Challenging and Emerging Conditions in Emergency Medicine

Edited by Arvind Venkat, MD, FACEP, Allegheny General Hospital, Pittsburgh, PA, and Drexel University College of Medicine, Philadelphia, PA, USA

With growing numbers of chronically ill patients surviving longer and receiving novel medical and surgical treatments, emergency departments are increasingly the venue for associated acute presentations. How can emergency physicians respond to these challenging and emerging conditions?

This book focuses on the unusual and complex disease presentations not covered in detail in the standard textbooks, helping you manage patients with conditions such as congenital heart disease, cystic fibrosis, morbid obesity, intellectual disability and intestinal failure.

Not only does this book provide guidance on evaluation and diagnosis, but it also addresses the practical issues of acute management and continuing referral. The individual chapters are written by high profile emergency physicians, in conjunction with appropriate specialists, and include authoritative evidence to back up the clinical information.

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Challenging and Emerging Conditions in Emergency Medicine
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Edited by

Arvind Venkat, MD, FACEP
Director of Research
Department of Emergency Medicine
Ethics Consultant
Allegheny General Hospital
Pittsburgh, PA;
Associate Professor of Emergency Medicine
Drexel University College of Medicine
Philadelphia, PA
USA
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List of contributors

Jonathan S. Anderson, MD
Attending Physician
Department of Emergency Medicine
Beth Israel Deaconess Medical Center and Milton Hospital;
Instructor in Medicine (Emergency Medicine)
Division of Emergency Medicine
Harvard Medical School
Boston, MA
USA

Melissa B. Bagloo, MD
Fellow
Section of Laparoscopic and Bariatric Surgery
Department of Surgery
Weill Medical College of Cornell University
New York Presbyterian Hospital
New York, NY
USA

Andra L. Blomkalns, MD, FACEP
Vice Chair, Academic Affairs
Associate Professor of Emergency Medicine
Department of Emergency Medicine
University of Cincinnati College of Medicine
Cincinnati, OH
USA

Clifton W. Callaway, MD, PhD, FACEP
Associate Professor and Executive Vice Chair of Emergency Medicine
Ronald D. Stewart Endowed Chair in Emergency Medicine
Department of Emergency Medicine
University of Pittsburgh School of Medicine
Pittsburgh, PA
USA
List of contributors

**Esther K. Choo, MD, MPH, FACEP**  
Attending Physician  
Department of Emergency Medicine  
Rhode Island Hospital and The Miriam Hospital;  
Assistant Professor of Emergency Medicine  
Department of Emergency Medicine  
Warren Alpert Medical School of Brown University  
Providence, RI  
USA

**Craig R. Cohen, MD, FACC**  
Medical Director  
Arizona Pediatric Cardiology Consultants  
Phoenix, AZ  
USA

**Moira Davenport, MD**  
Director of Simulation  
Associate Residency Program Director  
Department of Emergency Medicine  
Attending Physician  
Division of Sports Medicine  
Department of Orthopedic Surgery  
Allegheny General Hospital  
Pittsburgh, PA;  
Assistant Professor of Emergency Medicine and Clinical Instructor of Orthopedics  
Drexel University College of Medicine  
Philadelphia, PA  
USA

**Ankur A. Doshi, MD, FACEP**  
Attending Physician  
Department of Emergency Medicine  
UPMC Presbyterian and Mercy Hospitals;  
Assistant Professor of Emergency Medicine  
Department of Emergency Medicine  
University of Pittsburgh School of Medicine  
Pittsburgh, PA  
USA
List of contributors

Shamai A. Grossman, MD, MS
Attending Physician
Department of Emergency Medicine
Beth Israel Deaconess Medical Center;
Assistant Professor of Medicine (Emergency Medicine)
Division of Emergency Medicine
Harvard Medical School
Boston, MA
USA

Ward Hagar, MD
Director, Adult Sickle Cell Center
Children’s Hospital and Research Center
Oakland, CA
USA

Mary Ann Howland, PharmD, DABAT, FAACT
Clinical Professor of Pharmacy
College of Pharmacy
St. John’s University;
Adjunct Professor of Emergency Medicine
New York University School of Medicine
Bellevue Hospital Center and
New York University Langone Medical Center;
Senior Consultant in Residence
New York City Poison Center
New York, NY
USA

Kara M. Iskyan, MD, MPH
Clinical Faculty
Department of Emergency Medicine
Maricopa Medical Center
Phoenix, AZ
USA

Elan Jeremitsky, MD, FACS
Attending Physician
Division of Trauma Surgery
Department of Surgery
Allegheny General Hospital
Pittsburgh, PA
USA
List of contributors

Daniel M. Lindberg, MD, FACEP
Attending Physician
Department of Emergency Medicine
Brigham and Women’s Hospital;
Instructor in Medicine (Emergency Medicine)
Division of Emergency Medicine
Harvard Medical School
Boston, MA
USA

Joseph B. Miller, MD
Senior Staff Physician
Department of Emergency Medicine
Henry Ford Hospital;
Clinical Assistant Professor of Emergency Medicine
Wayne State University School of Medicine
Detroit, MI
USA

Claudia R. Morris, MD, FAAP
Attending Physician
Director of Fellowship Research
Department of Emergency Medicine
Children’s Hospital and Research Center
Oakland, CA
USA

John M. O’Neill, MD
Assistant Director, Combined Emergency Medicine/Internal Medicine Residency Program
Trauma Liaison
Department of Emergency Medicine
Attending Physician
Department of Medicine
Allegheny General Hospital
Pittsburgh, PA;
Clinical Instructor of Emergency Medicine
Drexel University College of Medicine
Philadelphia, PA
USA
List of contributors

Joseph M. Pilewski, MD
Co-Director, Adult Cystic Fibrosis Program
Antonio J and Janet Palumbo Cystic Fibrosis Center
Children’s Hospital of Pittsburgh of UPMC;
Associate Professor of Medicine, Cell Biology,
Physiology and Pediatrics
University of Pittsburgh School of Medicine
Pittsburgh, PA
USA

Alfons Pomp, MD, FACS, FRCSC
Leon C. Hirsch Professor of Surgery
Vice Chair for Quality and Patient Safety
Department of Surgery
Chief
Section of Laparoscopic and Bariatric Surgery
Weill Medical College of Cornell University
New York Presbyterian Hospital
New York, NY
USA

Jeffrey A. Rudolph, MD
Attending Physician
Children’s Hospital of Pittsburgh of UPMC;
Assistant Professor of Pediatrics
Division of Pediatric Gastroenterology
Department of Pediatrics
University of Pittsburgh School of Medicine
Pittsburgh, PA
USA

Richard A. Saladino, MD
Medical Director
Emergency Department
Children’s Hospital of Pittsburgh of UPMC;
Associate Professor of Pediatrics
Chief
Division of Pediatric Emergency Medicine
Department of Pediatrics
University of Pittsburgh School of Medicine
Pittsburgh, PA
USA
List of contributors

David W. Silver
Resident
Department of Emergency Medicine
University of Cincinnati College of Medicine
Cincinnati, OH
USA

Lauren T. Southerland, MD
Resident
Division of Emergency Medicine
Department of Surgery
Duke University School of Medicine
Durham, NC
USA

Sukhjit S. Takhar, MD
Attending Physician
Department of Emergency Medicine
Brigham and Women’s Hospital;
Instructor in Medicine (Emergency Medicine)
Division of Emergency Medicine
Harvard Medical School
Boston, MA
USA

Victoria L. Thornton, MD, MBA, FACEP
Director, Pain Management and Palliative Care
in Emergency Medicine
Attending Physician
Department of Emergency Medicine
Duke University Medical Center;
Assistant Professor of Surgery (Emergency Medicine)
Division of Emergency Medicine
Department of Surgery
Duke University School of Medicine
Durham, NC
USA
List of contributors

**Arvind Venkat, MD, FACEP**
Director of Research
Department of Emergency Medicine
Ethics Consultant
Allegheny General Hospital
Pittsburgh, PA;
Associate Professor of Emergency Medicine
Drexel University College of Medicine
Philadelphia, PA
USA

**K.K. Venkat, MD**
Senior Staff Physician
Division of Nephrology and Hypertension
Department of Medicine
Henry Ford Hospital;
Clinical Associate Professor of Medicine (Nephrology)
Wayne State University School of Medicine
Detroit, MI
USA

**Melissa A. Vitale, MD**
Attending Physician
Children’s Hospital of Pittsburgh of UPMC;
Assistant Professor of Pediatrics
Division of Pediatric Emergency Medicine
Department of Pediatrics
University of Pittsburgh School of Medicine
Pittsburgh, PA
USA
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CHAPTER 1

Introduction

Arvind Venkat
Allegheny General Hospital, Pittsburgh, PA, USA; Drexel University College of Medicine, Philadelphia, PA, USA

The emergency department (ED) serves as the gateway for medical care for the preponderance of acutely ill patients. Whether due to medical, surgical, pediatric, obstetric, neurologic, or psychiatric conditions, patients presenting with acute ailments expect that the ED and emergency physicians in particular will be able to diagnose and initiate management of critical conditions. In the United States, as of 2007, there were 117 million annual visits to the ED with 39.4 visits/100 persons [1]. Worldwide, there has been increasing recognition of the need for quality emergency care and the resultant recognition of emergency medicine as a medical specialty in nations as diverse as India, Turkey, and Malaysia. With this explosive growth in emergency care, it is increasingly common for patients to view the ED as the location for entrance into the health care system when confronted with unexpected and severe medical complaints.

This recognition of the ED is well warranted, but it does create a dilemma for emergency physicians who in their practice must be aware of the vast complexities of ailments that can cause patients to present for emergency care. While emergency physicians are clearly well trained to deal with the most common diseases that require emergency interventions, such as cardiovascular disease and trauma, providers in the ED must now become facile with managing patients whose disease entities are either only now being recognized and treated or whose therapies have only recently been developed. During a typical clinical shift, an emergency physician may have to manage acute issues in patients whose co-morbid illnesses may include transplantation, congenital heart disease, end-stage renal disease or cancer. Without awareness of the new treatments and procedures in these areas as well as the implications of increased longevity in patients who previously may have never required emergency care in the past, it is
easily foreseeable that emergency physicians may not correctly diagnose and initiate treatment in conditions that require acute intervention with resultant detriment to the patient.

At the same time, the literature and educational process in emergency medicine has understandably largely focused upon patients who present most commonly for ED care. Research in emergency medicine largely, though not exclusively, focuses on the most prevalent conditions, such as acute coronary syndromes, pulmonary embolism, stroke, trauma, and sepsis, while textbooks in emergency medicine are largely comprehensive surveys of the entire gamut of diseases that can cause presentation to the ED. Similarly, the core curriculum in emergency medicine for residency training in the United States attempts to cover the entire range of conditions to the ED, but in the process does not allow for more in-depth consideration by trainees of patient populations that are either on the horizon or whose therapies are quickly evolving to result in increased longevity and changed pathophysiology.

This book attempts to address this educational need for emergency physicians to understand patient populations whose ailments either are being treated in new ways or to rectify a lack of common recognition both in diagnosis and the implications of increasing longevity. In selecting topics for inclusion, three themes emerge that underline the challenge facing emergency physicians.

**Increased longevity**

As seen in the chapters on adults with congenital heart disease, the geriatric trauma patient, adults with cystic fibrosis, the intellectually disabled patient, adults with sickle cell disease, and children with intestinal failure, evolving medical care and understanding of the pathophysiology of disease has resulted in a vast improvement in the life expectancy of patients who previously have not survived to adulthood or whose survival to late adulthood has resulted in their exposure to illnesses that will now require ED care. For emergency physicians, this increased longevity will result in the need to reconsider the pathologic processes that can result in illness as well as new complications of late-stage disease. For example, survival to adulthood of patients with congenital heart disease means that emergency physicians will have to recognize the late complications of surgical procedures that were used to correct these defects in infancy as well as the late cardiovascular and pulmonary issues that may not arise until adulthood. The aging of the general population means that emergency physicians will have to understand the more complex pathophysiology of trauma when interacting with other age-related illnesses. Children with intestinal failure may now survive for longer periods of time and present with complications that were only seen in the past in specialized centers shortly after
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For all the patient populations discussed in these chapters, the underlying theme is that the emergency physicians have to conceive of these patients as surviving well beyond what was previously recognized in day-to-day medical practice and consider how that may cause these individuals to present with novel complications not seen in the past.

Novel treatment modalities

As seen in the chapters on the bariatric surgery patient, HIV-positive adults on highly active antiretroviral therapy, emergency complications of chemotherapeutic regimens, the post-cardiac arrest patient, renal dialysis patients and renal transplant patients, evolving medical and surgical care for patients who previously either had different or ineffective treatment modalities has resulted in emergency complications that require recognition by ED providers. Such treatments have often provided wonderful benefits to these patient populations in terms of quality of life and longevity, but have made the ED the venue in which acute diagnosis of treatment failures or complications will take place. For example, the astromic growth of bariatric surgical procedures requires emergency physicians to recognize the resultant anatomic and physiological changes that take place post-operatively and the side effects and treatment issues that can arise. The increased longevity of HIV-positive adults on highly active antiretroviral therapy has resulted in completely new disease processes that more commonly affect this patient population. With the development of hypothermia treatment post-cardiac arrest, emergency physicians are being called upon to manage patients previously thought to be neurologically devastated in a novel and potentially life-changing way. For all these patient populations, the underlying theme is that new and evolving therapies have created a novel set of disease processes and treatments with which emergency physicians must become familiar.

Complications of social pathologies and lack of medical resources

As seen in the chapters on conditions causing chronic pain, family violence, and the obese patient, the ED also serves as the “canary in the mine” for pathologies that often extend beyond the medical realm [2]. To some extent, this may be seen as the dark side of the increased recognition of the ED as the gateway to the health care system. As such, emergency physicians now must contend with the consequences of failures in our medical system and complexities that result from the breakdown in family relationships or societal forces well beyond their control. For example, the growth in the number of patients with conditions that cause chronic pain coupled with a lack of medical training in pain management and a shortage
of pain management physicians has left the ED as the venue of last resort for patients who require analgesia, perhaps best managed ideally in the outpatient setting. Increased recognition of child abuse and intimate partner violence has imposed a burden on emergency physicians to treat the medical and social dangers imposed by these conditions. The epidemic of obesity has profound implications for the diagnostic assessment and therapeutic management of patients in the ED. Together, these emerging patient populations represent a profound challenge for emergency care in the twenty-first century.

Chapters in this book are structured so that the reader will have an understanding of the epidemiology, procedural interventions, and disease presentation and management in these patient populations in the ED. Each chapter concludes with a section entitled “The next five years” which is meant to provide the reader with a prediction of where these fields will likely evolve in the near future and the implications of those changes for emergency practice. The contributing authors to this book and I hope that the reader will find that this serves as a starting point for consideration in training programs and clinical EDs as to how best to address the numerous challenging and emerging conditions that will cause patients to present for emergency care.

References

CHAPTER 2
The post-cardiac arrest patient

Ankur A. Doshi1,2 and Clifton W. Callaway2
1UPMC Presbyterian and Mercy Hospitals, Pittsburgh, PA, USA
2University of Pittsburgh School of Medicine, Pittsburgh, PA, USA

Introduction
Heart disease is the leading cause of death in the industrialized world [1]. Consequently, the presentation of end-stage heart disease—cardiac arrest—is well known to emergency physicians. Similarly, emergency providers, both in the prehospital setting and in the emergency department (ED), are well versed in the treatment algorithms for patients during cardiac arrest. Over the past 35 years, organizations such as the American Heart Association (AHA) and International Liaison Committee on Resuscitation (ILCOR) have developed recommendations for care of patients in cardiac arrest [2, 3]. These references, including Advanced Cardiovascular Life Support (ACLS), provide standardized care of patients in cardiac arrest. Even though the baseline characteristics of patients in cardiac arrest are fairly uniform, the rates of survival for these patients still vary geographically [4]. Moreover, from the 1970s through the early 2000s, despite a variety of newly researched and implemented interventions, there was no change in long-term survival of cardiac arrest patients [5–10].

In the past 10 years, scientists have begun to better describe the pathophysiology of cardiac arrest leading to research that has demonstrated that physiologic derangements occur not only during but also after cardiac arrest [11]. Consequently, clinicians have begun to recognize the need to coordinate care of patients during and after cardiac arrest to maximize patients’ survival [11]. In many cases, early, aggressive treatment directed at the specific pathology after cardiac arrest (post-cardiac arrest care) is essential to allow patients the maximum likelihood of beneficial neurological outcomes [11]. This strategy of beginning post-cardiac arrest care promptly is now advocated by guidelines published by the AHA and ILCOR such as...
ACLS [2, 3]. Yet, less than 20% of US emergency physicians have treated patients with post-cardiac arrest care [12]. This chapter outlines the evidence supporting aggressive post-cardiac arrest care in the ED, protocols for performing efficient post-cardiac arrest resuscitation in the acute setting, and future directions in the evolution of care of the post-cardiac arrest patient.

**Epidemiology and pathophysiology**

An estimated two-thirds of US citizens are at high lifetime predicted risk for atherosclerotic cardiovascular disease [13]. Consequently, cardiovascular disease was the cause of one in six deaths in the United States in 2006 [1]. The end point of cardiovascular disease is sudden cardiac arrest, which most often occurs in the out-of-hospital setting [14]. The incidence of out-of-hospital cardiac arrest is estimated to range from 55 to 120 events per 100,000 persons per year [14, 15]. A recent North American sample demonstrated the median incidence of out-of-hospital cardiac arrest to be 52.1 events per 100,000 persons per year. The mean survival in this cohort was 8.4% [4]. As expected, the demographics of cardiac arrest mirror those of other coronary heart disease. The mean age for patients with sudden cardiac arrest is between 65 and 70 years of age, and death from sudden cardiac arrest is more common in men than women [4, 14]. Patients with ventricular fibrillation arrests, those who received bystander CPR (cardiopulmonary resuscitation), and those with rapid return of spontaneous circulation, survive at a greater rate than those who do not meet these criteria [11]. However, there is great variability in survival, with some regions of North America reporting overall survival after out-of-hospital cardiac arrest to be greater than 15% and others reporting survival of less than 2% [4]. This variation persists even after controlling for patient and resuscitation variables, such as witnessed collapse, bystander CPR, ambulance response times, and initial rhythm [4]. Part of this variation may be explained by differing ED and in-hospital care [16].

The causes of death for patients after cardiac arrest can be broadly divided into two categories—“cardiac death” and “neurological death.” Cardiac death is due to intrinsic cardiac failure, either the inability to restart spontaneous cardiac contraction or the inability to maintain systemic perfusion after significant myocardial damage. Neurological death is due to accumulated cellular damage to the central nervous system (CNS). Standard care of the cardiac arrest patient prior to 2002 focused only on the restoration of circulation and did not address the continued pathology of cardiac arrest after return of spontaneous circulation [17]. For the past 30 years, despite newer medications and devices to treat out-of-hospital cardiac arrest, only approximately one-third of patients have return of spontaneous circulation long enough to be admitted to the hospital [14]. Almost by
The post-cardiac arrest patient

definition, patients who do not survive to hospital admission are considered to have cardiac death [11].

Of the out-of-hospital cardiac arrest patients who survive to hospital admission, another two-thirds will die prior to hospital discharge. Although some patients develop secondary cardiac failure or other complications of severe illness, the primary etiology of in-hospital mortality is severe neurological injury [18,19]. The CNS cellular damage in this group is not simply due to ischemic cell necrosis but also due to reperfusion injury. Reperfusion injury is a second wave of cellular damage that is characterized by dysregulation of CNS protective mechanisms and plays out for hours to days after return of spontaneous circulation. Consequently, the previous treatment of cardiac arrest, as limited to the achievement of return of spontaneous circulation, did not address this secondary neurological injury [11]. Although CNS reperfusion injury had been identified for a number of years, until recently, no therapy had been found to minimize its effect. Randomized trials tested treatment such as calcium-channel blockers, benzodiazepines, and even specific antibodies without demonstrating benefit in humans [20–22]. The first successful clinical trials demonstrating successful treatment of CNS reperfusion injury after out-of-hospital cardiac arrest were published in 2001 and 2002 [23–25]. These three trials evaluated the use of induced therapeutic hypothermia (ITH) in patients after return of spontaneous circulation from out-of-hospital cardiac arrest.

Over the past 20 years, ITH has been used to treat neurologic disorders, such as traumatic brain injury and stroke, and has been shown to be of benefit to prevent brain injury during cardiac bypass surgery [26–28]. ITH is theorized to minimize CNS reperfusion injury via a number of mechanisms. Possibilities include decreasing cerebral metabolism, reducing brain edema, and therefore, increasing perfusion, diminishing free radical production, suppressing neuroexcitatory toxins, controlling apoptosis, improving brain glucose metabolism, and reducing seizures. This multifactorial effect on the CNS is likely why ITH has shown benefit, when other specific therapies have not [11].

The three original cardiac arrest ITH trials demonstrated that the use of ITH after out-of-hospital cardiac arrest could significantly increase patient survival to hospital discharge. A meta-analysis of these studies showed an almost 30% relative risk reduction for death or poor neurological outcome in survivors of cardiac arrest treated with ITH [29]. Early use of ITH (within the first 8 hours of cardiac arrest) increased patients’ absolute survival to discharge with good neurologic functioning by 16% [24,25,29]. Additionally, the early use of ITH resulted in the same magnitude of benefit at 6 months post-arrest [24]. A number of further studies have confirmed this benefit [30, 31]. On the basis of these data, the AHA and ILCOR recommend that patients after out-of-hospital cardiac arrest receive specific post-arrest management [2, 3].
Supportive treatment to minimize secondary CNS injury during reperfusion has also been associated with improved patient survival with good neurological status. These therapies have been modeled after successful treatments in the intensive care setting, such as minimizing hypoxia, hypotension, and hyperglycemia, in other critical illnesses [32–34]. Bundling these interventions with ITH into treatment pathways has been labeled as “post-cardiac arrest care.” A truly integrated protocol to neuroresuscitation for the post-cardiac arrest patient should include these interventions. There have been several small studies that have demonstrated the efficacy of integrated post-cardiac arrest care in the intensive care setting to increase patient survival [35–37].

Despite the known benefits of post-cardiac arrest care, there remains large local variability of in-hospital care after out-of-hospital cardiac arrest. For example, when similar out-of-hospital cardiac arrest patients were taken by ambulance to different hospitals, their survival to hospital discharge differed simply based upon the hospital where treatment occurred [35, 38]. This difference was postulated to be due to the different in-hospital care patients received, including the use, or lack thereof, of ITH. It is postulated, then, that regional variability in out-of-hospital cardiac arrest survival is also partially due to in-hospital care differences [16, 39]. When hospitals are polled, a majority self-report the capacity to provide post-cardiac arrest care. However, many of these institutions do not provide this type of care on a routine basis (Martin-Gill: unpublished data). Even when an institution has a post-cardiac arrest care system in place, treatment is often delayed until admission, because of the perception that such care is too time and resource intensive for the ED [12].

Indications and contraindications

There are a number of potential barriers to the use of post-cardiac arrest care in the ED. As a result, a majority of emergency physicians report that they do not provide post-cardiac arrest care routinely [12]. Yet, just as early treatment of other critical illnesses, such as stroke, myocardial infarction, or trauma, in the ED improves patient outcomes, emergent treatment of post-cardiac arrest patients can improve survival to hospital discharge [11]. Reperfusion injury of the brain begins at the moment of return of spontaneous circulation; the earlier that treatment is begun, the more the potential benefit for improved neurological outcome [11]. Therefore, post-cardiac arrest care should be initiated in the ED for all out-of-hospital cardiac arrest patients without contraindications detailed in Table 2.1.

Although no one has reported on specific barriers to post-cardiac arrest care in the ED, research has identified the barriers to the use of ITH—the most novel component of post-cardiac arrest care. A survey of North American and European physicians identified the most common barriers
Table 2.1 Contraindications to induced therapeutic hypothermia and post-cardiac arrest care

**Absolute contraindications**
- Active uncontrolled or noncompressible hemorrhage
- Do not resuscitate/do not intubate status
- Rapid neurologic improvement (i.e., following commands with 60 minutes of return of spontaneous circulation)

**Relative contraindications**
- Multisystem trauma
- Shock from GI bleed or sepsis
- Intracranial hemorrhage (unless cleared by neurosurgery)

to the use of ITH in the ED [12]. Reasons for not using ITH include the following: “not enough data” (49% of North American physicians and 41% of European physicians), “too technically difficult” (35% North American physicians and 32% European physicians), and “have not considered it” (34% North American physicians and 23% European physicians) [12].

However, new data address these concerns of emergency physicians. For example, respondents in the Merchant study indicating “not enough data” pointed to the fact that studies only demonstrated a benefit to ITH only after out-of-hospital cardiac arrest due to ventricular fibrillation or pulseless ventricular tachycardia [12]. However, there is no evidence that the pathophysiology of CNS injury from out-of-hospital ventricular fibrillation arrest is different from that of any other type of cardiac arrest. A number of case series have found a benefit to the use of ITH in all cardiac arrest patients (in-hospital and out-of-hospital) and with all initial rhythms [40–43]. Large registries have shown that ITH provides outcome benefit to patients after cardiac arrests because of non-cardiac causes, such as asphyxiation or drug overdose [36, 40, 44]. Therefore, presently, there is sufficient data to support the use of ITH, and in association, post-cardiac arrest care, after all types of cardiac arrest.

Another frequent reason why physicians do not provide ITH is that they “have not considered it” [12]. This is despite these physicians understanding the benefit of ITH after out-of-hospital cardiac arrest; 34% of North American respondents felt that the data for ITH was compelling enough to make a trial randomizing some patients to normothermia unethical. The reason for this disparity may lie in the fact that emergency physicians provide care in a scarce resource model. They attempt to provide the best care possible to the most number of patients with the available time and resources. Consequently, emergency physicians may not consider the use of hypothermia in contrast to other lifesaving interventions such as intravenous thrombolytics for acute stroke or immediate coronary intervention in the setting of ST-segment elevation myocardial infarction. In short,
many emergency physicians may feel that, despite the proven benefit of ITH after cardiac arrest, the magnitude of this benefit does not outweigh the risk incurred to other patients when ITH is initiated for cardiac arrest. Pessimism about the survivability from cardiac arrest may bias physicians into believing that aggressive post-cardiac arrest care is rarely useful. One study demonstrated that a majority (63%) of in-hospital cardiac arrest patients after return of spontaneous circulation were placed in “do not resuscitate” status. Of these patients, 43% had mechanical support actively discontinued during the hospitalization [45]. The fact that many patients after return of spontaneous circulation seem neurologically devastated may contribute to this pessimism. However, neurological assessment in the ED is not predictive of final neurological outcome after cardiac arrest [46–49]. Many patients receive medications during cardiac arrest that may make neurological assessment invalid. Atropine, for instance, will cause pupillary dilation because of its anticholinergic effect, in the absence of brain injury. Additionally, all patients after cardiac arrest suffer some degree of neurological stunning. The depth of this stunning is likely proportional to the ischemic injury suffered during the arrest. A patient with very little injury, such as one rapidly defibrillated in the field, may seem almost neurologically normal in the ED. However, even patients who demonstrate no neurological function shortly after a prolonged cardiac arrest will improve neurologically during the next 48–72 hours [49]. Because of this initial inability to predict final neurological outcome of out-of-hospital cardiac arrest patients, the benefit of post-cardiac arrest care may be less obvious to the emergency physician than the benefit from interventions for ST-segment elevation myocardial infarction or stroke.

The final barrier to care identified—that ITH is “too technically difficult”—probably has two components. First, many physicians may not feel comfortable with the mechanical steps needed to implement ITH. Additionally, physicians may be concerned about the possible side effects of ITH that could lead to worse patient outcomes. As with any new therapy or medication, there is a learning curve to the implementation of post-cardiac arrest care. Providing an algorithm for emergency physicians to manage patients after cardiac arrest may clarify both how post-cardiac arrest care can be integrated into the patient’s ED care as well as for which patients such care is contraindicated.

The algorithm we recommend is the “ABCs” of resuscitation—already well known to all emergency physicians. After return of spontaneous circulation, the emergency physician should return back to the beginning of the ABCs and ensure that the patient’s airway is secured, followed by assessments of oxygenation, ventilation, perfusion, and focal neurological disability. This algorithm helps the physician provide post-cardiac arrest care using a well-known pathway. In addition, by following this pathway, those few patients in whom post-cardiac arrest care is
contraindicated will be evident. For instance, patients in shock due to multisystem trauma, hemorrhage, or sepsis should have their ED resuscitation focused on correction of these illnesses or injuries processes prior to specialized post-cardiac arrest care. However, the presence of one of these medical problems is only a relative contraindication to treatment with post-cardiac arrest care. Similarly, a brief focused neurological assessment, with the addition of neuroimaging via CT, should identify patients with intracranial hemorrhage. If cleared by neurosurgery, these patients may still be treated with post-cardiac arrest care. Initial resuscitation may focus on correction of the intracranial hemorrhage first. Previously accepted contraindications to post-cardiac arrest care, such as pregnancy or under-18 age, do not have physiologic bases. There have been published case reports detailing successful use of ITH in these settings [50–52].

It is true that despite optimal care, a patient’s final neurological status can only be maximized to the level of functioning prior to the recent cardiac arrest. In an ED setting, therefore, it may be reasonable to withhold aggressive post-cardiac arrest care for a patient whose neurological status prior to cardiac arrest is poor. Some patients, in addition, have made their decision on the use of lifesaving technology clear prior to their event in the form of an advanced directive. As post-cardiac arrest care is a therapy that may take days to accomplish, it is reasonable for the emergency physician to not aggressively treat a post-cardiac arrest patient who has specifically declined intensive care therapy. However, in other cases, aggressive post-cardiac arrest care in the ED can make a profound difference in patients’ final neurological outcome.

Nuts and bolts

The application of post-cardiac arrest care in the ED is straightforward and requires resources that are already available in most institutions. However, some preparation is needed to ensure that the needed equipment is resourced to the ED to minimize time delay from return of spontaneous circulation to the beginning of neuroresuscitation. It is also imperative that the care provided in the ED not be labor or resource intensive. This will ensure that providers, even in a chaotic ED environment, are able to provide maximum care to the post-arrest patient without compromising care to other patients. The familiar “ABCs” algorithm is a useful framework for defining the care to be provided with some modification (Table 2.2).

Airway

The beginning of post-cardiac arrest care in the ED is an assessment of the patient’s airway. If, after return of spontaneous circulation, the patient is alert and protecting his or her own airway, further interventions may not be necessary. However, most survivors of cardiac arrest will be neurologically injured and require placement of an artificial airway. Recent
data on patients in cardiac arrest has demonstrated a significant association between the amount of time chest compressions were performed and survival [53, 54]. Endotracheal intubation often requires lengthy interruption in chest compressions [55]. Supraglottic airways, in contrast, can be more easily placed without an interruption of chest compressions [56]. For
The post-cardiac arrest patient

this reason, some local protocols have begun using these airways for rescue when endotracheal intubation cannot be accomplished or favoring immediate supraglottic airway placement over standard intubation [57, 58]. If a rescue airway or primary supraglottic airway was placed during resuscitation, it should be converted to a standard endotracheal tube in the ED, as most patients will require mechanical ventilation for at least 24 hours. Additionally, a common complication of out-of-hospital cardiac arrest is aspiration; therefore, intubation should be accomplished with an endotracheal tube large enough to allow for frequent suctioning [24, 25].

Pulmonary function

The next step in post-cardiac arrest care is an evaluation and management of pulmonary function. The patient should be placed on continuous pulse oximetry. An arterial blood gas (ABG) should be assessed to evaluate the partial pressure of oxygen (PaO₂) and carbon dioxide (PaCO₂). If possible, a continuous end-tidal carbon dioxide detector may be used as well. Immediately after return of spontaneous circulation, the CNS is extremely vulnerable to hypoxia. Therefore, ED care should ensure that adequate oxygenation is maintained throughout [32]. In addition, early hyperoxia is also associated with poorer final neurologic outcomes. Patients with initial PaO₂ less than 60 or greater than 300 had worse outcomes than those with PaO₂ between 61 and 299 [59]. Therefore, the emergency physician should titrate supplemental oxygen to avoid these extremes of blood oxygenation. To avoid repetitive ABG sampling, continuous pulse oximetry can be used to reduce oxygen levels to the lowest possible to maintain saturations >94%.

Management of ventilation is also an important component of post-cardiac arrest care. Hypocapnia decreases cerebral blood flow. Therefore, maintenance of normal PaCO₂ (between 35–45 mm Hg) is useful in preventing a secondary reduction in CNS perfusion. Initial management can be done on the basis of the ABG, with subsequent changes being performed using the end-tidal carbon dioxide monitor.

Systemic and cerebral perfusion

Thirdly, the emergency physician should continually evaluate the adequacy of circulation and CNS perfusion after return of spontaneous circulation. In the first 24 hours after return of spontaneous circulation, myocardial dysfunction is common, likely due to primary cardiac disease or coronary reperfusion injury. This cellular damage results in decreased cardiac stroke volume and decreased cardiac output and cerebral blood flow [60]. Also, during initial resuscitation, vasopressors, such as epinephrine, are often used, which artificially increase systemic blood pressure. The effect of these vasopressors is quite transient; as their effect wanes, hemodynamic compromise may occur. Without an adequate mean arterial
pressure, CNS perfusion will not be maintained [11]. After reperfusion, autoregulation of the cerebral circulation causes further decrease in cerebral blood flow due to cerebral vasoconstriction. Due to this, mean arterial pressures after return of spontaneous circulation should be kept on the higher end of normal (80–100 mm Hg) to avoid secondary CNS injury [61]. One study demonstrated that even transient hypotension in the first 2 hours following return of spontaneous circulation was associated with poor neurologic recovery [33]. Therefore, emergency care of the post-cardiac arrest patient should include frequent assessments of global perfusion. At a minimum, measurement of blood pressure should be made frequently (every 5 minutes) using noninvasive measures. Ideally, arterial cannulation should be performed to measure blood pressure continuously. Adequate fluid resuscitation is needed to maintain adequate cardiac filling pressures. Either inotropes, such as dopamine, or vasopressors, such as norepinephrine, may be needed to ensure adequate perfusion.

Other cardiac therapy within post-cardiac arrest care must include an evaluation of the need for coronary intervention. All patients after out-of-hospital cardiac arrest should have a 12-lead ECG obtained. The presence of ischemia should be aggressively managed including a low threshold for percutaneous coronary intervention. Routine cardiac catheterization may lead to increased survival from cardiac arrest, and standard criteria for direct coronary intervention, such as the presence of ST-segment elevations, should prompt immediate intervention [62, 63]. One series showed that 48% of patients with return of spontaneous circulation after cardiac arrest had some coronary artery occlusion [64]. Another study showed that 51% of resuscitated patients had either cardiac enzyme elevation or electrocardiographic evidence of acute myocardial infarction [65]. Multiple studies have shown that emergent coronary intervention with angioplasty or coronary stenting is safe in patients during ITH [66,67]. Patients in cardiogenic shock, even without ST-segment elevation, may also benefit from coronary intervention or the placement of an intra-aortic balloon pump, although the evidence for this benefit is more from correlation rather than clinical trials [63]. ITH has been used with concomitant fibrinolytic therapy after cardiac arrest and stroke, with no more bleeding than usually expected after fibrinolysis [68, 69]. Therefore, ITH should be considered for patients regardless of the reperfusion strategy selected. From the standpoint of the emergency physician, the data supports that a strong partnership with cardiology and an ability to carry out immediate coronary intervention are needed to care for post-cardiac arrest patients.

**Neuroresuscitation**

The most novel portion of post-cardiac arrest care is the focus on neuroresuscitation. The major component of this is ITH. However, other therapies, such as the use of benzodiazapine or barbiturate sedation and control of