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Therapeutic Hypothermia after Perioperative Cardiac Arrest in Cardiac Surgical Patients

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Abstract

BACKGROUND—Therapeutic hypothermia (TH) has been established as an effective treatment for preserving neurological function after out of hospital cardiac arrest (CA). Use of TH has been limited in cardiac surgery patients in particular because of concern about adverse effects such as hemorrhage and dysrhythmia. Little published data describe efficacy or safety of TH in cardiac surgical patients who suffer unintentional CA. However, the benefits of TH are such as may suggest clinical equipoise, even in this high risk patient population.

OBJECTIVE—To report a series of three patients in our institution's cardiac surgery intensive care unit who suffered unintentional CA within 48 hours of cardiac surgery and were treated with TH.

METHODS—After institutional review board approval, study patients were identified by diagnosis of undesired intraoperative CA or arrest on ICU days 1–2, as well as having documented TH. The institution's electronic medical record and the Society of Thoracic Surgeons database were retrospectively reviewed for demographic information, comorbid diagnoses, surgical procedure, and outcomes including hemorrhage, re-warming dysrhythmias, infection, in-hospital mortality, and neurologic outcome were assessed. TH was initiated and monitored using active cooling pads according to written institutional protocol.

RESULTS—Four patients received TH after perioperative arrest. One patient was inadequately cooled and had massive surgical bleeding, and was therefore excluded from this review. The remaining three patients had a predicted mortality of 14.6% (±13.3) based on Euroscore calculation, and were cooled for 17.6±4.0 hours after CA. Coagulopathy, hypovolemia, severe electrolyte abnormalities, and re-warming dysrhythmias were not identified in any patient. 2 patients were discharged home and 1 was discharged to a long-term care facility.

CONCLUSION—Herein we report the safe and successful use of TH after unintentional perioperative CA in 3 cardiac surgery patients. These data suggest that further investigation of this therapy may be warranted given the potential benefit and apparent safety in a small series.

Keywords

therapeutic hypothermia; cardiac surgery; cardiac arrest; outcome

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Conflicts of Interest: None

INTRODUCTION

Therapeutic hypothermia (TH) is effective for the preservation of neurological function after out-of-hospital cardiac arrest (CA)[1–3], but to date there is little specific data reporting safety or efficacy of TH in cardiac surgery patients who experience unintentional CA. Indeed, TH is underutilized even in those populations with substantial supporting evidence[4,5]. TH may have serious complications, thus it is understandable that practitioners may be reluctant to adopt it without strong evidence, particularly in high risk patient populations.

Induction of hypothermia in the immediate postoperative period can be complicated by hypovolemia, electrolyte disorders, hyperglycemia, shivering, bradycardia, and vasodilatory shock[6–8]. Maintenance of TH can be associated with coagulopathy due to platelet dysfunction and clotting factor deficits, symptomatic vasoconstriction of severely atherosclerotic coronary arteries, and increased risk for nosocomial infections[9]. Rewarming of the mildly hypothermic patient can precipitate arrhythmias, electrolyte disturbances, and hypoglycemia[6]. Convincing data exists to prove the benefit of TH for patients suffering out of hospital CA, but few studies to date have examined the utility of TH in perioperative cardiac surgical patients suffering CA. The data supporting use of TH in out-of-hospital arrest, however, is so substantial as to suggest clinical equipoise for use of this therapy in other populations, such as has been demonstrated in pediatric patients, patients suffering in-hospital cardiac arrest, and patients seen in the emergency department after anoxic brain injury from near-hanging[10–13]. We here describe 3 patients who underwent TH after CA within 2 days of cardiac surgery.

MATERIALS AND METHODS

Upon institutional ethics review board approval, study patients were identified from our institution's cardiac surgery intensive care unit database by a diagnosis of unintentional intraoperative CA or arrest on ICU days 1–2, as well as documentation of controlled TH during the period from April 2007 and April 2010. After identifying patients, institutional electronic medical record and Society of Thoracic Surgeons (STS) data were collected and reviewed for demographic information, comorbid diagnoses, surgical procedure, and other information (Table 1). Outcomes including hemorrhage, laboratory abnormalities, re-warming dysrhythmias, infection, in-ICU and in-hospital mortality, and length of stay were assessed. Neurologic outcome was assessed using the five-point Glasgow-Pittsburgh Cerebral Performance Category (GP-CPS) [14] and discharge destination[3]. Discharge to home or to a rehabilitation center, and a GP-CPS of 1–2 were considered favorable neurologic outcomes, whereas death, discharge to a long-term nursing facility, or GP-CPS of 3–5 were considered unfavorable. The decision to implement TH was made by the attending intensivist in conjunction with the attending cardiac surgeon, and in part was based on the duration of CA, time to return of spontaneous circulation and the estimated risk of bleeding. TH was initiated and monitored using active cooling pads (ArcticSun, Medivance, Inc., Louisville, Colorado) according to an institutional protocol. All team members were experienced in the institutional TH protocol. Statistical analysis was performed using SigmaPlot (Systat Software Inc., San Jose, California). Data are reported as mean +/− SEM.

RESULTS

During the review period, there were 4 cases of perioperative arrest within 48 hours of cardiac surgery after which cooling was initiated. There were 6 additional patients who experienced CA within 48 hours of cardiac surgery, but did not undergo cooling. Of these patients, 4 died after being unsuccessfully resuscitated, and therefore not cooled, while two more were rapidly noted to be neurologically intact, and not cooled.

One patient who was cooled during the trial period was involved in a massive transfusion protocol prior to cooling, and subsequently experienced a suspected transfusion related coagulopathy, and was therefore excluded from analysis. The excluded patient was taken to the OR for re-operation due to massive mediastinal hemorrhage from the right internal mammary artery after suffering CA on post-operative day # 1. Although this source of bleeding was controlled prior to initiation of TH, evaluation of coagulopathy based on blood product administration and chest tube output in a patient with a known previous bleed requiring ongoing transfusion would be difficult, and transport to the OR suite and transfusion of blood products was thought to have affected her cooling times. In this patient it took 16 hours to achieve the target cooling temperature, time at target temperature was 8 hours, and re-warming was achieved after 18.75 hours. Although this patient eventually had a favorable neurologic outcome and was discharged home, the patient was excluded from the study due to the fact that the cooling period was prolonged to the extremes of previously reported TH studies, but also because the time at target temperature was significantly less than the 12–24 hour recommended in the 2003 ILCOR consensus statement[15]. Furthermore, one of the primary outcomes we examined was the effect of TH on coagulopathy and blood product administration, which was confounded by this patient's complicated peri-cooling events, including initiation of massive transfusion protocol, and a suspected transfusion related coagulopathy.

The remaining patients reached target temperatures within target times and were included in data analysis. Patient demographics and co-morbidities as identified prior to surgery are listed in Table 1. All patients were male. Two of the three patients (patients 1 and 3) had not had previous cardiac surgery, and one patient (patient 2) underwent re-sternotomy following a prior Ross procedure. One patient had previously identified co-morbidities (patient 1): non-insulin dependent diabetes mellitus, hypertension, and peripheral vascular disease.

Patient 1 underwent elective 4-vessel CABG and suffered pre-cardiopulmonary bypass ventricular fibrillation and arrest. Documented time until return of spontaneous circulation was 3.5 minutes. Upon successful resuscitation with return of spontaneous circulation in the OR, cardiopulmonary bypass (CPB) was initiated via stab cannulation of the ascending aorta just proximal to the brachiocephalic artery, and placing venous return in right atrium. After the patient was stabilized, a retrograde cardioplegia site was placed., the patient was cooled to 31°C and cardioplegia was administered. Upon completion of the surgery, the patient was rewarmed on CPB to 37°C, CPB was discontinued without difficulties, and surgical hemostasis was obtained. The patient was transferred to the cardiac ICU, where TH was immediately implemented (Table 2). The patient underwent 17 hours of TH at 33°C, and suffered no dysrhythmia during cooling or re-warming. Total chest tube output was 1595 ml over 6 days (average 266 ml/day). This patient was noted to have lack of left lower extremity pulses during cooling, but no further workup was pursued because of similar findings pre-cooling. After rewarming, the left lower extremity was noted to be cold and mottled, and the patient underwent left above knee amputation 20 hours after initiation of rewarming. After return to the ICU following his initial amputation, he required transfusion of 3 units packed red blood cells. One day after his initial amputation, the intensive care team noted swelling at the surgical site and increased creatinine kinase values, so the patient returned to the operating room for revision of his amputation. He sustained no other in-ICU or in-hospital morbidity. His total hospital stay was 28 days, and ICU stay was 14 days. He was discharged home at the end of his hospital course, and his GP-CPC was one.

Patient 2 underwent emergent ascending aortic aneurysm repair and sustained intraoperative pulseless electrical activity (PEA) after sternotomy but before initiation of CPB, with time-to-ROSC of 13 minutes. After initiation of CPB, the patient was cooled to 28°C on CPB. After surgical repair, the patient was rewarmed to 36°C, separated from CPB, and surgical

hemostasis was achieved. The patient was transferred to the cardiac/surgical ICU, and TH was initiated. The patient underwent uneventful cooling and re-warming, and spent a total of 22 hours at 33°C. While on TH, he developed symptomatic bradycardia, and was treated with isoproterenol with good clinical response. Total chest tube output was 1430 ml/5 days (286 ml/day), and the patient required no blood products. The patient suffered no in-hospital morbidity, and his total hospital stay was 12 days, with his ICU stay lasting 5 days. The patient went home upon hospital discharge, with GP-CPC of one.

Patient 3 experienced ventricular fibrillation and arrest on post-operative day two after emergent pulmonary embolectomy. The patient was resuscitated for a total of 15 minutes, after which he was immediately cooled to a temperature of 35°C for 14 hours. While the patient did not have any complications during cooling or re-warming, the patient did develop ventilator associated pneumonia (VAP) from a suspected aspiration event prior to TH, with a prolonged ventilator course (Table 4). Chest tube output was 1630 ml over 4 days (407.5 ml/day), and the patient received no transfusion. He required a tracheotomy on POD 12 to facilitate liberation from mechanical ventilation. The patient's hospital stay was 28 days, 19 of those days in the ICU. He was ultimately discharged to a long-term care facility and due to severe epilepsy as a result of hypoxic brain injury, he was categorized as GP-CPC 3.

DISCUSSION

The main finding of this study is that TH was applied in a small series of perioperative cardiac surgery patients without life-threatening hemorrhage, electrolyte disturbance, or dysrhythmia. In this population there are no specific guidelines to guide post cardiac arrest treatment. Cardiac surgical patients are at risk for dysrhythmia, electrolyte disturbances, and coagulopathy in the immediate postoperative period [16–19], and it is reasonable to hypothesize that TH might add additional risk. That said, to date few published case series have examined the use of TH in cardiac surgical patients suffering perioperative CA. Thus, we retrospectively reviewed our application of an existing TH protocol in three patients on a case-by-case basis accounting for the severity of the clinical scenario following CA in cardiac surgical patients, and reviewed incidence of dysrhythmias, hemorrhage, and electrolyte disturbance associated with TH.

During the period of retrospective review, 4 patients were identified that were cooled following CA. As previously mentioned, one female patient was excluded from the review due to inadequacy of cooling and transfusion due to lack of surgical hemostasis. The remaining 3 patients were all male (23–48 years old), with diverse surgical procedures, pre-operative risk factors, as well as mechanisms of CA. All 3 patients survived their operative course, and 2 patients were discharged home, with one patient requiring skilled nursing care upon discharge.

Unintentional cardiac arrest occurs in 0.7%–2.9% of cardiac surgical patients[20–23]. In one series reported by Anthi et al, survival to hospital discharge in cardiac surgical patients suffering CA was 79% without TH[20]. High quality, randomized, controlled trials document known complications of TH in other patient populations. Significant bleeding is reported in up to 26% of patients, with arrhythmia occurring in 26–36% of patients. The incidence of nosocomial pneumonia is 37%–70%, and hypokalemia occurs in 81% of patients. Poor neurologic outcome occurs in 45–59% of patients undergoing TH for CA[1,2].

During cooling, one patient required initiation of isoproterenol for bradycardia accompanied by hypotension. Isoproterenol was quickly weaned during rewarming. No patient experienced any other dysrhythmia at any point during cooling, rewarming, or the remainder

of the hospital stay. Laboratory evidence of coagulopathy was absent during cooling (table 6), and neither physical exam nor chest tube drainage revealed any significant increase in postoperative bleeding (Table 3). Only patient #1 required transfusion of blood products and was given 3 units of packed red blood cells post-operatively. This was likely due to loss of blood during limb amputation and revison procedures.

Hemodynamics were stable throughout cooling in all patients, and no patient required an increase of vasopressor or inotropic infusion to doses above standard regimens in our ICU to maintain hemodynamic stability. Patient # 1 required an increase in both epinephrine and vasopressin therapy while on TH, and patient # 2 required initiation of isoproteronol for symptomatic bradycardia (Table 4).

Patient #1 suffered a significant in-hospital morbidity requiring above the knee amputation. It is possible that existing severe limb ischemia was masked by TH, thus delaying limb-saving therapy. Another possibility is that vasoconstriction induced by TH worsened this patient's limb ischemia. Finally, it is possible that limb ischemia and TH were coincident; regardless, this complication represents an important cautionary note when applying TH in patient with severe peripheral vascular disease. Patient #3 had a suspected aspiration event resulting in hypoxia and CA prior to initiation of TH, with subsequent development of ventilator associated pneumonia, and prolonged ventilator dependence (Table 5). This patient ultimately required a tracheotomy before being successfully liberated from mechanical ventilation. Additionally, this patient was the only patient of the three to experience a poor neurologic outcome by our criteria. While it should be noted that this patient had the longest observed time until ROSC (15 minutes), he was also cooled to a goal temperature of 35° due to difficulties achieving a lower set point, and practitioner preference for this higher set point. While it is difficult to infer causality from these observations, it is possible that lower temperature during TH (32–34 °C) as established and published in large cohort studies, and recommended by expert consensus[1,3,15] could have resulted in better neurologic recovery.

Although several international groups have issued guidelines regarding TH application, many questions remain. Perhaps the largest paucity of data related to the cases presented here is optimal length of time to cooling. Published guidelines generally state "sooner is better"[15] with initiation of cooling, but no studies have compared outcomes as a function of time taken to reach goal temperature[24]. A recently published study which aimed to investigate the feasibility of TH administered via intra-nasal cooling assigned intra-arrest TH to one arm of the study[25]. Although inadequately powered to detect a significant difference in outcomes, the intra-arrest TH did not appear to worsen survival or neurologic outcomes for any patients, and subgroup analysis of patients receiving CPR revealed intra-arrest TH was associated with improved survival and neurologic salvage. Investigations such as these raise questions about the potential use of TH for intra-operative cardiac arrest; specifically, transporting the patient cooled to the ICU instead of re-warming patients after CPB. Such an early intervention would be novel, and could be a powerful way to compare outcomes to application of TH at a later time in the post-arrest period. However, no data yet supports the "sooner is better" hypothesis, and the inflammatory and transcriptional events following CA occur over hours to days, it is possible that "later is better", if warming patients conventionally after CPB allows hemostasis to occur prior to initiation of TH. With regard to standard practice of CPB, this question should be explored.

Time at target temperature is also controversial, with some studies cooling 12 hours, and some cooling for 24 hours with similar outcomes. No comparative studies have been undertaken.

The current data supporting TH for in-hospital patients suffering CA is minimal. Further, data regarding safety of cooling perioperative cardiac surgical patients is non-existent. Our critical care teams elected to institute TH in four cardiac surgical patients over a period of three years after weighing the potential for improvement in neurologic outcome against the risk of adverse effects on a case-by-case basis. After retrospectively reviewing these cases, incidence of complications, neurologic outcomes, and hospital morbidity have been presented in an attempt to further the application of TH in critically ill patients.

CONCLUSIONS

This case series suggests that it is possible to safely administer TH in a controlled intensive care unit setting to high-risk surgical patients in the immediate postoperative period. However, patients with severe peripheral vascular disease may suffer increased risk of prolonged ischemia secondary to hypothermia. Additionally, the rapidly expanding role of TH for inhospital and perioperative patients deserves continued investigation to fully realize the potential utility in patients with diverse mechanisms of cardiac arrest.

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Table 1

Patient Demographics

Patient	**1**	**2**	**3**
Age	48	23	38
Sex	Male	Male	Male
BMI	24.5	42.7	32.8
ASA Class	4	4E	5E
EUROSCORE	1.54	28.05	14.25
Surgical Procedure	CABG x 4	Aortic aneurysm repair	Pulmonary Embolectomy
Diabetes	Yes	No	No
Hyperlipidemia	No	No	No
Hypertension	Yes	No	No
PVD	Yes	No	No
Pre-op TTE	Mild segmental LV dysfunction, mild concentric LV hypertrophy, mild mitral annular calcification. EF 45–50%	Mildly reduced LV systolic function, severely/reduced RV function, large hematoma, small pericardial effusion. EF 50–55%	Mildly decreased LV systolic function, severely enlarged right ventricle, severely reduced RV systolic function. EF 40–45%
Mechanism of CA	Intraop V. fib arrest	Intraop PEA	V. fib arrest

Table 2

Characteristics of therapeutic hypothermia

Patient	**1**	**2**	**3**
Duration of resuscitation	3.5 minutes	13 minutes	15 minutes
Goal temperature	33°	33°	35°
Time to reach goal temperature (hours)	5	5.5	6.5
Time at Target Temperature (hours)	17	22	14
Time from rewarming to normothermia (hours)	3.5	6	3

Table 3

Chest tube output, blood variables.

Patient	**1**	**2**	**3**
Chest tube drainage (ml)	1595	1430	1630
Number of days	6	5	4
Chest tube output per day (ml)	265.8	286	407.5
pRBC units received	3	0	0

Table 4

Vasopressor and Inotrope support during cooling.

Patient	1	2	3
Pre-Cooling	Epinephrine 0.02–0.04 mcg/kg/min Vasopressin 1.5 units/hr	None	None
Cooling	Epinephrine 0.03–0.05 mcg/kg/min Vasopressin 4–5 units/hr Milrinone 0.3 mcg/kg/min	Isoproterenol 0.005–0.01 mcg/kg/min	None
Post Cooling	Epinephrine 0.05 mcg/kg/min Vasopressin 3 units/hr Milrinone 0.3 mcg/kg/min	Isoproterenol 0.01 mcg/kg/min Norepinephrine 0.01–0.02 mcg/kg/min	None

Table 5

Ventilator Settings and characteristics, length of hospitalization.

Patient	**1**	**2**	**3**
Ventilator Duration [h]	134.75	35.5	603
Time to first spontaneous breathing trial [h]	39.5	34	22
Tracheostomy	No	No	Yes
Length of ICU stay [d]	14	5	19
Length of hospital stay [d]	28	12	28
Outcome	Home	Home	Long term care
GC-CPC	1	1	3

Table 6

Laboratory values.

Patient	1			2			3		
Time	**Pre**	**Cooling**	**Post**	**Pre**	**Cooling**	**Post**	**Pre**	**Cooling**	**Post**
ABG									
pH	7.2	7.28±0.07	7.39	7.48	7.5±0.05	7.5	7.22	7.49±0.04	7.42
pCO_2	59	35.5±5.8	38	38	37±4.4	36	55	36.7±8.08	44
pO_2	162	80±14.9	83	156	98.6±72.4	98	224	72.7±11.9	89
BE	−6.0	−10±2.5	−1.4	4.6	5.5±1.5	4.2	−6.5	1.2±0.6	3.6
HCO_3	22	17±2.0	23	28	29.5±1.4	27	21	24.67±0.58	28
SaO_2	98.8	96.9±0.7	97.5	99.8	95.9±4.4	98.6	99	93.6±4.6	96.8
Basic Metabolic Panel									
Na^+	139	139±1.8	140	144	145±0.75	142	146	142.5±2.1	146
K^+	5.2	4.2±0.18	4.0	4.4	3.5±0.63	4	5.3	3.6±0.14	3.7
Cl^-	108	113±1.41	106	106	109±0.75	110	111	111±0.0	112
Total CO_2	24	18±2.16	27	27	29.8±0.98	28	23	26.5±0.7	29
BUN	24	21±1.63	18	14	15±0.81	15	22	17.5±0.7	16
Creatinine	1.7	1.4±0.08	1.2	1.7	1.3±0.15	1.5	1.8	1.04±0.02	1.3
Glucose	92	164±28.9	78	161	85±11.1	75	191	127.5±33.2	85
Ca^{2+}	6.6	8.05±0.3	7.7	11.1	9.6±0.74	8.9	7.5	7.9±0.35	7.8
Mg^{2+}	2.8	2.5±0.07	N/A	2.4	N/A	4.9	2.7	N/A	2.7
Phosphorous	3.1	3.85±1.3	N/A	4.9	N/A	N/A	5.4	3.5±0.57	3.9
CBC									
WBC	21.1	19.4±1.20	19.2	22.9	11.3±0.66	13.2	18.8	8.15±0.57	9.4
Hb	11.4	11.9±0.07	9.2	11.3	9.27±0.23	8.9	12.1	10.3±0.28	8.7
Hct	32.3	34.3±0.57	26.7	32.7	26.5±0.59	25.6	35.9	30.9±1	26.2
Plt	148	116±2.8	98	236	163±18.2	206	235	218±4.2	247
Coagulation									
INR	1.39	N/A	1.23	1.53	1.45±0.02	N/A	1.63	1.34	N/A

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Patient	1			2			3		
Time	Pre	Cooling	Post	Pre	Cooling	Post	Pre	Cooling	Post
APTT	40.7	N/A	44.1	58.9	36.0±4.8	26.4	44.8	45.7	48.5