References

1. Janshon GP, Dudziak R: Interactions of dry soda lime with enflurane and sevoflurane. Clinical report on two unusual anesthesias. Anaesthesist 1997; 46:1050-3
2. Woehlck HJ, Dunning M III, Connolly LA: Reduction in the incidence of carbon monoxide exposures in humans undergoing general anesthesia. ANESTHESIOLOGY 1997; 87:228-34
3. Woehlck HJ, Dunning M III, Gandhi S, Chang D, Milosavljevic D: Indirect detection of intraoperative carbon monoxide exposure by mass spectrometry during isoflurane anesthesia. ANESTHESIOLOGY 1995; 83:213-7
4. Fang ZX, Eger EI II, Laster MJ, Chortkoff BS, Kandel L, Ionescu P: Carbon monoxide production from degradation of desflurane, enflurane, isoflurane, halothane, and sevoflurane by soda lime and Baralyme. Anesth Analg 1995; 80:1187-93
5. Frink EJ Jr, Nogami WM, Morgan SE, Salmon RC: High carboxyhemoglobin concentrations occur in swine during desflurane anesthesia in the presence of partially dried carbon dioxide absorbents. ANESTHESIOLOGY 1997; 87:308-16
6. Woehlck HJ, Dunning MB III, Kulier AH, Sasse FJ, Nithipataikom K, Henry DW: The response of anesthetic agent monitors to trifluoromethane warns of the presence of carbon monoxide from anesthetic breakdown. J Clin Monit 1997; 13:149-55
7. Thom SR, Keim LW: Carbon monoxide poisoning: A review epidemiology, pathophysiology, clinical findings, and treatment options including hyperbaric oxygen therapy. J Toxicol Clin Toxicol 1989; 27:141-56
8. Stewart RD: The effect of carbon monoxide on humans. Annu Rev Pharmacol 1975; 15:409-23
9. Anderson EW, Andelman RJ, Strauch JM, Fortuin NJ, Knelson JH: Effect of low-level carbon monoxide exposure on onset and duration of angina pectoris. A study in ten patients with ischemic heart disease. Ann Intern Med 1973; 79:46-50
10. Allred EN, Bleecker ER, Chaitman BR, Dahms TE, Gottlieb SO, Hackney JD, Pagano M, Selvester RH, Walden SM, Warren J: Short-term effects of carbon monoxide exposure on the exercise performance of subjects with coronary artery disease. N Engl J Med 1989; 321:1426-32
11. Seger D, Welch L: Carbon monoxide controversies: Neuropsychologic testing, mechanism of toxicity, and hyperbaric oxygen. Ann Emerg Med 1994; 24:242-8
12. Bonome C, Belda F, Refojo M, Soro C, Gotti H, Aymerich H: Carbon monoxide production in rebreathing circuits: The halogenated concentration effect (abstract). Br J Anaesth 1998; 80:A86
13. Woehlck HJ, Dunning MB III: Predicting the severity of carbon monoxide poisoning at varying FIO2. ANESTHESIOLOGY 1998; 88:1126-7
14. Vegfors M, Lennmarken C: Carboxyhaemoglobinaemia and pulse oximetry. Br J Anaesth 1991; 66:625-6
15. Buckley RG, Aks SE, Eshom JL, Rydman R, Schaider J, Shayne P: The pulse oximetry gap in carbon monoxide intoxication. Ann Emerg Med 1994; 24:252-5
16. Barker SJ, Tremper KK: The effect of carbon monoxide inhalation on pulse oximetry and transcutaneous pO2. ANESTHESIOLOGY 1987; 66:677-9
17. Baum J, Sachs G, v.d. Driesch C, Stanke HG: Carbon monoxide generation in carbon dioxide absorbents. Anesth Analg 1995; 81:144-6

Anesthesiology
1999; 90:616-8

Intraoperative Burns Secondary to Warmed IV Bags: A Warning

Lorne K. Rosenfield, M.D., Paul J. Pitlyk, M.D.†*

IN positioning for cervical spinal surgery, it is common to place a bolstering device to help support and position the operative site, specifically the anterior aspect of the neck. Ordinarily a 10-lb leather-bound sandbag or a liter bag of intravenous fluid is used for this purpose. During a lengthy surgery, warmed intravenous fluid bags also have been used to treat hypothermia. The purpose of the following two case reports is to promulgate a strong awareness of a hazard in using warmed intravenous fluid bags in such circumstances.

* Chief, Department of Plastic Surgery.

† Assistant Clinical Professor of Neurosurgery, University of California, San Francisco.

Received from the Department of Surgery, Section of Neurosurgery, Mills-Peninsula Hospital, Burlingame, California. Submitted for publication October 13, 1997. Accepted for publication July 6, 1998.

Address reprint requests to Dr. Rosenfield: Department of Plastic Surgery, Mills-Peninsula Hospital, Burlingame, California, 1750 El Camino Real, Suite 405, Burlingame, California 94010.

Key words: Force; heat; lesions; sedation.

Case Reports

Case 1

A 53-yr-old man was taken to surgery on January 6, 1996 for resection of cervical osteophytes *via* the anterior approach. The patient was placed in the supine position, and a liter bag of intravenous fluid was used to position his head in an extended posture. The intravenous fluid bag was obtained from the blanket warmer (set at 114°F) for patient comfort. It was wrapped in a towel and placed between the scapulae


Fig. 1. Upper back burn wounds.


Fig. 2. Right inner arm and axillary–chest burn wounds.

at the upper thoracic and lower cervical region of his back. The surgery proceeded without complication in the ensuing 1.5 h.

After the procedure the patient reported severe pain in the upper thoracic region. Inspection of this site revealed "islands" of second- and third-degree burns overlying the spine and the scapulae (fig. 1). Plastic surgical consultation was obtained, and successful debridement and skin grafting was accomplished in subsequent days. The patient healed satisfactorily without functional impairment.

Case 2

On January 1, 1992 a 44-yr-old woman, in preparation for a lumbar laminectomy, was placed on the operating room table in the knee-chest position. The procedure required 4.5 h. During this time, progressive hypothermia developed that was resistant to standard warming measures, such as heated inspiratory gasses, warm intravenous fluids, and warming blankets. For this reason, warmed intravenous bags were taken from the blanket warmer and placed in the axillary areas.

After operation the patient was noted to have sustained burns in the regions of the inner arm and upper lateral chest, including the breasts bilaterally (figs. 2, 3). Debridement and grafting were subsequently conducted to care for the second- and third-degree burns. The patient healed without functional impairment.

Discussion

Burns secondary to "warmed" supporting and bolstering devices in anesthetized patients have been reported. Kokate *et al.*[1] performed an animal study wherein a disc-shaped pressure device generating 100 mmHg pressure was applied for 5 h at different positions on the dorsal surface of the animal. The pressure device contained a heating element, which allowed the effects of the applied pressure at temperatures of 25, 35, 40, and 45°C to be studied. Table 1 outlines the histopathologic observations at various tissue levels for the fixed pressure device at the temperatures noted during a 5-h period. There was increasing tissue damage between 35 and 45°C; at 25°C, tissues remained free of injury. At 35°C, muscle damage deep to the epidermis and dermis predominated, whereas at 40°C there was a mixture of epidermal necrosis and moderate muscle damage. At 45°C, full epidermal necrosis with moderate dermal and severe muscle damage was observed.

Salisbury reported that a temperature even less than 44°C results in tissue death if exposure is prolonged.[2] Between 44 and 51°C the rate of cell destruction doubles with each increased temperature degree. Thus, the higher the temperature with constant pressure and time the more likely and deeper will be the burn depth.

Wounds from hypothermic animals during the same conditions were compared with those of normothermic animals. No difference was identified, indicating that generalized hypothermia is not protective. However, Iaizzo *et al.*[3] showed that decreasing tissue temperatures


Fig. 3. Left inner arm and axillary–chest burn wounds.

Table 1. Summary of Tissue Status after the Application of 100 mmHg for 5 h at 25, 35, or 45°C

Site Temperature	Status
25°C	All tissues normal
35°C	Moderate muscle damage
40°C	Partial epidermal necrosis, moderate muscle damage
45°C	Full epidermal necrosis, moderate dermal and severe muscle damage

From Kokate et al.[1]

to less than 30°C minimizes tissue damage, which would normally result from an applied pressure of 100 mmHg for durations of 5 h or more. A mechanism by which this phenomena occurs was offered by Morris and Field,[4] who identified a difference between heating time and injury in normal and ischemic tissue. These results were interpreted in terms of a decreased tissue *p*H.

The frequent need for bolstering and supporting devices during surgical procedures makes the contour-forming aspect of a liter bag of intravenous fluid ideal for certain body regions. Such containers are also readily available in the operating theater. Raising its temperature, however, to more than room temperature for protection from the risk of hypothermia or for patient comfort is unsound practice. Litigation involving patients undergoing surgery who sustained burns as a result of either designed warming devices or other heated devices used for patient positioning and bolstering have been reviewed by Cheney *et al.*[5] Burns from either of these two sources represented 1% of a total of 3,000 medicolegal claims studied. In their review, intravenous fluid bags specifically used as warming devices represented 64% of the burn claims, compared with 19% of the claims when the intravenous fluid bag was used strictly for body positioning and bolstering.

Hypothermia during surgical procedures is best prevented using equipment designed for such purposes. The factors affecting the genesis of such lesions include temperature magnitude, points of bony prominence, duration of pressure-temperature exposure, skin protection, and patient weight. This time-temperature-pressure relation is critical in the development and depth of a burn.

Therefore, we can appreciate how a bag of intravenous fluid warmed nominally and placed in contact with skin and under pressure for a few hours can produce severe burns. In the first patient described, the significant temperature and pressure of the warm fluid bag produced by the bulk of the patient and the scapular spinous process protuberances accelerated the thermal damage. In the second patient, the thin skin of the inner arm, axilla, and ribs in a similar temperature-pressure environment were contributing factors in the production of a severe thermal burn.

Conclusion

Two cases were presented wherein minimally invasive warming of a patient during surgery resulted in significant thermal burns. Given the potential for thermal injury, warmed bags should not be used as bolsters or warming devices during surgical procedures. Because of the potentially protracted contact time under some pressure, even a bag minimally warm to the touch can cause a full-thickness thermal burn. All health-care providers managing anesthesia and postanesthesia care should be made aware of this risk, and department procedures should be modified appropriately.

References

1. Kokate JY, Leland KJ, Held AM, Hansen GL, Kveen GL, Johnson BA, Wilke MS, Sparrow EM, Iaizzo PA: Temperature-modulated pressure ulcers: A porcine model. Arch Phys Med Rehabil 1995; 76:666-73

2. McCarthy JG, May JW, Littler JW: Burns, Plastic Surgery. Philadelphia, WB Saunders, 1990, pp 787-813

3. Iaizzo PA, Kveen GL, Kokate JY, Leland KJ, Hansen GL, Sparrow EM: Prevention of pressure ulcers by focal cooling: Histological assessment in a porcine model. Wounds: A Compendium of Clinical Research and Practice 1995; 7:161-9

4. Morris CC, Field SB: The relationship between heating time and temperature for rat tail necrosis with and without occlusion of the blood supply. Int J Radiat Biol 1985; 47:41-8

5. Cheney FW, Posner KL, Caplan RA, Gild WM: Burns from warming devices in anesthesia: A closed claims analysis. Anesthesia 1994; 80:806-10