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Out of hospital cardiac arrest: Concise review of strategies to improve outcome☆

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ABSTRACT

Despite decades of research, cardiac arrest remains a major cause of morbidity and mortality, with frustratingly poor survival rates of approximately 10% to hospital discharge. Various strategies have been shown to improve survival, but differing degrees of implementation have led to a disparity in survival rates. These improvements, however, are balanced against the increasing age of patients presenting with out of hospital cardiac arrest and decreasing incidence of ventricular fibrillation, the rhythm with the best outcome. In this review, we will summarize the most up-to-date literature on key questions in the management pathway and recommend evidence based strategies to improve care.

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1. Introduction

Out of hospital cardiac arrest (OOHCA) remains a major cause of death in the Western world. [1] Despite the remarkable improvements in survival in patients with ST elevation myocardial infarction, OOHCA survival remains frustratingly poor with overall rates of approximately 10% to hospital discharge. [1,2] Developments in the management of OOHCA patients, both in pre-hospital (bystander cardiopulmonary resuscitation (CPR) and early defibrillation), in-hospital care (targeted temperature management, cardiac assessment), and post-arrest rehabilitation, have had varying degrees of success in real world cohorts. [3] These improvements, however, are balanced against the increasing age of patients presenting with OOHCA and decreasing incidence of ventricular fibrillation, the rhythm with the best outcome. [4,5] In this publication, we focus on the management of OOHCAs from a clinician's perspective.

2. Etiology and epidemiology of cardiac arrest

Cardiac arrest is the sudden cessation of cardiac mechanical activity, due to asystole, pulseless electrical activity (PEA), or the development of ventricular fibrillation/sustained ventricular tachycardia. Wide ranging incidences of cardiac arrest data have been reported, due in part to

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differences in definitions and variability in recording cardiac arrest data. [6] In the USA, each year approximately 360,000 people develop OOHCA and overall it has been estimated to be the third leading cause of death. [6,7] In a systematic review of 67 studies, the global average incidence of OOHCAs with a presumed cardiac cause was 54.6 per 100,000 person-years. Ventricular fibrillation was the presenting rhythm in 27% of patients, with average post arrest survival of only 7%. [1] In one cohort study in America, the incidence of OOHCA was found to be higher and survival lower in Blacks than in Whites, predominantly due to a lower prevalence of ventricular fibrillation as the presenting rhythm. [8] Well-known risk factors for cardiac arrest include prior history of cardiac disease and family history in a first degree relative. In addition, those risk factors commonly associated with the development of coronary artery disease are also similar for cardiac arrest. [6].

In younger patients, structural heart disease is a common cause. For example, in an autopsy study of over 900 patients with presumed sudden cardiac death, approximately 80% were due to cardiac pathology, and coronary artery disease was the primary cause in 56.6%. However, atherosclerotic artery disease was much less common in patients <35 years (23.2% vs 73.2% [>35 years]). [9,10] Pulseless electrical activity is a less common presenting rhythm of cardiac arrest and causes include pulmonary embolism, hypoxia, hypovolemia, and myocardial ischemia. [11] Other potentially reversible causes of cardiac arrest include tension pneumothorax, electrolyte abnormalities, medication, toxins and cardiac tamponade. [12].

3. Ground zero: Strategies to improve care

The "chain of survival" concept, developed over 20 years ago, helped coordinate and standardize OOHCA protocols worldwide with the aim of improving survival. [13] It involved a series of critical steps in management: early recognition of symptoms and activation of an emergency response system; early bystander cardiopulmonary resuscitation; rapid defibrillation, if needed; early advanced cardiac life support and integrated post-resuscitation care. If these steps are implemented effectively, a larger proportion of patients achieve return of spontaneous circulation (ROSC) prior to hospital (40–60%). [14] Critical to the success of ROSC is time to defibrillation in cases of VF falling within the 5-min threshold. [15] In addition, if ROSC is achieved prior to hospital arrival, outcome improves, with nearly a quarter surviving to discharge. [16,17] Various successful public health strategies have been implemented in different healthcare systems. Specifically, the implementation of publicly available automatic external defibrillators (AEDs) and training of public bystanders to both deliver CPR and use AEDs have improved outcomes. [18] This is especially true in some parts of Europe, with bystander CPR rates of 60% and 73% in North Holland and Norway respectively. [19] In contrast, in the United States of America, a median of 2.39% of the population is CPR trained. [20] Recently, emergency non-medical first responders (e.g. police, firefighters) have been equipped and trained to use AEDs. [21,22] In addition, the use of cellular phone apps which can alert the public to find the nearest CPR trained individual have become available and has further improved rates of bystander CPR. [23].

4. Question 1: Where should the patient go?

If circulation is not restored, then the chance of success is low. In this case, it is generally recommended to take the patient to the nearest Emergency Department. However, with ROSC from a witnessed arrest [24] with initial rhythm of ventricular fibrillation [18], and with bystander CPR ongoing [25], transfer to a tertiary center capable of immediate cardiac and neurological/intensive care support is likely to produce the best survival regardless of the ECG post arrest. [3,18] This is advantageous for the patient as treatment at larger, more experienced, centers is associated with better survival. [26,27] Several factors may account for this, including familiarity with management, medical expertise and hospital facilities.

5. Question 2: To cool or not to cool?

Early initiation of targeted temperature management (TTM) is thought to be crucial in helping to prevent the neurological effects of sustained hypoperfusion and limiting tissue injury from the effects of ischemia/reperfusion. Indeed, delaying cooling increases mortality by up to 20% for each hour without initiation. [28] The most common cause of death in patients with cardiac arrest is neurological and therefore performing immediate angiography/angioplasty should not preclude the initiation of cooling and can be performed in conjunction with angioplasty by using ice packs for surface cooling, and cold IV fluid administration for intravascular temperature reduction. [28,29] Several other mechanisms are available to cool patients including intranasal spray devices and intravascular catheters. These are more complex and less available. No method however has been shown to affect outcomes more than another. [30,31] A recent large randomized trial found no difference in mortality or neurological function outcome between patients cooled to 33 °C or 36 °C. [32] A follow-up study of survivors performed at 6 months reported no significant difference in cognitive function between the two treatment groups. [33] Thus, recent American and European guidelines recommend that all comatose adult patients with ROSC after cardiac arrest should have TTM, with a target temperature between 32 °C and 36 °C for at least 24 h. The higher temperature range may be more suitable for those in whom lower temperatures convey risk (e.g. bleeding), with the lower range for those more likely to derive a benefit (e.g. seizure activity or cerebral edema). [34,35].

6. Question 3: Should all patients be assessed by cardiologists on arrival?

The implementation of a strategy of early angiography and targeted temperature management in ST-elevation myocardial infarction (STEMI) patients who have had return of circulation ROSC has mirrored a dramatic increase in survival rates with up to 60% surviving to hospital discharge, and importantly, with favorable neurological outcomes in over 80% of these survivors. [36–38] It has become clear that most patients with OOHCA presenting with ventricular fibrillation have significant coronary artery disease independent of ECG findings; therefore, it seems reasonable that cardiologists should be involved from the moment the patient reaches hospital. While the treatment recommendation of OOHCA patients presenting to hospital with ST elevation after ROSC is clear, with European guidelines recommending immediate coronary angiography with a view to primary percutaneous coronary intervention, the optimal treatment pathways of patients without ST elevation and no other clear non-cardiac cause remain less certain. [39,40] Differentiating between cardiac and non-cardiac causes, often with the lack of a clear history of chest pain and equivocal ECG changes, can be challenging. Therefore, while STEMI patients should be transported directly to the catheterization laboratory, patients without ST elevation have a short "Emergency Room" stop where they can be assessed for obvious non-cardiac etiology and then transferred to the catheterization laboratory if indicated. [41] The Emergency Team with immediate access to diagnostic facilities including computed tomography scanning along with early cardiology input and bedside echocardiography can help determine the etiology and therefore triage the patient to the appropriate locations (Intensive Care Unit/Catheterization laboratory). Patients can be investigated for causes of OOCHA including metabolic derangements, respiratory failure, neurological causes and tamponade (Table 1). Echocardiography is useful in ruling out tamponade, dissection or the presence of a regional wall motion abnormality. In the absence of an obvious non-cardiac cause the patient should be transferred to a catheterization laboratory for coronary angiography with a view to PCI as soon as possible (Fig. 1). Thrombolytic therapy should only be considered in patients

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