Etiology of In-hospital Cardiopulmonary Arrest as a Predictor of Outcome

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Introduction

Cardiopulmonary arrest (CPA) is a major contributor to overall mortality as well as years-of-productive-life lost.[Roger] Despite advances in the field of resuscitation science, little progress has been observed with regard to CPA outcomes both in and out of the hospital. Much of this lack of progress has been attributed to the influence of factors outside of the control of rescuers, including down time and the performance of bystander cardiopulmonary resuscitation (CPR).[Rea] The Utstein template was developed to standardize out-of-hospital cardiac arrest (OOHCA) reporting.[Cummins] The multiple covariates included as part of the Utstein template have consistently demonstrated good predictive ability with regard to OOHCA survival.[Jacobs] However, little is known about in-hospital CPA and the covariates that affect survival-to-discharge and neurological outcomes.

As part of the Advanced Resuscitation Training (ART) program at the University of California at San Diego (UCSD), a complex taxonomy is applied to CPA victims. This "ART Matrix" was designed to direct continuous quality improvement (CQI) efforts and guide training.[Davis] The ability of this classification scheme to predict outcomes from in-hospital CPA has not yet been evaluated. This analysis evaluates the ability of the various ART arrest taxonomies, or "ART Matrix," to accurately predict outcome from in-hospital CPA and to serve as Inpatient Utstein Criteria. In addition, we also explored the impact of the ART program on the incidence and outcome of various arrest etiologies.

Methods

Design

This analysis utilized our institutional resuscitation database, which is described in detail below. Waiver of informed consent was granted from our Human Research Protection Program.

Setting

The University of California at San Diego Healthcare System includes two urban hospitals with a total of 600 licensed beds and approximately 25,000 annual patient discharges. Both facilities receive ambulance admissions and are designated as emergency percutaneous coronary interventional facilities. A designated Code Blue team (senior Internal Medicine resident or Critical Care fellow, critical care nurse, airway physician, respiratory therapist, pharmacist) responds to critical resuscitation events within each hospital. The Code Blue team nurse and respiratory therapist also respond to less critical resuscitation events as part of a Rapid Response Team.

Until Spring 2007, all in-hospital providers were required to maintain Basic Life Support (BLS) certification. Critical care providers were also required to maintain Advanced Cardiac Life Support (ACLS) certification. In Spring 2007, the ART program was implemented, consisting of five specific elements: 1) an institutional treatment algorithm for both CPA and non-CPA resuscitations; 2) semi-annual and annual courses for critical care and non-critical care providers respectively with flexible content/format and adaptive training to allow providers to assume anticipated resuscitation roles using actual equipment; 3) a Rapid Response Team; 4) links between CQI efforts, training, and

competency assessments; and 5) new defibrillators (Zoll E Series, Zoll Medical Inc, Chelmsford, MA) with universal capability for CPR process measurement, ECG filtration, and quantitative capnometry.

Data Collection

All Code Blue and Rapid Response activations are entered into an electronic database (electronic quality variance report or eQVR) by the Code Blue nurse immediately following completion of resuscitation efforts. Three levels of clinical review occur following the initial input of data. First, dedicated data analysts review entries for input errors and completeness and to link events to clinical outcomes. The telecommunications and cardiac care unit logs are cross-referenced to assure complete capture of resuscitation events. Second, a dedicated critical care nurse and the Resuscitation Director classify each CPA based on the taxonomy described below. These determinations are based on narratives entered into the eQVR system, clinical data retrieved from the electronic patient care record (PCR), and interviews with hospital providers. This classification triggers additional data collection to guide CQI efforts as part of the ART program. Finally, members of the Code Blue Committee review all resuscitation events to identify CQI issues for follow-up.

To develop the ART matrix taxonomy, all arrests are subsequently defined by the precipitating etiology – cardiovascular, respiratory or neurologic deterioration. Each of the classifications triggers additional CQI pre-arrest data collection in addition to the standard intra- and post-arrest data elements. Summary data are presented to resuscitation team members as feedback. The taxonomy data are also summarized to

identify trends and opportunities for intervention through changes to the treatment algorithm, training, and initiation of new initiatives.

Data Analysis

All in-hospital cardiopulmonary arrests from July 2005 through June 2009 were included in this analysis. This includes approximately 2 years of data before and after the introduction of the ART program. A cardiopulmonary arrest was defined by the absence of a palpable pulse, the performance of chest compressions, or a defibrillation attempt. Cardiopulmonary arrests occurring in non-admitted patients, the operating room or emergency department, or in patients with active "Do Not Attempt Resuscitation" (DNAR) orders were excluded.

Clinical and demographic data were collected from the institutional resuscitation database, which includes data elements from the eQVR system and the electronic PCR. In addition, the ART Matrix taxonomic classifications were included. A simplified version of the taxonomy was used for this analysis (Table 1).

The impact of the ART program with regard to the incidence of various etiologies of arrest was explored graphically and by comparing the incidence of each (per 1,000 patient discharges). In addition, the ART impact with regard to arrest outcomes was also assessed by comparing pre- and post-ART survival-to-discharge rates for each taxonomic category.

The primary analysis was intended to evaluate the predictive ability of the ART Matrix taxonomies. We hypothesized that etiology of arrest would be highly predictive of outcome, even after adjusting for multiple covariates. Multivariate logistic regression

was used with both survival-to-hospital discharge and good neurological outcome as outcome variables. A good outcome was defined as a Cerebral Performance Category (CPC) score of 1 or 2. The following covariates were included in the analysis: age, gender, arrest during working hours (0700-1700), initial shockable rhythm, taxonomic classification, and location of arrest (ICU/non-ICU). Hosmer-Lemeshow test was used to determine goodness-of-fit. SPSS (IBM Corp, Armonk, NY) was used for all comparisons. P-values less than 0.05 were considered statistically significant.

Results

A total of 332 in-hospitals suffered cardiopulmonary arrest during the study period (Table 2). This included 182 patients in the pre-intervention period and 150 patients in the post-intervention period. Survival-to-hospital discharge increased following ART implementation from 20.8 percent (38/182) to 36.0 percent (54/150), $p<0.01$. Good neurological outcome (CPC 0-2) also increased following ART implementation from 10.4 percent (19/182) to 21.3 percent (32/150), $p<0.01$.

The incidence of respiratory arrests decreased following ART program implementation (Table 3) yet remained a common cause. Survival to discharge was more likely with a cardiac arrest secondary to a respiratory event or hemorrhage. Survival-to-discharge increased for respiratory, hemorrhagic, and vagal-mediated arrests after ART implementation, but did not change for VF/PVT, neurological, or septic arrests (Table 4). Multivariate logistic regression revealed an independent association between CPA etiology and outcome. Of note, improved outcomes were observed following ART implementation with regard to both survival-to-discharge ($p<0.01$) and good neurological outcomes ($p<0.01$) across all etiologies of arrest.

Discussion

While our understanding of CPA has expanded exponentially in recent years, much of this reflects research performed in the out-of-hospital environment. While the physiology of arrest and performance of CPR may be universal, the in-hospital environment affords the unique opportunity for intervention prior to arrest. Furthermore, down times are generally shorter and the etiologies of arrest would be expected to reflect the admission diagnoses and resultant therapies. Here we explore a unique taxonomy to describe the etiologies of in-hospital CPA and thus focus preventative therapies, early recognition and interventions upon arrest. This appears to be a useful approach for several reasons. First, the etiology of arrest had a strong association with outcome despite adjusting for multiple traditional covariates. In addition, these etiologies are part of a broader, more detailed taxonomy that serves as the nucleus of the ART program, guiding CQI efforts and providing a structure for treatment guidelines and training efforts. The impact of the program is evident in the differential impact on various arrest etiologies with regard to both the incidence and survival-to-hospital discharge.

The Utstein template represents one of the first major efforts to describe various factors that influence outcome from CPA. While the intent was to standardize reporting of results from OOHCA investigations, development of the Utstein template required systematic consideration of multiple covariates affecting survival. Although arrest etiology is important, prehospital providers are rarely able to determine this from the limited data available, and initial rhythm is used as a surrogate to differentiate primary dysrhythmias from other causes of arrest.

While the Utstein template has remained a valid strategy for reporting OOHCA data, we advocate a different approach for in-hospital arrest for several reasons. First, shockable rhythms are relatively less common than reported for OOHCA and because non-shockable rhythms contribute substantially to the cohort of survivors for in-hospital – but not out-of-hospital – arrest. In addition, the presence of hospital providers during the period of deterioration affords the unique opportunity to intervene and prevent arrest, a concept that forms the basis for Rapid Response Teams. Of paramount importance to their success, however, is a thorough understanding of the various etiologies that lead to arrest. This requires an objective and reproducible taxonomy that demonstrates consistent association with arrest outcomes.

The UCSD ART program is successful in both preventing arrest and improving survival-to-discharge from in-hospital CPA. We believe this reflects the unique approach in considering various arrest etiologies and addressing each with different strategies. This is particularly important in arrest prevention, with the opportunity to implement multiple targeted interventions. Once an arrest state is reached, the opportunities to "customize" therapies are limited due to time constraints and the urgency of the situation.

Several examples are apparent within the current analysis. Non-intubated respiratory arrest victims had a dramatic increase in survival after the implementation of ART. While the ART program includes the use of continuous chest compressions, we also employ a unique interposed ventilation strategy that appears to provide adequate tidal

volumes without interrupting compressions. In addition, we emphasize the early and repeated administration of vasopressin, which appears to carry pharmacological advantages in hemorrhagic arrest and may explain the improved outcomes in this group. More recently, we have addressed the lack of response in our VF/PVT patients by successfully implementing an alternative initial resuscitation strategy for this subset of patients.

The non-intubated respiratory arrest victims warrant additional discussion. The vast majority of these patients did not have underlying pulmonary disease but appear to suffer an apneic event preceding their cardiac arrest. This may be a consequence of analgesics or sedatives, obesity and un-diagnosed obstructive sleep apnea, or a vulnerable post-operative period following the use of general anesthetics. In most of these cases, there is not an underlying physiological instability prior to the arrest, which may explain their response to optimal resuscitation efforts. It also appears to be a subgroup amenable to early detection and intervention, as shown by the decrease in incidence following ART implementation.

One of the important observations from these data is the lack of improvement in certain arrest subgroups, particularly those with poor predicted survival at baseline. Patients with devastating neurological injuries did not benefit from the improved resuscitation performance observed following ART implementation. Similarly, patients who arrest while on maximum therapy for sepsis or suffer arrests from pulmonary emboli do not appear to have good outcomes regardless of the resuscitation strategy. These findings

suggest opportunities for end-of-life discussions with family to avoid futile resuscitation attempts, as well as policies to avoid futile care.

There are several limitations to this analysis that should be considered when interpreting these data. The taxonomy described here was designed for CQI and is a subset of a more complex grid than represented here. It is possible that other strategies for categorizing patients would be even more predictive of outcome. In addition, certain etiologies were under-represented, which limits statistical power. Lastly, we did not incorporate CPR quality into this analysis, which has emerged as an important variable influencing outcome. While the ART program routinely collects data regarding CPR depth and rate as well as chest compression fraction, these were not collected in the pre-ART period.

In conclusion, our comprehensive taxonomy categorizing cardiac arrests by etiology lead to interventions which may prevent arrests, informed conversations with patients and their surrogates in regard to outcome in the event of an arrest, and led to changes in resuscitation strategy (ventilations, defibrillation timing and pressor use) that contributed to improved survival.

Table 1. Arrest etiologies

Cardiovascular
Sepsis
Hemorrhage
Pulmonary embolus
Heart failure
Dysrhythmic
VF/PVT
Vagal/block
Neurological
Respiratory
Intubated respiratory
Non-intubated respiratory
Unknown

Table 2. Clinical and demographic data regarding study population (n=332).

Parameter	Mean or % (95% CI)
Age (years)	58 (56-59)
Male gender (%)	63 (58-68)
Initial shockable rhythm (%)	14 (10-17)
Non-ICU location (%)	48 (43-54)
Working hours (0700-1700) (%)	42 (37-48)
Compressions prior to Code Blue team arrival (%)	84 (79-88)

Table 3. Arrest incidence (per 1,000 patient discharges) based on ART Matrix etiologies of cardiopulmonary arrest.

Arrest Etiology	Pre-ART	Post-ART	Odds Ratio (95% C.I.)
Sepsis	0.66	0.53	0.8 (0.5-1.4)
Hemorrhage	0.42	0.16	0.4 (0.2-0.9)*
Pulmonary embolus	0.08	0.11	1.5 (0.4-6.3)
Heart failure	0.10	0.07	0.7 (0.2-3.0)
VF/PVT	0.51	0.53	1.0 (0.6-1.9)
Vagal/block	0.25	0.42	1.7 (0.8-3.7)
Neurological	0.05	0.09	1.8 (0.3-9.9)
Intubated respiratory	0.17	0.11	0.6 (0.2-2.0)
Non-intubated respiratory	2.13	1.18	0.6 (0.4-0.8)**
Unknown	0.17	0.13	0.8 (0.3-2.3)

VF/PVT = ventricular fibrillation/pulseless ventricular tachycardia

*$p<0.05$

**$p<0.01$

Table 4. Survival-to-hospital discharge based on ART Matrix etiologies of cardiopulmonary arrest.

Arrest Etiology	Pre-ART	Post-ART	Odds Ratio (95% C.I.)
Sepsis	3/27 (11)	0/24 (0)	--
Hemorrhage	1/17 (6)	5/7 (71)	40.0 (3.0-539.7)**
Pulmonary embolus	0/3 (0)	1/5 (20)	--
Heart failure	0/4 (0)	0/3 (0)	--
VF/PVT	12/21 (57)	8/24 (33)	0.4 (0.1-1.3)
Vagal/block	3/10 (30)	10/19 (53)	2.6 (0.5-13.2)
Neurological	0/4 (0)	0/3 (0)	--
Intubated respiratory	0/7 (0)	2/5 (40)	--
Non-intubated respiratory	19/87 (22)	25/53 (47)	3.2 (1.5-6.7)**
Unknown	0/7 (0)	2/6 (33)	--

VF/PVT = ventricular fibrillation/pulseless ventricular tachycardia

*$p<0.05$

**$p<0.01$

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