

Hypothermia (26.9°C) in a Polytrauma Patient: Case Report of Survival and Review of Science

Aparna Vijayasekaran^{1*}, Julie Wynne^{1,2}, Terence O'Keeffe^{1,2}, Randall Friese^{1,2}, Bellal Joseph^{1,2} and Peter Rhee^{1,2}

¹Department of Surgery, University of Arizona, Arizona, USA

²Division of Acute Care Surgery, University of Arizona, Arizona, USA

Abstract

Hypothermia is beneficial in certain circumstances; the single most advantageous aspect of hypothermia is the reduction in metabolic demand. However, induced hypothermia differs from post-traumatic hypothermia. It is acknowledged that hypothermic polytrauma patients have poorer outcomes than normothermic polytrauma patients.

Post-traumatic hypothermia has worse outcomes, likely due to it being a sequel of hemorrhagic shock, and the attendant failure to meet metabolic demands. Hemorrhagic shock produces the "lethal triad" of trauma: hypothermia, acidosis and coagulopathy. Studies of post-traumatic hypothermia have shown that core body temperature of 32°C predicts fatality [1-4]. We present a case report of a trauma patient with documented presenting core temperature of 26.9°C in the context of treatable hemorrhagic shock along with a systematic literature review of post-traumatic hypothermia.

Clinical Presentation

A twenty-four year old male was ejected from his jeep while driving in the Sonoran Desert of southern Arizona. It was estimated that the patient had been exposed to temperatures of 3°C for almost 7 hours before being found by EMS. On primary survey he had poor respiratory effort with diminished left breath sounds, oxygen saturation was not documentable secondary to absence of peripheral pulses, and was hypotensive with a blood pressure of 46/36 mmHg. Glasgow Coma Scale (GCS) was 5 (E1, V2, M2), and temperature by tympanic membrane was 28.1°C. He was immediately intubated and transfused warmed normal saline and 2 Units of warmed packed red blood cells (PRBC's). A left chest tube was placed to relieve his pneumothorax. Chest X Ray revealed a left 10th rib fracture and a residual left pneumothorax after placement of the chest tube. His blood pressure improved to 90/64 mmHg and he was taken to the CT scanner suite, covered with blankets. Laboratory studies were significant for a pH of 6.8 and an arterial lactate of 15 mMol/L. Imaging studies revealed no evidence for brain injury, a residual left pneumothorax, grade V kidney laceration, C7 transverse process fracture, T12 non-displaced vertebral body fracture, L2 transverse process fracture, and left superior pubic ramus fracture. EKG showed widened QRS complex, atrial fibrillation, right bundle branch block and a ventricular rate of 87. The patient was taken emergently to the operating room for exploratory laparotomy, with findings of contained left retroperitoneal hematoma and a shattered right kidney. The right renal hilum was clamped and nephrectomy performed. He received a total of 7 Liters of crystalloids and 2U PRBC's during the procedure. The estimated blood loss was 300 cc although this number could be confounded by the extensive intra-abdominal irrigation that was performed.

Intra-cavity warming measures were undertaken. The patient's lowest core temperature by esophageal probe in the operating room was 26.9°C. Intra-operative warming measures included a Bair hugger air blanket, warmed humidified air via the ventilator, warmed fluids and transfusions, and warmed irrigation through bilateral chest tubes. Intra-abdominal warming was performed by placing 36F chest tubes in the lateral gutters; the abdominal wall was loosely closed over surgical lap sponges with towel clips. Warmed fluid was aggressively infused into the bilateral intra-abdominal chest tubes. The core temperature rose to 32°C within 22 minutes, at which time the patient was re-explored. The renal vascular bundle was ligated and the abdomen was closed. The patient was then transported to the Surgical Intensive Care Unit. Arterial lactate was > 15 mMol/L (pH 6.8) on presentation,

8.3mMol/L (pH 7.1) at the end of the procedure and 2.8mMol/L (pH 7.3) after 6 hours of continued resuscitation in the ICU. INR improved from 1.9 on presentation to 1.2.

He achieved normo-thermia within nine hours of presentation to our facility. His neurologic exam improved and he was extubated the next day with full neurologic capabilities. He was transferred to the ward in stable condition on post-operative day four, and had no memory of the day of injury or the first two days of his hospitalization. As he was an active member of the armed forces, he was transferred to a military facility, and made a complete recovery.

Discussion

Hypothermia can be both beneficial and detrimental. Cold-water near-drowning victims have higher survival rates, thanks to preservation of the brain and other vital organs. This concept of cooling in order to slow metabolism is also the rationale behind using hypothermia to decrease ischemia during cardiac, transplant, pediatric, and neurologic surgery [5-8]. The Advanced Life Support Task Force of the International Liaison Committee of Resuscitation now recommends cooling, to 32°C to 34°C, unconscious adults who have spontaneous circulation after out-of-hospital ventricular fibrillation arrests [9].

Induced hypothermia differs markedly from spontaneous hypothermia, which typically results from shock or inadequate tissue perfusion, and this likely explains the variance in outcomes (Table 1). The survival rates after accidental hypothermia range from 12% to 39%. The lowest recorded temperature in a survivor of accidental hypothermia (13.7°C, or 56.7°F) was in an extreme skier in Norway; she was trapped under the ice and, eventually, fully recovered neurologically after resuscitation [10]. The extreme hypothermia in

***Corresponding author:** Aparna Vijayasekaran, Department of Surgery, University of Arizona, 1501 N Campbell Avenue, PO Box: Tucson, Arizona, 85704, USA, Tel: 520-626-7747; E-mail: aparnav@email.arizona.edu

Received March 21, 2012; Accepted March 30, 2012 Published April 02, 2012

Citation: Vijayasekaran A, Wynne J, O'Keeffe T, Friese R, Joseph B, et al. (2012) Hypothermia (26.9°C) in a Polytrauma Patient: Case Report of Survival and Review of Science. Emergency Medicine S2:001. doi:10.4172/2165-7548.S2-001

Copyright: © 2012 Vijayasekaran A, et al. 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.

	Trauma	Accidental
Mild	36-34°C	35-32°C
Moderate	34-32°C	32-28°C
Severe	<32°C (<90F)	<28°C (<82F)

Table 1: Classification of hypothermia depending on whether it is from trauma or accidental.

our patient was precipitated due to a combination of blood loss from polytrauma along with low environmental temperature. Our patient had intra-abdominal free fluid and a large retroperitoneal hematoma that resulted in hypovolemia. Decreased circulating volume contributes to hypothermia due to decreased tissue perfusion.

Hypothermia and its association with mortality and morbidity in trauma patients is well known [4,11]. Whether in the emergency room, operating room, or intensive care unit, survival rate falls dramatically with core temperature. There are multiple theories as to why there is a difference between trauma and non-trauma patients. The main reason is thought to be that non traumatic hypothermia is induced whereas traumatic hypothermia is spontaneous. Non injured patients do not have blood loss and the hypothermia is mostly due to the cold environment which takes away the body’s heat and actually preserves tissues. In this state the body has reserves and with the decreased metabolic demand due to hypothermia, the body upon restoration of normothermia can meet the metabolic demand.

In trauma patients, hypothermia is due to shock and perpetuates uncontrolled bleeding because of the associated coagulopathy. Both of these mechanisms worsen hemorrhagic shock and lactic acidosis. Thus during shock state, the body will gravitate towards ambient temperature as it cannot produce enough heat to maintain optimal temperature. Often for trauma patients, ambient temperature is 25°C, the average temperature inside hospitals. In addition, trauma patients are often resuscitated with cold fluids, further cooling the patient as well as causing dilutional coagulopathy. A triad of acidosis, coagulopathy and hypothermia ensues and perpetuates this metabolic catastrophe. Trauma patients with a postoperative core temperature of less than 35° C have a four-fold increase in death; less than 33°C, a seven-fold increase in death. Hypothermic trauma patients tend to be more severely injured and older, with bleeding as indicated by blood loss and transfusions [12]. A small number of trauma patients with a recorded core temperature of less than 32° C have survived [2].

To understand hypothermia, we have to remember that humans are homeothermic (warm-blooded) animals, in contrast to poikilothermic (cold-blooded) animals such as snakes and fish. To maintain a body temperature of 37° C, our hypothalamus taps a variety of mechanisms to tightly control core temperature. We use oxygen as the key ingredient or fuel to generate heat in the mitochondria in the form of Adenosine triphosphate (ATP). When ATP production is below its lowest threshold, one of the side effects is the lowering of body temperature to the ambient temperature. When we produce excess heat during exercise, we use more oxygen and release heat in the form of radiation and by the cooling process of evaporation as we sweat.

The patient in this case report demonstrated the lethal triad: acidosis with a pH of 6.8, coagulopathy with an INR of 1.9, and hypothermia. Of note is that the measured state of coagulopathy is not truly reflective of the degree of coagulopathy. A normal coagulation profile does not necessarily represent the body’s actual physiologic state. Cold affects coagulopathy by decreasing enzyme activity, enhancing fibrinolytic activity, and causing platelet dysfunction. Platelets are affected by the inhibition of thromboxane B2 production, resulting in decreased aggregation [13]. A heparin-like substance is released, causing diffuse

intravascular coagulation (DIC)-like syndrome. Hageman factor and thromboplastin are some of the enzymes most affected. A drop in core temperature of just a few degrees results in 40% inefficiency in some of the enzymes. When blood is drawn in cold patients and sent to the laboratory, the sample is heated to 37° C, because cold delays clotting and renders test results inaccurate. Thus, in a cold and coagulopathic patient, if the coagulation profile obtained from the laboratory shows an abnormality, the result represents the level of coagulopathy as if the patient (and not just the sample) had been warmed to 37° C. Therefore, a cold patient is typically more coagulopathic than indicated by the coagulation profile.

The reason for the survival in our patient is that although the patient is technically a trauma patient, the ambient temperature in the chilly desert also induced hypothermia. The hypotension on presentation was probably due to the combination of hypovolemia from his abdominal trauma, pneumothorax as well as his profound hypothermia. Although tension pneumothorax could have caused the decreased cardiac output which may have contributed to the hypotension as well as the hypothermia, the diagnosis and treatment of the pneumothorax was rapid on arrival and hence the role of the pneumothorax in either of these conditions cannot be accurately quantified.

In this patient external active rewarming measures were started as soon as the patient arrived in the ED. Rewarming techniques are classified as either passive or active. Active warming is further classified as either external or internal (Table 2). Passive warming involves preventing heat loss, by drying the patient to minimize evaporative cooling, giving warm fluids to prevent cooling, or covering the patient so that the ambient air temperature immediately around the patient is higher than the room temperature. Covering the patient’s head helps reduce a tremendous amount of heat loss. Using aluminum-lined head covers is preferred: they reflect back the infrared radiation that is normally lost through the scalp. Warming the room technically helps reduce the heat loss gradient, but the surgical staff is usually unable to work in a humidified room of 37°C. Passive warming also includes closing open body cavities, such as the chest or abdomen, in order to prevent evaporative heat loss. The most important way to prevent heat loss is to treat hemorrhagic shock by controlling bleeding. Once shock has been treated, metabolism will heat the patient from his or her own core. This point cannot be overemphasized.

Active warming actively transfers calories to the patient, either externally through the skin or internally. Skin and fat are designed to be highly efficient in preventing heat transfer, so active external warming is inefficient, as compared with internal warming. Forced-air heating, such as with Bair Hugger temperature management therapy (Arizant Healthcare Inc., Eden Prairie, MN), is technically classified as active warming, but air is an inefficient medium, so not many calories are provided to patients. Forced-air heating only increases the patient’s ambient temperature, but it can actually cool the patient, because it increases evaporative heat loss if the patient is wet from blood, fluids, clothes, or sweat. Warming the skin may feel good to the patient and the

PASSIVE	ACTIVE EXTERNAL	ACTIVE INTERNAL
Dry the patient	Bear hugger	Warmed fluids
Warm fluids	Heated warmers	Heat ventilator
Warm blankets / sheets	lamps	Cavity lavage. - Chest tube / abdomen / bladder
Head covers	Radiant warmers	Continuous arterial or venous rewarming
Warm the room	Clinitron bed	Full or partial bypass

Table 2: Classification of warming techniques.

surgeon, but it actually decreases shivering (a highly efficient method of internal warming that tricks the thermoregulatory nerve input on the skin). Since forced-air heating uses convection, the actual amount of active warming is estimated to be only 10 Kcal per hour.

Heat is measured in calories. One calorie is the amount of energy required to raise the temperature of one cc of water (which has, by definition, a specific heat of 1.0). One kilocalorie is required to raise the temperature of one liter of water by one degree. If an average human (75 kg) consisted of pure water, then it would take 75 Kcal to raise his or her temperature by one degree. However, we are not made of pure water; blood has a specific heat coefficient of 0.87. The human body as a whole has a specific heat of 0.83. Thus, it actually takes 62.25 Kcal (75 kg times 0.83) to raise body temperature by one degree. If a patient were to lose 62.25 Kcal, his or her temperature would drop by one degree.

The normal basal metabolic heat generation is about 70 Kcal per hour; shivering can increase this to 250 Kcal per hour. Heat is transferred by conduction (as in a frying pan and Jacuzzi), convection (as in an oven and sauna), radiation, and evaporation. Convection is an extremely inefficient way to transfer heat: the air molecules are far apart, as compared with liquids and solids. Conduction and radiation are the most efficient ways to transfer heat. However, transferring radiation to the body is fraught with inconsistencies and is difficult to apply clinically. So, we rely on conduction to transfer energy efficiently.

Although IV fluids can be warmed, the U.S. Food and Drug Administration (FDA) allows fluid warmers to be set at a maximum of 40°C. Therefore, the differential between a cold trauma patient (34°C) and warmed fluid is only 6 degrees. Thus, one liter of warmed fluids can only transfer 6 Kcal to the patient. We need about 62 Kcal to raise the core temperature by one degree, so we need 10.4 liters of warmed fluids to raise the core temperature by one degree to 35°C [14]. Once that has been achieved, the differential is now only five degrees between the patient and the warmed fluid; so it actually now takes 12.5 liters of warmed fluids to raise the patient from 35°C to 36°C. Note that a liter of fluid must be given at the highest rate possible, because it cools to room temperature as it enters the IV line. To avoid IV line cooling, devices should be used that warm the fluid just before it enters the skin.

Warming patients by infusing warmed fluids is difficult, but fluid warmers are still critical. Cold fluids would very quickly cool patients. Thus, the main reason for using fluid warmers is not necessarily to warm patients but to prevent cooling them during resuscitation. The fluids that are typically infused are either at room temperature (22°C) or at the temperature of packed red blood cells that have been refrigerated (4°C). It takes five liters of 22°C fluid or two liters of cold blood to cool a patient by one degree. A cold patient at 32°C needs to be given 311 Kcal (75 kg times 0.83) to warm to 37°C.

Active external warming is best performed by placing patients on heating pads, which use conduction to transfer heat. Beds are available that can expedite warming, such as the “Clinitron bed” (Hill-Rom, Batesville, IN), which uses heated air-fluidized beads. Such beds are not practical in the emergency department or the operating room, but are applicable in the ICU.

The best method to warm patients is to deliver the calories internally (Table 3). Heating the air used for ventilators is technically internal active warming, but it is inefficient. The surface area of the lungs is massive, but the energy is mainly transferred through humidified water droplets, using mostly convection and not conduction. This method of warming was used in the case being presented.

The main method of warming used in this case was cavitory lavage.

METHOD	KCAL/ hour
Airway from vent	9
Overhead radiant warmers	17
Heating blankets	20
Convective warmers	15-26
Body cavity lavages	35
CAVR	92-140
Cardiopulmonary bypass	710

Table 3: Calories delivered by active warming.

Body cavities can be lavaged by infusing warmed fluids through chest tubes or by merely irrigating the abdominal cavity with warmed fluids [15,16]. Although it has been calculated that cavitory lavage only produces approximately 30 Kcal per hour, the fact that we were able to warm the patient more rapidly in this case (three degrees in 22 minutes) suggests that it can be more than predicted. For this patient of approximately 80 kg weight, the calculated amount of heat provided with the three cavity lavage (bilateral chest and abdomen) was over 600 Kcal. Once the critical temperature of 32°C was reached surgical exploration was commenced. At the same time the patient’s hemodynamics were maintained by infusion of warmed crystalloids. The post-operative temperature was 34.9°C. The total OR time of the patient was 115 minutes and hence this patient was rapidly warmed at a rate of 2.75°C /hr.

Much has been discussed comparing intra-cavitory rewarming measures to extracorporeal methods. Gentilello and colleagues have proven that extracorporeal methods reduce the requirement of resuscitation volumes and decrease mortality [17,18]. The best means to deliver heat is through countercurrent exchange of fluids, using conduction to transfer calories. Full cardiopulmonary bypass is unmatched: it delivers over five liters per minute of heated blood to every capillary bed [19-23]. If full cardiopulmonary bypass is not available or not desired, alternatives include continuous venous or arterial rewarming [24,25]. Venovenous rewarming is most easily accomplished using the roller pump of a dialysis machine (which is often more available to the average surgeon) [26]. A prospective study showed arterial-venous rewarming to be highly effective. It can warm patients to 37°C in about 39 minutes, as compared with an average warming time of 3.2 hours using standard techniques. Special “Gentilello arterial warming catheters” are inserted into the femoral artery, and a second line is inserted into the opposite femoral vein. The pressure from the artery produces flow, which is then directed to a fluid warmer and back into the vein. This method depends highly on the patient’s blood pressure, because flow is directly related to blood pressure. Other means written about but rarely used in practice include gastric lavage and esophageal lavage with special tubes [27-29]. If gastric lavage is desired, one method is to place two nasogastric tubes and infuse warm fluids through one while suctioning the other. Bladder irrigation with an irrigating Foley catheter is useful. Instruments to warm the hand through conduction show much promise but are not yet readily available. The benefits of mild hypothermia during resuscitation in a hemorrhagic setting are controversial. It has been shown in a rat model that mild controlled hypothermia improves immediate but not long term survival and also helps in maintaining arterial pressures during resuscitation [30].

One of major factors that contributed to the successful resuscitation of this patient was the prompt diagnosis and initiation of treatment of his hypothermia on arrival in the emergency room. The other factors that influenced the positive outcome were the contained nature of his intra-abdominal hemorrhage (retroperitoneal hematoma contained

within the Gerota's fascia), rapid surgical exploration and the optimal usage of both active and passive rewarming measures.

Conclusion

In conclusion, hypothermia in trauma patients contributes to the lethality of the injury and must be rapidly addressed by the physician. All aspects of passive and active warming should be considered and used. Warming should be started at the earliest, preferably in the field or as soon as the patient arrives in the emergency room. The knowledge of the science behind transfer of heat will help the clinician understand causes of hypothermia, and avoid heat loss. Trauma patients have worse outcomes than patients with non-traumatic induced hypothermia, and this is probably due to the associated tissue injury and hemorrhagic shock. Although hypothermia can be used to protect tissue and reduce inflammation, it is associated with poor outcome following trauma due to the unwanted coagulopathy which will worsen hemorrhagic shock and its lactic acidosis.

References

1. Arthurs Z, Cuadrado D, Beekley A, Grathwohl K, Perkins J, et al. (2006) The impact of hypothermia on trauma care at the 31st combat support hospital. *Am J Surg* 191: 610-614.
2. Martin RS, Kilgo PD, Miller PR, Hoth JJ, Meredith JW, et al. (2005) Injury-associated hypothermia: an analysis of the 2004 National Trauma Data Bank. *Shock* 24: 114-118.
3. Shafi S, Elliott AC, Gentilello L (2005) Is hypothermia simply a marker of shock and injury severity or an independent risk factor for mortality in trauma patients? Analysis of a large national trauma registry. *J Trauma* 59: 1081-1085.
4. Wang HE, Callaway CW, Peitzman AB, Tisherman SA (2005) Admission hypothermia and outcome after major trauma. *Crit Care Med* 33: 1296-1301.
5. Pacini D, Di Marco L, Leone A, Di Bartolomeo R, Sodeck G, et al. (2012) Antegrade selective cerebral perfusion and moderate hypothermia in aortic arch surgery: clinical outcomes in elderly patients. *Eur J Cardiothorac Surg*.
6. Lin H, Mo A, Zhang F, Huang A, Wen Z, et al. (2010) Donor heart preservation in an empty beating state under mild hypothermia. *Ann Thorac Surg* 89: 1518-1523.
7. Saliba E, Debillon T (2010) Hypothermia for hypoxic-ischemic encephalopathy in fullterm newborns. *Arch Pediatr* 3: S67-S77.
8. Yenari MA, Hemmen TM (2010) Therapeutic hypothermia for brain ischemia: where have we come and where do we go? *Stroke* 41: S72-74.
9. Nolan JP, Morley PT, Vanden Hoek TL, Hickey RW, Kloeck WG, et al. (2003) Therapeutic hypothermia after cardiac arrest: an advisory statement by the advanced life support task force of the International Liaison Committee on Resuscitation. *Circulation* 108: 118-121.
10. Gilbert M, Busund R, Skagseth A, Nilsen PA, Solbo JP (2000) Resuscitation from accidental hypothermia of 13.7 degrees C with circulatory arrest. *Lancet* 355: 375-376.
11. Beilman GJ, Blondet JJ, Nelson TR, Nathens AB, Moore FA, et al. (2009) Early hypothermia in severely injured trauma patients is a significant risk factor for multiple organ dysfunction syndrome but not mortality. *Ann Surg* 249: 845-850.
12. Inaba K, Teixeira PG, Rhee P, Brown C, Salim A, et al. (2009) Mortality impact of hypothermia after cavity explorations in trauma. *World J Surg* 33: 864-869.
13. Wolberg AS, Meng ZH, Monroe DM 3rd, Hoffman M (2004) A systematic evaluation of the effect of temperature on coagulation enzyme activity and platelet function. *J Trauma* 56:1221-1228.
14. Gentilello LM, Pierson DJ (2001) Trauma critical care. *Am J Respir Crit Care Med* 163: 604-607.
15. Plaisier BR (2005) Thoracic lavage in accidental hypothermia with cardiac arrest—report of a case and review of the literature. *Resuscitation* 66: 99-104.
16. Talbot SG, Davidson MJ, Javid S, Patel AN, Fitzgerald D, et al. (2010) Hypothermic cardiac arrest rescued with cardiopulmonary bypass and decompressive laparotomy. *Emerg Med J* 27: 958-959.
17. Gentilello LM, Cobean RA, Offner PJ, Soderberg RW, Jurkovich GJ (1992) Continuous arteriovenous rewarming: rapid reversal of hypothermia in critically ill patients. *J Trauma* 32: 316-325.
18. Gentilello LM, Jurkovich GJ, Stark MS, Hassantash SA, O'Keefe GE (1997) Is hypothermia in the victim of major trauma protective or harmful? A randomized, prospective study. *Ann Surg* 226: 439-449.
19. Vretenar DF, Urschel JD, Parrott JCW, Unruh HW (1994) Cardiopulmonary bypass resuscitation for accidental hypothermia. *Ann Thorac Surg* 58: 895-898.
20. Bolgiano E, Sykes L, Barish RA, Zickler R, Eastridge B (1992) Accidental hypothermia with cardiac arrest: recovery following rewarming by cardiopulmonary bypass. *J Emerg Med* 10: 427-433.
21. Walpoth BH, Locher T, Leupi F, Schüpbach P, Mühlemann W, et al. (1990) Accidental deep hypothermia with cardiopulmonary arrest: extracorporeal blood rewarming in 11 patients. *Eur J Cardiothorac Surg* 4: 390-393.
22. Oberhammer R, Beikircher W, Hörmann C, Lorenz I, Pycha R, et al. (2008) Full recovery of an avalanche victim with profound hypothermia and prolonged cardiac arrest treated by extracorporeal re-warming. *Resuscitation* 76: 474-480.
23. Morita S, Inokuchi S, Inoue S, Akieda K, Umezawa K, et al. (2008) The efficacy of rewarming with a portable and percutaneous cardiopulmonary bypass system in accidental deep hypothermia patients with hemodynamic instability. *J Trauma* 65: 1391-1395.
24. Gregory JS, Bergstein JM, Aprahamian C, Wittmann DH, Quebbeman EJ (1991) Comparison of three methods of rewarming from hypothermia: advantages of extracorporeal blood warming. *J Trauma* 31: 1247-1251.
25. Laniewicz M, Lyn-Kew K, Silbergleit R (2008) Rapid endovascular warming for profound hypothermia. *Ann Emerg Med* 51: 160-163.
26. Caluwe R, Vanholder R, Dhondt A (2010) Hemodialysis as a treatment of severe accidental hypothermia. *Artif Organs* 34: 237-239.
27. Lloyd EL (1973) Accidental hypothermia treated by central rewarming through the airway. *Br J Anaesth* 45: 41-48.
28. Marcus P (1979) The treatment of acute accidental hypothermia: proceedings of a symposium held at the RAF Institute of Aviation Medicine. *Aviat Space Environ Med* 50: 834-843.
29. Sperling DR, Beran AV (1976) Hypothermia and rewarming by peritoneal dialysis and temperature-controlled inhalate. *Ann Thorac Surg* 21: 151-157.
30. Wu X, Stezoski J, Safar P, Nozari A, Tisherman SA (2003) After spontaneous hypothermia during hemorrhagic shock, continuing mild hypothermia (34 degrees C) improves early but not late survival in rats. *J Trauma* 55: 308-316.

This article was originally published in a special issue, **Trauma & Accidental Surgery** handled by Editor(s), Dr. Antonios G Angoules, Educational Institute of Athens, Greece