

Basic of fluid considerations in neonates with burn – Current perspectives

Pramod Gupta^{1*} and Ridhima Sharma²

^{1,2}Department of Anesthesia, Safdarjung Hospital / Vardhman Mahavir Medical College, New Delhi, India

Abstract

The uniqueness of the neonate physiology and the peculiarities in the various body compositions indicate the need of special emphasis on the management of accidental burns in this vulnerable cohort. Neonatal burns trigger a sequel of pathophysiological response resulting in an additional detrimental outcome. Out of the total burns admission in India, pediatric burn, is 17-25 % [1]. Specific concerns in pediatric patients include disproportionately thin skin, three times the body surface area to body mass ratio than adults requiring aggressive fluid losses resuscitation. Although, burn in neonate is rare, despite there is literature published that documents the increasing trend in recent years. Furthermore, because of the rarity of thermal injury, there exist no robust guidelines regarding the choice of fluid management.

The current review is intended to highlight inherent challenges in terms of fluid choices in the resuscitation of neonatal burn.

Keywords: Burns; Neonates; Fluid; Resuscitation; Fluid Guidelines in Neonates;

Search strategies

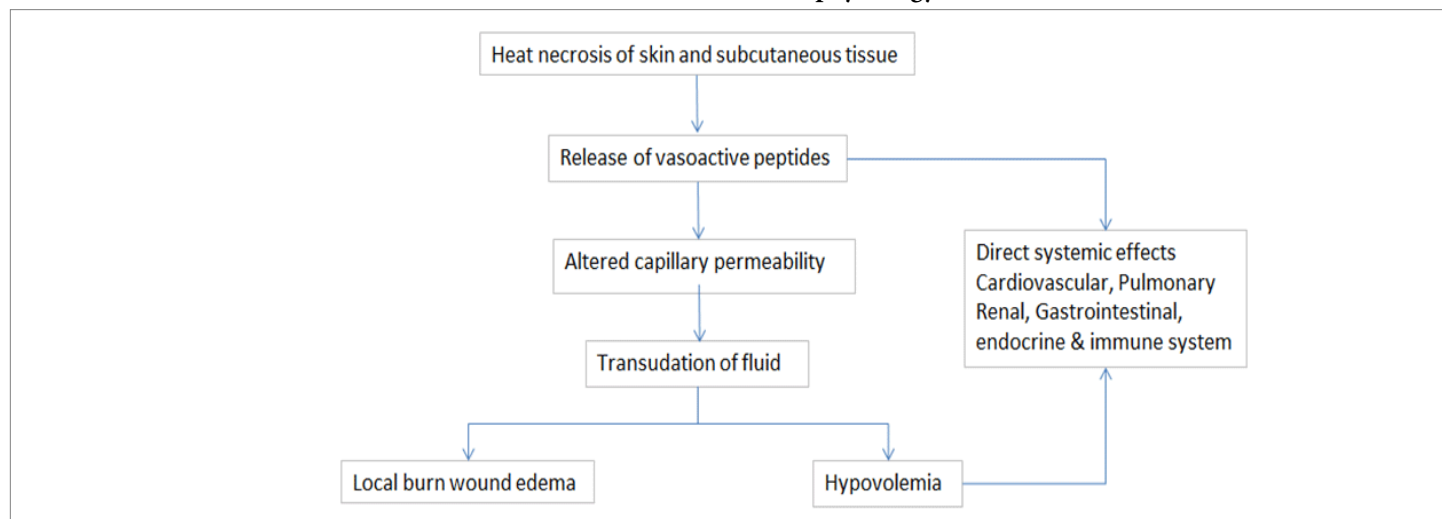
Caudal block is useful as an adjunct during general anaesthesia and for providing post-operative analgesia after infraumbilical operations [1]. Fentanyl has been widely used as an analgesic adjuvant to epidural analgesia. However it has undesirable side effects as respiratory depression, itching and vomiting [2,3]. Nalbuphine has relatively potent μ -antagonist and κ -agonist activity. The respiratory depression induced by opioids is primarily mediated by the μ -receptor agonist activity. The μ -antagonist properties of the nalbuphine produce fewer μ -mediated side effects such as respiratory depression, pruritus, nausea and vomiting [4-6]. In addition, presently there are no studies reporting the efficacy of caudal nalbuphine

versus fentanyl. The aim of this prospective, randomized, double-blind study, therefore, was to investigate the non-inferiority of nalbuphine to fentanyl and to assess efficacy and side-effects of caudal nalbuphine as an adjuvant by single shot technique in children.

Epidemiology

Burn associated mortality in children rank third among 1 to 9 years of age [2]. One study showed no survivors in 112 children younger than 3 years with more than 60% burns [3]. Benmeir, et al. concluded triad of risk factors, victim, cause and conditions to be kept away from each other [4].

Pathophysiology of Burn



Received date: April 06, 2018; Accepted date: June 25, 2018; Published date: July 06, 2018.

*Corresponding Author: Prof. Pramod Gupta, Department of Anesthesia, Safdarjung Hospital / Vardhman Mahavir Medical College, New Delhi, India, E- mail: guptapdnb@yahoo.co.in

Citation: Pramod G, Ridhima S, (2018) Basic of fluid considerations in neonates with burn – Current perspectives. J Anesthe Advan Res 1(1)

Copyright: © 2018 Pramod G. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Criteria for admission

- More than 10% TBSA burn in a child
- Full thickness burns (> 2% TBSA)
- Very young child
- Burns involving hands, feet, face and perineum
- Inhalational/ Chemical
- Suspicion of child abuse
- Circumferential extremity burns.

Citation: Pramod G, Ridhima S, (2018) Basic of fluid considerations in neonates with burn – Current perspectives. J Anesthe Advan Res 1(1)

Antibiotic Prophylaxis

Sepsis with burn wound infection can be a fatal condition in a child [5]. Immediately wound swab, blood culture and sensitivity should be done and appropriate antibiotic cover to be started as soon as possible. In case of refractory shock, colloid / albumin up to 20 ml/kg and inotropes can be started according to the clinical scenario.

The pathogen that can be the cause of burn wound infection with evidence

The hallmark of thermal injury is the breached skin, due to the thermal injury a state of immunosuppression is noted predisposing the patient to infectious complication. The interruption of systemic and local humoral and host cellular immune response are the cardinal factors contributing to infection in patients of severe burn [1]. Immediately burn surfaces are sterile, then also later on to become colonized with microorganisms [2]. Initially Gram-positive bacteria (staphylococci) colonize the wound surface within 48 hrs and subsequently with other microbes (Gram-negative bacteria, yeast from hosts normal respiratory flora and gastrointestinal tract, also from the health care personnel and hospital environment within 5-7 day [3].

Group A beta Hemolytic streptococci (*Streptococcus pyogenes*) was the predominant pathogen before the start of antibiotic era, and could lead to a devastating catastrophic sequel leading to high rates of mortality [4]. After the introduction of Penicillin G, *Staphylococcus aureus* became the predominant organism [5].

Pseudomonas aeruginosa is the most common cause of wound infection derived from environmental source or from patient gut flora. Less common infection encountered are due to anaerobic bacteria electrical burns or due to open wound dressings [6].

Due to the broad spectrum systemic and topical antibiotics fungal infections (*Candida* spp) and other opportunistic pathogen have been emerging as serious pathogen, also MRSA (methicillin-resistant Coagulase-negative staphylococci, vancomycin-resistant enterococci, and various resistant gram-negative bacteria have been seen in burn wound infection [7].

Empirically Antibiotics before Cultural test

Effectual topical antimicrobial therapy consequently reduces the risk of infection and microbial load. Topical antimicrobial should be applied first to the patients' dressings to reduce contamination by burns, wound flora that eventually prevent infection and conversion to full thickness from partial thickness wounds.

Silver nanocrystalline dressings (bactericidal) based on silver ion inhibitory action and interaction with thiol groups [8], interact with structural proteins and bind to the DNA nucleic acid base to prevent interaction. [9] {Also silver is highly toxic to keratinocytes and fibroblast, may delay wound (burn) healing if applied to healthy tissue area [10]. Recently, Moist exposure therapy using a moisture-retentive ointment has been used as an effective antibacterial agent, causing autolysis debridement and moist wound (optimal) healing in partial thickness wound, resulting in decreased scar formation and improved healing [11]. Silver nitrate ointment has bacteriostatic activity against *Pseudomonas aeruginosa* and *Escherichia coli*, also has less antifungal activity (nystatin to be used simultaneously) [12]. Silver sulfadiazine contains sodium sulfadiazine and silver nitrate. Silver ion binds to nucleic acid of microorganisms, interfering with microbe's metabolism by releasing sulfadiazine [13].

Mafenic acetate cream (topical) can be applied on open wound after cleansing the debris. It is (Sulfamylon) cream has a broad spectrum activity against particularly *Pseudomonas aeruginosa*, *Staphylococcus aureus* (to

a lesser extend), *Clostridium* spp. It favors of the fungi (*candida albicans* and other sp) hence to be used concomitant with nystatin. Mafenic acid compound is converted to *p*-sulfamylvanzoic acid by monoamide oxidase, (carbonic anhydrase inhibitor) causing metabolic acidosis in the burn patient. Acticoat A.B. dressing/Silverlo is a specialized dressing that consists of two sheets of high-density polyethylene mesh coated with nanocrystalline silver [14]. Mupirocin (pseudomonic acid A) is a fermentation product of *Pseudomonas fluorescens*, has potent inhibitory activity against gram-positive skin flora such as coagulase-negative staphylococci and *Staphylococcus aureus*, including MRSA [15].

Nystatin is produced by *Streptomyces noursei* and has potent antifungal agent by binding to the sterols in the fungal cell membrane [16].

Systemic Antibiotics

In a study of 77 pediatric burn patients, 47 received prophylactic antibiotics and the rest no prophylaxis, the administration of antibiotics did not prevent the burn wound infection. The group of children that received prophylactic antibiotics had a higher burn wound infection rate (17) (21.3% versus 16.7%). Systemic antibiotic therapy may also cause antibiotic-associated diarrhea due to the overgrowth of toxigenic strains of *Clostridium difficile* [18]. The liberal use of prophylactic antibiotics can cause other secondary infections also increase the resistance of endogenous and pathogenic bacterial culture should be used as a guide for the selection of effective antimicrobial agents for the treatment of overt clinical infections [19].

The different kind of incident can be more aggressive for neonate to get severe burn with any correlation with TBSA

The neonate skin has less keratinization and is relatively thinner and more prone to thermal insults, resulting in full thickness burns. Uguro AO et al. [20] reported 21 neonatal burn injuries, all resulting from domestic accidents, with flame burns, scalds and chemical burns and have documented a mean TBSA of $26.00 \pm 5.53\%$ in their series. Cox SG et al. [21], in a review of 37 years, reported 86 neonates under 4 months of age, with scalds, flame burns and 2 cases of burns due to primitive heating devices, Reported a mean TBSA of 11.5%, with 62.7% of the neonates having had less than 10% TBSA involvement.

In the study of 11 neonates [22] (aged ranged from 01 day to 28 days), TBSA burnt ranged from 3%-55% with a mean of $18.72 \pm 17.13\%$. The lowest TBSA involved was observed in a neonate with scald burns to the face, while the highest TBSA involved was found in a neonate injured by an accidental fall on a room heater. Among the body areas affected, the most common was face/head and neck. Rimdeika R, et al. [23] has reported one neonate with 20% TBSA burns and another with 14% TBSA involvement. There are various mechanisms of iatrogenic injury, have been reported from developed countries regarding causes of neonatal burn, including

- Warm baths [24]
- Devices for keeping babies warm [25]
- Topical disinfectants such as chlorhexidine and alcohol, [21,22] malfunctioning pulse oxymeters [26]
- Laryngoscopes [27]
- Phototherapy [28]
- Infra-red heating lamps, alcohol lamps defective transillumination device [29]
- Monitoring devices such as temperature probes, electrode jelly [30]

Fluid resuscitation

The basic norms of resuscitation are similar that of adults recognizing the fact that “children are not small adult”. Even with half an hour delay in resuscitation due to failure to recognize the severity and size of the burn can lead to increased mortality [6].

Larger burns (>15%TBSA) can initiate systemic inflammatory syndrome that necessitate immediate i.v fluid resuscitation to prevent burn shock and mortality. Earlier the same formulas were used in burned children as an adult with comparatives “smaller quantities”. Due to peculiarities in neonates physiology, including large body surface area than adult it was of paramount importance to have specific formulas.

Kyle and Wallace elucidated the first distinct formulas for pediatric burns [9]. Their approach was based on percentage of total burn surface area, age and weight specific requirement and depth of burn.

- **Eagle [10]** discovered the formula using body surface area, in 48 h of injury, i.e. 30ml/%TBSA burn plus 10% of body weight (kg) plus 4000ml/m² BSA of o. 66 normal saline along with 5% dextrose and 20g of albumin/liter. In current scenario, Cincinnati and Galveston formulas are used in pediatric burns.
- **Cincinnati** is akin to parkland with prepending maintenance fluid calculation depending on body surface area [11]. The formula is 4ml/kg/%TBSA burn plus 1500ml/m² total BSA over 24 h. The first 8h fluid composition was lactated Ringers with 50mEq of sodium bicarbonate. In the second 8h only lactated Ringer and in the next 8 h, 12.5 g of 25% albumin per liter plus lactated Ringer
- **Galveston** formula [12] relies on BSA, provides resuscitation fluid (5000 ml/m² BSA burn) and maintenance fluid (2000 ml/m² total BSA). The fluid composition includes lactated Ringer’s solution with 12.5 g of 25% albumin per liter plus 5% dextrose as required. Half is given in first 8h and rest over the next 16h.
- **Parkland formula** is based on burn percentage and is used in 78% of burn unit in United Kingdom, Ireland and Canada, according to the survey done recently [13]. The formula provides maintenance requirements PLUS burn requirements of 4 ml/kg/% TBSA burn.

Fluid Creep

Pruitt¹⁴ contrived the term “fluid creep”, patients with major burns receive excessive fluid than recommended by Parkland formula and is generally associated with many complications including, acute respiratory distress syndrome (ARDS) increased risk of infectious complications, abdominal compartment syndrome and death.

- Brooke’s formula: maintenance fluids PLUS 2–3 ml/kg/% TBSA burn for children <20 kg.
- Warden [15] amended the parkland formula for children with inclusion of maintenance fluid ensuring adequate resuscitation. It provided the total requirement for first 24 hours = 4 ml/kg/% TBSA + 1500 ml/m² BSA, half in first 8hrs and rest in half over the next 16 hrs.

Tug of War - Over the Preferred fluid

Although, there exists no level 1 or 2, class of publication regarding the absolute choice of fluid, traditional dogma have preferred Isotonic solutions such as Hartmann’s, Lactate Ringers or normal saline. Despite being preferred Lactate ringer has its own unique pitfalls, racemic mixture of d-lactate and l-lactate isomers in ringer lactate are accountable for increased production of reactive oxygen species (ROS) [19].

According to one survey, most frequently used fluid for burn resuscitation in UK, Ireland is Hartmann’s solution [18] (75% paediatric unit) another study, conducted in the USA and Canada showed Ringer

lactate to be used commonly [19].

A multicentre trial in 66 neonates used BS-G1 isotonic balanced solution at the rate of 10ml/kg/h (according to blood glucose levels), demonstrated no episodes of hypernatremia or hyponatremia [20].

Role of glucose containing solutions- Hypoglycemia versus Hyperglycemia

Neonate ability to utilize glucose as well as ketones as substrate in brain prevent hypoglycemic damage, however, due to sparse glycogen stores, prolonged hypoglycemia (2.6mmol/l) can lead to deleterious neurological outcomes, as a result of not giving glucose as a part of resuscitation fluid in burn neonates.

In hypoglycemic prone infants, glucose infusion (120-300 mg/kg/h) is adequate to perpetuate glucose level and prevent mobilization of lipid [21].

In view of the cataclysmic sequel of hypoglycemia in comparison to hyperglycemia, glucose concentration in maintenance solutions to be decreased cautiously in neonates resuscitation fluid.

Colloid

The use of colloid in immediate post burn in neonate is contentious. An RCT (randomized controlled trial) advocated that saline is equally effective as 5% albumin and decreases fluid retention in the initial 48h in premature infant [22]. In a radioisotope studies conducted by Baxter and shires [23] challenged the use of colloid in an early phase and found that plasma expansion was unhampered of the type of fluid used, as the capillary integrity is inadequate to permit the colloid impact the oncotic pressure (intravascular).

The literature supporting the use of starch containing solution in neonates is not adequate to establish any long term safety outcome. Lawrence [24] et al suggested that the incorporation of the colloid to parkland formula restored the normal resuscitation ratio and ameliorated fluid requirement (hourly) and fluid creep.

The current recommendation is to bound the use of dextran to 20mg/kg/day in pediatric group [22], although there exist no clear cut guidelines for its use in neonates.

The Association of Paediatric Anaesthesia (90%) and the French-language Society of Paediatric Anaesthesiologists (81%) recommended albumin during perioperative fluid losses as a replacement fluid in term and pre-term neonates [26]. A literature search was performed in the PubMed database using search term *neonates burn, children fluid resuscitation*, consideration in burn pediatric cohort. More than 30 articles were selected for review, the majority of pediatric anesthesia, general anesthesia, and general emergency medicine literature. Five articles specifically addressed in pediatric neonate burns resuscitation and fluids. Due to the relatively recent implementation, the majority of literature included is from the previous decade. The measures adopted included extensive scrutiny of literary evidence from internet resources, journals and textbooks of pediatrics, plastic surgery, fluid management, anesthesiology, and intensive care. The strategies included exploration of full text articles and abstracts from various search engines such as PubMed, Medscape, Google Scholar, Medline, Scopus, Science Direct, Yahoo and many others.

Adequate Resuscitation

The most commonly used Terminus for resuscitation is urine output in neonates with the aim of 1-1.5 ml/kg/h and any variation (increase or decrease) in urine output require adjustment in fluid resuscitation. The ventilation –induced variation in aortic flow velocity measured by

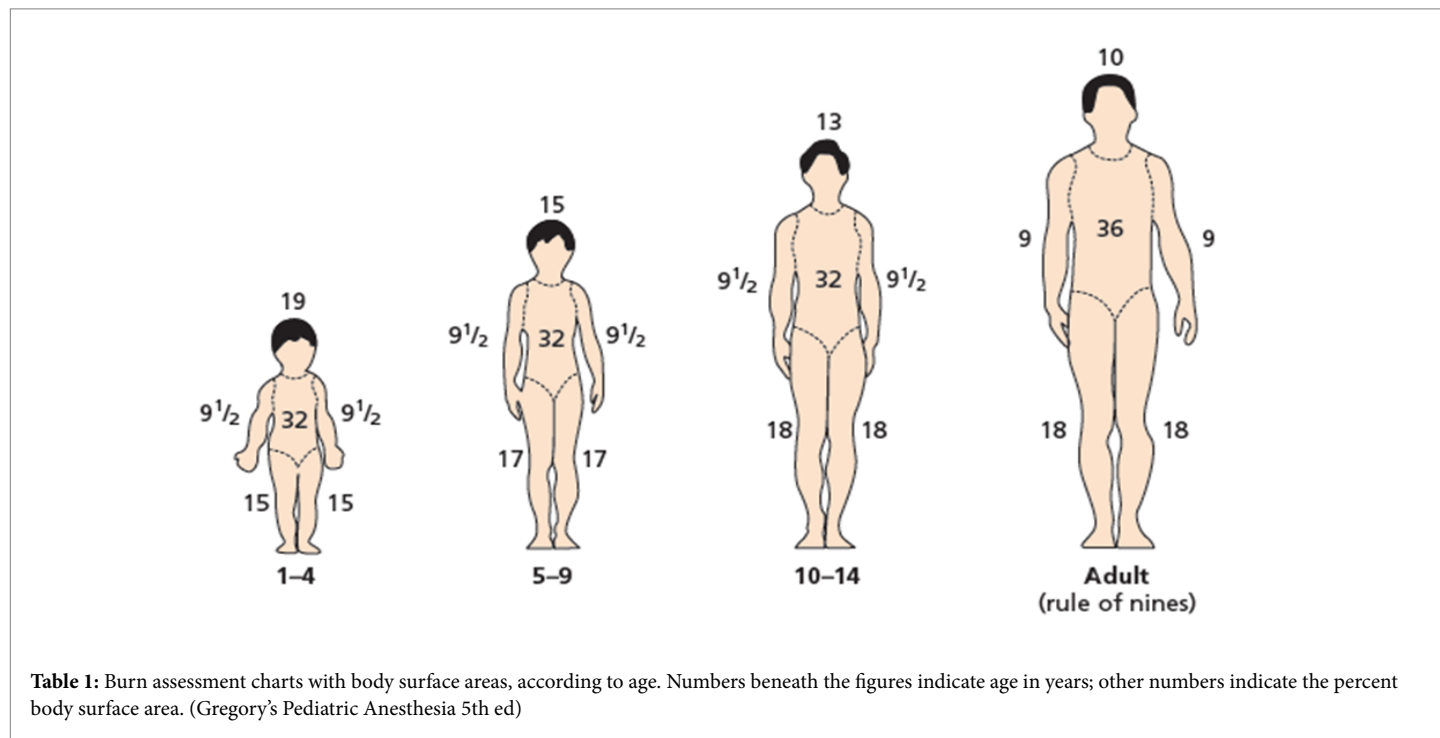
transthoracic/oesophageal echocardiogram is the most useful parameter in predicting fluid responsiveness in neonates.

Take Home Message

Neonates Do Not Follow- Rule of nine

In neonates, the head and neck, body surface areas are large when

compared to adults, and this necessitates some modifications in rule of nine for determining burn size. Lund and browder⁹ provide a precise formula for calculating the percentage of body surface area, also exist the percentage of body surface area according to age. (Table 1, 2)



Newborn	
Head	18%
Trunk	40%
Arms	16%
Legs	26%

Conclusion

Prompt resuscitation is critical in neonates and can decrease complications and mortality although multiple fluids and regimens available for infants and children can be misleading, clinical endpoints alteration of fluid is the mainstay of resuscitation. Maintenance fluid to be given in addition to neonates in addition to calculated fluid in burn resuscitation.

References

- Alexander, J. W. Mechanism of immunologic suppression in burn injury. *J Trauma*. 1990;30:S70-S75.
- Barret, J. P., and D. N. Herndon. Effects of burn wound excision on bacterial colonization and invasion. *Plast Reconstr Surg*. 2003;111:744-750. doi: 10.1097/01.PRS.0000041445.76730.23
- Altoparlak, U., S. Erol, M. N. Akcay, F. Celebi, and A. Kadanali. The time-related changes of antimicrobial resistance patterns and predominant bacterial profiles of burn wounds and body flora of burned patients. *Burns*. 2004;30:660-664. doi: 10.1016/j.burns.2004.03.005
- Bang, R. L., R. K. Gang, S. C. Sanyal, E. M. Mokaddas, and A. R. Lari. Beta-haemolytic *Streptococcus* infection in burns. *Burns* 1999;25:242-246.
- Lilly, H. A., E. J. Lowbury, M. D. Wilkins, J. S. Cason. Staphylococcal sepsis in a burns unit. *J Hyg (London)*. 1979;83:429-435.
- Barret, J. P., D. N. Herndon. Modulation of inflammatory and catabolic responses in severely burned children by early burn wound excision in the first 24 hours. *Arch Surg*. 2003;138:127-132.
- Clark, N. M., J. Patterson, and J. P. Lynch III. Antimicrobial resistance among gram-negative organisms in the intensive care unit. *Curr Opin Crit Care*. 2003;9:413-423.
- Lansdown, A. B. Silver. 1. Its antibacterial properties and mechanism of action. *J Wound Care*. 2002;11:125-130. doi: 10.12968/jowc.2002.11.4.26389
- Lansdown, A. B. 2002. Silver. 2. Toxicity in mammals and how its products aid wound repair. *J. Wound Care* 2002;11:173-177. doi: 10.12968/jowc.2002.11.5.26398.
- Boyce, S. T., A. P. Supp, V. B. Swope, and G. D. Warden. Topical sulfamylon reduces engraftment of cultured skin substitutes on athymic mice. *J. Burn Care Rehabil*. 1999;20:33-36.
- Atiyeh, B. S., K. A. El-Musa, and R. Dham. Scar quality and physiologic barrier function restoration after moist and moist-exposed dressings of partial-thickness wounds. *Dermatol Surg*. 2003;29:14-20
- Hegggers, J. P., M. C. Robson, D. N. Herndon, and M. H. Desai. The efficacy of nystatin combined with topical microbial agents in the treatment of burn wound sepsis. *J. Burn Care Rehabil*. 1989;10:508-511.

13. McCormack, P. L., and C. M. Perry. Caspofungin: a review of its use in the treatment of fungal infections. *Drugs*. 2005;**65**:2049–2068
14. Dunn, K., and V. Edwards-Jones. The role of Acticoat with nanocrystalline silver in the management of burns. *Burns*. 2004;**30**(Suppl. 1):S1–S9.
15. Meier, P. A., C. D. Carter, S. E. Wallace, R. J. Hollis, M. A. Pfaller, and L. A. Herwaldt. A prolonged outbreak of methicillin-resistant *Staphylococcus aureus* in the burn unit of a tertiary medical center. *Infect Control Hosp Epidemiol*. 1996;**17**(12):798–802.
16. Hegggers, J. P., H. Hawkins, P. Edgar, C. Villarreal, and D. N. Herndon. 2002. Treatment of infections in burns, p. 120–169. *In* D. N. Herndon (ed.), *Total burn care*. Saunders, London, England.
17. Ergun, O., A. Celik, G. Ergun, and G. Ozok. Prophylactic antibiotic use in pediatric burn units. *Eur J Pediatr Surg*. 2004;**14**:422–426. DOI: 10.1055/s-2004-821065.
18. Grube, B. J., D. M. Heimbach, and J. A. Marvin. *Clostridium difficile* diarrhea in critically ill burned patients. *Arch Surg*. 1987;**122**(6):655–661.
19. Murphy, K. D., J. O. Lee, and D. N. Herndon. Current pharmacotherapy for the treatment of severe burns. *Expert Opin Pharmacother*. 2003;**4**(3):369–384.
20. Uguro AO, Fadeyibi IO, Mofikoya BO, Akanmu ON, Temiye EO, Kanu OO, et al. Neonatal burns in Lagos, South-Western Nigeria: Epidemiology and outcome of management. *Burns*. 2013;**39**:483–492. doi: 10.1016/j.burns.2012.07.025.
21. Cox SG, Rode H, Darani AN, Fitzpatrick-Swallow VL. Thermal injury within the first 4 months of life. *Burns*. 2011;**37**:828–834. doi: 10.1016/j.burns.2011.02.003.
22. Saaiq M, Ahmad S, Zaib S. Neonatal burn injuries: an agony for the newborn as well as the burn care team. *Annals of Burns and Fire Disasters*. 2013;**26**(4):175–181.
23. Rimdeika R, Bagdonas R. Major full thickness skin burn injuries in premature neonate twins. *Burns*. 2005;**31**:76–84. doi: 10.1016/j.burns.2004.04.009.
24. Sun B, Zhou X, Xia C, Chen Y. Management of severe burn injuries in neonates. *J Burn Care Rehabil*. 2004;**25**(3):219–223.
25. Mohrenschlager M, Weigl LB, Haug S, Schnopp C, Cremer H, Ring J, et al. Iatrogenic burns by warming bottles in the neonatal period report of two cases and review of the literature. *J Burn Care Rehabil*. 2003;**24**:52–55. doi: 10.1097/01.BCR.0000045660.50162.FE.
26. Lin CW, Wang HZ, Hsieh KS. Pulse oximeter-associated toe injuries in a premature neonate: A case report. *Zhonghua Yi Xue Za Zhi Taipei*. 1999;**62**:914–916.
27. Toung TJ, Donham RT, Shipley R. Thermal burn caused by a laryngoscope. *Anesthesiology*. 1981;**55**(2):184–185.
28. Siegfried EC, Stone MS, Madison KC. Ultraviolet light burn: A cutaneous complication of visible light phototherapy of neonatal jaundice. *Pediatr Dermatol*. 1992;**9**(3):278–282.
29. Ho WS, Ying SY. Iatrogenic burn caused by an alcohol lamp. *Burns*. 2000;**26**(8):757–759.
30. Saaiq M, Zaib S, Ahmad S. Early excision and grafting versus delayed excision and grafting of deep thermal burns up to 40% total body surface area: A comparison of outcome. *Annals of Burns and Fire Disasters*. 2012;**25**(3):143–147.