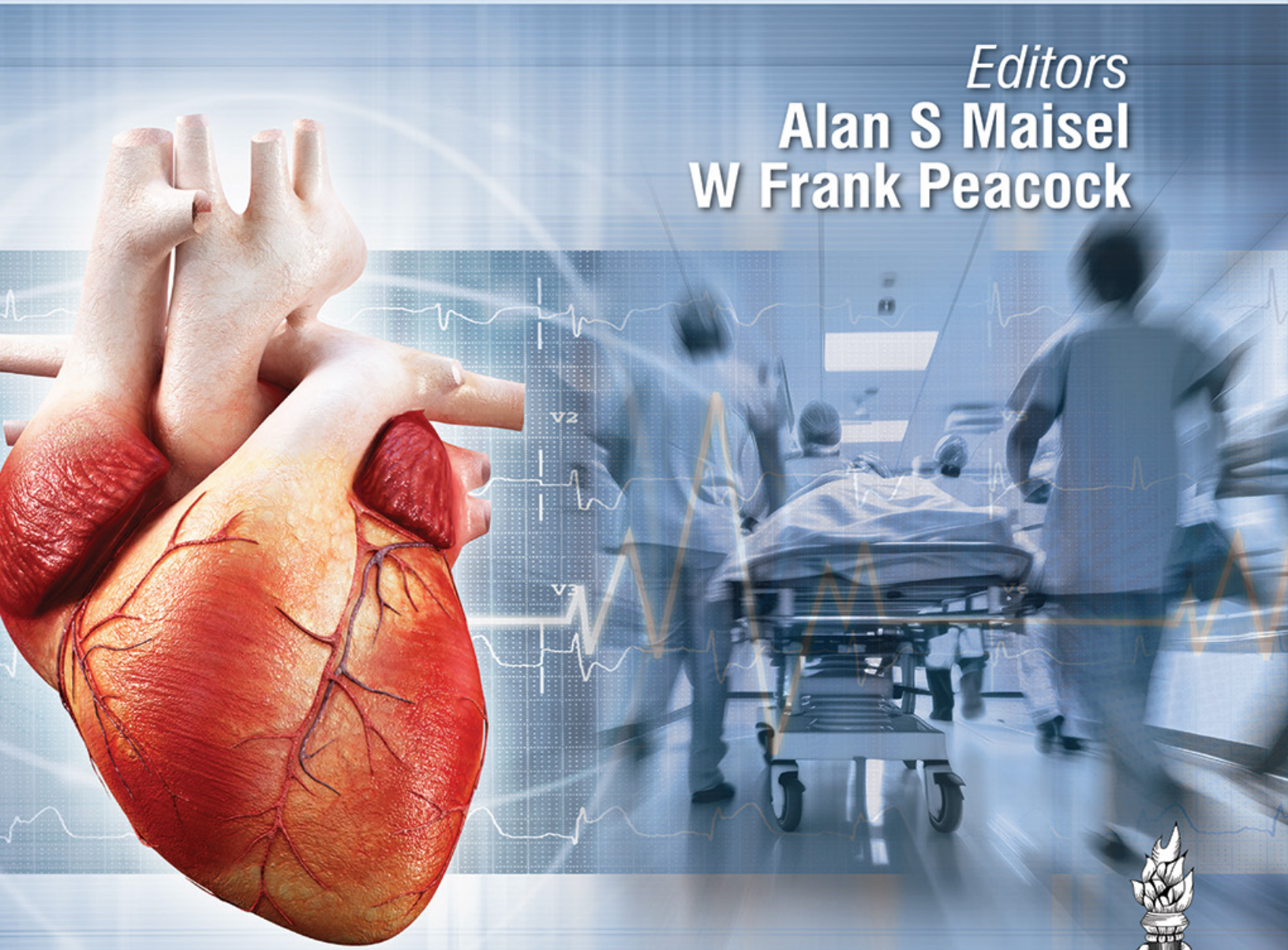
visit our website www.ejaypee.com, for detailed information on cardiology books, visit our website www.ejaypee.com, for detailed information on cardiology books

Textbook of EMERGENCY CARDIOLOGY

Editors
Alan S Maisel
W Frank Peacock



JAYPEE

Contents

Section 1: Cardiac Arrest

1. **Cardiac Resuscitation** 3
   Jillian B Horning, Caitlin M Kibbey, Joe T Kofoed, Deborah B Diercks
2. **Postcardiac Arrest Syndrome and Therapeutic Hypothermia** 16
   Nathan S Deal
3. **Biomarkers in Acute Care** 23
   Kevin S Shah, Vaishal M Tolia
4. **Electrocardiography in the Emergency Department** 30
   Semhar Z Tewelde, Amal Mattu
5. **Echocardiography** 49
   Darrin J Wong, Daniel G Blanchard
6. **Magnetic Resonance Imaging** 64
   Kimberly C Atianzar, Joel R Wilson
7. **Coronary Computed Tomography Angiography** 68
   Anna Marie Chang, Judd E Hollander
8. **Clinical Evaluation of Chest Pain** 76
   Matthew Noble, Anna Marie Chang
9. **Syncope** 90
   Sean-Xavier Neath
10. **Clinical Evaluation of Palpitations** 103
    Jeremy Egnatios, Nicholas A Marston

Section 2: Acute Myocardial Infarction and Acute Coronary Syndrome

11. **Acute Coronary Syndrome: Diagnosis of Acute Myocardial Infarction and Acute Coronary Syndrome** 113
    Francesca R Civitarese, Ava E Pierce, Deborah B Diercks
12. **Strategies to Rule-out Acute Myocardial Infarction in the Emergency Department: Who Can Go Home?** 127
    Martin Than
13. **Acute Coronary Syndrome: Risk Stratification** 141
    Lane M Smith, Chadwick D Miller, Simon A Mahler

14. **Treatment of Acute Coronary Syndrome in the Emergency Department** 150
Navdeep S Sekhon

15. **Noninvasive Testing** 161
Alexander T Limkakeng, Traci L Thoureen

Section 3: Acute Heart Failure

16. **How to Use Natriuretic Peptides in the Emergency Department?** 177
Boris Arbit, Alan S Maisel

17. **Other Biomarkers** 185
Nikola A Kozhuharov, Zaid Sabti, Samyut Shrestha, Christian Müller

18. **Bioimpedance Vector Analysis in Acute Heart Failure** 196
Salvatore Di Somma, Silvia Navarin

19. **Echocardiographic Assessment of Acute Heart Failure in the Emergency Department** 202
Jayant K Raikhelkar, Brian D Hoit

20. **Emergency Department Acute Heart Failure Treatment** 215
Dick C Kuo, Tyson Pillow

21. **The Role of Observation Units in Acute Heart Failure** 227
Edgardo Ordonez

22. **Cardiogenic Shock** 239
W Barton Campbell

23. **Acute Pericarditis** 250
Jacqueline J Denysiak, Daniel C Walters, Lori B Daniels

24. **Cardiac Tamponade** 257
Nicholas W Wettersten, Denise D Barnard

25. **Hypertensive Emergencies** 269
Phillip D Levy, Aaron M Brody

26. **Aortic Dissection** 284
D Mark Courtney

27. **Cardiac Trauma** 290
Michael Jaung, Zubaid R Rafique

Section 4: Arrhythmias

28. **Atrial Fibrillation and Atrial Flutter** 299
David E Krummen, Amir A Schricker

29. **Ventricular Tachycardia and Ventricular Fibrillation** 309
David E Krummen, Gautam G Lalan, Amir A Schricker

30. **Emergency Bradycardias** 320
Amir A Schricker, David E Krummen

31. ABC of Pacing: Temporary and Permanent **326**
Kay-Won Chang, Boris Arbit, Alan S Maisel

32. Supraventricular Tachycardia **339**
David E Krummen, Amir A Schricker

33. Use of Anticoagulation in Arrhythmias **348**
Nicholas Phreaner, Jonathan Harrison, Alex Pearce

34. Cardiac Toxicity Due to Drugs **371**
Spencer C Greene, Stephen Sanders

35. Acute Pulmonary Embolism **384**
Adam J Singer

36. Kidney Injury in the Face of Heart Disease **393**
Nicholas W Wettersten, Sutton R Fox

Index ***413***

2 CHAPTER

Postcardiac Arrest Syndrome and Therapeutic Hypothermia

Nathan S Deal

JAYPEE BROTHERS

INTRODUCTION

Medical history is full of attempts to understand the physiology of cardiac arrest and to identify maneuvers that could assist in the restoration of blood flow. This early work has evolved into the familiar chest compressions used today as part of cardiopulmonary resuscitation. Despite the success of CPR, our understanding of cardiac arrest physiology remained limited and robust post-arrest therapies were largely absent. However, in the last couple of decades, the field of post-arrest management has blossomed with the advent of therapeutic hypothermia. Today, therapeutic hypothermia, provides patients with the opportunity to survive cardiac arrest with meaningful neurologic recovery.

CARDIAC ARREST INCIDENCE AND SURVIVAL

It is estimated that out-of-hospital cardiac arrest (OHCA) affects roughly 325,000 patients per year in the United States,[1,2] with another 210,000 patients suffering cardiac arrest while hospitalized.[3] Despite advances in the area of intra-arrest resuscitation, out-of-hospital, nontraumatic cardiac arrest survival to hospital discharge remains low, estimated to be 10.6% nationally.[4] Those patients who do survive are often burdened with neurologic impairment due to the hypoxic insult endured during the arrest. Postarrest neurologic function is often classified using the Cerebral performance category (CPC) system (Table 1), with overall survival with a CPC score of 1 or 2 as low as 8.3% of patients.

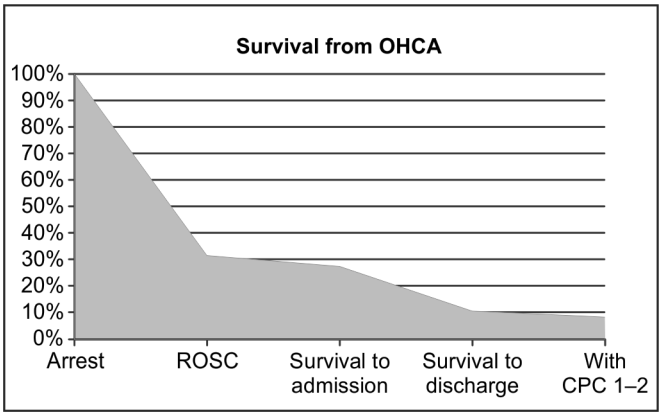
Cardiac arrest survival data may be further delineated based on intermediate goals along the path towards survival to hospital discharge, including return of spontaneous circulation (ROSC), and survival to hospital admission.[6] Sustained ROSC in the prehospital arena, estimated at approximately 30%, has been demonstrated to have the greatest correlation with survival [odds ratio (OR ≥35)].[7] Less than 1% of patients with no prehospital ROSC survived to discharge.[8] This declining survival data suggests that while patients may survive the acute emergency of cardiac arrest, irreversible damage sustained during the event, and/or ongoing cardiovascular collapse results in further mortality (Fig. 1).

The incidence of cardiac arrest, with such a high overall mortality, paired with the additional neurologic impair-

TABLE 1: Cerebral Performance Category Scale[5]

CPC 1	Good cerebral performance: Conscious, alert, able to work, might have mild neurologic or psychological deficit
CPC 2	Moderate cerebral disability: Conscious, sufficient cerebral function for independent activities of daily life. Able to work in sheltered environment
CPC 3	Severe cerebral disability: Conscious, dependent on others for daily support because of impaired brain function. Ranges from ambulatory state to severe dementia or paralysis
CPC 4	Coma or vegetative state: Any degree of coma without the presence of all brain death criteria. Unawareness, even if appears awake (vegetative state) without interaction with environment; may have spontaneous eye opening and sleep/awake cycles. Cerebral unresponsiveness
CPC 5	Brain death: Apnea, areflexia, EEG silence, etc.

CPC, cerebral performance category; EEG, electroencephalogram.



ROSC, return of spontaneous circulation; CPC, Cerebral Performance Category.

FIG. 1: Data from Cardiac Arrest Registry to Enhance Survival (CARES) Summary Report. 31,127 out-of-hospital cardiac arrest patients, 01/01/13 – 12/31/13. mycares.net

ment faced by those who do survive has engendered a great deal of research into the pathophysiology of ischemic/hypoxic injury.

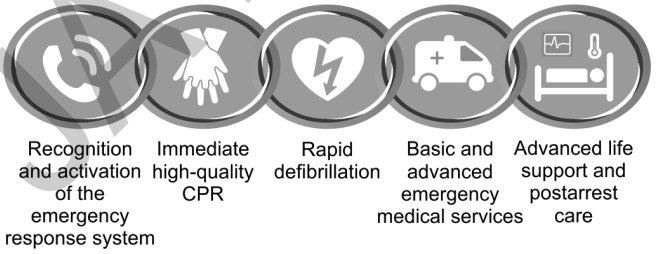
PATHOPHYSIOLOGY MODEL OF ISCHEMIC/HYPOXIC INJURY

It was recognized early in cardiac arrest research that even minutes of ischemia and hypoxia can result in deleterious effects throughout the body.[9,10] Subsequent reperfusion compounds this injury, and is attributed to a multifactorial process that includes cell membrane injury,[11] calcium dysregulation,[12] resulting in mitochondria-mediated ROS formation,[13] and subsequent cellular apoptosis.[14] These mechanisms affect multiple organ systems, but are most pronounced in the central nervous system. It is this injury to the brain that accounts for two-thirds of the mortality and a significant amount of morbidity in the postarrest patient.[15]

Recognizing the significance of ischemia/reperfusion pathology and the continued cellular death occurring in this phase, the American Heart Association renewed focus on the support of the postarrest patient. In 2010, a new link in the chain of survival was added, specifically focusing on the support required to aid in the recovery of these patients (Fig. 2).[16]

EMERGENCE OF THERAPEUTIC HYPOTHERMIA

To date, limited therapies have been demonstrated to be effective in minimizing the deleterious effects seen following cardiac arrest.[17] Two landmark studies, published in 2002, introduced the utility of therapeutic hypothermia (TH) in postarrest survivors.[18,19] In these studies, patients who experienced OHCA, and whose initial rhythm was identified as a nonperfusing ventricular arrhythmia (ventricular fibrillation and ventricular tachycardia in one study, and only ventricular fibrillation in the other), and who experienced ROSC were rapidly cooled to a target temperature of 32–34°C.



ACLS, advanced cardiac life support; CPR, cardiopulmonary resuscitation.

FIG. 2: American Heart Association 2015 Out-of-Hospital-Cardiac-Arrest (OHCA) Chain of Survival

This hypothermia was maintained for 12–24 hours before the patient was rewarmed to 37°C. These patients were compared to a similar cohort of patients in which no active temperature management was initiated.

Using survival to hospital discharge as an endpoint of the studies, both groups were able to demonstrate a significant improvement in the patients receiving hypothermia. In addition, neurologic recovery, measured by a CPC score of 1 or 2 also was substantially higher in patients receiving hypothermia. Subsequent analysis of these two studies plus one similar study of OHCA survivors reviewed survival with good neurologic outcome data at 6 month postarrest and found significant improvement in the hypothermia cohorts. Based on this analysis, it is calculated that the number needed to treat (NNT) patients with OHCA with TH to result in one more survivor with good neurologic outcome is six.[20]

A variety of mechanisms have been proposed to explain the remarkable improvement exhibited by TH.[21] Therapeutic hypothermia results in reduction of global cerebral metabolism and oxygen demand, likely reducing the overall production of ROS.[22] Excitatory neurotransmitter release is blunted by TH and may limit the excitotoxicity seen in cerebral ischemia.[23,24] Proapoptotic pathways are also blunted with the application of TH.[25,26] Global inflammatory markers are also suppressed following the application of TH.[27] The body's response to ischemia/reperfusion injury is complex, with cellular damage resulting from a collection of metabolic pathways. While TH should be viewed as a blunt tool for this complicated biochemical process, its success in improving neurologic survival is profound.

Inclusion of Asystole and Pulseless Electrical Activity Patients

Initial work focused on TH for the treatment of OHCA patients in whom initial rhythm analysis demonstrated ventricular fibrillation/ventricular tachycardia (VF/VT). Subsequent work attempted to determine what benefit this therapy could provide to patients with asystole or pulseless electrical activity (PEA) as the initial nonperfusing rhythm. Some institutions have included all OHCA patients, including asystole and PEA patients, in their TH protocols with demonstrated improvements in all cohorts.[28,29]

A meta-analysis of 12 nonrandomized control trials using TH for asystole and PEA patients demonstrates statistically significant improvement in survival to hospital discharge.[30] Only sparse data exists from randomized control trials with few patients enrolled. While the data cautiously demonstrates a trend towards long-term improvement in survival,[31,32] the small numbers of patients prevent any conclusive statements from being

JAYPEE BROTHERS

made. Based primarily on the observational data, the most recent AHA recommendations state that TH may be considered for patients suffering from OHCA with asystole or PEA.[17]

TIME TO TARGET TEMPERATURE

Most data evaluating the speed with which cardiac arrest survivors should be cooled is based on work in animal models.[33-38] By reaching the goal temperature of 33°C within 4 hours of arrest, animal models demonstrated improved outcomes compared to normothermic controls. However, this benefit was absent if cooling was delayed more than 8 hours.[39] This work in animal models supports the need for rapid cooling in postarrest survivors.

Additional analysis of human cardiac arrest data has provided mixed results regarding the conclusions of earlier animal work in which a delay in initiation of hypothermia and delays in achieving the desired target temperature resulted in worsening outcomes for patients. In a registry-based study of approximately 1,000 OHCA patients, treated with TH by a variety of modalities, time to initiation of TH (mean of 90 min) and time to target temperature (mean of 260 min) were not associated with any difference in patient outcome.[40]

However, additional data in human survivors supports the conclusion that time to target temperature is critical. On study of 49 consecutive OHCA patients, it demonstrated time to target temperature (mean of 410 min) as an independent predictor of good outcome.[41] In a more recent study of 172 OHCA patients receiving TH, each 30 minute delay in time to target temperature resulted in a 17% increase in poor neurologic outcome.[42] Based on a wealth of animal models and complementary human data, many experts recommended that TH be initiated as quickly as possible, although this continues to be an area of continued investigation.

Target Temperature: 32–34°C

Initial identification of a target temperature between 32°C and 34°C was guided primarily by earlier work in animal models. For this reason, initial studies[18,19] used this range for early human trials. Subsequent work has investigated whether very mild hypothermia of 36°C is sufficient to achieve the desired benefits. In one large, multicenter trial, patients were randomized to a goal temperature of 33°C versus 36°C and demonstrated no difference in overall outcomes.[43] However, in this study, the mean time to achieving the target temperature in the 33°C cohort was roughly 8 hours. Any beneficial effect that may have been conferred in this 33°C cohort may have been blunted by the delay in time to target temperature.[44] The optimal target temperature remains an active area of research.

Following cardiac arrest, it has been noted that patients frequently develop hyperthermia if no active temperature management in performed.[18] It is thought that this hyperthermia is a result of the global inflammation that occurs following ischemia/reperfusion injury.[45] This hyperthermia may be exhibited soon after the arrest or a few days later, requiring continued vigilance and intervention as needed.

THERAPEUTIC HYPOTHERMIA PROTOCOLS AND METHODOLOGY

Since the publication of successful TH trials in 2002, this therapy for OHCA patients has experienced continued adoption. Institutions have taken the parameters outlined in early trials and adapted them to their own environments, with the creation of predetermined TH protocols being critical in improving adoption and implementation rates.[46,47] Some centers have augmented their protocols by creating specialized response teams to support primary care providers in the use of this therapy.[48]

Hypothermia treatment protocols differ from institution to institution but the overarching principles remain consistent.[49] Patients suffering from OHCA who achieve ROSC are cooled to a target temperature of 32–34°C as quickly as possible. Once the patient's temperature reaches the target range, hypothermia is maintained for 24 hours before rewarming. The rewarming phase occurs much more slowly with a goal rewarming rate no faster than 0.25–0.5°C per hour.

Multiple temperature management modalities have been developed since the advent of TH. Initial studies used externally applied cold packs or a device that circulated cold air over the patient to achieve the desired temperature.[18,19] Since that time, additional techniques have been developed that allow for greater control of the patient's temperature. In an endovascular approach, a catheter is placed within the central vasculature and chilled saline is circulated in a closed-loop system within the catheter. Blood passing over the catheter is rapidly cooled and this technique has been shown to safely, accurately and quickly reduce the patient's core temperature. External cooling modalities including ice-water immersion, circulation of cold air, and surface cooling devices have been described. The use of adhesive pads that are cooled using circulating water is commonly referenced and also has demonstrated rapid and accurate temperature management.[51] Additional modalities including cooling helmets,[52] and intranasal cooling devices[53] have been described but are less commonly used when compared to surface or endovascular methods (Box 1).

JAYPEE BROTHERS

BOX 1 Sample therapeutic hypothermia criteria and protocol developed primarily from University of Chicago Medical Center Protocol

Sample therapeutic hypothermia criteria

Inclusion criteria (all must apply)

- Cardiac arrest (any rhythm)
- Return of spontaneous circulation (ROSC)
- Comatose
  - Does not open eyes to pain
  - Does not follow commands
- Age ≥18 years old

Absolute contraindications

- Refractory shock
  - Mean arterial pressure (MAP) <65 mm Hg despite fluids and vasoactive agents
- Life-threatening coagulopathy or uncontrollable bleeding
  - Disseminated intravascular coagulation
  - Severe thrombocytopenia, liver failure
  - Vaso-occlusive disease (e.g., Hgb-SS)
- End-stage terminal illness

Relative contraindications

- Refractory ventricular arrhythmia
  - Ventricular tachycardia, ventricular fibrillation
- Pregnancy
  - Obstetrics/gynecology consultation recommended
- Significant preexisting neurologic impairment

Sample therapeutic hypothermia protocol

Target temperature: 32–34°C

- General principles
  - Proper sedation should be titrated for a Richmond Agitation Sedation score of –4 or –5
  - Oxygenation and ventilation techniques should be focused on minimizing hypoxia while also avoiding unnecessarily high oxygen concentrations
  - Cardiovascular support with fluid administration, vasoactive, medications or mechanical support e.g., intra-aortic balloon pump, should be targeted to a MAP ≥65 mm Hg
- Induction phase
  - Cooling should be initiated, as quickly as possible, following ROSC
  - Apply ice packs to axilla, groin, and neck
  - Chilled intravenous saline or chilled nasogastric saline lavage may be considered
  - Primary maintenance modality (e.g., endovascular, surface cooling) should be applied promptly
- Maintenance phase
  - Continuous monitoring of core temperature should be continued with appropriate temperature probes (e.g., bladder, orogastric/nasogastric)
  - Once target temperature range is reached, it should be maintained for 24 h
- Rewarming phase
  - Rewarming should occur no faster than 0.25–0.5°C per hour
  - Once 37°C has been achieved, temperature management should continue to prevent rebound hyperthermia

NEUROCOGNITIVE RECOVERY AND PROGNOSTICATION

Due to the complex global injury process that occurs and the multifactorial manner in which TH supports cardiac arrest survivors, early signs of meaningful neurologic recovery may take several days to manifest. Providers are often motivated to provide family members with some kind of prognostication of patient recovery early on in the treatment period. In one study, greater than 50% of patients were given a "poor or grave" prognosis while undergoing TH, with roughly one-fifth of these patients surviving with good neurologic outcomes.[54] Additional work suggested that neurologic examinations to assess for meaningful recovery may be inaccurate if performed before 72 hours after TH has been completed.[55] For this reason, it is recommended that any prognostication be cautiously withheld until at least 3 days after the completion of the TH protocol (Table 2).

TABLE 2: General guidelines for therapeutic hypothermia

What is the goal temperature?	Between 32°C and 34°C
How fast should the patient be cooled?	As fast as possible
How long should the patient remain at the target temperature?	24 h
What modality should I use to maintain hypothermia?	Many modalities have been shown to be effective with the most common strategies using endovascular or surface cooling approaches
How quickly can I rewarm the patient?	No faster than 0.25–0.5°C per hour
Can patients with an initial rhythm of asystole or PEA benefit from TH?	Yes, while the potential benefit for VF/VT patients is greater, asystole and PEA can be included
When should the care team begin to prognosticate long-term recovery?	No earlier than 72 h after the completion of the hypothermia protocol

PEA, pulseless electrical activity; TH, therapeutic hypothermia; VF, ventricular fibrillation; VT, ventricular tachycardia.

REGIONAL SYSTEMS OF POSTARREST CARE

Recognizing the many resources that postarrest patients require for optimal care, the multidisciplinary team that must be assembled to provide that care and the technical challenges associated with postarrest therapies, there is ongoing interest in the development of postarrest treatment centers. These regional centers would be structured in a similar fashion to the regional centers for

JAYPEE BROTHERS

other high-acuity, time-sensitive diseases such as trauma, ST-segment elevation myocardial infarction (STEMI) and stroke.

Initial work using the Nationwide Inpatient Sample, a large United States-based database, demonstrated varying survival data in patients suffering cardiac arrest based on hospital factors. It was found that large, urban, academic hospitals demonstrated substantially improved survival.[56] Building on these data, a few communities have developed cardiac arrest centers of excellence, in which TH as well as complementary cardiac interventions [percutaneous coronary intervention (PCI), automated implantable cardioverter defibrillator placement, intra-aortic balloon pump, etc.] are available. Transport of OHCA patients to these centers, paired with other improvements in cardiac arrest care has demonstrated improved patient survival.[57]

In Japan, hospitals may be designated as critical care medical centers in which the therapies discussed herein are available. In a large registry of approximately 10,000 OHCA patients transported to various facilities, it was demonstrated that neurologically favorable survival was substantially higher if a patient was transported to a critical care-designated facility.[58] With the documented success of centers specializing in the care of cardiac arrest patients, it is anticipated that more communities will begin to develop regional systems akin to the framework currently used from trauma, STEMI, and stroke to further improve the medical care provided to postarrest patients.

AREAS OF FUTURE RESEARCH

While the last 10–15 years has brought a considerable amount of interest and investigation into the field of cardiac arrest and postarrest therapy, a great deal of questions remain. Currently, there is great interest in determining the optimal temperature to which patients should be cooled during TH. While cooler temperatures may be more effective in blunting the global inflammation and ROS-associated damage that characterizes postarrest pathophysiology, cooler temperatures present their own side effects and technical limitations. This field of optimal target temperature is expected to receiving increasing interest in the upcoming years.

In addition to defining the optimal temperature target, investigators are also examining the link between the time to target temperature. Analogous to other life-saving, time-sensitive therapies, like PCI in STEMI and tissue plasminogen activator (tPA) in stroke, it is expected that TH will have the greatest impact when provided as quickly as possible. Attempts to correlate time to hypothermia and long-term outcomes have produced mixed results and for that reason, studies including larger patient cohorts are underway.

Closely related to the question of the timing of hypothermia is the possibility of prehospital initiation of cooling. Assuming that there is advantage to providing this therapy as quickly as possible, interest exists in initiating cooling by paramedics and first-responders. A small collection of studies has begun to look at the feasibility of this with limited results. Barriers to successful implementation include provider training and familiarity with the intervention, decisions on which cooling modalities can be successfully used in a prehospital setting and the anticipated manpower needed to initiate and maintain this treatment. Studies in large emergency medical service agencies are in process to further investigate the feasibility of prehospital hypothermia.

A critical and overarching concern for many cardiac arrest and hypothermia researchers is the great complexity associated with postarrest physiology. As described earlier, a multitude of cellular changes occur in the postarrest setting, ultimately cascading towards cell death. The ability to further parse these biochemical and cellular pathways and to understand which are most relevant will be critical in development of better therapies. While TH remains the most successful intervention for postarrest survivors, it is, in its essence, a blunt tool. As complex as is the cellular response to ischemia/reperfusion injury,[45] the effect of hypothermia is just of multifaceted and opaque. Better understanding at the cellular level of the processes involved may lead to more targeted interventions aimed at the central biochemical pathways.

CONCLUSION

Affecting over 300,000 patients in the United States each year, OHCA is associated with significant mortality and morbidity. The complex biochemical pathways that are associated with the ischemia/reperfusion injury of this disease are collectively known as the postcardiac arrest syndrome. Although few interventions exist for this disease, TH has proven to reduce mortality and allow for a greater degree of meaningful neurologic recovery. Several modalities including endovascular and surface cooling techniques exist but most institutions in the United States follow a similar framework for the TH protocols. Patients with any initial rhythm can be cooled to a targeted temperature of 32–34°C, as rapidly as possible, and maintained at this level for 24 hours. Support for the postarrest patient is increasing, being provided as regional centers that specialize in this complex care. Because of this therapy, a growing community of cardiac arrest survivors exists. With a great deal of research still needed to further develop this therapy and other interventions for postcardiac arrest syndrome, this field represents an exciting area of discovery.

JAYPEE BROTHERS

REFERENCES

1. Mozaffarian D, Benjamin EJ, Go AS, Arnett DK, Blaha MJ, Cushman M, et al. Heart disease and stroke statistics--2015 update: a report from the American Heart Association. Circulation. 2015;131(4):e29-322.
2. Nichol G, Thomas E, Callaway CW, Hedges J, Powell JL, Aufderheide TP, et al. Regional variation in out-of-hospital cardiac arrest incidence and outcome [published correction appears in JAMA. 2008;300:1763]. JAMA. 2008;300:1423-31.
3. Merchant RM, Yang L, Becker LB, Berg RA, Nadkarni V, Nichol G, et al. Incidence of treated cardiac arrest in hospitalized patients in the United States. Crit Care Med. 2011;39:2401-6.
4. Centers for Disease Control and Prevention. 2013 Cardiac Arrest Registry to Enhance Survival (CARES) National Summary Report. Available from: https://mycares.net/sitepages/uploads/2014/2013CARESNationalSummaryReport.pdf.
5. Safar P. Resuscitation after brain ischemia. In: Grenvik A, Safar P (Eds). Brain Failure and Resuscitation. New York: Churchill Livingstone; 1981. pp. 155-84.
6. Weil MH, Tang W. CPR: Resuscitation of the Arrested Heart. Philadelphia: W.B. Saunders Co.; 1999.
7. Sasson C, Rogers MA, Dahl J, Kellermann AL. Predictors of survival from out-of-hospital cardiac arrest: a systematic review and meta-analysis. Circ Cardiovasc Qual Outcomes. 2010;3:63-81.
8. Drennan IR, Lin S, Sidalak DE, Morrison LJ. Survival rates in out-of-hospital cardiac arrest patients transported without prehospital return of spontaneous circulation: an observational cohort study. Resuscitation. 2014;85(11):1488-93.
9. Weisfeldt ML, BeckerLB. Resuscitation after cardiac arrest: a 3-phase time-sensitive model. JAMA. 2002;288(23):3035-8.
10. Nolan JP, Neumar RW, Adrie C, Aibiki M, Berg RA, Bottiger BW, et al. Post-cardiac arrest syndrome: Epidemiology, pathophysiology, treatment, and prognostication. A Scientific Statement from the International Liaison Committee on Resuscitation; the American Heart Association Emergency Cardiovascular Care Committee; the Council on Cardiovascular Surgery and Anesthesia; the Council on Cardiopulmonary, Perioperative, and Critical Care; the Council on Clinical Cardiology; the Council on Stroke. Resuscitation. 2008;79:350-79.
11. Vanden Hoek TL, Shao Z, Li C, Zak R, Schumacker PT, Becker LB. Reperfusion injury in cardiac myocytes after simulated ischemia. Am J Physiol. 1996;270:H1334-41.
12. Kristián T, Siesjö BK. Calcium in ischemic cell death. Stroke. 1998;29: 705-18.
13. Loor G, Kondapalli J, Iwase H, Chandel NS, Waypa GB, Guzy RD, et al. Mitochondrial oxidant stress triggers cell death in simulated ischemia-reperfusion. Biochim Biophys Acta. 2011;1813(7):1382-94.
14. Vanden Hoek TL, Qin Y, Wojcik K, Li CQ, Shao ZH, Anderson T, et al. Reperfusion, not simulated ischemia, initiates intrinsic apoptosis injury in chick cardiomyocytes. Am J Physiol Heart Circ Physiol. 2003;284(1):H141-50.
15. Laver S, Farrow C, Turner D, Nolan J. Mode of death after admission to an intensive care unit following cardiac arrest. Intensive Care Med. 2004;30:2126-8.
16. Field JM, Hazinski MF, Sayre MR, Chameides L, Schexnayder SM, Hemphill R, et al. Part 1: executive summary: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2010;122:S640-56.
17. Peberdy MA, Callaway CW, Neumar RW, Geocadin RG, Zimmerman JL, Donnino M, et al. Part 9: post-cardiac arrest care: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2010;122:S768-86.
18. Hypothermia after Cardiac Arrest Study Group. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. N Engl J Med. 2002;346:549-56.
19. Bernard SA, Gray TW, Buist MD, Jones BM, Silvester W, Gutteridge G, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. N Engl J Med. 2002;346:557-63.
20. Holzer M, Bernard SA, Hachimi-Idrissi S, RoineRO, SterzF, Müllner M, et al. Hypothermia for neuroprotection after cardiac arrest: Systematic review and individual patient data meta-analysis. Crit Care Med. 2005;33(2):414-8.
21. González-Ibarra FP, Varon J, López-Meza EG. Therapeutic hypothermia: critical review of the molecular mechanisms of action. Front Neurol. 2011;2:4.
22. Sakoh M, Gjedde A. Neuroprotection in hypothermia linked to redistribution of oxygen in brain. Am J Physiol Heart Circ Physiol. 2003;285(1):H17-25.
23. Illievich UM, Zornow MH, Choi KT, Scheller MS, Strnat MA. Effects of hypothermic metabolic suppression on hippocampal glutamate concentrations after transient global cerebral ischemia. Anesth Analg. 1994;78:905-11.
Ooboshi H, Ibayashi S, Takano K, Sadoshima S, Kondo A, Uchimura H, et al. Hypothermia inhibits ischemia-induced efflux of amino acids and neuronal damage in the hippocampus of aged rats. Brain Res. 2000;884:23-30.
25. Bargonetti J, Manfredi JJ. Multiple roles of the tumor suppressor p53. Curr Opin Oncol. 2002;14:86-91.
26. Ji X, Luo Y, Ling F, Stetler RA, Lan J, Cao G, et al. Mild hypothermia diminishes oxidative DNA damage and pro-death signaling events after cerebral ischemia: a mechanism for neuroprotection. Front Biosci. 2007;12:1737-47.
27. Deng H, Han HS, Cheng D, Sun GH, Yenari MA. Mild hypothermia inhibits inflammation after experimental stroke and brain inflammation. Stroke. 2003; 34(10):2495-501.
28. Sunde K, Pytte M, Jacobsen D, Mangschau A, Jensen LP, Smedsrud C, et al. Implementation of a standardised treatment protocol for post resuscitation care after out-of-hospital cardiac arrest. Resuscitation. 2007;73:29-39.
29. Busch M, Soreide E, Lossius HM, Lexow K, Dickstein K. Rapid implementation of therapeutic hypothermia in comatose out-of-hospital cardiac arrest survivors. Acta Anaesthesiol Scand. 2006;50:1277-83
30. Kim YM, Yim HW, Jeong SH, Klem ML, Callaway CW. Does therapeutic hypothermia benefit adult cardiac arrest patients presenting with non-shockable initial rhythms?: A systematic review and meta-analysis of randomized and non-randomized studies. Resuscitation. 2012;83(2): 188-96.
31. Hachimi-Idrissi S, Zizi M, Nguyen DN, Schiettecate J, Ebinger G, Michotte Y, et al. The evolution of serum astroglial S-100 beta protein in patients with cardiac arrest treated with mild hypothermia. Resuscitation. 2005;64(2): 187-92.
32. Laurent I, Adrie C, Vinsonneau C, Cariou A, Chiche JD, Ohanessian A, et al. High-volume hemofiltration after out-of-hospital cardiac arrest: a randomized study. J Am Coll Cardiol. 2005;46(3):432-7.
33. Busto R, Dietrich WD, Globus MY, Ginsberg MD. Postischemic moderate hypothermia inhibits CA1 hippocampal ischemic neuronal injury. Neurosci Lett. 1989;101:299-304.
34. Kuboyama K, Safar P, Radovsky A, Tisherman SA, Stezoski SW, Alexander H. Delay in cooling negates the beneficial effect of mild resuscitative cerebral hypothermia after cardiac arrest in dogs: a prospective, randomized study. Crit Care Med. 1993;21(9):1348-58.
35. Markgraf CG, Clifton GL, Moody MR. Treatment window for hypothermia in brain injury. J Neurosurg. 2001;95(6):979-83.
36. Alam HB, Chen Z, Honma K, Koustova E, Querol RI, Jaskille A, et al. The rate of induction of hypothermic arrest determines the outcome in a Swine model of lethal hemorrhage. J Trauma. 2004;57(5):961-9.
37. Shao ZH, Chang WT, Chan KC, Wojcik KR, Hsu CW, Li CQ, et al. Hypothermia-induced cardioprotection using extended ischemia and early reperfusion cooling. Am J Physiol Heart Circ Physiol. 2007;292(4):H1995-2003.
38. Zhao H, Steinberg G. Limited Therapeutic Time Windows of Mild-to-Moderate Hypothermia in a Focal Ischemia Model in Rat. Stroke Res Treat. 2011;2011:131834
39. Che D, Li L, Kopil CM, Liu Z, Guo W, Neumar RW. Impact of therapeutic hypothermia onset and duration on survival, neurologic function, and neurodegeneration after cardiac arrest. Crit Care Med. 2011;39(6):1423-30.
40. Nielsen N, Hovdenes J, Nilsson F, Rubertsson S, Stammet P, Sunde K, et al. Outcome, timing and adverse events in therapeutic hypothermia after out-of-hospital cardiac arrest. Acta Anaesthesiol Scand. 2009;53(7):926-34.
41. Wolff B, Machill K, Schumacher D, Schulzki I, Werner D. Early achievement of mild therapeutic hypothermia and the neurologic outcome after cardiac arrest. Int J Cardiol. 2009;133(2):223-8.
42. Sendelbach S, Hearst MO, Johnson PJ, Unger BT, Mooney MR. Effects of variation in temperature management on cerebral performance category scores in patients who received therapeutic hypothermia post cardiac arrest. Resuscitation. 2012;83(7):829-34.
43. Nielsen N, Wetterslev J, Cronberg T, Erlinge D, Gasche Y, Hassager C, et al. Targeted temperature management at 33°C versus 36°C after cardiac arrest. N Engl J Med. 2013;369(23):2197-206.
44. Peacock WF, Deal NS. Is time to cooling target temperature important? Crit Pathw Cardiol. 2014;13(2):82-3.
45. Adrie C, Adib-Conquy M, Laurent I, Monchi M, Vinsonneau C, Fitting C, et al. Successful cardiopulmonary resuscitation after cardiac arrest as a "sepsis-like" syndrome. Circulation. 2002;106(5):562-8.
46. RittenbergerJC, Guyette FX, Tisherman SA, DeVita MA, Alvarez RJ, Callaway CW. Outcomes of a hospital-wide plan to improve care of comatose survivors of cardiac arrest. Resuscitation. 2008;79(2):198-204.

JAYPEE BROTHERS

47. Sunde K, Pytte M, Jacobsen D, Mangschau A, Jensen LP, Smedsrud C, et al. Implementation of a standardized treatment protocol for post resuscitation care after out-of-hospital cardiac arrest. Resuscitation. 2007;73(1):29-39.
48. Deal NS, Sharp WW, Orbelyan GA, Borak MH, Friant J, Shah AP, et al. The emergency cardiac arrest response team (eCART): a novel strategy for improving therapeutic hypothermia utilization following out-of-hospital cardiac arrest. Resuscitation. 2014;85(12):1775-8.
49. Center for Resuscitation Science. University of Pennsylvania. (2014). Hypothermia Protocols. [online] Available from: https://www.med.upenn.edu/resuscitation/hypothermia/protocols.shtml [Accessed Jan, 2016].
50. Gillies MA, Pratt R, Whiteley C, Borg J, Beale RJ, Tibby SM. Therapeutic hypothermia after cardiac arrest: a retrospective comparison of surface and endovascular cooling techniques. Resuscitation. 2010;81(9):1117-22.
51. Shinada T, Hata N, Yokoyama S, Kobayashi N, Tomita K, Shirakabe A, et al. Usefulness of a surface cooling device (Arctic Sun®) for therapeutic hypothermia following cardiac arrest. J Cardiol. 2014;63(1):46-52.
52. Hachimi-Idrissi S, Corne L, Ebinger G, Michotte Y, Huyghens L. Mild hypothermia induced by a helmet device: a clinical feasibility study. Resuscitation. 2001; 51(3):275-81.
53. Castren M, Nordberg P, Svensson L, Taccone F, Vincent J-L, Desruelles D, et al. Intra-arrest transnasal evaporative cooling: a randomized, prehospital, multicenter study (PRINCE: Pre-ROSC IntraNasal Cooling Effectiveness). Circulation. 2010;122(7):729-36.
54. Perman SM, Kirkpatrick JN, Reitsma AM, Gaieski DF, Lau B, Smith TM, et al. Timing of neuroprognostication in post-cardiac arrest therapeutic hypothermia. Crit Care Med. 2012;40(3):719-24.
55. Al Thenayan E, Savard M, Sharpe M, Norton L, Young B. Predictors of poor neurologic outcome after induced mild hypothermia following cardiac arrest. Neurology. 2008;71(19):1535-7.
56. Carr BG, Goyal M, Band RA, Gaieski DF, Abella BS, Merchant RM, et al. A national analysis of the relationship between hospital factors and post-cardiac arrest mortality. Intensive Care Med. 2009;35(3):505-11.
57. Lurie K, Steinkamp J, Lick C, Aufderheide T, Sayre M, White L, et al. Take Heart AmericaTM: a community-based sudden cardiac arrest survival initiative is saving lives by implementing the most highly recommended 2005 American Heart Association resuscitation guidelines. Circulation. 2008;118:S1464.
58. Kajino K, Iwami T, Daya M, Nishiuchi T, Hayashi Y, Kitamura T, et al. Impact of transport to critical care medical centers on outcomes after out-of-hospital cardiac arrest. Resuscitation. 2010;81(5):549-54.

JAYPEE BROTHERS

information on cardiology books, visit our website www.ejaypee.com, for detailed information on cardiology books, visit our website www.ejaypee.com, for detailed

Textbook of EMERGENCY CARDIOLOGY

Key Features

- In summary, a must-read reference book with information that is useful in every day clinical practice for cardiologists
- Provides easy access to acute cardiovascular problems incorporating both their diagnosis and management
- Special emphasis on common cardiac conditions, including coronary artery disease, arrhythmias, valvular and congenital heart disease as well as acute heart failure
- Provides essential advice for the rapid diagnosis and management of cardiac emergencies both common and rare.

Alan S Maisel MD
Professor of Medicine
Director, CCU and HF Program
Department of Medicine and Cardiology
VA Medical Center, University of California
San Diego, California, USA

W Frank Peacock MD FACEP FACC
Professor, Emergency Medicine
Associate Chair and Research Director
Baylor College of Medicine
Houston, Texas, USA

Shelving Recommendation
CARDIOLOGY

ISBN 978-93-86322-24-1
9 789386 322241

Buy from ejaypee

JAYPEE BROTHERS