (2005). However, it is refuted by other studies that did find an association with school holidays (Tse et al., 2006a; Tung et al., 2005). Although most of the patients were pre-schoolers, other related influences might have contributed to injury risk in school holidays. For instance, it is possible that the increase in numbers of family members present in the household might increase risk due to less parental supervision, or otherwise affect risk in either direction due to increased sibling supervision or influence, depending on the ages of the siblings. When the admissions are assessed by day of the week, a change in pattern between 1998 and 2008 is noted. The most common time for a scald injury in 1998 was Mondays and Tuesdays.

In 2008 there was a tendency for less scald injuries mid week, with a spike in injury occurrence on a Friday. This change was interesting but not statistically significant.

Overall, the injuries had a bimodal pattern, peaking at breakfast time and supper or bath time. The agent causing injury varied by time of day: Scald injury from hot drinks did not show a specific pattern, starting around breakfast time. Hot water scalds showed a moderate breakfast and tea-time increase. Injuries associated with other agents tended to occur in the afternoons and evenings, especially those involving soup, which only occurred at this time (Figure 15). The injuries to six children between 10 and 11pm, were mostly due to hot drinks. Of the two injuries between 11pm and 6 am both were in older children; one from tap water, and one from a steam inhaler. In 1998, more children were sustaining a scald injury at breakfast time, with a steady likelihood of scald injury during their waking hours. In 2008, the children were more likely to be injured in the early evening, at around tea-time. The increase of injury around the mealtimes corresponds with the findings of Lin et al (2005).

In summary, the biggest group of patients remain the 12-24 month old children, who commonly get injured by hot water or adult's hot drinks, and these injuries are consistent across the day. Hot water scalds in this age group have had the greatest reduction in admission rates, seemingly due to recent changes in medical and nursing management. However, scald injury from hot drinks has not reduced. There seems to be a trend for younger children to be scalded by other scald agents, particularly noodles. Increased vigilance is needed at breakfast and dinner time.

During the study period was there a change in the severity of scald injury between 1998 and 2008?

Severity of injury

An assessment of how severely the children were injured was conducted by exploring the size and depth of their injuries. The aim was to assess whether the admitted scalds were more severe. Estimation of size of their injury was taken from the assessment given by the most senior member of the burns team, recorded as TBSAB (total body surface area burnt). As erythema is not included in the TBSAB, and erythematous areas can resolve or develop, the injury may not fully declare itself for 48 hours. The resultant wound depth may depend upon the immediate care given, and the resultant scar, in turn, depends on wound depth. This is why timely and optimal

intervention is the key to good scar outcomes, especially if given within the first 24 hours of injury. Therefore, early assessment of TBSAB, which does not include areas of erythema only, is often over or under estimated, especially by the untrained eye. The Lund and Browder chart (Appendix 1) is the assessment tool used in children.

For all patients, the mean TBSAB in 1998 was 4.5% compared to 5.4% in 2008. This rise neared statistical significance ($p=0.0659$ on 2-tailed t-test) and further analysis did not show statistical differences in mean TBSAB between 1998 and 2008 for gender, Aboriginality, mode of injury and area of residence. The analysis reveals a statistically significant increase in wound size in admitted scalds in those between one and two years old only ($p=0.0207$). In this age group the mean TBSAB increased from 5% in 1998 to 6.5% in 2008. Thus the overall trend was for 2008 scalds to be larger than those treated in 1998. This finding is most likely associated with the fact that smaller injuries are being managed in an outpatient clinic or via Telehealth. There has been a statistically significant increase in size of injury in the 12-24 month old sub-group. This is the group that had a statistically significant reduction in admissions. These two findings, in conjunction with each other, suggest that the clinical management of these patients might be better for the patient and more resourceful. Identification of why will help determine how this knowledge can be extended to benefit other groups.

As discussed, depth of injury is difficult to assess. The optimal time to assess depth is at 48 hours, when the wound has fully declared itself. There are clinical indicators that will suggest injury depth at these early stages, and accurate assessment of depth is important as it will indicate time to healing, if surgery will be required and determine resultant scarring. To complicate matters, scald wounds are often of variable depth. If a burn wound will heal within 10-14 days there will be minimal scarring, if the wound takes longer to heal than this, the local practice would be to aid healing via surgical techniques such as full or split thickness skin grafting and/or autologous cell suspension therapies. Therefore, when collecting retrospective data, a good indicator of injury depth is need for surgery, and so this is used as an indicator of a deep burn wound.

Overall, surgery rates, although lower in 2008 compared to 1998, were not statistically different. Further analysis of the need for surgical intervention to heal was repeated for age, gender, indigenous status, area of residence and mode of injury and do differ for any specific group. The larger wounds have a higher rate of surgical need to heal, therefore are not only larger but also are deeper. The mean TBSAB was larger in the surgical group in both years and in 2008 the mean was 4.3% in those who did not

require surgery compared to 7.8% in those who did, this difference was highly significant (p=0.0001).

It is interesting to notice that surgery rates have not increased. This would be contrary to predictive thought, as it would be logical to rationalise that more patients with wounds that are less deep are managed by Telehealth or as outpatients until surgery was required. On these grounds, it would be likely that those living further from Perth, and who would be managed via Telehealth in 2008, would have higher surgery rates. Therefore, this demonstrates that the criteria for admission are not dependent on the need for surgery.

In summary, the size of the admitted wounds has increased, which was an expected finding that supports the idea that incidence has not decreased, but more of the smaller wounds are treated as outpatients. The accurate estimation of TBSAB is an important admission criterion as the extent of skin loss is directly related to the amount of fluid lost from the body. The calculation of intravenous resuscitation required is dependent on this. However, assessment of injury depth at presentation, which is when the clinical decision is taken whether or not to admit, does not feature as a criterion for this decision. So, if severity of injury is used as a clinical decision making tool for scald wound management via admission or not; the assessment of TBSAB has remained consistent. However, the assessment of depth and the prediction of need for surgery is often difficult on acute presentation and currently is not used as a criterion for admission.

During the study period was there an improvement in first aid given to children with scalds during the pre-hospital period and were there changes in the actions of the carers present at the time of injury?

Prevention is the best message to teach, but if an injury has not been prevented the next best message to deliver is that prompt cooling of the burn will minimise injury. As described in the literature this involves the application of cool, but not icy cold, water for twenty minutes or more to cool the burn without causing local vasoconstriction and without causing systemic hypothermia (Australia and New Zealand Burns Association, 2010). The aim of this section is to assess the initial actions of the carer so that the level of public consciousness can be ascertained; whether an understanding of burns first aid is embedded in public knowledge, and whether there was a change between 1998 and 2008. First aid received by the child, and factors that affect this, choice of

first medical attention, any delays incurred, and use of emergency services, such as the State ambulance service or the flying doctor service is explored.

Interpretation of first aid data was limited by lack of documentation. Only three quarters of the 1998 cohort had first aid documented, improving to almost nine out of ten of the 2008 patients. Cases for which first aid was documented; approximately 10% of carers claimed to have given 20 mins of cool water in 1998, compared to 14% in 2008, although this improvement was not statistically significant. Another two thirds of the patients received cool water in 1998, although for half of these the length of administration in unknown, and in 2008 just over half of the remaining patients who had had first aid recorded, received cool water, one sixth of these for an unknown duration.

Therefore, in total, 75% in 1998 and 70% in 2008 of those admissions where first aid was documented received cool water to their wound. Of these patients 11% (9/83) in 1998, and 12% (10/85) in 2008 received very inadequate first aid, receiving less than five minutes of cool water. Thus, the administration of cold water by the carer is unchanged and that knowledge about appropriate first aid action is not reaching those who need to know. It is unknown from this study if the application of cool fluid is an intuitive reaction to an injury by hot fluid, or if it is based on the need for analgesia, or if it is based on prior knowledge of correct first aid. The importance of continuing cool water application beyond initial analgesic action, and prioritising it a little above the need for hospital transfer needs to be taught and emphasised.

The number of patients who received no first aid remained unchanged between the years at 7% and 9% respectively. There was no difference in patient characteristics for age, gender or ethnicity; although a larger sample size might reveal differences. For instance, different cultures demonstrate different levels of stoicism and this could impact on the type and duration first aid interventions that are used to relieve pain. These wounds were larger, but not statistically so. The wounds were often deeper, more often requiring surgery although this did not quite reach statistical significance on a sample of this size ($p=0.0829$), but this difference might be regarded as being clinically significant. This means that although the numbers do not reveal statistically harmful effects of first aid not being given, the increase in surgical rate has practical implications both to health care providers and patients. The health care providers are subjected to greater use of resources, and the patients are subjected to surgical intervention, and therefore exposure to the associated risks of surgery and general anaesthetic.

Although more children received ice to their wounds in 2008 than 1998, this increase was statistically unchanged, increasing from 4% to 6%. However, it might be indicative of an increased awareness of the need to cool the wound, but no knowledge of how to do this in an appropriate manner. There was a cultural difference in this group; 50% had not been born in Australia, compared to over 90% of all patients, and none of the patients who received ice were Aboriginal. The data shows that three of the eight patients were near the median age of 19 months, and the remaining five patients were all over 7y 8m. More boys received ice than girls; 62% of the patients who received ice were boys, compared to 52% in the whole sample, although this was not significant. The wounds that received ice were significantly smaller than average with a mean TBSAB of 2.1% ($p=0.021$), but healing times and surgery rates were unchanged compared to the whole sample. This suggests that ice might be applied to less severe wounds in the first instance, and the effect of the ice application on depth is unknown in this sample, which might not be representative of the whole.

Other substances to scald wounds were applied in 6% and 3% of patients in the respective years and included a variety of agents commonly found in the home, often from the kitchen cupboard (Table 14). Choices included emulsions such as butter or vegetable oil, which soothe but do not cool, alkalis such as sodium bicarbonate, sugar, acids such as lemon juice and vinegar, proteins such as egg white and soothing creams and gels such as antiseptic cream or aloe vera. More than half (55%) of these patients were born outside Australia, therefore demonstrating cultural difference in first aid choice. This fits in with the current literature, which noticed a strong cultural element to the first aid choices made by parents (Blair & Carter, 2005; Cavanagh & Wilkinson, 2005; Johnson & Coleman, 2000). There was no statistical difference in wound severity or other characteristics. The sample sizes in the groups who received no first aid, other substances than water or ice to their wounds were too small to reveal statistical significance, but indicate trends of interest in both groups, which could warrant further investigation.

It is interesting to notice that the time of day in which the injury occurs had a bearing on first aid given. There were two peak times for scald occurring; breakfast time and tea time. There was a tendency to give less adequate or inappropriate first aid later in the day. A surprising finding was that there was no association between area of residence and first aid. If the patient was living remotely, with less and slower access to medical management, it would be logical that there was more time to give adequate first aid in the form of 20 minutes of cool running water. It might be that there is less water available in rural areas, or the available water is not cool enough to provide benefit.

Chronologically, the next step in the patient's journey is the treatment and care received at the first professional medical interface. The nature of this interface depends upon the actions taken by the carer in the acute situation. There was increased use of emergency services in 2008 compared to 1998, and less patients presented initially to their General Practitioner. This might show an increased awareness of the importance of prompt action amongst the public. In 2008, with larger scald injury, carers were more likely to call 000 in response to injury occurrence. However, this was not evident in 1998. Those that called 000 were more likely to receive 20 minutes of cool water to their injury in 2008. This might indicate improved communication and advice from ambulance and ambulance control personnel, and better prioritisation of the need for burns first aid before removing the child to hospital. Alternatively, it might indicate that the decision to transfer the child to the hospital has been lifted from the shoulders of the carer and continuation of first aid whilst waiting for the arrival of ambulance personnel is commonsense. It seems that the public are starting to understand that immediate action is required, but the knowledge of how requires improvement.

First aid given by the emergency services is hard to determine due to lack of available documentation and small sample size. No child who attended a GP first received 20 minutes of cool water, but further analysis was limited, also due to small sample size. Choice of GP as first medical attender was not related to delayed presentation; thus GPs need good burns first aid knowledge. GPs did not delay in transferring patients to hospital. A minimal improvement in cool water application by EDs other than PMH showed a small improvement in the numbers of children who received wound cooling in 2008 compared to 1998. This increased in total from 35% in 1998 to about 45% in 2008, and consisted of a combination of cool water or a wet wrap. The use of a wet wrap can be a practical alternative depending on the part of the body that has been burnt, for maintaining water application to transfer or for maintenance of body temperature. This data is limited by the fact that there was no documentation about first aid treatments given in ED in 62% of cases in 1998 and 45% in 2008.

In summary, there was an improvement in medical documentation between 1998 and 2008. This might reflect a better awareness of the importance of burns first aid and increased knowledge amongst health professionals. For the patients who had information available, there was no improvement in the first aid given between 1998 and 2008. Approximately 8% of patients did not receive any first aid. About three quarters received cool water to the burn but only about 10% received it for long enough. A small number of children received ice to their burns, showing that

knowledge about how to cool a burn is poor. The few patients that received other substances to their scald injury for first aid treatment had a variety of ethnic backgrounds. As scald injury is caused by hot fluid, washing it off with cold fluid might be intuitive, and this might account for the large proportion of patients who received some amount of water as first aid. This would account for the lack of further knowledge about how long to apply it for, and how cold the water should be. It might be that the duration of water given is directly related to the analgesic effect of the same. Once the pain has eased, the cool water administration ceases, allowing the carer to take the next action, which is often transporting the child to the nearest emergency department in their own vehicle.

If adequate first aid has not been performed by the carer at the scene, it is important that this is ascertained, documented and remedied at the earliest opportunity. Cooling the wound with running water is considered to be of benefit until three hours post injury (Australia and New Zealand Burns Association, 2010). There has been a trend towards the application of water by emergency services and in local emergency departments, however, sample size is too small to show meaningful statistical significance; therefore a descriptive analysis was used. Burns first aid given by GPs is insufficient to benefit the child, although most presentations occurred within 3 hours of injury and GPs referred the children immediately to hospital.

Therefore, appropriate burns first education is required by all those likely to come into early contact with the scalded child. These individuals can be separated into two groups. Firstly, parents, carers and general public make up one group, and secondly, all medical professionals that provide medical services at the public interface make up the second group. The importance of the duration of cooling techniques, while keeping the patient warm, needs emphasis; and that adequate first aid is not just soothing, but that it is believed to impact on the resultant scar.

During the study period were there changes in medical treatment and nursing care of paediatric patients with scald injury?

Time spent in PMH ED

Those who presented via other EDs or the emergency services spent the shortest time in PMH ED, and the longest time spent in ED is by those who self-present or are referred by their GPs. All patients have longer average stays in ED in 2008 compared to 1998, although those presenting via SJA or RFDS represented the only group for whom the increase was not statistically significant. However, there was a correlation with TBSAB; the length of time spent in ED was inversely related to the size of the wound. Therefore, the larger the wound, the quicker ward admission was enabled.

Patients stayed in PMH ED longer in 2008 compared with 1998 for the patients who were admitted directly to the ward. A number of factors may have impacted on this. The ED might have been busier, with increased nursing and medical workloads. Also, workload has increased for the ward nurses; the admitted burn patients have larger wounds, but there are less of them. This means that the bed occupancy from burns patients has reduced, and instead the beds are used for time-intensive non burn patients.

Analgesia

Optimal pain management in young children is paramount for effective care. As described in the literature review, pain has an impact on long term outcomes such as post traumatic stress disorder. In a scald injury, which can have lasting scars, minimising psychological sequelae is important. First line medical intervention focuses on pain relief that is geared to immediate and short term outcomes; calming a distressed infant, steadying a child to allow intravenous cannulation for life-saving resuscitation fluids, and to allow painful debridement and dressing procedures. The ideal agent needs to be effective and fast acting. The aim of this section is to assess analgesic choice at each stage in the patient journey and how these have changed between 1998 and 2008. Some children received more than one type of pain, and therefore these analgesic groups are not independent of each other.

No carer gave pain relief before presenting themselves to PMH ED. For those who had medical attention elsewhere first; in 1998 two thirds of patients received one or more intramuscular (IM) injection to relieve pain from the injury. Forty-four percent of

patients received IM Pethidine, and 20% of patients received IM Morphine. Just over 40% of scalded children received the second most common choice of pain relief; Painstop (or Liquegesic), with some children receiving both.

The pattern had changed by 2008; the use of IM analgesia had reduced (p=0.008), and Pethidine had almost been eliminated. Oral agents had taken over as first choice, with 55% of patients receiving them. Two new oral agents emerged; Ibuprofen was given to 14% of patients, and oral morphine was given to 16% of patients. Intranasal (IN) fentanyl was another new agent, adopted mostly by the emergency services and a small number of metropolitan EDs and given to one third of patients. The emergency services did not give any oral pain relief, possibly because they do not have these available for use. The move towards non invasive routes of drug administration was apparent, both clinically and statistically significant, with intramuscular administration occurring less often (p=0.008) and intranasal administration occurring more often (p=0.000). The number of children who received intravenous analgesia for pain relief before PMH presentation was largely unchanged at 11% and 12% respectively. In both years, only 50% of the children who had IV cannulae inserted before PMH presentation had analgesia given by this route. This might have been because pain relief was necessary before IV cannulation was possible in a distressed child.

A similar pattern is apparent in the analysis of the type and route of analgesia administered in the Emergency Department at PMH for those who required analgesia at this time. Patients, who had been seen at other emergency departments or by the emergency services, often had already received analgesia and did not stay long in PMH ED before transfer to the ward. So although most patients passed through PMH ED, their required treatments varied depending on where they had come from, and whether they were destined for the ward or for outpatient follow up.

There has been almost a complete cessation in the use of intramuscular medication since 1998. In 1998, the most common medication was IM morphine. Pethidine IV was used once in 1998 and Pethidine IM was not used at all. There was statistically significant rise in the use of IV morphine (p=0.002) and a drop in oral morphine (p=0.005). Intranasal fentanyl had been introduced by 2008 and then was in regular use (p=0.000). The increase in the use of oral medication was statistically significant (p=0.000) overall. In 1998, just over a quarter of patients received Morphine orally, the only other oral drug used was Painstop, which was given to 10% of patients. In 2008, almost three quarters of patients had at least one oral analgesic; the choice had expanded to included paracetamol, ibuprofen and occasionally, codeine. These

findings fit with the PMH guidelines 'medications for use in children with burns' introduced in 2002 that advocated that the use of IM injections should be avoided if possible (Appendix 6). They also correspond to the findings of a study conducted in PMH ED that found that intranasal fentanyl was a suitable agent for use in paediatric burns (Borland, Bergesio, Pascoe, Turner, & Woodger, 2005).

Once admitted to the ward, pain was managed almost exclusively with a combination of oral agents; morphine, paracetamol, painstop/liquegesic, and ibuprofen. For patients whose pain was difficult to control, IV drugs were used. These numbers are too small for useful analysis, but in 1998 included morphine and pethidine and in 2008 included morphine, fentanyl (IV) and ketamine. Inhaled entonox was used more commonly in 1998 than 2008, providing a further option for analgesia.

Play coordinators are employed to distract patients with toys and conversation during dressing procedures. This adjunct to pain control is not documented in the medical notes, and therefore a factor that cannot be accounted for in this study. The literature review discusses the influence that distraction techniques have on pain thresholds. It appears that the more mindful attention that the patient gives the pain and discomfort, the higher the pain sensitivity. Pain coping mechanisms vary in individuals, but positive self-talk and minimisation of catastrophising thought patterns and behaviours in the children is beneficial.

In summary, there has been a noticeable move away from using painful intramuscular administration of drugs to relieve injury pain. The introduction of intranasal fentanyl has been embraced by many in the emergency situation, it is easy to administer, potent and fast acting. It is not suitable for ongoing pain management as it has a short half-life and requires close and continuous monitoring of the patient. The move has been towards this, and also towards oral agents. The management of pain on the ward was mostly by oral agents in both 1998 and 2008. The biggest change has been in PMH ED where the changes of analgesic choice and route have changed greatly over the 10 year study period. Change is also apparent in the patients who present by ambulance or RFDS, whilst other pain relief given before arrival in PMH ED seems to indicate a trend for the better. It is interesting that a commonly held misconception by the public is that they should not medicate their own child before presenting. While it is true that some medications might limit the immediate use of another drug, as long as the details and time of medication is recorded by the person giving the medication, there is no reason for a child to suffer pain longer than necessary if pain relief can be available early after injury.

Intravenous access and hydration

Appropriate identification of patients needing fluid resuscitation is important to ensure that major scalds are optimally treated and that patients with minor scalds are not subjected to unnecessary intervention. The aim of this section is to assess the balance of these needs.

There was no change in numbers of children in 2008 (13%; 13/97) who met the criteria for fluid resuscitation compared to 1998 (11%; 12/113). Two children in 1998 and one child in 2008 did not receive resuscitation fluids, but all three were borderline for the guidelines, i.e. estimated at 8%, and very near to 18 months old. Most patients received an IV cannula and fluids before ward admission. The correctness of the calculations and administration of fluids was not assessed in this study.

Four children (1 in 1998, 3 in 2008) had an IV cannula inserted prior to arrival at PMH, who did not meet the TBSAB criteria for fluid resuscitation. Each of these children received IV analgesia. A further 11 children (5 in 1998, 6 in 2008) did not meet the criteria for fluid resuscitation, but had fluids started prior to arrival at PMH ED, three of whom also received IV analgesia.

To summarise, despite the difficulties inherent in assessment of TBSAB, it seems that generally the right patients are getting IV fluids. About 5% of children get an IV cannula which is not required. If the non-specialist practitioners are erring on the side of caution by cannulating a small number of children unnecessarily, this is better than the reverse. If an IV cannula is inserted and not required for fluid resuscitation, using it for IV analgesic drugs makes a painful procedure useful. Cannulae are not left in unnecessarily because of infection risk (Easterlow et al., 2011).

Dressings

Early application of silver dressings is thought to be important for control of microbiological colonisation. The recommendations of silver dressing type have changed since 1998, and by 2008 differences were apparent. Acticoat™ was not used in 1998, but has been increasingly adopted for use since it was first marketed in June 2006. The aim of this section is to assess if there has was an increased use of silver dressings as recommended for bacterial control (Fong et al., 2005), especially before ward admission, and which dressings are chosen.

Of those who had dressings applied before PMH ED presentation, there was an increase in those who received silver based dressings from 20% to nearly 50% in 2008

(p=0.002); 10% of these 2008 dressings were Acticoat™. There was also an increase in the application of silver based dressings for those who were attended in PMH ED (p=0.004) from 4% in 1998 to 18% in 2008. These numbers are small because the patient would commonly wait until they were admitted to the ward for dressing application. The rise of dressing application in 2008 might be accounted for by the increase in number of patients who were initially referred to the outpatient clinic prior to admission. The remainder of patients had silver based dressings on the ward if they were not contraindicated by site (e.g. silver is not applied to facial scald burns). Eventually, 95% of patients in both years received a silver dressing to their wounds; in 1998 SSD was the only silver dressing used, in 2008 Acticoat™ was used on two thirds of patients, sometimes in combination with SSD depending on the bodily location of the injury. This shift towards the use of Acticoat™ was highly significant (p=0.0000).

The improvement in the application of silver dressings between 1998 and 2008 at the pre-hospital stage is an important finding. The early use of topical antimicrobials to combat potential or actual wound infection is essential for two main reasons. Firstly wound infection delays healing and worsens scar outcome, secondly minimising oral antibiotic use in these children will assist in reducing the advent of more antibiotic resistant bacteria (Bull et al., 2006; de Macedo & Santos, 2006; Edwards-Jones & Greenwood, 2003). It is of particular benefit that these children are receiving silver dressings to their wounds at the place of first medical intervention in Western Australia because transfer times can be several hours due to the distances that need to be travelled, and ambient temperatures are high. These two factors allow the perfect conditions for bacteria to colonise and proliferate within the wound bed, which is the perfect environment bacterial growth.

There has been a positive improvement in the use of Acticoat™ as preferred dressing, especially when consideration is given to the fact that it had been available for use in Australia for only 18 months by the beginning of 2008. This dressing is not always available at rural centres, prohibited by its comparative expense to SSD. The adoption of novel specialist dressings, such as Acticoat™, by generalists in the rural community is a process that can take time.

Microbiology

Wound infection is thought to be an important factor in healing delays and scarring. These might be directly related, or other factors might augment this. For example, does colonisation without infection delay healing, or increase the likelihood of wound breakdown? Prevention of wound infection is paramount in burn care, and it is important to ascertain the risk factors for this, and whether current treatments are changing the rate of wound infection. The aim of this section is to assess the rates of infection in 1998 and 2008, and suggest contributing factors.

Overall, 10% of patients had a wound infection in 1998 compared to 20% in 2008. This increase is significant, both statistically and clinically (p=0.042). When stratified by type, increase in wound infection was only significant in the rates of wound infection acquired following discharge (p=0.05).

If a clinical infection developed soon after admission from the same bacteria that were colonising the wound on admission, or if the patient presented with clinical infection, these cases were categorised as preadmission infections. The factors increasing risk of infection acquired before admission were a delay of more than 3 hours to first presentation (p=0.010), Aboriginality (p=0.003) and nearing significance for non-metropolitan living (p=0.059). Silver dressings applied prior to PMH arrival did not alter this outcome; this might have been due to delays to presentation. When these factors are assessed for changes between 1998 and 2008 data there were no significant changes. However, if the use of Acticoat™ has increased since 2008, this might start to show an impact. Thus, although the pre-PMH application of silver dressings has risen significantly (p=0.002) from 20% to nearly 50%, this intervention needs to be completed early.

85% (180/210) patients had wound swabs taken on admission. *S aureus* and *S. pyogenes* were the most likely colonising bacteria to develop into clinical infection. *B. cereus* and *S. pneumonaie,* if colonising a wound on admission, were also likely to develop into clinical infection, but were less common colonisers.

The sample size was not large enough for logistic regression analysis. Based on a power calculation a sample size of 400 patients with scald injury is required to investigate these factors. Thus, a suggestion would be to repeat this analysis with a larger cohort. It would also be interesting to see if the increasing use of Acticoat™, and the time of application of an Acticoat™ dressing affects these numbers.

The patients whose admission wound swabs did not reveal colonisation on admission, and later developed an infection whilst an inpatient, or who developed a wound infection from different bacteria to the initial colonising bacteria, were categorised as acquiring a wound infection as an inpatient. There was no increase in numbers in 2008 compared to 1998 for this category of patients. Patients with an infection acquired in hospital were likely to have larger wounds than average (p= 0.0065), and longer hospital stays (p=0.0137). Larger wounds are a risk factor for infection as previously stated, and both severity and infection are associated with longer patient stay.

Aboriginal patients were more likely to get a wound infection whilst inpatients (p=0.036), and those living in non-metropolitan areas (p=0.020) but no other factors were significant. Season did not affect infection rate in any group; this is interesting as warm summer weather might have increased the risk. For colonising bacteria; *S. aureus*, *S. pyogenes,* and *P. aeruginosa* were all highly likely to be associated with infection. There was no change between the years.

The increase in wound infection acquired post discharge between 1998 and 2008 reached significance (p=0.050). All infections were due to *S. aureus* and all were in an upper limb or shoulder. The length of hospital stay was not found to be shorter in these patients, so the patients were not getting wound infections because the patients were discharged earlier, although the wounds took longer to heal (p=0.0029). It might be that silver dressings have prevented infection, but the risk of infection returns when the use of silver has ceased. As the wound heals, dressings can be switched to other non-silver types, such as Algoderm. It might be that discharge information given to parents has changed; chlorhexidine washes were routine practice in 1998, and although still used in 2008, less so. To assess these possible factors a prospective observational study is required.

The colonising bacteria, regardless of whether they caused a wound infection, seemed to delay healing for *S. aureus* (p=0.0034) and *B. cereus* (p= 0.0492) only. However, because the wounds in the *S. aureus group* were larger (p=0.02) and more likely to need surgery to heal (p=0.002), analysis of a larger cohort is needed us needed to determine the odds ratio for each variable, and whether *S. aureus* is a causative factor for slow healing or not. The group colonised with *B. cereus* was too small for statistical analysis (n=3), but did not seem to have larger or deeper wounds, and had delayed healing. More investigation into this type of bacterial infection is warranted, particularly as empirical observation seems to indicate that these patients also have more painful wounds.

To summarise, infection increased in 2008. For the infections acquired during admission, this might be because the wounds were larger in 2008, and increased TBSAB is a known risk factor for developing wound infection. Aboriginality seems to increase risk of wound infection except for those acquired after discharge. Delays of more than three hours to first medical attention, or living in non-metropolitan areas seem to increase risk of preadmission wound infection. The role of silver dressings needs more investigation, particularly in relation to the advent of post-discharge *S. aureus* infection of unhealed wounds. Bacteria likely to cause infection are *S. aureus*, *S. pyogenes,* and *P. aeruginosa;* all of which are commensal organisms in WA.

Surgery

As previously identified, surgery rates between the years had reduced, but this was not statistically significant. The cohort of patients in 2008 had wounds that were larger and deeper, therefore it might be expected that surgery rates would increase between 1998 and 2008.

The shift to Acticoat as the dressing of choice, which demonstrates excellent microbiological control, might shorten healing time and therefore could reduce surgical rate in 2008 compared to 1998. However, this cannot be confirmed in this study. In 1998, the use of SSD meant that dressing needed to be changed on a daily basis, whereas in 2008 the use of Acticoat reduced the necessity for frequent dressing changes to every 2-3 days. This would negate any tendency to perform early surgery to reduce the need for painful dressing changes. The provision for taking patients to theatre sooner after injury is now possible, and it is interesting to notice that although there has been no real change in this timing statistically, the median time to theatre was actually longer in 2008 compared to 1998.

The process of the application of cells has changed over the study period. In 1998, the method of cell spray synthesis was to biopsy several days before they were required for use in theatre. The epithelial cells would then be cultured in the laboratory to be made into a spray for application (CEA). The development of cultured autologous sprays since 1998 has allowed for the cells to be taken and the spray prepared in theatre for immediate use (ReCell). The number of patients who required surgery, and only received a split skin graft (SSG) to their wounds declined from 21% to 10% between the years, although this was not statistically significant. The use of cells applied to a wound, whether with or without SSG, was statistically unchanged, although the trend for use increased. There was a statistically significant rise in size of wounds

that ReCell alone was used on compared to CEA alone in 1998 (p=0.0208). This shows increased confidence in the use of autologous cell spray by 2008.

Currently, a recent local study has brought about a change in practice for those who have a scald injury that is thought unlikely to heal within 10 days (Wood et al., 2011). If this were evaluated in 2011 or later, it is likely there would be a trend for quicker surgery times. However, caution is exercised not to take patients to theatre for surgery to close the wound unnecessarily, and a close watch on those wounds for which healing time is not known is needed for timely surgical intervention.

The fact that those patients who required surgery had longer hospital stays is not surprising. Surgical patients who lived in non metropolitan areas stayed the longest, although these patients had longer hospital stays in 1998 compared to 2008. This is expected. The stays would be longer to ensure more healing had occurred. In 2008, the Telehealth service was closely reviewing and advising care for these patients on discharge, allowing their earlier discharge. It is expected that length of stay is longer for those receiving surgery, however it is interesting to note that the stays for outer metropolitan patients were no different for those who had had surgery or not in 1998. This suggests earlier discharge, possibly with management in the outpatient clinic, although this result might have been expected for in the inner metropolitan patients as well.

In summary, surgery rates and types are unchanged statistically, although there are technical differences in the process of autologous cell culture and application. These cells are now applied to larger wounds, indicating an increased confidence in their use. Children who have surgery stay in hospital longer, except for the outer metropolitan patients in 2008, whose length of stay is not affected by surgical intervention. The reason for this is unclear, particularly as the inner metropolitan patients do not follow this pattern. This might be revealed by data analysis on the outpatients. The non metropolitan surgical patients stay the longest, but this group have shorter hospital stays in 2008, possibly because of the ability to monitor them by Telehealth on discharge.

Scar management

The difference in scar management use did not show significant change in 2008 compared to 1998. However, when comparing surgical patients to non-surgical patients there were differences noted. There was a greater use of pressure garments and silicone in both 1998 and 2008 for the surgical patients, and greater use of tubigrip only in 1998 for the surgical patients. Only surgical patients needed steroid injections. Scar reconstruction was required in one 1998 non-surgical patient only. Physiotherapy need was unrelated to incidence to surgery, but was related to the need for scar reconstruction. This is expected as scar management often involves contracture release.

Scar evaluation was not available via a specific validated tool. Some descriptive evidence was available from annotations by medical and nursing staff in the medical notes. The amount of scar management used in each year was a measure of the scar outcome in this study. However, there might have been differences in practice between the years in assessment of acceptability of scar outcome and the treatment of the scar. Therefore, comparing use between two separate years is difficult. Different surgeons, occupational therapists, physiotherapists and nurses can have differing views on scar prevention and treatment, and what level of scarring is acceptable. It is easier to assess differences between groups within the years, as the treatment is likely to be consistent. Also, the criteria for routine scar treatment might have changed. For example, the provision of pressure garments might have risen for the 2008 surgical patients because the prevention of scar by the use of these might have been routine practice if the location of the injury on the body allowed effective use of them.

Patients who required surgery for wound closure also needed more scar management techniques. The association cannot be defined further based on the available data. It is the larger and deeper injuries that require surgery, and are therefore more likely to scar. Early wound closure reduces scar, as described in the literature review (Deitch et al., 1983) so if surgery enables early wound closure it might be thought to reduce scarring. However, scar appears to be worse in this group, if the degree and number of scar management techniques are an adequate way to assess this, which probably it is not. So does surgery improve or worsen the resultant scar? And if the latter, which surgical methods result in better scar outcomes, and why? Is it due to judicious tissue salvage? It might be hypothesised that early closure prevents colonisation, infection and complication such as overgranulation. Identification of other factors that affect scar outcome need to be taken into account; for example, itch is a problem not recorded in

the notes or measures, apart from the record of medication given. Itch is something that can have extrinsic damaging effects on the wound because it causes patients to scratch, but also is associated with inflammatory processes, which may cause intrinsic cell damage . Evaluation of this parameter was outside the scope of this study, but is put forward as an example of the complexity of the factors affecting scar formation.

Routine evaluation of scar using a reliable validated assessment tool would enable an outcome measure of the effectiveness of all interventions received by the patients from the moment of injury. This would allow comparison of surgical techniques, for both dermabrasion and grafting methods, for scar result. Investigation to assess if degree of itch and associated factors is related to scar outcome would be made possible.

Conclusion

The primary objective of the study was to assess changes to the profile of hospital admissions for children with scald injury in Western Australia between 1998 and 2008. Initially, the incidence and patterns of scald injury were evaluated. Furthermore, factors that increased risk of injury, poor first aid, or increased risk of complications were analysed, with the aim of identifying specific sub-groups so that targeted prevention and education strategies could be developed. These objectives had not previously been addressed in Western Australian studies of paediatric scald injury. Finally, the epidemiology, treatment and outcomes of Western Australian paediatric scald injuries admitted to the Total Care Burns Unit, PMH in 1998 and 2008 were assessed.

In this study the incidence of burn injury remained unchanged, and scald injury *per se* seems to have increased. This finding was masked by the reducing incidence of scald admissions due to different treatment pathways such as the introduction of the burns outpatient clinic and the provision of the Telehealth service.

The 12-24 month old patients, which comprised the largest group, had statistically larger wounds in 2008. Overall, the wounds were also larger, but not statistically so. This seems to be because the smaller wounds were managed as outpatients. The admitted wounds did not seem to be deeper in 2008, and surgery rates were unchanged. This demonstrates the difficulties inherent in depth assessment, and highlights the fact that depth or predicted surgical need did not seem to feature in decisions whether or not to admit. The numbers of scalds from hot water were reduced in 2008, and those from other agents increased. The prevalence of hot drink burns among 1-2 year olds remained unchanged. The indigenous children are at higher risk of sustaining a burn, and together with children from rural WA are at higher risk from wound infections which might impact on scar outcome.

The findings of this study challenges the belief that the incidence of injury is declining, and that the prevention message is reaching the community. Targeting the parents of young children, new parents and parents-to-be with effective education is necessary to make an impact on incidence. It should be noted however, in the design of such a campaign that the prevention campaigns previously run in Western Australia have had no apparent impact on numbers. It is interesting to note that legislation appears to have been more effective in reducing scalds from tap water. The indigenous children are at risk from injury, and any campaign designed to help this group, needs to be

designed by indigenous people with experienced culturally sensitive health care professionals.

There was no improvement over the study period in the appropriateness of first aid treatment rendered by parents or carers at the scene. It was not possible to assess first aid rendered by health care professionals before the patient arrived at PMH due to poor documentation. In 2008 more carers called the ambulance or Royal Flying Doctor Service and less scald injuries were presenting to GPs in the first instance.

Over the study period hospital based analgesic administration and dressing use changed. Surgical techniques also changed, but the amount of comparative surgery did not. Future resourcing aimed at reducing the incidence severity and scar outcomes associated with scald injury among WA children should focus on the most affected suburbs; those which have the highest incidence and those which have the highest admission rate. Parents and carers of toddlers should be targeted for the prevention of hot drink scalds.

Recommendations

1. The implementation of an outpatient data collection program. The amalgamation of the outpatient data is essential to obtain a true picture of the patients that are managed by the paediatric burns service.
    a. to identify if the scald injury incidence in 1-2 year old patients has reduced overall, or if the management has changed.
    b. to assess the incidence of hot water scalds originating from tap water or kettle/pan water.
    c. to evaluate the effectiveness of the new hot water system legislation
    d. to identify if TBSAB is unchanged overall.
2. An ethically approved socio-economic study of admitted patients. These data can be used to help plan health promotion interventions by stratifying by type of burn; scald, contact, flame, chemical etc
    a. to compare socio-economic circumstances of individual patients to assess the effect of socioeconomic status on burn injury risk in children.

b. to investigate family structure such as the number of single parents, the number of siblings, and position of the child within the family structure

c. to investigate recent changes within the family to identify flux; for example, recent change of residence, would identify further risk factors.

3. A prevention program that targets:

    a. suburbs from which incidence and admissions are highest

    b. toddlers who are frequently scalded by hot drinks

4. An education program targeting the burns first aid message.

5. The development of novel wound depth analysis tools.

Further research is required for the development of depth analysis tools to allow for the earliest possible assessment of depth to predict surgical requirement. If the depth of the wound can be ascertained, then early surgical management of the patients with deeper wounds will result in an improved scar outcome. If the wounds are expected to heal within 10 days then this can be used as part of the clinical decision making process about admission vs outpatient management

6. The development of information for parents and carers with practical advice regarding coping strategies with children.

7. The provision of electronic distraction games for use during painful dressing procedures. Novel distraction methods are emerging with virtual reality and electronic games used for this purpose; these need to be tried and evaluated for their effectiveness in this setting to augment side effects from analgesic agents

8. A prospective observational study to investigate all types of bacterial colonisation and wound infection and current dressing use is needed to investigate:

    a. if preadmission wound infections are affected by area of residence and/or delays to first medical attention or silver dressing application

    b. if Aboriginal status has a bearing on incidence of wound infection

    c. if cessation of silver dressing before healing has occurred is contributing to post discharge infection.

d. if early application of silver helps prevent wound infection

9. The routine assessment and documentation of burn scars with a validated assessment tool for comparative outcome data analysis. Once implemented, routine data collection of key indicators can be evaluated in respect to scar outcome. Further studies of the relationship of scar outcome of itch or surgical techniques can then be undertaken. It is not possible to isolate and prioritise the factors affecting scarring without this.

Limitations

Retrospective data taken from medical notes is limited by the clinical experience and documentation skills of the writer. Clinical judgement taken from these data is subject to error, for example whether the wound infection data meet Peck's criteria, which is designed to eliminate false positives. First aid and preadmission data are often not documented.

Data is local to Western Australia and might not be generaliseable beyond the WA population. This was exploratory data study that used a multiplicity of testing methods and variables of subgroups, which were identified through analytical processes. This could have increased the likelihood of false-positive results and all results should be regarded with caution until confirmed by further research.

Knowledge contribution

This study has defined the status of paediatric scald injury in WA. It challenges some commonly held beliefs, such as:

- Burn injury is thought to be declining, but it seems that the incidence is unchanged and that scald injury might be increasing
- Socio-economic disadvantage is thought to increase injury risk, but it seems as though this, might be false for paediatric scald injury *per se*
- The time of injury might influence the first aid given by the carer
- It confirming the notion that the main risk factors for preadmission wound infection are 1) delay to presentation 2) non-metropolitan living and 3) Aboriginality
- New dressing types need further evaluation to assess their effect on wound infection rates

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APPENDIX 1

LUND AND BROWDER CHART FOR THE ASSESSMENT OF TOTAL BODY SURFACE AREA BURNED IN CHILDREN

BURNS SURFACE AREA SHEET

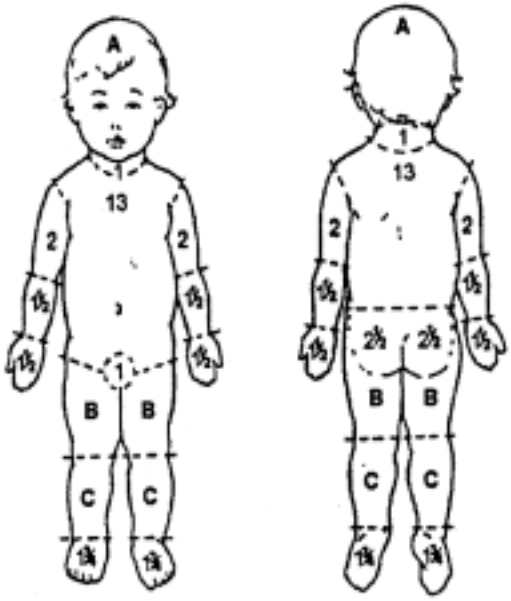
BURNS SURFACE AREA SHEET

Med. Rec. No: .....
Surname: .....
Forename: .....
Sex: ..... D.O.B. .....

AFFIX LABEL HERE

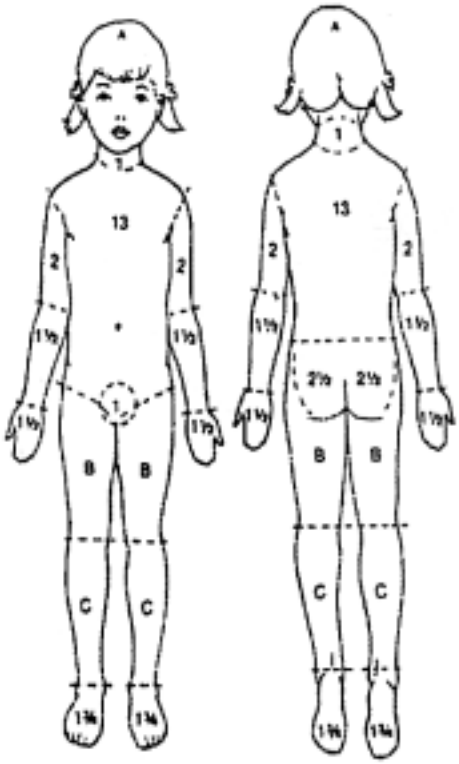
INSTRUCTIONS

1. Choose the figure most accurately resembling the child.
2. Shade the areas corresponding to the burn. DO NOT INCLUDE ERYTHEMA.
3. Use the charts to calculate the body surface area (BSA).
4. GUIDE: The patient's palm is equal to approx. 1% BSA.
5. See over for alternative charts.



RELATIVE PERCENTAGE OF BODY SURFACE AREA AFFECTED BY GROWTH

AREA / AGE	0 yr	1 yr	5 yrs	10 yrs	15 yrs	ADULT
A = ½ of head	9½	8½	6½	5½	4½	3½
B = ½ of one thigh	2¾	3¼	4	4½	4½	4¾
C = ½ of one leg	2½	2½	2¾	3	3¼	3½



REGION	%
HEAD	
NECK	
ANT. TRUNK	
POST. TRUNK	
RIGHT ARM	
LEFT ARM	
BUTTOCKS	
GENITALIA	
RIGHT LEG	
LEFT LEG	
TOTAL BURN	

Name:.....

Signature:.....

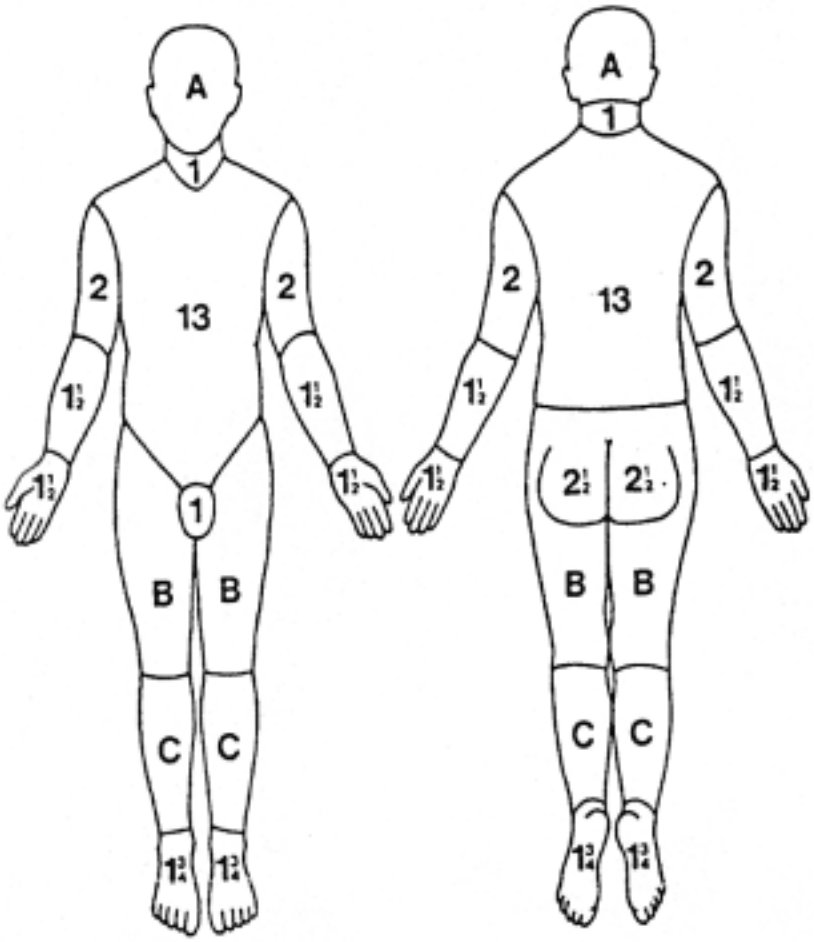
Date:.....

PRINCESS MARGARET HOSPITAL FOR CHILDREN

FEB 00
PM2024
MR96
19154L

Modified from: Wong, D. 1995 Whaley and Wong's Nursing Care of Infants and Children (5th ed) Mosby St Louis

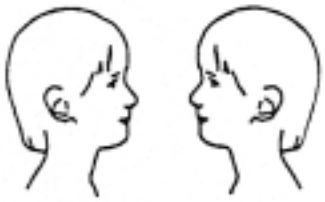
LUND AND BROWDER CHART



IGNORE SIMPLE ERYTHEMA

REGION	%
HEAD	
NECK	
ANT. TRUNK	
POST. TRUNK	
RIGHT ARM	
LEFT ARM	
BUTTOCKS	
GENITALIA	
RIGHT LEG	
LEFT LEG	
TOTAL BURN	

AREA / AGE	0 yr	1 yr	5 yrs	10 yrs	15 yrs	ADULT
A = ½ of head	9½	8½	6½	5½	4½	3½
B = ½ of one thigh	2¾	3¼	4	4½	4½	4¾
C = ½ of one leg	2½	2½	2¾	3	3¼	3½



Name:.....

Signature:..... **Date:**.....

APPENDIX 2

FLUID REUSCITATION CALCULATION GUIDELINES FOLLOWING SEVERE BURN INJURY IN CHILDREN

Burns
IV Fluid Management
(Burns Resuscitation fluids)

Management Guidelines
Emergency Department
Princess Margaret Hospital for Children
Perth, Western Australia
Last reviewed October 2010
Dr Gareth Kameron
PMH Burns Team

These guidelines are the approved Emergency Department management of all children with burns treated at PMH. Any variation of management must be approved by the burns consultant in charge of the patient.

Aims

- To rehydrate the child with a major body surface area (BSA) burn:

0 – 18 months:	8% and over	***Excludes** simple erythema*
Older children:	10% and over	

- To achieve adequate perfusion of all potentially viable tissue and to maintain function of all vital organs, as evidenced by adequate, and not excessive, urine output.

Resuscitation Fluids

- Please refer to ED IV fluids guideline

Burns Fluids according to Parkland Formula

(to be added to the child's normal maintenance fluids)

- Calculate the volume required for fluid replacement using the following formula:

% BSA x weight (in kg) x 2

- This gives an estimate of the volume of *replacement fluid* required in the first 24 hours **from the time of the burn** (not from time of arrival in hospital).

- Administer this calculated volume using **Hartmann's solution** as follows:

  50% within the first 8 hours
  50% over the next 16 hours

- Any resuscitation 0.9% saline boluses are ignored in this calculation.

NB:

Adjustments may be required based on the ongoing assessment of the child.
The Burns registrar will re calculate estimated BSA during their initial assessment

To this calculated volume, **add** the child's normal daily maintenance requirements (see below for details on calculating maintenance fluid).

Total fluid = rehydration fluids + maintenance fluid given as an hourly rate

- Any volume of fluid given and tolerated orally or by nasogastric tube should be deducted from the IV fluid volume that is required to maintain the desired urine output.
- Monitor urine output closely after first 2 hours.

Maintenance fluid:

This can be calculated as follows:

Term neonate: < 28 days

Day 1:	60 mL/kg/day	
2:	80	"
3:	80	"
4-5:	100	
6-7:	120	
8-28	150	

Other Children: Calculate maintenance fluids using the 4:2:1 Method

Up to 10kg 4mls/kg/hr

10-20kg 40mls/hr **AND** 2mls/kg/hr over 10 kg

Over 20kg 60mls/hr **AND** 1ml/kg/hr over 20 kg

Urine output

- As a rule, if a burn is severe enough to require IV fluid resuscitation, then urine output should be properly monitored with a catheter. Aim for 0.5- 1ml/kg/hr urine output. Adjust fluid rate to compensate.

Neurovascular Observations

The burnt area must be **elevated** to reduce swelling (especially important in circumferential burns), and the neurovascular status must be observed closely. Excessive fluids may result in increasing oedema, possibly compromising the circulation and necessitating an escarotomy. Early review by the Registrar or Consultant is vital if there are any concerns regarding vascular compromise.

Blood chemistry

The following need to be measured, if a cannula has been inserted for fluids

- Baseline tests:
  1. FBC, LFT, EUC, BSL
  2. Blood gases (if inhalation burns or carbon monoxide poisoning is suspected)
  3. Group and Hold (if other injuries present secondary to trauma)

APPENDIX 3

DIAGNOSTIC CRITERIA FOR WOUND INFECTION

Definitions of Burn Wound Infection

Peck et al (1998)

1. Burn wound impetigo:
    - loss of epithelium from a previously reepithelialised area – eg grafted burn, partial thickness burn wound healed by secondary intention, or healed donor site
    - not related to inadequate excision, mechanical disruption of graft, or haematoma formation
    - requires change or addition of antimicrobial therapy
2. Open burn related surgical wound infection
    - Occurring in surgically created wounds e.g. excised burns and donor sites that have not yet epithelialised
    - Has a culture-positive purulent exudate
    - Requires change in treatment e.g. a change or addition of antimicrobial therapy, removal of wound covering, or increased frequency for dressing changes and at least one of the following:
        i. Loss of synthetic or biologic wound covering
        ii. Changes in wound appearance such as hyperaemia
        iii. Erythema in the uninjured tissue around the wound
        iv. Systemic sighs e.g. pyrexia, leukocytosis
3. Burn wound cellulitis:
    - Infection occurs in uninjured skin around the wound or donor site
    - Associated with erythema that is more than expected burn inflammation
    - Not associated with other signs of burn wound infection
    - requires change or addition of antimicrobial therapy
    - and is associated with one or more of the following

i. localised pain, swelling, tenderness and heat

ii. systemic signs of infection

iii. progression of erythema and swelling

iv. lymphangitis and/or lymphadenitis

4. Invasive infection in unexcised burn wounds

- In deep partial or full thickness non surgically excised burn wound
- Associated with a change in appearance eg rapid eschar separation, brown, black, or violaceous discolouration of the eschar
- Requires surgical excision and systemic antimicrobials
- Associated with, but not dependent on one of the following

i. Inflammation of surrounding uninjured skin

ii. Histological examination of the burn biopsy specimen shows invasive infectious organism in adjacent viable tissue

iii. The organism is isolated in blood culture in absence of other identifiable infection

iv. Systemic signs of infection

APPENDIX 4

VANCOUVER SCAR SCALE

The Vancouver Scar Scale

1. Vascularity	Normal	0
	Pink	1
	Red	2
	Purple	3
2. Pigmentation	Normal	0
	Hypopigmentation	1
	Mixed	2
	Hyperpigmentation	3
3. Pliability	Normal	0
	Supple	1
	Yielding	2
	Firm	3
	Ropes	4
	Contracture	5
4. Height	Flat	0
	< 2mm	1
	2-5mm	2
	> 5mm	3

Vancouver Scar Scale modified according to Baryza and Baryza, 1995

APPENDIX 5

BURNS MINIMUM DATA SET

No.

PRINCESS MARGARET HOSPITAL DATA COLLECTION SHEET FOR BURN MINIMUM DATA SET

Addressograph

Age: _____(if <2years, write as ....months)

Country of birth: _____

Aboriginal/TSI Y N

Workers compensation Y N

Occupation:_____ *Funding source:* _____

Admission Data

Date and time of burn injury: ___/___/___ ____:____

Place of first attendance: SJA/RFDS GP/AMS/Clinic Local hospital PMH ED

Specify (if appropriate/known) _____

Place of second attendance: SJA/RFDS GP/AMS/Clinic Local hospital PMH ED

Referral source: PMH ED OPC Telehealth

Date and time of presentation to PMH ED:___/___/___ ___:____

Date and time of presentation to PMH OPC:___/___/___ ___:____

Date and time of admission ____/___/___ ____:____

Delay Y N

If Yes:

Delayed presentation	Y	N	Delayed referral	Y	N
Rural/remote	Y	N	Refusal/non-compliance	Y	N
Treatment in OPC	Y	N	Stabilisation at other ED	Y	N

Referral source: ED GP rural hospital metro hospital OPC Telehealth Other: _____

Category: Acute □ Non-acute

Burn wound:

Burn type: Chemical Contact Scald Electrical Explosion/flash Flame Friction Hot gas Radiant heat Cooling No cutaneous burn Other: _____

Specify agent: _____

TBSA (%): _____ Documented/estimated/unable to estimate

Total % Deep partial & full thickness burns: _____ Total % full thickness burns: _____

Location of burn (site): _____

Depth of burn (deepest area on clinical assessment):

Superficial ☐ Superficial/Partial ☐ Deep partial ☐ Full-thickness ☐

Circumstance of injury:

Accident (work) ☐ **Accident (non-work)** ☐ (non-intentional)

Self-harm Y N **Intentional harm** Yes / No /Possible (intentional)

Risky behaviour Y N **Alcohol/drug involvement** Y N

CPU Y N **DCP** Y N

Post code of injury (or, if unknown, residence): _____ Indoors/outdoors

Place of injury occurrence: home other residence school workplace other unknown

Geographical location: Metro□ Regional□ Remote□ Offshore□ Interstate□ Overseas□

 Acknowledgement: Robyn Silla Revised: T McWilliams, L Martin, C Jeeves

First aid:

Cool water Y N **Running water** Y N **Cool water immersion** Y N

Total duration: 0-5 min 5-10 min 10-20 min >20min

Wet towels Y N **Burn Aid** Y N

Inappropriate fist aid Y N Specify _____

No first aid Y N

First aid adequate (cool running water~20min within 3hrs of injury) Y N

Kept warm? Y N **First temp recorded**_____

Silver dressing during transfer to PMH Y N **Glad wrap during transfer to PMH** Y N

Transport to PMH > 2hrs? Y N

Transfer dressing to PMH (circle one only):

Nil SSD Acticoat other antimicrobial emollient Wet wrap Burn Aid

Other Not stated

Initial dressing on admission (circle one only):

Nil SSD Acticoat other antimicrobial emollient Wet wrap Burn Aid

Other Not stated

Co-morbidities : **Y** **N** **If yes:**

Universal **Y** **N**

Renal Y N CVS Y N Liver disease Y N

Resp Y N CNS Y N Diabetes Y N Neoplasm Y N

Psych Y N PVD Y N Dementia Y N

Paed specific **Y** **N**

Ear infection Y N URTI Y N Asthma Y N Eczema Y N

Giardia/parasites Y N FTT Y N Scabies Y N Head Lice Y N

Other: (specify) _____

Wt percentile: _____ Ht percentile _____

Concurrent injuries:

Inhalation injury **Y** **N** Corneal burn(s) **Y** **N** Perforated eardrum/s **Y** **N**

Lacerations **Y** **N** Abdominal injury **Y** **N** Fracture/dislocation **Y** **N**

Head injury **Y** **N** Drug overdose **Y** **N** Other: specify:_____

Initial intervention:

Admission ward: ICU □ BU □ Other _____

IV **Y** **N** Hydration **Y** **N** Resus fluids **Y** **N**

IDC **Y** **N** NGT **Y** **N** Escharotomy **Y** **N**

CVC **Y** **N** Intubated **Y** **N** Femoral line **Y** **N**

ADT **Y** **UTD** **N**

Screening swabs **Y** **N** Type**: Wound/Other** **(List results in micro section)**

Time from injury to IV access if fluid resus required: _____

Mean urine output = 0.5 – 1ml/kg/hr in first 36hours from time of injury? **Y** **N**

Blood sodium level at 36 hours post injury: _____

NGT inserted within 24 hrs from time of injury? **Y** **N**

Enteral feeds commenced within 24 hrs from time of injury? **Y** **N**

Escharotomy within 6 hrs of injury if compartment syndrome present**?** **Y** **N**

 Acknowledgement: Robyn Silla Revised: T McWilliams, L Martin, C Jeeves

Acute management up to 6 weeks post injury

NGT feeds **Y** **N** Number of days_____

TPN feeds **Y** **N** Number of days_____

Pre-op biopsy **Y** **N**

Surgery **Y** **N** Number surgical procedures to close wound _____

Number of surgical procedures (total) _____

Surgery

Date	Procedure	Graft failure?	Admission episode

CEA/cell spray **Y** **N** Recell **Y** **N** SSG **Y** **N** FTSG **Y** **N**

Glaborous cells **Y** **N** Biobrane **Y** **N** Integra® **Y** **N**

Blood Products Details

Blood Product Type	Date	Amount	Where given (Theatre/Ward/ICU)

FFP **Y** **N** Platelets **Y** **N** Albumin **Y** **N** Blood **Y** **N**

Systemic complications: **Y** **N**

If Yes:

ABx for PUO □ UTI □ Pneumonia □ Sepsis □ Other: _____

Local complications: **Y** **N**

Impetigo □ Overgranulation □ Other: _____

Graft loss **Y** **N** **Partial**

If yes: Healed by: Primary intent □ Additional surgery □ SSG / CEA (ward) □

Cellulitis □ Burn wound infection □ (pre admission / during admission / post-discharge)

Microbiology:

Number of different organisms isolated from wound _____

Were there concurrent multiple organisms in a single wound site **Y** **N**

Did the patient receive a course of antibiotic therapy for wound colonisation or infection? **Y** **N**

Was antibiotic prophylaxis given in theatre? **Y** **N**

For following table complete all wound swab results and other relevant positive microbiological tests

Date	WOUND	ISOLATE	SENSITIVITY
Date	OTHER SOURCE	ISOLATE	SENSITIVITY

Antibiotic Therapy: **Y** **N** Allergies (specify)_____

AGENT	DOSE	ROUTE	FREQ	START DATE	FINISH DATE	DURATION (days)

Scar management:

Pressure garments **Y N** Tubigrips **Y N** Splint **Y N** Steroid injection **Y N**

Discharge details:

Home ☐ Interhospital transfer ☐ Own risk ☐ Death ☐ Other ☐

Post discharge follow up: OPC ☐ Telehealth ☐ Booked admission ☐

Date of discharge: ___/___/___

Disposition on discharge: Alive ☐ Deceased ☐ Deceased date: ___/___/_____

Cause of death: _____ Survival time (days) _____

Disposition on discharge: Home ☐ Acute care facility ☐ Rehab facility ☐ Prison ☐ Death ☐

LOS total (days) _____ ICU ____BU__

ALLIED HEALTH SERVICES

Pain Team	**Y**	**N**	Physiotherapy	**Y**	**N**	Dietetics	**Y**	**N**
Occupational Therapy	**Y**	**N**	Social Work	**Y**	**N**	Speech pathology	**Y**	**N**
Audiology	**Y**	**N**	Orthotics	**Y**	**N**	Psychiatry/Psychology	**Y**	**N**
HITH	**Y**	**N**	Aboriginal Liaison	**Y**	**N**	Child Activity Coordinator	**Y**	**N**
Other	**Y**	**N**	(specify) _____					

COMMENTS

_____

_____
_____
_____
_____
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_____
_____

DEFINITIONS

BU	Burns Unit	ICU	Intensive Care Unit
CEA	Cultured Epithelial Autograft	LOS	Length of Stay
CNS	Central Nervous System	NGT	Naso-gastric Tube
CVC	Central Venous Catheter	SSG	Split Skin Graft
FFP	Fresh Frozen Plasma	TBSA	Total Body Surface Area
IDC	Indwelling Catheter	UTI	Urinary Tract Infection