

MANAGEMENT OF BURNS IN A MODERN CONFLICT – THE MITCHINER MEMORIAL LECTURE 2007

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Introduction

It is little known that General Mitchiner had an interest in burns. The subject, featured in his Hunterian Lecture to the Royal College of Surgeons of England in 1933, was followed by an entire textbook, "The Modern Treatment of Burns and Scalds," two years later [1].

In Mitchiner's lecture "Surgery in Two Wars" delivered before the Royal Faculty of Physicians and Surgeons of Glasgow, on January 29, 1947, he concluded, "*Burns have been more common and more extensive in the recent war. It is difficult to obtain any figures of the mortality, since so many died before reaching medical aid or, after first-aid treatment, before getting back to base hospital*" [2].

Today, mortality has been drastically reduced. Improvements in initial resuscitation, control of infection and reconstructive surgery, enable patients with burns in excess of 95% not only to survive but to expect a reasonable quality of life. Unfortunately, however, the figures coming out of Iraq show that the mortality rate for combat burn patients with a total burn size over 50% is only 52.5% [3]. The reasons for poor survival statistics can be understood by examining medical care under battle conditions. The handling of mass casualties is compounded in war by primitive facilities and the constant threat of enemy fire. This was illustrated by the dressing station at Ajax Bay during the Falklands campaign where there were no static medical facilities, inadequate numbers of staff and equipment, the problem of sheer numbers and the uncertainty and confusion of the "fog of war".

This paper discusses the advances in burn care, built on General Mitchiner's contributions to the welfare of the injured soldier.

Mitchiner's View

In 1935 Mitchiner wrote "injurious and often fatal results from burns fall into four categories" [1]:

Immediate Shock

He used the word 'shock' in the lay sense, "*an upset in the nervous system resulting from the accident,*" but he also included in this category inhalation injury as a major contributor to fire related deaths referring to it as "*pulmonary congestion and suffocation from exposure to the smoke of the conflagration*" [1].

The Collapse

Now known as burn shock, Mitchiner reported this to be responsible for 80% of deaths in his series. He correctly attributed this phase to loss of serum from the burn wound, and observed that it became manifest at half an hour to two hours from the

accident. He noted that its severity was related to the size of the burn but independent of depth and recognised that the leak was due to loss of serum from the wound, hypothesising that this was caused by histamine release. Interestingly, his recommendation for treatment centred on the use of tannic acid, rather than aggressive fluid replacement, commenting "*it is better to try and prevent the drain by coagulation of the damaged vessels and their contents*" [1].

The stage of septic intoxication

This was regarded as a serious menace in those few cases that survived the previous stage of collapse. Mitchiner described wounds that were cellulitic and purulent and patients deteriorating with fever, rigors and delirium; perforated duodenal ulceration not uncommonly found at post mortem. A major advance reported by him in the treatment of the burn wound was the development of antibiotics during the Second World War, since sulphonamides available at the beginning, had no effect on staphylococcal infections. In 1943 the introduction of penicillin appeared miraculous in combating dread infections such as gas gangrene. "*No longer were they seeing the thin, anaemic, emaciated individuals with their septic wounds, commonplace in the 1914-18 war.*" [2]

Scarring

The fourth category of Mitchiner was illustrated in his book with a soldier who had a cordite burn to the head and face. His final sentence explained why scarring and deformity were included in a chapter on mortality, "*scarring though seldom directly a cause of death may lead to suicide.*" [1]. And he raised an important point; disruption of facial appearance may constitute a major life crisis. Ultimately, if we have someone who cannot be integrated into society, we will have achieved little for our patients.

After Mitchiner

Inhalation injury

The evening of November 28, 1942, marked one of the worst civilian catastrophes in American history when fire blazed through Boston's Coconut Grove nightclub [4]. Within 15 minutes the entire club was consumed, culminating in the deaths of 491 victims. Survivors were treated at two neighbouring medical facilities where subsequent research hugely advanced the understanding of burn physiology.

Patients, often with minimal burns, were noted to have high carbon monoxide levels, their faces covered with soot, and survivors demonstrated hoarseness, cough and dyspnoea. Walking wounded were observed to collapse suddenly and die, reminiscent of mustard and phosgene poisoning in World War 1. Autopsies frequently revealed extensive necrotizing tracheobronchitis [5].

Sixty years later we recognise that these clinical findings were due to inhalation of dense and choking smoke. Combustion of wood and plastic is now known to contain a host of respiratory toxins, including carbon monoxide, hydrogen cyanide and

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phosgene amongst others. It is a sanguine fact that if this room were to ignite, the burning of the upholstery alone could produce numerous lethal gases, a situation recently brought home following the admission of six patients to our burn unit when a polyvinyl chloride chemical plant exploded, producing hydrogen chloride [6]. Each of them suffered severe respiratory damage.

Smoke causes sloughing of the mucosa, accumulation of epithelium and fibrin [casts] in the airways and margination of leukocytes in the pulmonary capillaries with release of inflammatory mediators [7]. The net result is airway obstruction, bronchospasm, diminished lung compliance, tracheobronchitis and pneumonia and ultimately respiratory failure. The mainstay of treatment is still control and security of the airway, although controversy exists regarding the role of prophylactic steroids and antibiotics and specific modes of mechanical ventilation [8]. Despite numerous innovative methods of support, smoke inhalation remains a main determinant of burn mortality.

Burn Shock

The Cocoanut Grove fire also stimulated advances in the treatment of burn shock. Studies by Cope and Moore at the Massachusetts General Hospital, clarified the concept of burn oedema. This led to the development of the first comprehensive formula for fluid resuscitation based on estimation of burn size aided by the simultaneous development of an accurate method for calculating the percentage surface area burned, by Lund and Browder, working at the nearby Boston City Hospital [9,10].

We now understand that histamine is only one of several inflammatory mediators modulating vascular permeability and microvascular hydrostatic pressures. The net result is an imbalance of physical forces controlling fluid flux across the capillary as fluid passes from the vascular to the interstitial compartments. Failure to correct the fluid losses will result in decreased cardiac output, renal failure and cardiovascular collapse [11].

Medical experts for the British military stated before the start of the First Gulf War that "Our burns and plastic surgery units might be overwhelmed with thousands of casualties," with estimates reaching 4,500 [12]. Indeed, to accommodate such casualties, the world's largest burn unit was set up in the basement of an unfinished terminal at King Khaled international airport, Riyadh. These figures appeared realistic considering that in recent wars one in four casualties was burned [13] and could be expected to be even higher in the Gulf War conflict, in which heavy tank combat was anticipated, and the threat of oil well ignition and chemical warfare loomed.

In preparation, military exercises were carried out at 33 Field Hospital, Al Jubayl, where simulated patients were passed through a chain of evacuation. Gross errors in calculating fluid requirements were made, occasionally by a factor of 10. Difficulties arose because of the variable quantities of fluid administered in transit, protracted transit times as well as lack of familiarity with the standard formulae. It is naïve to think that by using a mathematical formula and producing a regimen to the nearest millilitre or even a fraction of a millilitre that this is the most accurate way of assessing the fluid requirements after a burn: at best it is only a guide. After all, it is easy to forget that the weight of most ill patients will be guessed. Estimating the surface area burned is also an approximation; it is rare for a burn to fit precisely an area on a Lund Browder Chart.

Further chaos ensued when real casualties arrived. Fluid balance charts of some of the wounded soldiers showed urine outputs in excess of 700 ml. per hour, a problem not helped by faulty Combat Treatment Regimens which proved too complex to be understood by most medical personnel.

Delayed initiation of correct fluid replacement sets the stage for hypovolaemia and renal failure; whilst fear of under resuscitation has led to over-resuscitation which has now become a pressing

concern in the present Iraq theatre. A new term "resuscitation morbidity" has been coined to include a host of sinister complications, such as abdominal compartment syndrome [14].

Simpler regimens have been suggested to treat mass casualties under conditions of war. The "British Army Burns Formula" fails to take into consideration the weight of the patient. Although probably sufficient for young servicemen it may be inaccurate in slightly older soldiers, or less healthy civilians, who are seen not infrequently in recent conflicts.

The "Uganda Rule" was named after the hospital ship in the Falklands conflict whereby the drip rate was adjusted to hourly hematocrit measurement [15]. Haematocrit, however, as a ratio of blood cells to blood volume is an exceedingly poor monitor in the presence of large burns where blood is destroyed in the burned capillaries, or in the presence of associated haemorrhage; a likely possibility, since burn injury does not always occur in isolation.

It was therefore clear that a system was needed to calculate accurately and rapidly, fluid requirements in the burn patient during battle. The first step in the evolution of such a system was to simplify mathematical calculations by assuming the patients to be of standard weight [appropriate for a military setting] and computing the fluid volumes in advance. The information was then displayed in a simple tabular form (Battlefield Burns Table), which was displayed on the sides of the tent flaps [16] (Figure 1). Later this concept was expanded for use in the civilian population and included children. Multiple tables were represented in disc form, which progressed to the 'Burn Wheel [17,18]. The process was subsequently automated by a computer program, which ran on a hand held device [19].



Figure 1. Battlefield Burns Tables

Sepsis

Sepsis is still the major cause of death among burn patients. The burn wound is an ideal substrate for bacterial growth and provides a portal for microbial invasion. Loss of the skin's physical barrier and translocation of bacteria across the gut favour bacterial invasion. The activation of a pro-inflammatory cascade after a burn appears to be important in the development of subsequent immune dysfunction, typified by reduced lymphocyte subpopulations, reduced macrophage and neutrophil killing, and decreased levels of immunoglobulins, opsonins and chemotactic factors [20]. Therefore, a major factor in improving outcomes has been the control of infection. The introduction of topical antimicrobial agents in the mid 1960s significantly decreased bacterial sepsis. Despite our efforts to limit the use of systemic antibiotics to specifically identified pathogens, strains of bacteria have evolved to become highly resistant to all antibiotics, including vancomycin-resistant *Enterococcus*, *Pseudomonas* and *Serratia* [21].

In 1987, surgery on African clawed frogs in the laboratory, led to the observation that despite a total lack of sterile procedure their wounds healed without infection. The deduction that the skin of the frog produced a substance that had antibiotic qualities proved correct when two related peptides were isolated, which were called

magainins (Hebrew = shield), able to kill a wide range of bacteria and fungi [22].

A second observation reported in Nature, that patients with psoriasis have fewer skin infections than expected, lead to an analysis of psoriatic scales and their isolation and purification of an inducible transcriptionally regulated, antibiotic peptide that protects human skin in a similar way, which they named Human beta Defensin-2 (HBD-2)[23]. It is now clear that these peptides form part of a group whose microbiocidal and cytotoxic properties are most likely a consequence of their detergent-like properties with their ability to insert into biological membranes and generate pores.

The existence of a shield of endogenous peptide antibiotics, operative in skin was of major interest to us. Did this represent a third immunological defence system of the human body that was independent of lymphoid cells, and might disruption of this shield be a reason for invasive burn sepsis?

In an attempt to address these questions, our first series of experiments showed that HBD-2 levels are decreased in burned skin following thermal injury [24], altered in lung fluid following inhalation injury [25], and absent in burn blister fluid [26]. Using fluorescence deconvolution microscopy we later localized HBD2 protein in the basal layer of normal skin and, in a number of other cell types and structures, such as hair follicles and sweat gland acini [27]. The results suggest that these sites may be capable of being upregulated as a treatment of burn infection.

More recently, new roles for antimicrobial peptides have been proposed. In addition to their antimicrobial properties they have been shown to play a role in adaptive immunity being chemotactic for dendritic cells and T-lymphocytes and also can promote angiogenesis and wound healing. We recently demonstrated the presence of intranuclear HBD-1 in keratinocytes throughout the stratum spinosum, suggesting a role for this peptide in gene expression and providing new data that may help to determine mechanisms of defensin functions [28,29].

Replacement in the form of a topical agent or by gene transfection may also provide a practical therapy in the future.

Treatment of the burn wound

Tannic acid

Throughout his career, Mitchiner was a keen advocate of tannic acid. The acid was credited with the ability to bind to some toxin, said to be produced as a bi-product of burned skin, a concept supported by strong experimental evidence at the time. Precipitation of protein formed a dense leathery eschar, said to reduce plasma loss, decrease pain, speed up wound healing and promote recovery. The coagulum on the surface was even said to act as a scaffold for growth of epithelial cells over the wound and reduce scarring. Mitchiner reported a miraculous reduction in mortality to an all time low of 2.4% [30].

Subsequently, however, the use of tannic acid fell out of favour [31]. Its use on hands led to stiffness of the fingers, and tanning of facial burns often led to immobilisation of the eyelids resulting in dehydration and even blindness. Cytotoxicity was thought capable of converting burns from superficial to deep. Its poor antibacterial capacity also carried the risk of spread of infection, seen in the Second World War when Porritt, reporting on the desert wars in the Middle East, found that after tanning, 90% of his patients were septic with infected eschars. The final nail in the coffin occurred in the early nineteen forties when reports on the potential hepatotoxic effects of tannic acid started to appear.

Excisional Therapy

It is understood that superficial burns will heal spontaneously. By keeping the wound moist and free of infection, we can preserve viable cells in the dermis and encourage regeneration of skin and obtain a perfect result. Unfortunately if we apply this conservative

approach to deeper burns, devastating scar contractures may result.

The treatment of deep burns was for a long time the centre of debate. Traditional wound management whereby topical antibiotics were applied daily or twice daily until the eschar was allowed to slough by auto-digestion was abandoned for a more aggressive approach. Early excision of dead tissue and immediate closure with skin grafts became the standard of care following the demonstration by Zora Jankecovic in a review of 2615 patients. Here, the early tangential excision, the shaving of parallel layers of burned skin until bleeding is reached, and immediate skin grafting produced better cosmetic and functional results. Additionally, the number of operations and length of hospital stay were reduced [32].

Today, the knife may be our worst enemy, with the fear that the pendulum has swung too far. Our approach to burn wounds is often akin to treating all gastric ulcers by gastrectomy. The challenge is that excision of necrotic burn tissue inadvertently removes viable keratinocytes and progenitor stem cells that have the potential to regenerate new skin.

Having seen that superficial burns heal with minimal scarring; can we promote spontaneous healing in deeper 2nd degree burns?

We recently treated a baby, who had both cheeks cruelly burned with an iron. Instead of excising and grafting the burn, which would have produced unsightly patches on her face, the wound was lightly debrided and then covered with Transcyte, a human-cell derived tissue engineered biological wound covering. The contained neonatal fibroblasts which secrete growth factors, have stimulated complete regeneration of epithelium with no scarring.

Massive burns (over 60%) where autologous donor sites are limited, present a further challenge. One approach that emerged was the growth of epidermal cells in culture [33]. A sample of unburned skin was harvested from the axilla and processed in the laboratory to produce sheets of autologous keratinocytes, 2-8 cells thick. Unfortunately, optimism for this technique has waned, not least due to poor durability of skin which lacks dermis and the high cost of its production. Skin substitutes such as Integra, a bi-layered product consisting of an epidermal and dermal analogue have been used with success. After 2-3 weeks the dermal matrix is vascularised and the sialastic layer is removed to be replaced with ultra thin autografts [34].

Scarring and facial deformity

The final question is how do you give back a human face to a young person whose face has been totally disfigured [35]? Current methods of facial reconstruction emerged from the trenches of the First World War. Mitchiner would have been familiar with such patients whose shattered faces were been repaired utilising the patient's own tissue in the form of grafts and novel flaps (Figure 2). More recently, this patient of mine (Figure 3), suffered similar wounds following a shotgun injury. The principles of treatment have changed little, with perhaps one advance being the replacement of the mandible and chin in one stage using the fibula as a free tissue transfer.

Newer techniques for correction of post burn hypertrophic scarring of the face have surfaced. For example the use of tissue expansion has allowed the creation of supple adjacent flaps that match the reconstructed area in colour and texture [36], Integra™ [37], has also proved helpful. Nevertheless it can be argued that present reconstructive techniques are not always possible and after numerous operations may produce inferior, mask like results associated with huge donor site defects.

The superior cosmetic results of facial reattachment following traumatic avulsion of the face [38] coupled with the encouraging results in human hand transplantation laid the ethical groundwork for human face transplantation [39], and in November 2005, a 38 year-old woman after being savaged by a



Figure 2a and b. Gun shot wound during the First World War. Treated over a 2 year period by methods pioneered by Gillies et al.



Figure 3a and b. Extensive gun shot wound to the face treated by contemporary surgical techniques

dog had her nose, lips and chin replaced, in one piece, from a corpse, by a surgical team in France [40]. This unleashed media frenzy. Public reaction was decidedly mixed. One argument was that surgeons were entering an undignified race, taking a fairly healthy person with a facial disfigurement and transforming them into a morbidly ill individual who must endure a toxic regimen of drugs for the remainder of his/her life.

The technical feasibility of the operation uses well established micro vascular techniques and our own injection studies have confirmed that vascularisation of the entire face relies on the terminal branches of the external carotid arteries [41].

Thus, on one hand we have devastating psychological affects of severe disfigurement against which the patient must balance huge risks. Unless donor and recipient are identical twins, all patients who receive a transplant will have to be treated with lifelong immunosuppression to prevent rejection. Modern drug regimens are likely to be the same as that used for hand transplants and all are problematic [42].

Immunosuppression increases the risk of most forms of cancer; the incidence of colorectal and lung cancer is increased two to four fold and in those tumours associated with a viral cause such as Kaposi's sarcoma or lymphomas the risk may be increased 50 times. Half the patients will develop recurrent squamous cell carcinomas. Long-term therapy also increases the risk of infection with particular susceptibility to viruses and fungi [43]. Although studies show that the newer drugs mentioned have improved acute rejection-free survival, it is not possible to predict accurately the likelihood of immunologic rejection after facial transplantation, but a graft loss of approximately 10% from acute rejection within

the first year might be a reasonable estimate. While an episode of rejection might be treatable by increasing dosage of immunosuppressive agents, surgical removal of the transplant might well be necessary. This would require additional surgeries, and at best return the patient to preoperative levels of disfigurement with possible further scarring [43].

Most of the arguments against transplantation, however, would vanish if it proved feasible to achieve antigen specific tolerance? This absence of graft rejection without the use of immunosuppressive agents is the holy grail of transplant immunological research and at present the only clinically applicable strategies for producing transplant tolerance involves bone marrow transplantation, which would be unacceptable in this context [44,45].

Surprising as it may seem, burn surgeons perform more transplants than anyone else. Although not as romantic an organ as the heart or lungs, cadaver skin or allograft is the "gold standard" for temporary cover of large, full thickness burns [46]. A 19 year old man presented to our burn unit with a 75% total body surface area deep second and third degree burn from accidental ignition of his toy aeroplane fuel. Due to lack of donor sites, his chest, abdomen and arms were resurfaced with allograft. Four weeks later, the cadaver skin showed no signs of rejection and at seven weeks, the graft continued to provide stable wound cover. HLA testing and histological analysis suggested that the mechanism of allograft persistence was not survival of donor keratinocytes but repopulation of the allograft by recipient cells. Clearly the challenge of skin antigenicity and risk of life-long immunosuppression, might be solved if methods to repopulate allograft skin are pursued.

Now that face transplantation has moved into the clinical phase it is important to consider the physical, cosmetic, psychological and social aspects of facial disfigurement [46]. In essence, the ethical issues are complex, but boil down to the following: Proponents argue that a new face will give those individuals who are significantly disfigured a better quality of life if not a "new life." Opponents believe that the risks outweigh the benefits.

Conclusion

And so we have come full circle. We return to two quotations from General Mitchiner: "The plastic surgeon is all too prone to judge the results of burns treatment by the scars he produces or rather their absence, and to lose sight of their mortality resulting from the burn itself before a patient comes to his care." And secondly "Scarring, though seldom directly a cause of death, may later lead to suicide."

Mitchiner seems to be torn between our duty, on the one hand, to save life and, on the other, to prevent deformity. Modern advances may enable us to realise, in the interest of the troops, his efforts to achieve both.

References:

1. Mitchiner PH. The Modern Treatment of Burns and Scalds 1935. Baltimore; William Wood and Company.
2. Mitchiner PH. Surgery in two wars. Br Med J. 1947; 2[4518]: 219-20.
3. Wolfe SE. Modern burn care. J Trauma. 2007;62:S67.
4. Saffle JR. The 1942 fire at Boston's Coconut Grove Nightclub. Edgar J Poth Memorial Lecture. Am J Surg 1993; 581-91.
5. Davies JW. Toxic chemicals versus lung tissue – an aspect of inhalation injury revisited. The Everett Idris Evans Memorial Lecture – 1986. J Burn Care Rehabil 1986;7:213-22.
6. Einhorn IN. Physiological and toxicological aspects of smoke produced during the combination of polymeric materials. Environ Health Perspect 1975;11:163-89.
7. Walker HL, McLeod Jr CG, McManus WF. Experimental inhalation injury in the goat. J Trauma 1981;21:962-4.
8. Mleak RP, Suman OE, Herndon DN. Respiratory management of inhalation injury. Burns 2007; 33:2-13.
9. Cope O, Moore FD. The redistribution of body water. Ann Surg 1947;126:1016.
10. Lund CC, Browder NC. The estimation of areas of burns. Surg Gyn Obstet

- 1944;79:352-8.
11. Demling RH. The burn edema process: current concepts. *J Burn Care Rehabil* 2005;26:35-45.
 12. Hansard. Gulf War Casualties. *HC Deb* 05 February 1991; 185145.
 13. Williams JG, Riley TRD, Moody RA. Resuscitation in the Falklands Campaign. *Br Med J [Clin Res Ed]* 1983;286:6367.
 14. Chung KK, Blackburne LH, Wolf SE, White CE, Renz EM, Cancio LC, Holcomb JB, Barillo DJ. Evolution of burn resuscitation in operation Iraqi freedom. *J Burn Care Res* 2006;27:606-11.
 15. Richards T. Medical lessons from the Falklands. *Br J Hosp* 1983;286:790-1.
 16. Milner SM, Rylah LT. War burns: a simplified resuscitation protocol. *Br J Hosp Med*. 1993;50:163-7.
 17. Milner SM, Hodgetts TJ, Rylah LT. The Burns Calculator: a simple proposed guide for fluid resuscitation. *Lancet*. 1993;342:1089-91.
 18. Milner SM, Rylah LT, Bennett JD. The Burn Wheel: a practical guide to fluid resuscitation. *Burns*. 1995;21:288-90.
 19. Roth AC, Leon MA, Milner SM, Herting RL Jr, Hahn AW. A personal digital assistant for determination of fluid needs for burn patients. *Biomed Sci Instrum* 1997;34:186-90
 20. Nguyen TT, Gilpin DA, Meyer NA, Herndon DN. Current treatment of severely burned patients. *Ann Surg* 1996;223:14-25.
 21. Erol S, Altopariak U, Akcay MN, Calebi F, Pariak M. Changes of microbial flora and wound colonization in burned patients. *Burns* 2004;30:357-61.
 22. Zasloff M. Magainins, a class of antimicrobial peptides from *Xenopus* skin: isolation, characterization of two active forms, and partial cDNA sequence of a precursor. *Proceedings of the National Academy of Sciences [USA]* 1987;84:5449-53.
 23. Harder J, Bartels J, Christophers E, Schröder JM. A peptide antibiotic from human skin. *Nature*. 1997;387:861.
 24. Ortega MR, Milner SM. Reduced antimicrobial peptide expression in human burn wounds. *Burns*. 1999;25:411-3.
 25. Milner SM, Cole A, Ortega MR, Bakir MH, Gulati S, Bhat S, Ganz T. Inducibility of HBD-2 in acute burns and chronic conditions of the lung. *Burns* 2003;29:553-5.
 26. Ortega MR, Ganz T, Milner SM. Human beta defensin is absent in burn blister fluid. *Burns*. 2000 Dec;26:724-46.
 27. Poindexter BJ, Bhat S, Buja LM, Bick RJ, Milner SM. Localization of antimicrobial peptides in normal and burned skin. *Burns* 2006;32:402-7.
 28. Bhat S, Milner S. Antimicrobial peptides in burns and wounds. *Curr Protein Pept Sci* 2007;8:506-20.
 29. Bick RJ, Poindexter BJ, Buja LM, Lawyer CH, Milner SM, Bhat S. Nuclear Localization of HBD-1 in Human Keratinocytes. *J Burns Wounds*. 2007;24;7:e3.
 30. Maisel AQ. *Miracles of Military Medicine* 2006. Kessinger Publishing, MT.106.
 31. The use of tannic acid in the local treatment of burn wounds; intriguing old and new perspectives. *Wounds* 2001;13:144-58.
 32. Janzekovic Z. A new concept in the early excision and immediate skin grafting of burns. *J Trauma* 1975; 15:42-62.
 33. Gallico GG, O'Connor, Compton CC et al. Permanent coverage of large burn wounds with autologous cultured human epithelium. *N Engl J Med* 1984;311:448-51.
 34. Burke JF, Yannas IV, Quinby WC, Bondoc CC, Jung WK. Successful use of a physiologically acceptable artificial skin in the treatment of extensive burn injury. *Ann Surg* 1981;194:413-28.
 35. Bradbury E. Understanding the problems. In: Landsdowne R, Rumsey N, Bradbury E, Carr A, Partridge J, eds. *Visibly different: Coping with disfigurement*. London: Butterworth-Heinman;1987.
 36. Spence RJ. Expanded Transposition Flap Technique for Total and Subtotal Resurfacing of the Face and Neck. *J Burns Wounds* 2007;6:e8.
 37. Klein MB, Engrav LH, Holmes JH, Friedrich JB, Costa BA, Honari S, Gibran NS. Management of facial burns with a collagen/glycosaminoglycan skin substitute-prospective experience with 12 consecutive patients with large, deep facial burns. *Burns*. 2005 May;31:257-61.
 38. Wilhelm BJ, Kang RH, Movassaghi K, Ganchi PA, Lee WP. First successful replantation of face and scalp with single-artery repair: model for face and scalp transplantation. *Ann Plast Surg* 2003 May;50:535-40.
 39. Jones JW, Usturner ET, Zdichavsky M, et al. Long-term survival of an extremely composite tissue allograft with FK506-mycophenolate mofetil therapy. *Surgery* 1999; 126: 348-
 40. The first facial transplant. *Lancet* 2005;10[9502]:366.
 41. Siemionow M, Agaoglu G, Unal S. A cadaver study in preparation for facial allograft transplantation in humans: part II. Mock facial transplantation. *Plast Reconstr Surg* 2006;117:876-85.
 42. Jones JW, Gruber SA, Barker JH, Breidenbach WC. Successful hand transplantation. One year follow-up. *Louisville Hand Transplantation Team*. *N Engl J Med* 2000;342:468-73.
 43. Knechtle SJ, et al. Primate renal transplants using immunotoxin. *Surgery* 1998. 124:438-46.
 44. Auchincloss H Jr. In search of the elusive Holy Grail: the mechanisms and prospects for achieving clinical transplantation tolerance. *Am J Transplant* 2001;1:6-12.
 45. Morris P, Bradley A, Doyal et al. Transplantation 2007;83:109-28.
 46. Herndon DN. Perspectives in the use of allograft. *J Burn Care Rehabil*. 1997 Jan-Feb;18[1 Pt 2]:S6.
 47. Salma AD, Remuzzi G, Harmon WE, Sayegh MH. Challenges to achieving clinical transplantation tolerance. *J Clin Invest* 2001;108:943-8.
 48. Banks ND, Milner SM. Persistence of human skin allograft in a burn patient without exogenous immunosuppression. *Plast Reconstr Surg*. 2008;121:230e-1e.
 49. Wiggins OP, Barker JH, Martinez S et al. On the ethics of facial transplantation research. *Am J Bioeth* 2004;4:1-12.

THE TRI-SERVICE EMERGENCY MEDICINE CONFERENCE 2009

The Tri-Service Emergency Medicine Conference 2009 was held at HMS DRAKE in Plymouth from 9-11 June. Over 140 delegates from all three services attended this year's multidisciplinary event, with keynote speeches from Colonel Ian Greaves, Defence Consultant Adviser in Emergency Medicine, and Colonel Tim Hodgetts, Defence Professor of Emergency Medicine. The following abstracts were delivered as oral presentations during the conference.

Combat trauma survival: where is the proof of good outcomes?

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Aim: To quantify the number of unexpected survivors from major trauma who are treated within the UK military trauma system in Afghanistan and Iraq. **Population:** Survivors of major trauma [ISS \geq 16] or traumatic cardiac arrest ISS <16 recorded on the UK military's Joint Theatre Trauma Registry (JTTR) from 02 April 2006 to 30 July 2008. **Methods:** "Mathematical unexpected survivors" were identified from those who survived traumatic injury with ISS 60-75 and/or NISS 60-75 and/or TRISS Ps <50% and/or ASCOT Pd \geq 50% and/or documented cardiac arrest.

"Mathematical unexpected survivors" were subject to peer review. "Clinical unexpected survivors" were identified by independent peer review (expert panel of 3) of all additional survivors ISS \geq 16. These were characterised as either "civilian unexpected, but military expected" or "civilian and military unexpected" survivors to account for the raised expectations within the deployed military trauma system. **Results:** There were 1474 patients on UK JTTR; 530 were ISS \geq 16 and 296 were survivors. A total of 44 patients were "mathematical unexpected survivors" and 34 of these were validated by peer review. Within this group, there were 6 survivors from traumatic cardiac arrest. An additional 41 cases were identified as "clinical unexpected survivors"; 26 were "civilian unexpected, but military expected" and 15 were "civilian and military unexpected". The sensitivity of mathematical models to predict unexpected survivors of combat trauma is 45% [34/75];

the specificity is 77% [34/44]. Of the clinical unexpected survivor group, 36/41 [88%] had their unexpected outcome attributed to the advanced resuscitation strategies in the military to arrest and treat catastrophic haemorrhage following combat trauma. **Conclusions:** Mathematical modelling underestimates the unexpected survivors of combat trauma within the current UK military trauma system. Clinical peer review is essential if the value of advances in the military trauma system are to be recognised.

A comparison of civilian trauma standards [NCEPOD] with current practice in a deployed field hospital in Afghanistan

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Background: The National Confidential Enquiry into Patient Outcome and Death (NCEPOD) report on trauma management, published in 2007, defined standards for UK hospitals dealing with trauma. This study compared the NCEPOD standards with the performance of a UK military field hospital in Afghanistan. **Setting:** Role 2 (Enhanced) medical facility, Camp Bastion, Helmand Province, Afghanistan. **Materials and methods:** Data were collected prospectively for all patients fulfilling trauma team activation criteria during the 3 months of Operation HERRICK IXa (mid-October 2008 to mid-January 2009), and combined with a retrospective review of pre-hospital documentation, trauma resuscitation notes, operations notes and transfer notes for these patients. **Results:** During the study period there were 226 trauma team activations. Of those patients brought to the medical facility at Camp Bastion by UK assets 93.7% were accompanied by a doctor with advanced airway skills, although only 6.2% of patients required such an intervention. Consultants in emergency medicine and anaesthesia were present in 100% of cases, and were directly involved [in either leading the team or performing airway management] in 63.5% and 77.6% of cases respectively. 98.1% of those patients requiring operative intervention had this performed by a consultant surgeon. Of those patients requiring computed tomography, 93.6% of cases had this performed within 1 hour of arrival. **Conclusions:** Trauma patients presenting to the medical facility at Camp Bastion during Operation HERRICK IXa, irrespective of their nationality or background, received a high standard of medical care when compared to NCEPOD standards.

The Incidence of Heat Illness on UK Deployed Operations in Iraq and Afghanistan 2003-2008

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Objective: To establish the numbers and characteristics of heat illness patients on deployed operations since the start of war fighting in Iraq in 2003 and Afghanistan in 2006. **Methods:** A retrospective database analysis was performed on OpEDAR to identify numbers and outcome of patients with heat illness presenting to the deployed field Hospital during Operations TELIC and Operations HERRICK. **Data Sources:** The patient information regarding patient status, admission status, disposal and outcome, was gathered from the Operational Emergency Department Attendance Register (OpEDAR). This database records all patients who have attended the Emergency Department or who were admitted to a UK Operational Hospital via the Emergency Department. The OpEDAR database includes

all patients i.e. UK Service Personnel, all other NATO forces, civilians (UK and other nationalities) and detainees. **Results:** In Iraq and Afghanistan from 2003-2008, there were 1425 patients presenting to the Emergency Department with heat illness. Of these 1425 patients, 1301 [91.29%] were serving personnel, 27 [1.89%] were admitted to ITU/HDU, 218 [15.29%] were returned to the UK and in 88 [6.17%] the outcome was not documented. There were no recorded deaths from heat illness during this period.

The number and rate of heat illness patients are expressed per six month tour. During Op TELIC the total number of attendances for serving personnel ranged from 8-751 with a rate range of 0.8-60.1 per 1000 personnel. During Op HERRICK the number of attendances for serving personnel ranged from 2-49 with a rate range of 0.3-7.1 per 1000 personnel. **Conclusion:** Heat Illness remains a major problem for UK Service Personnel on deployed operations in hot climates and continues to pose a threat to operational effectiveness.

OpEDAR – analysis of data from Operation HERRICK

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Background: The Operational Emergency Department Attendance Register [OpEDAR] was started in February 2003; before this there was no audited record of activity within a deployed Emergency Department (ED) to guide realistic and contemporary assumptions for manning, equipment, organisational processes and training. OpEDAR was initiated as a hand-kept record in a paper register that has since evolved to the electronic register held at ADMEM today. Support from the Defence Analytical Services Agency (DASA) ensures validation of the data. **Methods:** OpEDAR was established at the Field Hospital in Helmand Province, Afghanistan (Operation HERRICK 4) from its inception. A retrospective database analysis was performed. **Results:** Data analysis to date shows over 11 000 attendances to the Field Hospital, 4.5% with ISS>16. Sixty two percent of attendances were UK military personnel, 12% local civilians. Twenty five percent of attendances were attributed to hostile intent. There has been variation in rate of UK military attendances but averaging about 140 attendances per 1000 troops deployed. Leading classifications were consistently orthopaedic soft tissue injury, gastrointestinal disease, and surgical conditions followed by orthopaedic fracture / dislocations, ophthalmology and musculoskeletal conditions. IEDs increasingly featured as cause of injury. **Discussion:** Information from analysis of OpEDAR is used to focus individual and collective training, guide planning and reinforce knowledge on injury patterns in current warfare. The future involves aiming for real-time projections of evolving cases, ensuring dedicated staff both on operations and at ADMEM for data completion and interpretation, and extrapolation of data analyses for use in operational planning of manpower and equipment.

Key events analysis from Operation TELIC.

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Background: Since the end of the war fighting phase of Operation TELIC in early 2003, British forces were engaged in stability operations in Southern Iraq until withdrawal in 2009. **Aim:** The aim of this paper is to examine the relationship between

key events during British military operations in Southern Iraq and battle casualty numbers. **Methods:** A retrospective database analysis was performed of the Operational Emergency Department Attendance Register (OpEDAR) and the major trauma registry (JTTR) at the Academic Department of Military Emergency Medicine. Data were gathered on all battle injuries from British Forces since the start of Op TELIC 1 to February 2009. Overall attendances and major trauma cases were examined independently. Non-battle injuries were excluded.

Results: The results are contained in Figure 1.

Conclusion: Higher battle injury numbers occurred during the war fighting phase [1] and the highlighted handover period [2]. Points 3-5 illustrate the fluctuating nature of unconventional warfare with peaks and lows corresponding to the highlighted events. Point 6 illustrates the burden of major trauma cases despite an overall downward trend in battle injury attendances. The continuous requirement for accurate data collection and its interpretation is reinforced and should be communicated to all levels of the chain of command. Key event analysis holds historic interest and is an aid towards epidemiological data collection. More detailed key events analysis could have future medical planning implications.

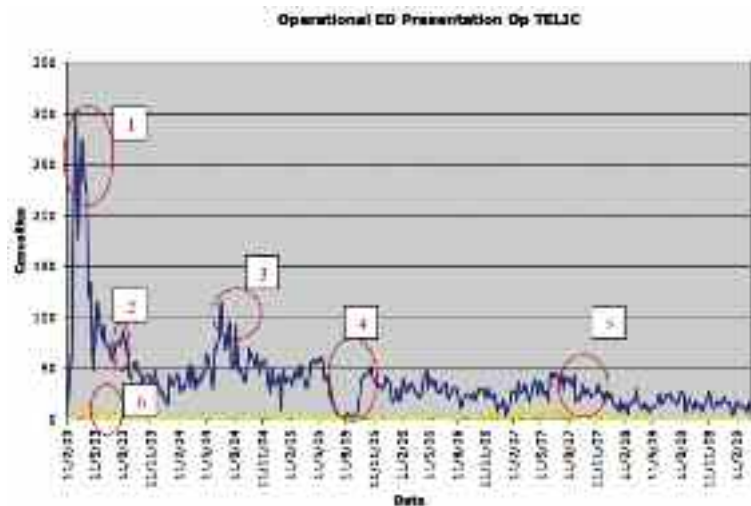


Figure 1: Operational ED presentation on OP TELIC Key: Blue – OpEDAR; Yellow – JTTR]. 1. February-May 2003 - war fighting phase. 2. July-August 2003 - 1 UK Armoured Division hand over to 3 [UK] Division in Basra. 3. June-August 2004 - sovereignty transfer from Coalition provincial authority to Iraqi government, the Defence of CIMIC House in Al Amarah, and the trial of Saddam Hussein begins. 4. June-November 2005 - few offensive operations, the Basra Prison incident, further activity in the Hussein trial, build up to Iraqi elections. 5. May-September 2007 - provincial handovers throughout MND(SE), handover of Basra Palace, Jaish Al Mahdi ceasefire. 6. June 2003 - closely spaced attacks in Majar Al Kabir.