

Treatment of white phosphorus and other chemical burn injuries at one burn center over a 51-year period[☆]

David J. Barillo^{a,b,*}, Leopoldo C. Cancio^a, Cleon W. Goodwin^{a,c}

^a US Army Institute of Surgical Research, 3400 Rowley Chambers Avenue Fort Sam Houston, San Antonio, TX 78234, USA

^b Shands Burn Center, University of Florida, Gainesville, FL, USA

^c The Johns Hopkins University, Baltimore, MD, USA

Accepted 21 January 2004

Abstract

Chemical burn injury meets the criteria of the American Burn Association for treatment at a specialized burn facility. Over a 51-year period, we have treated 276 patients with chemical burn injury including 146 white phosphorus injuries. In this study, we compare incidence, cause and outcome of chemical burn injury over time and review the management of white phosphorus injuries. *Methods:* Data for the period 1986–2000 was obtained by retrospective chart review. Data for the period 1950–1985 was obtained from previous studies and from retrospective chart review. *Results:* Chemical burn injury comprised 2.1% of all admissions between 1969 and 1985, and 2.07% between 1986 and 2000. The mean body surface area involved was 19.5% in the first 19 years of the study compared with 8.6% over the last 15 years. Mortality increased from 5.4% between 1950 and 1968 to 13.8% between 1969 and 1985. Mortality from 1986 to 2000 was 0%. Hospital length of stay decreased from a mean of 90 days in the first 19 years of the study to a mean of 15 days in the most recent 15-year period. The chemical responsible for injury was white phosphorus in 146 cases. *Conclusions:* Over time, the proportion of burn center admissions caused by chemical injury is constant, while the average total burn size, full thickness burn size, length of stay and mortality have decreased. During peacetime, the chemicals responsible are similar to those seen in civilian burn centers. The experience of this center with burns caused by white phosphorus is unique and needs to be maintained for future conflicts. Published by Elsevier Ltd and ISBI.

Keywords Chemical injury; Burn; White phosphorus

1. Introduction

Chemical burn injuries are those caused by exposure to acid, alkali or organic compounds. Burns caused by chemical exposure meet the criteria of the American Burn Association and the American College of Surgeons Committee on Trauma for referral to a burn treatment center [1,2]. The potential for chemical injury is large when one considers the number of chemical compounds that exist. Worldwide, there are at least 5–6 million known chemicals [3,4], with 10,000–20,000 new chemicals developed each year [5]. Annually, new development adds 1000–2000 chemicals to the existing 575,000 to 1.5 million chemical compounds present in the stream of commerce [3,5,6]. Of these, between 33,000

and 63,000 are classified as hazardous by one or more US government agencies [3,6]. There are approximately 4000 chemicals deemed both hazardous and used in sufficient quantity to make spillage likely. These chemicals are listed in an emergency responder guidebook published periodically by the US Department of Transportation to assist those called to mitigate spills of chemical product in transit [7]. Finally, there are over 300 common chemicals classified by the National Fire Protection Association as “extremely hazardous to health” or “too dangerous to health to expose fire fighters” [8].

Despite the large potential for injury, chemical burn injury appears to be either uncommon or under-referred. Several studies of chemical injury treated in burn centers indicate that chemical injury comprises only 2.1–6.5% of burn center admissions [6,9–11].

The US Army has maintained a burn research and treatment facility at Brooke Army Medical Center since 1947. As one of the oldest burn centers in continuous operation, the records of this center provide a large single-center ex-

[☆] Presented at the 11th Quadrennial Meeting of the International Society for Burn Injury, Seattle, WA, USA, August, 2002.

* Corresponding author. Present address: Department of Surgery, University of Florida, P.O. Box 100286, Gainesville, FL 32610-0286, USA. Tel.: +1-352-265-0262; fax: +1-843-881-9303.

E-mail address: barildj@surgery.ufl.edu (D.J. Barillo).

Report Documentation Page				Form Approved OMB No. 0704-0188	
Public reporting burden for the collection of information is estimated to average 1 hour per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden, to Washington Headquarters Services, Directorate for Information Operations and Reports, 1215 Jefferson Davis Highway, Suite 1204, Arlington VA 22202-4302. Respondents should be aware that notwithstanding any other provision of law, no person shall be subject to a penalty for failing to comply with a collection of information if it does not display a currently valid OMB control number.					
1. REPORT DATE 01 AUG 2004		2. REPORT TYPE N/A		3. DATES COVERED -	
4. TITLE AND SUBTITLE Treatment of white phosphorus and other chemical burn injuries at one burn center over a 51-year period.				5a. CONTRACT NUMBER	
				5b. GRANT NUMBER	
				5c. PROGRAM ELEMENT NUMBER	
6. AUTHOR(S) Barillo D. J., Cancio L. C., Goodwin C. W.,				5d. PROJECT NUMBER	
				5e. TASK NUMBER	
				5f. WORK UNIT NUMBER	
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES) United States Army Institute of Surgical Research, JBSA Fort Sam Houston, TX 78234				8. PERFORMING ORGANIZATION REPORT NUMBER	
9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)				10. SPONSOR/MONITOR'S ACRONYM(S)	
				11. SPONSOR/MONITOR'S REPORT NUMBER(S)	
12. DISTRIBUTION/AVAILABILITY STATEMENT Approved for public release, distribution unlimited					
13. SUPPLEMENTARY NOTES					
14. ABSTRACT					
15. SUBJECT TERMS					
16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT SAR	18. NUMBER OF PAGES 5	19a. NAME OF RESPONSIBLE PERSON
a REPORT unclassified	b ABSTRACT unclassified	c THIS PAGE unclassified			

perience with chemical injury, as well as an opportunity to evaluate change in incidence, cause and outcome over time. In this report, we detail the chemical burn experience of one center over a 51-year period.

2. Materials and methods

Two previous studies from this institution have addressed chemical burn injury. The first, published by Curreri et al. in 1970, documented experience with 111 patients treated for chemical burn injury between 1950 and 1968 [12]. The second, published by Mozingo et al. in 1988, documented treatment of 87 patients with chemical burn injury treated between 1969 and 1985 [13]. Data from these manuscripts, along with additional retrospective chart review was utilized in the present study.

For the years 1986–2000, retrospective chart review was undertaken of 3764 consecutive acute burn admissions. Data abstracted included demographics, chemical agent responsible, and measures of outcome. This study was approved by the Brooke Army Medical Center Institutional Review Board.

3. Results

Over a 51-year period, a total of 276 patients with chemical burn injury were admitted. Comparison of incidence, burn size and outcome is presented in Table 1.

The treatment of chemical burn injury at the Institute of Surgical Research between 1950 and 1986 has been previously described [12,13]. Between 1950 and 1968, the mean burn size was 19.5% total body surface area (TBSA) with a mean full thickness burn area of 10.5% and mean length of stay of 90 days. The mortality rate for patients admitted with chemical burn injury was 5.4%. The majority of patients treated for chemical burn injury within this timeframe sustained injuries related to combat, and 59 associated traumatic injuries were reported in this group. [12].

Between 1969 and 1985 there were 87 patients admitted, with a mean burn size of 25.4% TBSA and a mean full thickness burn injury of 13.8%. The mortality rate was 13.8%. The mean length of stay for patients with chemical burn injury was 67 days. A number of these patients were

combat casualties and a total of 35 associated injuries were diagnosed in this group [13].

Between the years 1986 and 2000, there were 78 patients admitted with chemical burn injury. The mean burn size was 8.6% TBSA with a mean full thickness burn of 2.06% TBSA. Mean length of stay was 15 days. There were no fatalities. In contrast to previous timeframes, only two injuries were war-related, both occurring during Operation Desert Shield/Desert Storm.

The chemicals responsible for burn injury are reported in Table 2. During the first 19 years of this study, 99 of 111 burn admissions (89.1%) resulted from contact with munitions, mostly white phosphorus. In the subsequent 17 years, 49 of 87 admissions (56.3%) were caused by chemical munitions. In the final 15 years, only three munitions-related chemical burns were admitted (3.8%).

Table 2

Category	Chemical	1950–1968	1969–1985	1986–2000	Total
Munition	White phosphorus	96	49	1	146
	Red phosphorus	0	0	1	1
	Mustard	3	0	0	3
	Other	0	0	1	1
Acid	Sulfuric	5	10	10	25
	Hydrofluoric	0	0	4	4
	Hydrochloric	0	1	2	3
	Nitric	0	0	2	2
	Acetic	0	0	2	2
	Formic	0	2	0	2
	Trichloric	0	0	1	1
	Other acid	0	0	3	3
Alkali	Lye/NaOH	3	5	10	18
	Cement/lime	0	0	5	5
	Calcium chloride	0	0	1	1
	Aluminum hydroxide	0	0	1	1
	Ammonia	0	1	2	3
	Bleach/chlorine	0	0	5	5
	Other alkali	0	4	6	10
Other	Gasoline	0	1	1	2
	Kerosene	0	0	1	1
	Diesel	0	0	1	1
	Oven cleaner	0	0	5	5
	JP4 fuel	0	0	1	1
	Phenol	0	2	2	4
	Fluorocarbon	0	2	1	3
	Paint thinner	0	1	0	1
	Triethylene glycol	0	1	0	1
	Sodium nitrate	0	2	0	2
	Magnesium	0	0	1	1
	Difluoroethane	0	0	1	1
	Other	4	6	7	17
Total		111	87	78	276

Table 1

	1950–1968	1969–1985	1986–2000
Number of patients	111	87	78
Acute admissions (%)	Not known	2.1	2.1
Mean burn size (%TBSA)	19.5	25.4	8.6
Mean full thickness burn (%)	10.5	13.8	2.1
Mean length of stay (days)	90	67	15
Mortality (%)	5.4	13.8	0

4. Discussion

Over a 51-year period at one burn center, the proportion of admissions resulting from chemical burn injury has remained constant, while the mean burn size, full thickness area, length of stay and mortality has decreased. The decreases in mortality and length of stay likely reflect general improvements in burn care over time. The constant and low percentage of admissions resulting from chemical injury is in agreement with other studies. The reason for this low percentage is unclear, but probably reflects under-referral of patients with chemical burn injury to specialized centers.

The chemicals responsible for burn injury have likewise changed over time. In the first timeframe, 89% of the chemicals were munitions such as white phosphorus and mustard gas. In the last 15 years of the study, the chemicals responsible for injury were similar to those seen in civilian practice and in other studies of chemical burn injury [9–11,14]. During this same timeframe, the US Environmental Protection Agency conducted one large-scale study of the chemicals encountered in hazardous materials spills [3]. Of 6928 nationwide incidents involving chemicals other than fuel, 10 chemicals were found to be responsible for 49.5% of all spills and 35.7% of all injuries (Table 3). These 10 and similar compounds were responsible for 31 of the 74 non-fuel chemical burn admissions to the Army Burn Center in the last 15 years of the study.

One difference between this and other studies of chemical burns is the high incidence of white phosphorus injuries. White phosphorus is an element of molecular weight 123.9. While thought of primarily as a munition, white phosphorus is also encountered in civilian practice as a component of fertilizers, insecticides, rodenticides and fireworks.

White phosphorus has a low melting point and converts from a solid to a liquid at 111 °F (44 °C) [8]. The auto-ignition temperature (temperature at which combustion can occur in the absence of an ignition source) is 86 °F (30 °C) [8]. Above the auto-ignition temperature, white phosphorus particles spontaneously ignite (oxidize) on contact with air, forming phosphorus pentoxide. In wounds,

particles of white phosphorus continue to oxidize until debrided, neutralized or consumed, producing a yellow flame with white smoke. Mendelson points out that white vapor issuing from a wound does not necessarily indicate ignition, but does point to ongoing formation of phosphoric acid, which must be stopped [15,16]. Because of the low melting point, wound irrigation with warm water facilitates conversion from solid to liquid and increases the fire risk as the auto-ignition temperature is reached [12,16]. The liquid form is also harder to recognize or remove. Ignition is prevented by excluding oxygen. When white phosphorus particles are embedded in a burn wound, the wound and particles should be kept wet with sterile saline or water. Once removed, particles will re-ignite if allowed to dry.

White phosphorus injury may produce profound physiologic changes. Life-threatening hypocalcemia and/or hyperphosphatemia can occur in certain individuals as early as 1 h post-burn [17]. Sudden and unexpected death can occur from burns of 10–15% TBSA [17,18]. There is no reliable predictor of who is at risk for electrolyte abnormalities [15]. Patients with white phosphorus injury should be monitored by electrocardiogram until electrolyte disturbances have been ruled out. Severe hypocalcemia may cause prolongation of QT interval, ST segment depression, T wave changes, progressive bradycardia, or sudden death [17–20]. Serum calcium and phosphorus levels should be monitored for at least 48–72 h [20,21].

White phosphorus injury produces a combined chemical and thermal burn (Fig. 1). The substance is highly fat-soluble and absorption may result in hepatic necrosis or renal damage [19,22]. Tissue damage also occurs secondary to the corrosive action of phosphoric acids (which form during combustion), from the heat of the chemical reaction producing phosphorus pentoxide, and from the hygroscopic actions of the phosphorus pentoxide itself [12,18,22]. White phosphorus burns take longer to heal than other forms of thermal injury [12,18,19]. Many white phosphorus injuries result from explosion of munitions, and associated injuries are common.

The initial management of white phosphorus burns is to stop the burning process. Wounds should be thoroughly irrigated and then covered with saline or saline-soaked pads. In animal studies, vigorous water irrigation of white phosphorus wounds proved superior to topical treatment with water soaked dressings, Water-Gel (Trilling Medical Technologies, Ridgefield Park, NJ), Kaltostat (BritCair Ltd., Aldershot, UK), 3% copper sulfate solution, copper sulfate emulsion, 0.2% KMnO₄ solution or intraperitoneal or intralesional superoxide dismutase injection [23]. Visible particles of white phosphorus should be removed and placed in cold water to prevent re-ignition [12]. Immediate surgical debridement is often necessary and is followed by repeated operative procedures until all phosphorous particles have been removed. Debrided wounds should be examined at least twice daily for new particles or smoking areas, which would indicate the need for re-operation [21]. Debrided areas can be covered in aqueous 5% mafenide acetate solution between operative

Table 3

Ten chemicals responsible for 49.5% of 6928 non-fuel hazardous material spills us environmental protection study

Chemical	Percentage of injuries	Percentage of spills
Chlorine	9.6	3.5
Anhydrous ammonia	6.8	3.7
Hydrochloric acid	5.6	3.1
Sulfuric acid	4.7	6.5
Polychlorinated biphenyls	2.8	23
Toluene	2.4	1.4
Sodium hydroxide	1.9	2.6
Nitric acid	1.5	1.7
Methyl alcohol	0.4	1.7
Methyl chloride	0.1	1.4
Total	35.7	49.5



Fig. 1. White phosphorus injury.

procedures to facilitate examination [21]. Definitive wound closure should be deferred until adequacy of debridement is assured, at which point split thickness skin grafts can be applied.

Considerable confusion remains regarding the role of topical copper sulfate solution in the treatment of white phosphorus burns. This practice evolved over 100 years ago and was resurrected during the Vietnam era as a method of identifying phosphorus particles in the wound [22]. Copper sulfate solution is not an antidote or neutralizing agent for white phosphorus injury [15], but rather, facilitates debridement by turning embedded phosphorus particles black. Unfortunately, this solution is easily absorbed through the burn wound. Absorbed copper can cause fatal massive intravascular hemolysis, hematuria, oliguria, acute renal failure, cardiovascular collapse and death [18,21,22]. Modifications of 5% copper sulfate solution to improve safety or efficacy have been advocated. These include decrease of concentration to 3, 1 or 0.5%, addition of 3 or 5% bicarbonate solution, addition of 1% hydroxyethyl cellulose or lauryl sulfate, avoidance of copper-sulfate soaked pads, and use of prompt water or saline rinse following application [19–22,24]. These modifications are largely unproven. One to three percent solutions of silver nitrate have also been utilized to identify embedded phosphorus particles in wounds, and may be safer than copper sulfate solution for this purpose [25].

Embedded white phosphorus particles are best identified using a Wood's lamp, which will cause retained particles to fluoresce [13,18,21]. In our opinion, copper sulfate so-

lution has no place in the contemporary management of white phosphorus burns, a point previously made in 1967 by Summerlin et al. [22]. Davis notes that 'rapid decontamination with water and rapid surgical debridement obviate the need for decontamination with copper-containing compounds' [18].

References

- [1] Committee on Trauma, American College of Surgeons. Advanced trauma life support program for physicians. Chicago: American College of Surgeons; 1997.
- [2] American Burn Association: advanced burn life support course. Chicago: American Burn Association; 2001.
- [3] Keffer, WJ. Beginning the hazard analysis process. In: Henry, M, editor. Hazardous materials response handbook. Quincy, MA: National Fire Protection Association; 1989.
- [4] Bowen, JE. Emergency management of hazardous materials incidents. Quincy MA: National Fire Protection Association; 1995.
- [5] Fire, FL. The common sense approach to hazardous materials. New York: Fire Engineering Inc.; 1986. p. xxvii.
- [6] Winfree J, Barillo DJ. Nonthermal injuries. *Nurs Clin North Am* 1997;32:275–96.
- [7] US Department of Transportation, Research and Special Programs Administration: 2000 emergency response guidebook.
- [8] National Fire Protection Association: fire protection guide to hazardous materials. 10th ed. Quincy MA: National Fire Protection Association; 1991.
- [9] Li YY, Liu Y. Analysis of 155 patients with chemical injury: a 5 year experience. *Burns* 1993;19:516–8.
- [10] Singer A, Sagi A, Ben Meir P, Rosenberg L. Chemical burns: our 10 year experience. *Burns* 1992;18:250–2.

- [11] Sykes RA, Mani MM, Hiebert JM. Chemical burns: retrospective review. *J Burn Care Rehabil* 1986;7:343–7.
- [12] Curreri PW, Asch MJ, Pruitt Jr BA. The treatment of chemical burns: specialized diagnostic, therapeutic, and prognostic considerations. *J Trauma* 1970;10:634–41.
- [13] Mozingo DW, Smith AA, McManus WF, Pruitt Jr BA, Mason Jr AD. Chemical burns. *J Trauma* 1988;28:642–7.
- [14] Moran KD, O'Reilly T, Munster AM. Chemical burns a ten year experience. *Am Surg* 1987;53:652–3.
- [15] Bellamy RF. Willie Peter. *Mil Med* 1988;153:76–9.
- [16] Mendelson JA. Some principles of protection against burns from flame and incendiary munitions. *J Trauma* 1971;11:286–94.
- [17] Bowen TE, Whelan TJ, Nelson TG. Sudden death after phosphorus burns. *Ann Surg* 1971;174:779–84.
- [18] Davis KG. Acute management of white phosphorus burn. *Mil Med* 2002;167:83–4.
- [19] Konjoyan TR. White phosphorus burns: case report and literature review. *Milit Med* 1983;148:881–4.
- [20] Chou TD, Lee TW, Chen SL, Tung YM, Dai NT, Chen SG, et al. The management of white phosphorus burns. *Burns* 2001;27:492–7.
- [21] Kaufman T, Ullman Y, Har-Shai Y. Phosphorus burns: a practical approach to local treatment. *JBCR* 1988;9:474–5.
- [22] Summerlin WT, Walder AI, Moncrief JA. White phosphorus burns and massive hemolysis. *J Trauma* 1967;7:476–84.
- [23] Eldad A, Wisoki M, Cohen H, Breiterman S, Chaouat M, Wexler MR, Ben-Bassat H. Phosphorus burns: evaluation of various modalities for primary treatment. *J Burn Care Rehabil* 1995;16:49–55.
- [24] Stewart CE. Chemical skin burns. *Am Fam Physician* 1985;31:149–57.
- [25] Zong-yue S, Yao-ping L, Xue-qi G. Treatment of yellow phosphorus skin burns with silver nitrate instead of copper sulfate. *Scand J Work Environ Health* 1985;11(Suppl 4):33.