

If undeliverable return to
The West Australian Wound Care Association
PO Box 1011
NEDLANDS WA 6909

West Australian Wound Care Association Newsletter
Print Post No PP639699/00027

SURFACE
MAIL

POSTAGE
PAID
AUSTRALIA

WEST AUSTRALIAN WOUND CARE ASSOCIATION

Newsletter

Edition No. 57

September, 2005

COMING EVENTS

7 September

Silver Chain 'Silver Symposium'
Venue: Parmelia Hilton

8-9 September

'Bugs & Bandages'
Combined State Conference
Venue: Parmelia Hilton

9 September, 2005

The WAWCA AGM will be held during the
'Bugs & Bandages' Conference
at 1300-1400

13-16 September

Australia and New Zealand
Burn Association (ANZBA) Conference, Sydney
anzba2005@tourhosts.com.au

Clinical Update

16 November 2005

Mary Lockett Theatre
(FJ Clarke Complex) SCGH

Disclaimer:- The opinions expressed by authors are their own and not necessarily those of the Editorial Board. The Board therefore accepts no responsibility for the accuracy of statements appearing herein.

Editorial comment

Keryln Carville RN STN(Cred) PhD

Silver Chain Nursing Association

September promises more excitement than the arrival of spring. On 8 and 9 September, 2005, the combined WAWCA and WA Infection Control Association Conference will be held at the Parmelia Hilton, Perth.

The planned program is an exciting blend of topics of interest to all delegates. Eminent international and national speakers have been invited and will enhance the two-day program. On 7 September, the Inaugural Australian Silver Symposium, which is being hosted by Silver Chain, is to be held at the same venue. The symposium affords an opportunity for scientists, clinicians, manufacturers and other professionals to meet as equal partners to discuss the scientific, clinical and ethical implications of using silver in clinical practice.

Late registrations are being accepted for both events and if further information is required please contact: Robyn Simcock on 9387 7265 or Keryln Carville 0402 792 324.

In this edition of the Newsletter, you will find an overview of a most interesting presentation that was given at the August WAWCA Clinical Update by Dr David Speers, Infectious Diseases Physician, Sir Charles Gairdner Hospital and Clinical Microbiologist, PathWest. He discusses the challenges associated with osteomyelitis. In addition, Robyn Rayner, RN from Silver Chain Bunbury, provides a comprehensive, but succinct review in table form of the different scar presentations and their defining features. The Editor would like to remind all members that the AGM will be held during the Bugs and Bandages Conference on Friday, 9 September at 1300-1400.

WAWCA Clinical Update

'Clinical case studies and Christmas cheer'

16 November, 2005

This will be the last clinical update for the year.

Come along and celebrate Christmas early.

Mary Lockett Theatre

(FJ Clarke Complex) SCGH

COMMITTEE MEMBERS

President:	Sue Hoskin	sue.hoskin@health.wa.gov.au
Vice President:	Pam Morey	(08) 9346 3266/3333 (page 4354) pam.morey@health.wa.gov.au
Secretary:	Beth Sperring	beth.sperring@health.wa.gov.au
Education Officer:	Donna Angel	donna.angel@health.wa.gov.au
Treasurer:	Laurie Foley	
Newsletter Editor:	Keryln Carver	
Medical Representative:	Genevieve Sadler	
Allied Health Representative:	Ron James	
State Representative to AWMA:	Pam Morey	
Committee members:	Barbara Pedersen, Jan Wright, Keryln Carville, Carmel Boylan and Tanya Baker	

Wound infections and osteomyelitis

Dr David Speers

Infectious Diseases Physician, Sir Charles Gairdner Hospital and Clinical Microbiologist, PathWest

Introduction

Only with the introduction of antibiotics to prevent and control wound infections could the modern era of surgery begin. However, it was soon recognised that not only was antisepsis of the surgical site critical but also that appropriately timed antibiotic prophylaxis significantly reduced the incidence of wound infection. These manoeuvres reduce the incidence of surgical wound infection but cannot eliminate it as heavily soiled wounds continue to have a low but defined wound infection rate (up to 10%).

Surgical wound infections

The determinants for the risk of surgical wound infection are listed in Table 1.

To help prevent wound infections bacterial contamination should be reduced by limiting the pre-hospital stay, treating any existing infections and minimising the use of drains and catheters. The local immunity at the wound site can be improved by containing any contaminating bacteria, minimising any wound dead space, removing devitalised tissue, draining haematomas and ensuring correct hydration and oxygenation. Treatment of malnutrition, obesity and diabetes and encouraging cessation of smoking will also promote the general immunity of the patient.

Antibiotics are best reserved for non-healing wounds with signs of infection, such as purulence, surrounding cellulitis and systemic toxicity as demonstrated by fever and malaise. Topical antibiotics are generally not recommended.

Wounds more likely to be complicated by osteomyelitis

The combination of contaminated wounds with proximity to underlying bones and joints is more likely to be complicated by osteomyelitis or septic arthritis. Examples include deep penetrating wounds such as animal bites, chronic ulcers such as diabetic foot ulcers and traumatic wounds such as compound fractures. Wounds such as clenched fist injuries where high level contamination occurs with little or no soft tissue covering of the underlying bones and joints are also at increased risk.

Diabetic patients with foot ulcers should always be assessed for the presence of underlying osteomyelitis as this is present in up to 50% of chronic ulcers and up to 90% of cases if the bone can be probed at the ulcer base ('probing for bone' test). The management of foot infections in diabetics is dependent on the severity of the infection as shown in Table 2.

Osteomyelitis

Osteomyelitis can be either haematogenous or contiguous to a local wound or ulcer. Acute haematogenous osteomyelitis is usually seen involving the bones around the large joints in children and can be treated with 3-4 weeks of antibiotics. Spinal osteomyelitis is seen in adults and can be either haematogenously seeded or occur following spinal surgery. It is usually due to *Staphylococcus aureus* but can be due to other organisms such as after complicated urinary tract infections. Contiguous osteomyelitis complicating infections such as foot ulcers in diabetics are due to the locally infecting organisms, once again most often *S. aureus*, but it can be polymicrobial.

Chronic osteomyelitis is defined by osteomyelitis present for at least 6 weeks. It should be suspected in non-healing ulcers and non-uniting fractures, and if sinuses are present. If necrotic bone is present (sequestrum) this must usually be debrided to attain cure. It is crucial to try and identify the causative organism prior to therapy due to the prolonged nature of the treatment. Blood cultures are often negative in acute osteomyelitis and usually so in chronic osteomyelitis. Bone biopsy or aspirates of associated soft tissue collections are superior to surface wound swabs as these specimens avoid contaminating organisms. X-ray changes occur late in osteomyelitis and nuclear medicine bone scans are sensitive but less specific, but MRI is the imaging modality of choice. Treatment involves a minimum of 6 weeks of antibiotic therapy, with the duration administered intravenously decided individually. Chronic osteomyelitis and spinal osteomyelitis are treated for longer, often for many months. Adequate surgical debridement is the key to the cure of chronic osteomyelitis.

Table 1: Surgical Wound Infection Risk

Increased Risk	Reduced Risk
Microbial contamination of the wound site	Appropriate antisepsis
Trauma to the wound site (devitalised tissue)	Prophylactic antibiotics
Presence of foreign material	Adequate debridement of wound
Presence of organisms resistant to prophylactic antibiotics	Improved local immunity at the wound site
Prolonged duration of operation	Improved general immunity of the host

Table 2. Management of Foot Infections in Diabetics

Severity	Characteristics	Therapy	Antibiotics
Mild	Superficial ulceration	Oral antibiotics Podiatry Care	Augmentin Dicloxacillin Clindamycin Cotrimoxazole
	Minimal cellulitis No osteomyelitis		
Moderate	Deep ulceration Cellulitis Systemic toxicity	IV antibiotics Surgical debridement	Timentin Tazocin Amoxycillin + Gentamicin + Metronidazole
	+/- Osteomyelitis	Assess vascular supply Metronidazole	Flucloxacillin + Gentamicin +
Severe	Deep ulceration Cellulitis	Urgent surgical debridement	Tazocin + Gentamicin
	Systemic toxicity +/- Osteomyelitis	IV broad spectrum antibiotics	Meropenem
	Necrosis/gangrene Bacteraemia	Assess vascular supply Metronidazole	Flucloxacillin + Gentamicin +

The above paper was presented by Dr Speers at the August WAWCA Clinical Update and has been reproduced in response to member interest.

Australian Wound Management Association
6th National Conference
Matrix of Wound Care
 National Convention Centre, Canberra
15-18 March, 2006

Keynote speakers:

Dr Sue Bale, Gwent Healthcare Trust; Mr Paul Banwell, FRCS Department of Plastic and Reconstructive Surgery, The Radcliffe Infirmary, Oxford; Dr Fiona Wood, Director, WA Burns Service; Mrs Susan P Morris, Vice President, Health Policy and Payment, Kinetics Concepts Inc. San Antonio, USA; Dr Keryln Carville, Silver Chain Nursing Association and Curtin University of Technology

For online registration or more information, visit
www.awma.com.au

Classification of keloid, hypertrophic, contracted and widespread scar

Robyn Rayner RN, BSc(Nsg), PG (Health Admin)

Wound Clinic, Silver Chain Bunbury

Scar formation is a normal physiological response to tissue injury and wound healing. Keloid, hypertrophic and contracted scar are however, demonstrations of aberrant healing. The following table outlines the clinical presentation, physiological composition, effects on healing times and predisposing factors associated with problematic scarring.

Table: Classification of keloid, hypertrophic, contracted and widespread scar

	Keloid scar	Hypertrophic scar	Contracted scar	Widespread (stretched) scar
Clinical presentation	<ul style="list-style-type: none"> • Raised indurated red or purple scar; spreads beyond wound margins^{2, 4, 8, 9, 12, 27} • Term applied after scar present at least a year but generally within 4 weeks^{1, 2} • May be inflamed, itchy and painful especially during growth-phase possibly from increased neural terminals or histamine level^{2, 9} • Site specific (ear lobe, deltoid and sternum), seen over bony prominence, thickened skin or where tension applied^{2, 4, 8} • Unique to humans, benign, occurs in response to injury (insect bites, acne, surgery ear piercing, lacerations, abrasions, tattoos, vaccinations, burns)^{1, 4, 8, 12, 18} • Genetic predisposition mainly dark races possibly from an abnormal melanocyte-stimulating hormone metabolism^{2, 3, 9} • 10:1 predilection in Afro-Caribbean women during pregnancy and puberty compared to cautions and 3:1 female / male ratio^{9, 17, 22} • Prevalent during increased hypothalamic activity^{9, 22} • Develop predominantly in 10-30 age group^{2, 4} • May remain symptomatic for years¹ • May be pruritic, tender and aggravated by secondary infections^{3, 19} 	<ul style="list-style-type: none"> • Raised scar that remains within wound boundaries^{2, 8, 15, 17} • Scar preceded by hyper-vascularity that may be hyper-pigmented, inflamed itchy and painful. Abates to pale or pink indurated tissue with maturity^{2, 4, 7, 9} • Occurs 6-8 weeks after epithelialisation, present earlier in erythematous tissue^{22, 25} • Occurs in wounds crossing lines of tension eg sternum (especially in females), upper back & major joints^{2, 4, 22, 25} • Generally occurs over joint or skin crease⁸ • 40-70% occur after surgery² • Approximately 91% occur after burn injury² • Occurs after delayed re-epithelialisation¹⁴ 	<ul style="list-style-type: none"> • Occurs at right angles over joint, skin crease or concavities² • Occurs when scars not fully matured² • Usually disfiguring & dysfunctional^{2, 16} • Occurs following burns² • Scar shorter than initial injury to restrict range of movement of skin and joint²³ 	<ul style="list-style-type: none"> • Fine surgical lines that stretch and widen² • Generally appears within 3 weeks of surgery² • Commonly present on trunk, arms & legs³ • Present as flat, pale, soft, symptomless scars frequently after shoulder or knee surgery²

	Keloid scar	Hypertrophic scar	Contracted scar	Widespread (stretched) scar
Physiological presentation	<ul style="list-style-type: none"> Histological – abundant chaotic orientated collagen fibres with acellular swirling nodular patterns located in deep epidermis^{2,4,12,20} Thickened, hyalinised collagen fibres with prominent mucoid matrix that is eosinophilic^{4,12} Benign, non-encapsulated hyperplastic fibrous growth^{9,12} Fibroblasts exhibit abnormal regulation of apoptosis^{19,20} Altered mRNA expression of apoptosis-associated genes¹⁰ In vitro fibroblasts produce high levels of collagen, fibronectin, proteoglycans²⁰ In vitro fibroblasts show atypical responses to regulation by metabolic modulators eg growth factors and hydrocortisone²⁰ Increased levels of inflammatory cytokines^{3,9,20} Increased deposition and insufficient degradation of collagen, extracellular matrix proteins (chondroitin-4-sulfate, fibronectin and elastin)^{4,12} Proliferation of collagen and glycosaminoglycan with increased collagen turnover in active growing phase^{4,9,27} Increased proline hydroxylase activity for collagen synthesis⁴ Increase collagenase activity, however, increased concentration of chondroitin-4-sulfate coats collagen fibres making them less susceptible to collagenase activity⁴ Lymphocytes persist compared to normal scars²⁵ Scar secondary to abnormal keratinocyte control over fibroblast²⁷ Epithelium generally thin⁷ Higher proliferation of keloid-derived human fibroblasts compared with normal scarring²⁷ Higher levels of immunoglobulins G, A & M compared to normal skin⁵ Anomalous expression of specific antigen by fibroblasts and keratinocytes with an increased level of epidermal CD1a+⁵ 	<ul style="list-style-type: none"> Over production collagen, fewer premature collagen fibres degraded^{7,9,22} Increase collagenase activity however, raise chondroitin-4-sulfate level coats collagen fibres making them insensitive to collagenase activity⁴ Fibroblastic level higher than keloid⁴ Collagen fibres disorganised with scanty mucoid matrix that run parallel to skin surface⁴ Scar highly vascular with large vessels^{3,22,24} Persistence of high density myofibroblasts cells^{21,24} Increased transforming growth factor-, (TGF-,) response & myofibroblast formation that temporary accumulate in wound²¹ TGF-b, constantly stimulate fibroblasts to secrete matrix proteins²¹ Increased TGF-, receptor over-expressing fibroblasts activity during remodelling causing over production of matrix proteins and fibrosis²¹ Persistence of lymphocytes compared to normal scarring²⁵ Increased number of Langerhans cells⁵ Constant keratinocytes activity indicate abnormal epidermal-mesenchymal interaction during prolonged inflammation delays healing & maturation^{14,25} Presence of keratinocyte (6, 16, 17) in interfollicular epidermis suggests epidermis pathogenesis in scar¹⁶ 	<ul style="list-style-type: none"> Myofibroblasts contract pulling epidermis inwards¹¹ Tropocollagen cross-links cleaved by collagenase causing contracture of collagen¹¹ 	<ul style="list-style-type: none"> Thin unbreached epidermis^{2,3} Scar flat but may be depressed³ No elevation, thickening or nodularity in scars² Scars possibly initiated by neutrophils & their neural proteases²⁵ Abdominal striae (stretch-marks) occur following injury to dermis and subcutaneous tissue² Elastin fibres failure to respond to tension³

Keloid scar	Hypertrophic scar	Contracted scar	Widespread (stretched) scar
<p>Physiological presentation</p> <ul style="list-style-type: none"> Possible epithelia-mesenchymal interactions contribute to formation keloid²⁷ Collagen synthesis 20 times that of normal skin and 3 times hypertrophic scar⁴ Fibroblasts fail to down-regulate growth and matrix synthesis in response to glucocorticoids⁶ 	<ul style="list-style-type: none"> Elevated levels of collagen propyl hydroxylase activity in collagen synthesis^{4,23} Increased testosterone receptor sites²⁰ 		
<p>Healing time and effect of time on healing</p> <ul style="list-style-type: none"> Increases with time^{2, 12, 20} Remains indefinitely, will not spontaneously regress^{2, 8, 12, 20} May not occur until sometime after wound healing⁹ 	<ul style="list-style-type: none"> Scar softens, flattens, blanches & regresses spontaneously after 1-2 years^{7, 8, 9, 22} May cause joint contraction and disfigurement^{9, 22, 24, 25} Improved by surgery⁸ 	<ul style="list-style-type: none"> Needs comprehensive long term therapy¹¹ Full mobility may never be achieved¹¹ 	<ul style="list-style-type: none"> Scar presents as flat highly vascular tissue that fades with time
<p>Predisposing factors for problematic scar</p> <ul style="list-style-type: none"> Increased occurrence after surgical excision^{2, 3, 8} Failure to use adjunctive therapies (radiotherapy or intralesional corticosteroid injection) following surgical revision^{3, 4, 8} Genetic predisposition with autosomal dominant and recessive modes of transmission^{4, 9, 20} Frequently occurrence in type A blood group¹ Prolonged inflammation from failing to clean wound, infection, anaemia, malnutrition, catabolism, impaired tissue perfusion, hepatic synthesis and diabetes⁹ Failure to provide appropriate support or reduce tension e.g. compression therapy, or low-load long-duration stress support (dynamic splints, serial casting, positional heat and stretching techniques)^{3, 4, 9, 11, 20} Injury to skin with high melanocyte levels^{3, 4} Extensive use or choice of sutures that act as a foreign body during scar revision and initiate an immune response^{4, 20} 	<ul style="list-style-type: none"> Deep dermis trauma inducing phenotypic epidermal changes¹⁶ Wound epithelialisation delayed due to prolonged inflammation from failing to clean wound, infection, anaemia, malnutrition, tension, catabolism, foreign material, impaired perfusion, hepatic synthesis & diabetes^{9, 16, 17, 24} 40-70% following surgery²⁰ 91% follow burn injuries, especially if epithelialisation delayed by >3 weeks²⁰ Present in 50-60% of children <5 years¹⁶ Skin with high melanin levels eg African, Asian and Hispanic races²⁰ Commencement of puberty or during pregnancy^{3, 20} Failure to reduce multi-directional mechanical tension particularly over sternum, deltoid and upper back wounds where presence skin cones high^{13, 17, 20, 22, 25} Failure to provide support & reduce longitudinal stretching^{9, 11, 25} Failure to excise burns < 20% of body surface¹⁹ Failure to provide appropriate support or treatment eg compression garments or early excision of scars^{3, 9, 11, 19} 	<ul style="list-style-type: none"> Failure to provide appropriate support eg compression therapy garments or low-load long-duration stress support (dynamic splints, serial casting, positional heat & stretching techniques) to increase tensile or shear forces^{3, 4, 9, 11, 20} Inappropriately applied or fitted pressure garments²⁶ Failure to apply skin graft to large joint wounds to reduce external tension^{3, 7}Continual tension in one axis & mobility of scar tissue^{3, 25} 	<ul style="list-style-type: none"> Elastin fibres failure to respond to tension³ Abdominal striae occurs after rapid weight gain or pregnancy Young predispose due to tight elastic skin that is unresponsive to tension^{3, 23}

References:

1. Baum, B.M. & Busuito, M.J. 1998, Use of a glycerin-based gel sheeting in scar management. *Advances in Wound Care*, 11, (1), pp. 40-43.
2. Bayat, A., McGrouther, D.A. & Ferguson, M. 2003, Skin scarring. *British Medical Journal*, 326, (7380), pp. 88-92
3. Beldon, P. 1999, Management of scarring. *Journal of Wound Care*, 9, (10), pp. 509-512.
4. Berman, B & Flores, F. 1998, The treatment of hypertrophic scars and keloids. *European Journal of Dermatology*, 8, (8), pp. 591-595.
5. Borgononi, L., Martini, L., Chiarugi, C., Gelli, R., Giannotti, V & Reali, U. 2000, Hypertrophic scars and keloids: immunophenotypic features and silicone sheets to prevent recurrences. *Annals of burns and fire Disasters*, 13, (3), pp. 164-169.
6. Davidson, J.M. 1998, Wound repair. *Journal of hand Therapy*, 11, (2), pp. 80-94.
7. Edwards, J. 2002, Scar management. *Journal of Community Nursing*, 12, pp. 1-5.
8. Edwards, J. 2003. Scar management. *Nursing Standard*. 17, (52), pp. 39-42.
9. Eisenbeiss, W., Peter, F.W., Bakhtiar, C., Frenz, C. 1998, hypertrophic scars and keloids. *Journal of Wound Care*. 7, (5), pp. 255-257.
10. Guttman, C. 2003, Keloid mechanics: apoptotic markers explored in scar development. *Dermatology Times*, 24, (5), pp. 28.
11. Hardy, M.A. 1989, The biology of scar formation. *Physical Therapy*, 19, (12), pp. 1018-1023
12. Hillmer, M.P. & Macleod, S.M. 2002, Experimental keloid scar models: a review of methodological issues. *Journal of Cutaneous Medicine and Surgery*, 6, (4), pp. 354-359.
13. Johnson, R.M. & Richard, R. 2003, Partial-thickness burns: identification and management. *Advances in Skin & Wound Care*, 16, (4), pp. 178-187.
14. Machesney, M., Tidman, N., Waseem, A., Kirby, L. & Leigh, I. 1998, Activated keratinocytes in the epidermis of hypertrophic scars. *The American Journal of Pathology*. 152, (5), pp. 1133-1141.
15. Matsumura, H., Engrav, L.H., Gibran, N.S., Yang, T-M., Grant, J.H., Yunusov, M.Y. Fang, P., Reichenbach, D.D., Heimbach, D.M. & Isik, F.F. 2001. Cones of skin occur where hypertrophic scar occurs. *Wound Repair and Regeneration*. 9, (4), pp. 269.
16. O'Kane, S. 2002, Wound remodelling and scarring. *Journal of Wound Care*, 11, (18), pp. 296-299.
17. Palmieri, b., Gozzi, G. & Palmieri, G. 1995, Vitamin E added silicon gel sheets for treatment of hypertrophic scars and keloids. *Internal Journal of Dermatology*, 34, (7), pp. 506-509.
18. Phipps, A. 1998, Evidence-based management of patients with burns. *Advances in Skin & Wound Care*, 7,(6), pp. 299-302.
19. Rekha, A. 2004. Keloids – a frustrating hurdle in wound healing. *International Wound Journal*, 1, (2), pp 145-148.
20. Roseborough, I., Grevious, M. & lee, R. 2004, Prevention and treatment of excessive dermal scarring. *Journal of the National Medical Association*, 96, (1), pp. 108-116.
21. Schmid, P., Itin, P., Cherry, G., Bi, C. & Cox, D. 1998, Enhanced expression of transforming growth factor- α , type I and type II receptors in wound granulation tissue and hypertrophic scar. *The American Journal of Pathology*, 152, (2), pp. 485-493.
22. Smith, F.R. 2005, Causes of and treatment options for abnormal scar tissue. *Journal of Wound Care*, 14, (2), pp. 49-52.
23. van den Helder, C.J.M. & Hage, J.J. 1994, Sense and nonsense of scar creams and gels. *Aesthetic Plastic Surgery*, 18, pp. 307-313.
24. Ward, S. 1991, Pressure therapy for control of hypertrophic scar formation after burn injury. *Journal of Burn Care and Rehabilitation*, 12 (3), pp. 257-262.
25. Widgerow, A.D., Chait, L.A, Stals, R. & Stals, P.J. 2000, New innovations in scar management. *Aesthetic Plastic Surgery*, 24, pp. 227-234.
26. Williams, F., Knapp, d. & Wallen, M. 1997, Comparison of the characteristics and features of pressure garments used in the management of burn scars. *Burns*, 24, pp. 329-335.
27. Yang, G.P., Lim, I.J., Phan, T., Lorenz, H.P. & Longaker, M.T. 2003, From scarless fetal wounds to keloids: molecular studies in wound healing. *Wound Repair and Regeneration*, November-December. Pp. 411-418.

This newsletter is produced with the generous support of

