

Systemic Hypothermia as Treatment for an Acute Cervical Spinal Cord Injury in a Professional Football Player: 9-Year Follow-Up

Andrew Cappuccino, MD, Leslie J. Bisson, MD, Bud Carpenter, ATC, Ken Snyder, MD, PhD, and Helen Cappuccino, MD, FACS

Abstract

The following report provides clinical follow-up on a National Football League player who sustained a complete cervical spinal cord injury (SCI) while tackling an opposing player in 2007. He received prompt medical and surgical care based on then-current recommendations, but was also treated with systemic hypothermia soon after his injury, which was controversial at the time. Since then, smaller randomized human studies have described the tolerable safety profile, efficacy, and potential benefits of this intervention in acute SCI in humans. Now, modest systemic hypothermia can be one of many tools considered in the treatment of acute SCI. Before it can become the standard of care, however, additional larger prospective randomized studies need to be completed. The patient described in this article had long-term excellent clinical results, with residual deficits of occasional tingling in fingertips and toe tips, although the patient continues to slowly improve.

In 2010, we reported the case of a professional American football player who sustained a complete cervical spinal cord injury (SCI) while tackling an opposing player.¹ He received prompt medical and surgical care based on then-current recommendations, but was also treated with systemic hypothermia soon after his injury. Although systemic hypothermia had been used in the management of other neurologic injuries at that time,

it had not been used in humans with acute SCI, except as described in 2 case reports.^{2,3} However, Dietrich⁴ described early emerging animal data on the efficacy of systemic hypothermia for acute SCI. We now provide a clinical update on our patient, who provided written informed consent for print and electronic publication of this case report.

Case Report

During a National Football League game, the player sustained a C3–C4 fracture-dislocation after a helmet-to-helmet hit on an opposing player. He fell face down on the ground and did not move. The team's physician and trainer rushed to the player's side, immediately assessed him, and initiated the emergency spinal resuscitation protocol.

As per protocol, the assigned team leader took charge of managing the player's head to maintain in-line traction with the helmet in place until the head was secured in place on a backboard designed to accommodate the helmet. In addition, 6 assistants were tasked with rolling the player to prone position with his head in traction. An equipment technician rapidly removed the player's mask, leaving the helmet in place. The trauma ABCs (airway, breathing, circulation) were assessed. Once the airway was stable, the 6-person roll technique was used, under the team leader's direction, to place the player on the backboard and secure him to it. Paramedics were ready to commence resuscitation, and an ambulance was driven onto the field (**Figures 1-3**).

Complete motor paralysis and

Take-Home Points

- Importance of on-field management.
- Preseason drilling of spinal injury management.
- Early and rapid intervention.
- Possible benefit of moderate systemic hypothermia as treatment for acute cervical injury.

Authors' Disclosure Statement: The authors report no actual or potential conflict of interest in relation to this article.



Figure 1. Player with proper in-line traction being applied.
Figure reprinted with permission from Danny Palumbo.



Figure 2. Player in supine position again, with traction applied and mask about to be removed.
Figure reprinted with permission from Danny Palumbo.



Figure 3. Player being loaded onto the ambulance on the field.
Figure reprinted with permission from Danny Palumbo.

sensory loss (American Spinal Injury Association [ASIA] level A) were noted below the clavicles during physical examination by the head athletic trainer and 2 independent physicians, and by self-report. The patient was breathing adequately despite feeling dyspneic. He was transferred to the ambulance within 15 minutes of injury, and there he received intravenous methylprednisolone in accordance with the National Acute Spinal Cord Injury Study (NASCIS) 2 protocol (Figure 3).⁵ With the goal being to achieve relative hypothermia, chilled saline was infused intravenously, rotating ice packs were applied to the axillae and groin, the patient's garments were removed, and the ambient temperature in the ambulance was lowered to 55°F. Repeated clinical examination in the ambulance revealed bulbocavernosus response, indicating resolution of spinal shock, though there was still no motor or sensory function below the clavicles (remained ASIA level A). During transportation, the patient was hemodynamically monitored and found to be stable, with no evidence of neurogenic shock. After 30 minutes of resuscitation, he showed slight improvement; by the time the

ambulance arrived at the emergency department, crude pressure and vibratory sensation in the extremities were apparent.

On arrival at the hospital, the patient had a core temperature of 98°F, which is substantially lower than the average core temperature ($\leq 101.7^\circ\text{F}$) of an active football player.⁶ He had a normal level of consciousness and normal cranial nerve function but remained without any voluntary motor function in the extremities and still had no sensation below the clavicles, except crude pressure sensation in one hand while in the emergency department. After the helmet and shoulder pads were removed, per National Athletic Trainers' Association (NATA) protocol⁷ (Figure 2), he was stabilized, and a hard cervical collar was placed. A lateral radiograph (Figure 4) showed a C3–C4 facet dislocation with about 46% anterior translation of C3 on C4 and obvious disruption of the facets. Computed tomography (CT) confirmed a left C3–C4 complete facet dislocation and a right C3–C4 facet subluxation with a "perched" facet, and Digital Imaging and Communications in Medicine (DICOM) measurements showed the same canal compromise ($\sim 55\%$) found with magnetic resonance imaging (MRI). After CT and before MRI, repeat clinical examination revealed persistent complete motor paralysis and rudimentary sensation to deep pressure below the nipples, with proprioception indicating anterior cord syndrome. At this point, hyperreflexia and nonsustained clonus were noted. MRI showed a small right central and paracentral disk herniation superimposed on signal change within the cord as it was draped over the postero-superior aspect of C4.

About 3 hours after injury, the patient was taken to the operating room. Although closed reduc-

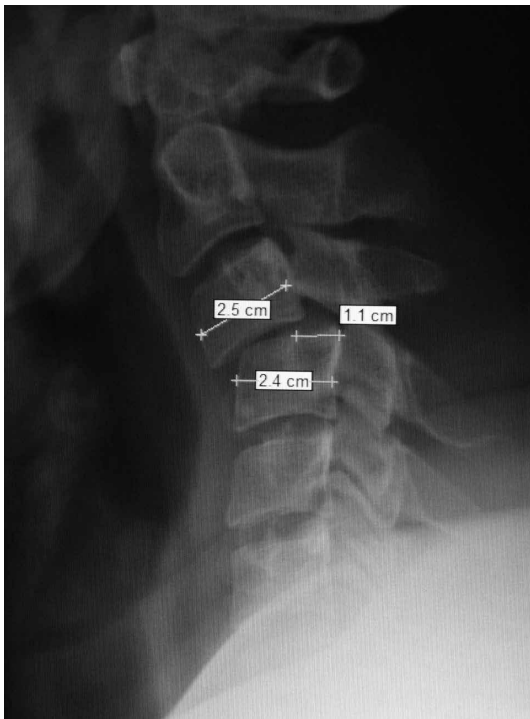


Figure 4. Emergency lateral radiograph of cervical spine.
Figure reprinted with permission from Danny Palumbo.

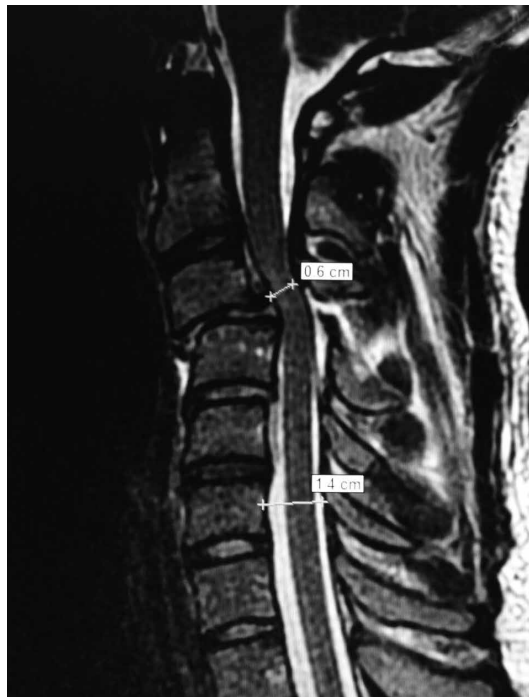


Figure 5. Magnetic resonance imaging of cervical spine after injury but before surgery.
Figure reprinted with permission from Danny Palumbo.

tion improved alignment dramatically, it failed to completely reduce the dislocated left C3–C4 facet. An hour later, anterior C3–C4 discectomy was performed from the front with instrumented anterior interbody fusion. This was immediately followed by posterior decompressive laminectomy, bilateral facet reduction, and fusion with instrumentation. Surgery was completed within about 4 hours, almost exactly 7 hours after injury. Anesthesia records indicated a core temperature range of 94.1°F to 95.3°F with passive cooling during surgery. CT and MRI performed within 4 hours after surgery showed excellent cord decompression.

The next morning, about 14.5 hours after injury, the patient demonstrated a flicker of the adductor muscles of the lower extremities. An examination an hour later revealed 1/5 quadriceps, 2/5 adductors, and 1/5 gastrocnemius/soleus. A nurse's hourly examinations and the surgeon's repeat examinations revealed no other motor function. Sensory function was more difficult to evaluate because of sedation, but rudimentary sensation was noted throughout the lower extremities, and proprioception and vibratory sensation were noted as well. With passive cooling, it was difficult to consistently maintain moderate hypothermia; the patient's core temperature ranged from 94.8°F to 98.8°F by 6:00 a.m. Therefore, the decision was made to place a Cordis sheath in the left femoral vein and introduce an intra-vena cava cooling catheter through it. This

catheter was highly effective in maintaining the patient's temperature at about 92.5°F.

Over the next 36 hours, the patient demonstrated increased motor activity in the upper and lower extremities: 1/5 biceps, 2-3/5 triceps, 3/5 quadriceps. He was slowly rewarmed and, on postoperative day 3, extubated. Continued intravascular cooling was used for several more days to maintain normal body temperature and avoid hyperthermia. During his 12-day hospital stay, the patient showed improvement in all leg muscle groups (3-4/5 motor strength) and minimal return of upper extremity motor function (0/5 deltoids, 1/5 biceps, 3/5 triceps, 1/5 first dorsal interosseous). After discharge to a rehabilitation facility, the patient improved dramatically, but discernible deficits in sensation, strength, coordination, and endurance (ASIA level D) lasted several months, improving only slowly. A year after injury, he was ambulating independently and having normal bowel, bladder, and sexual function but residual sensory deficits in the hands and feet (glove-and-stockings distribution) and significant deficits in fine motor movement.

At 2 years, the patient underwent another anterior-only cervical procedure: The inferior adjacent segment (C4–C5) was fused because of neck pain and deformity. Residual deficits now included only occasional tingling in fingertips and toe tips, and the patient continued to improve. Nine years after injury, he had a relapse of mechanical neck pain



Figure 6. Magnetic resonance imaging of cervical spine 2 weeks after surgery.

Figure reprinted with permission from Danny Palumbo.

only and was being treated for additional adjacent segment disease.

With respect to the original injury and the evolution in cord appearance, the patient had solid arthrodesis from C3–C5 with instrumentation in good position. There was evidence of loss of lordosis at C5–C6 with disk desiccation and broad-based bulging. The spinal cord had evidence of myelomalacia; this was noted when the patient was in rehabilitation, 1 month after injury. The 2-cm × 11-mm area of myelomalacia was directly posterior to the fused C3–C4 interval (original MRI, **Figure 5**; 2-week MRI, **Figure 6**).

Conclusion

At the time this player was injured, use of systemic hypothermia with standard therapy for acute SCI was unique and controversial. Since then, smaller randomized human studies have described the tolerable safety profile, efficacy, and potential benefits of this intervention in acute SCI in humans.⁸⁻¹⁰ Now, modest systemic hypothermia can be one of many tools considered in the treatment of acute SCI. Before it can become the standard of care, however, additional larger prospective randomized studies need to be completed.

Dr. A. Cappuccino is an Orthopedic Surgeon, Buffalo Spine Surgery, Lockport, New York, and Team Orthopedic Surgeon, Buffalo Bills, Buffalo, New York. Dr. Bisson is Eugene Mindell Professor and Chair, Department of Orthopedics, Jacobs School of Medicine and Biomedical Sciences, Buffalo, New York, and Medical Director, Buffalo Bills, Buffalo, New York. Mr. Carpenter is Director of Athletic Training Operations, Buffalo Bills, Buffalo, New York. Dr. Snyder is Associate Professor, Department of Neurosurgery, Jacobs School of Medicine and Biomedical Sciences, Buffalo, New York. Dr. H. Cappuccino is Clinical Associate Professor of Surgery, Jacobs School of Medicine and Biomedical Sciences, Buffalo, New York, and Associate Professor of Surgery, Breast Division, Department of Surgical Oncology, Roswell Park Cancer Institute, Buffalo, New York.

Address correspondence to: Andrew Cappuccino, MD, Buffalo Spine Surgery, 46 Davison Ct, Lockport, NY 14094 (tel, 716-438-2973; email, helencapp@aol.com).

Am J Orthop. 2017;46(2):E79-E82. Copyright Frontline Medical Communications Inc. 2017. All rights reserved.

References

1. Cappuccino A, Bisson LJ, Carpenter B, Marzo J, Dietrich WD 3rd, Cappuccino H. The use of systemic hypothermia for the treatment of an acute cervical spinal cord injury in a professional football player. *Spine.* 2010;35(2):E57-E62.
2. Goldstein J. Lowering body temp shows promise for trauma treatment. Spinal Cord Injury Information Pages news blog. <http://www.sci-info-pages.com/2006/05/lowering-body-temp-shows-promise-for.html>. Published May 3, 2006. Accessed March 19, 2009.
3. Hartemink KJ, Wisselink W, Rauwerda JA, Girbes AR, Polderman KH. Novel applications of therapeutic hypothermia: report of three cases. *Crit Care.* 2004;8(5):R343-R346.
4. Dietrich WD. Presidential address presented at: 34th Annual Meeting of the Cervical Spine Research Society; November 30, 2006; Palm Beach, FL.
5. Bracken MB, Shepard MJ, Collins WF, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the second National Acute Spinal Cord Injury Study. *N Engl J Med.* 1990;322(20):1405-1411.
6. Horodyski MB, LuCante K, Escobar E, et al. Intermittent Cool, Dry Air Underneath Football Shoulder Pads Assists in Temperature Homeostasis. In: The American Orthopaedic Society for Sports Medicine Proceedings 2008; 87-88.
7. Kleiner DM, Almqvist JL, Bales J, et al; Inter-Association Task Force for Appropriate Care of the Spine-Injured Athlete. *Prehospital Care of the Spine-Injured Athlete.* Dallas, TX: National Athletic Trainers' Association; 2001. <http://www.msata.org/Resources/Documents/PreHospitalCare4SpineInjuredAthlete.pdf>. Published March 2001. Accessed January 10, 2017.
8. Didizze M, Green BA, Dietrich WD, Vanni S, Wang MY, Levi AD. Systemic hypothermia in acute cervical spinal cord injury: a case-controlled study. *Spinal Cord.* 2013;51(5):395-400.
9. Levi AD, Casella G, Green BA, et al. Clinical outcomes using modest intravascular hypothermia after acute cervical spinal cord injury. *Neurosurgery.* 2010;66(4):670-677.
10. Levi AD, Green BA, Wang MY, et al. Clinical application of modest hypothermia after spinal cord injury. *J Neurotrauma.* 2009;26(3):407-415.