



AGENDA

ICEMA MEDICAL ADVISORY COMMITTEE

October 22, 2015

1300

Purpose: Information Sharing

Meeting Facilitator: Phong Nguyen

Timekeeper: Danielle Ogaz

Record Keeper: Danielle Ogaz

AGENDA ITEM		PERSON(S)	DISCUSSION/ACTION
I.	Welcome/Introductions	Phong Nguyen	
II.	Approval of Minutes	All	Discussion
III.	Discussion/Action Items		
	A. Standing EMS System Updates		
	<ol style="list-style-type: none"> 1. Review of Action Items 2. Trauma Program 3. STEMI Program: STEMI Data <ul style="list-style-type: none"> • Chest Pain Society Accreditation 4. Stroke Program: Stroke Data 5. CQI Report Update <ul style="list-style-type: none"> • Core Measures • Intubation and Capnography Data Task Force 6. SAC Update 	<ol style="list-style-type: none"> 1. Phong Nguyen 2. Chris Yoshida-McMath 3. Chris Yoshida-McMath 4. Chris Yoshida-McMath 5. Phong Nguyen <ul style="list-style-type: none"> • Ron Holk • Pam Martinez/ Joe Powell 6. Phong Nguyen 	<ol style="list-style-type: none"> 1. Discussion/Action 2. Discussion 3. Discussion 4. Discussion 5. Discussion
	B. EMS Trends		
	<ol style="list-style-type: none"> 1. TXA Study Update 2. Paramedicine Step I Research Update 3. Cardiac Arrest Survival Enhancement Project (CARES/ART) 	<ol style="list-style-type: none"> 1. Reza Vaezazizi/ Michael Neeki 2. Michael Neeki 3. Reza Vaezazizi 	<ol style="list-style-type: none"> 1. Discussion 2. Discussion 3. Discussion
	C. Task Force Report: ICEMA Protocol Survey	Henry Perez	Discussion
	D. Review of MAC membership	Ron Holk	Discussion
	E. ePCR Task Force	Ron Holk	Discussion
	F. Routine Use of Narcan and Glucose During Cardiac Arrest	Reza Vaezazizi	Discussion
	G. Protocol Review	Ron Holk	Discussion/Action
	<ol style="list-style-type: none"> 1. 7040 - Medication - Standard Orders 2. 9080 - Care of Minors in the Field 3. 10190 - ICEMA Approved Skills 		

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V.	Public Comment	All	Discussion
VI.	Round Table/Announcements	All	Discussion
VII.	Future Agenda Items	All	Discussion
VIII.	Next Meeting Date: December 17, 2015	All	Discussion
IX.	Adjournment	Phong Nguyen	Action
X.	Closed Session		
	A. Case Reviews		



MINUTES

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1300

	AGENDA ITEM	DISCUSSION/FOLLOW UP	RESPONSIBLE PERSON(S)
I.	WELCOME/INTRODUCTIONS	Meeting called to order at 1305.	Michael Neeki
II.	APPROVAL OF MINUTES	The April 23, 2015, minutes were approved. Motion to approve. MSC: Sam Chua/Joy Peters APPROVED Ayes: Sam Chua, Susie Moss, Michael Neeki, Phong Nguyen, Kevin Parkes, Joy Peters, Leslie Parham, Joe Powell, Aaron Rubin, Rosemary Sachs, Todd Sallenbach, Andrew Stevens, Andrea Thorp	
III.	DISCUSSION ITEMS		
	A. Standing EMS System Updates		
	1. Review of Action Items	Action items incorporated into the agenda.	Phong Nguyen
	2. Trauma Program	ARMC completed its ACS Level II Verification Survey. The next TSAC/TAC combo meeting is scheduled for September 22, 2015, at 1600, at ICEMA. The TSAC portion of the meeting is open to all of the EMS community, while the TAC portion is a closed session.	Chris Yoshida-McMath
	3. STEMI Program: STEMI Data	STEMI Receiving Centers (SRCs) will be required to obtain Society of Chest Pain Centers Accreditation as part of the new contracts. The committee is researching ECG transmission to SRCs, as well as considering developing a policy change for sustained ROSC patients to be transported to SRCs. The next STEMI meeting is November 19, 2015, at 1300, at ICEMA.	Chris Yoshida-McMath

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	4. Stroke Program: Stroke Data	California is one of seven (7) states to receive the CDC stroke registry grant. The next Stroke meeting is November 10, 2015, at 1300, at ICEMA.	Chris Yoshida-McMath
	5. CQI Report Update	Nothing new to update. The CQI plan remains under internal review.	Ron Holk
	<ul style="list-style-type: none"> • Core Measures 	Nothing to report.	Ron Holk
	<ul style="list-style-type: none"> • Intubation and Capnography Data Task Force 	<p>The task force reviewed the State core measures which were presented at the April meeting.</p> <p>The task force determined that there are approximately 10 different places where capnography results can be documented in the ePCR and concluded that the percentage of use is much higher than indicated in the core measures.</p> <p>The task force has identified several possible changes to the ePCR that may help to ensure data is entered correctly.</p> <p>MAC considered forming a special task force to review future recommendations to the input form and data changes. MAC deferred to future meeting.</p> <p>Pam Martinez was asked to forward the distribution list of attendees from the ePCR Working Group to MAC to be used as a pool of potential task force members.</p>	Pam Martinez/Joe Powell
	6. SAC Update	<p>The Active Shooter Task Force is reviewing draft regulations currently in public comment which is due September 18, 2015.</p> <p>The Triage Tag Task Force recommended mandatory education, which the task force developed, to start after the first of the year. ICEMA will review. The task force will provide recommendations regarding future hands-on training.</p> <p>APOD Task Force update was presented.</p>	Tom Lynch
	B. EMS Trends		
	1. TXA Study Update	<p>The TXA Study officially began on March 9, 2015.</p> <p>ARMC, LLUMC, and 4 trauma centers in Riverside, as well as 10 San Bernardino County EMS providers are participating in the</p>	Reza Vaezazizi/Michael Neeki

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		<p>study.</p> <p>To date, TXA has been administered for 8 blunt traumas, 17 penetrating traumas and four 4 non-traumas. A total of 14 of these met EMS inclusion criteria while 15 did not.</p> <p>An additional education piece was developed and participating providers were required to complete the education no later than July 31, 2015.</p>	
	2. Paramedicine Step I Research Update	A total of 280 evaluations have been completed to date. Anticipating terminating at a sample size of 1,000.	Michael Neeki
	3. Cardiac Arrest Survival Enhancement Project (CARES/ART)	<p>Application has been submitted to CARES. CARES will be adding hospitals and providers to the registry in regions.</p> <p>Loma Linda and Colton Fire Departments will be participating in Advanced Resuscitation Training (ART). Training dates for these departments have been scheduled.</p> <p>Due to financial reasons, the other providers that had expressed interest have decided not to participate. ICEMA encourages providers to consider the value of ART in patient care and hopes to have other providers participate.</p>	Reza Vaezazizi
	C. Appropriate Use of Oxygen	<p>Three articles on the use of oxygen were discussed at the meeting.</p> <p>EMDAC is reviewing protocols throughout the State to identify best practices. One of the items being supported is the titration of oxygen.</p> <p>There was a motion to develop a protocol for the titration of oxygen, but was not seconded.</p> <p>ICEMA will draft modifications to MSO protocol for approval at the October meeting.</p>	Kevin Parkes
	D. Task Force Report: ICEMA Protocol Survey	Tabled until October meeting.	Henry Perez
	E. IO Insertion Site	<p>IO insertion sites were discussed.</p> <p>ICEMA will present a modified protocol at the October meeting, in order for MAC to determine insertion sites.</p>	Ron Holk
	F. Review of Bylaws	Modified bylaws were presented to committee which lowered the quorum to 5 members instead of fifty percent plus one.	Ron Holk

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		<p>ICEMA will present the attendance log at the October meeting.</p> <p>Motion to change bylaws to state 7 members total, with a minimum of 3 physicians. MSC: Michael Neeki/Susie Moss APPROVED Ayes: Sam Chua, Susie Moss, Michael Neeki, Phong Nguyen, Joy Peters, Leslie Parham, Joe Powell, Aaron Rubin, Rosemary Sachs, Todd Sallenbach, Andrew Stevens, Andrea Thorp Nays: Kevin Parkes</p>	
	<p>G. Drug and Equipment List Review 1. Colorimetric and One-way Stop Cock</p>	<p>Tabled until oxygen change has been made.</p>	<p>Leigh Overton</p>
IV.	PUBLIC COMMENT	None	All
V.	ROUND TABLE/ ANNOUNCEMENTS	None	All
VI.	FUTURE AGENDA ITEMS	None	Danielle Ogaz
VII.	NEXT MEETING: October 22, 2015		
VIII.	ADJOURNMENT	The meeting adjourned at 1445.	Phong Nguyen
IX.	CLOSED SESSION	1450 - 1528	Phong Nguyen.
	A. Case Review	A total of two (2) cases were reviewed.	

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Attendees:

NAME	MAC POSITION	EMS AGENCY STAFF	POSITION
<input type="checkbox"/> VACANT <input type="checkbox"/> Jeff Grange - LLUMC	Trauma Hospital Physicians (2)	<input checked="" type="checkbox"/> Reza Vaezazizi, MD	Medical Director
<input checked="" type="checkbox"/> Phong Nguyen - RDCH <input checked="" type="checkbox"/> Todd Sallenbach - HDMC (Chair)	Non-Trauma Base Physicians (2)	<input checked="" type="checkbox"/> Tom Lynch	EMS Administrator
<input checked="" type="checkbox"/> Aaron Rubin - Kaiser	Non-Base Hospital Physician	<input type="checkbox"/> Denice Wicker-Stiles	Assist. Administrator
<input checked="" type="checkbox"/> Michael Neeki - Rialto FD	Public Transport Medical Director	<input type="checkbox"/> George Stone	Program Coordinator
<input checked="" type="checkbox"/> Sam Chua - AMR	Private Transport Medical Director	<input checked="" type="checkbox"/> Ron Holk	EMS Nurse Specialist
<input type="checkbox"/> Debbie Bervel - SB City FD	Fire Department Medical Director	<input checked="" type="checkbox"/> Chris Yoshida-McMath	EMS Nurse Specialist
<input checked="" type="checkbox"/> Joy Peters - ARMC	EMS Nurses	<input checked="" type="checkbox"/> Danielle Ogaz	EMS Specialist
<input checked="" type="checkbox"/> Joe Powell - Rialto FD	EMS Officers		
<input checked="" type="checkbox"/> Leslie Parham	Public Transport Medical Rep (Paramedic/RN)		
<input checked="" type="checkbox"/> Susie Moss	Private Transport Medical Rep (Paramedic/RN)		
<input type="checkbox"/> Lance Brown	Specialty Center Medical Director		
<input type="checkbox"/> Joanna Yang - LLUMC	Specialty Center Coordinator		
<input type="checkbox"/> Troy Pennington	Private Air Transport Medical Director		
<input type="checkbox"/> Stephen Patterson - Sheriff's Air Rescue	Public Air Transport Medical Director		
<input type="checkbox"/> Micheal Guirguis - SB Comm Center	PSAP Medical Director		
<input checked="" type="checkbox"/> Andrew Stevens	Inyo County Representative		
<input checked="" type="checkbox"/> Rosemary Sachs	Mono County Representative		
<input checked="" type="checkbox"/> Kevin Parkes	SAC Liaison		
<input checked="" type="checkbox"/> Andrea Thorp	Pediatric Critical Care Physician		

GUESTS	AGENCY
Sandy Carnes	Rancho Cucamonga FD
Carly Crews	SB City FD
Kevin Dearden	Rialto FD
Lisa Higuchi	AMR
Pam Martinez	Ontario FD
Sara Morning	RCH
Miranda Mulhull	SB County FD
Bob Tyson	Redlands FD

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Naloxone in cardiac arrest with suspected opioid overdoses

ARTICLE *in* RESUSCITATION · NOVEMBER 2009

Impact Factor: 4.17 · DOI: 10.1016/j.resuscitation.2009.09.016 · Source: PubMed

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Clinical paper

Naloxone in cardiac arrest with suspected opioid overdoses[☆]

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ABSTRACT

Introduction: Naloxone's use in cardiac arrest has been of recent interest, stimulated by conflicting results in both human case reports and animal studies demonstrating antiarrhythmic and positive inotropic effects. We hypothesized that naloxone administration during cardiac arrest, in suspected opioid overdosed patients, is associated with a change in cardiac rhythm.

Methods: From a database of 32,544 advanced life support (ALS) emergency medical dispatches between January 2003 and December 2007, a retrospective chart review was completed of patients receiving naloxone in cardiac arrest. Forty-two patients in non-traumatic cardiac arrest were identified. Each patient received naloxone because of suspicion by a paramedic of acute opioid use.

Results: Fifteen of the 36 (42%) (95% confidence interval [CI]: 26–58) patients in cardiac arrest who received naloxone in the pre-hospital setting had an improvement in electrocardiogram (EKG) rhythm. Of the participants who responded to naloxone, 47% (95% CI: 21–72) (19% [95% CI: 7–32] of all study subjects) demonstrated EKG rhythm changes immediately following the administration of naloxone.

Discussion: Although we cannot support the routine use of naloxone during cardiac arrest, we recommend its administration with any suspicion of opioid use. Due to low rates of return of spontaneous circulation and survival during cardiac arrest, any potential intervention leading to rhythm improvement is a reasonable treatment modality.

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1. Introduction

Naloxone has long had a presence in the armamentarium of emergency physicians caring for opioid poisoned patients.¹ Its use in cardiac arrest has been of recent interest, stimulated by conflicting results in both human case reports and animal studies demonstrating antiarrhythmic and positive inotropic effects.^{1–6} Naloxone alone in high doses has been shown to increase cardiopulmonary resuscitation (CPR) rates following asphyxia induced cardiac arrest in rats.⁷ Further animal data supports naloxone administration alone or in combination with epinephrine in simulated asphyxia induced cardiac arrest models.^{5,6} Naloxone with and without epinephrine resulted in increased incidence of return of spontaneous circulation (ROSC) as well as shorter resuscitation times. Additionally, a recent case report and literature review presented a patient with pulseless electrical activity

(PEA) who returned to spontaneous circulation after receiving naloxone, subsequently questioning the routine usage in cardiac arrest.¹

The interest in the utilization of naloxone in the non-overdosed opioid cardiac arrest patient stems from many hypotheses, one being that endogenous opioids are felt to have a myocardial depressant effect with a lowering of systemic blood pressure. Alternatively, naloxone may stimulate catecholamine release and increase sympathetic nerve activity significantly elevating heart rate and blood pressure.¹ Importantly, the safety profile of naloxone has been demonstrated in opioid toxicity as well as other non-poisoning scenarios such as spinal cord injury, shock, and acute ischemic stroke.^{8–22}

Naloxone has been demonstrated to reduce action potential upstroke in guinea pig, canine, rabbit, and sheep myocardium.^{8,18,19} The inhibition of action potential upstroke is correlated with the inhibition of fast inward sodium currents. In addition, an effect on repolarizing potassium currents has been shown to suppress re-entrant rhythms by prolonging action potential duration and increasing the refractory period.²³ Therefore, naloxone's antiarrhythmic activity appears to be similar to both class I and III antiarrhythmics.²³

[☆] A Spanish translated version of the abstract of this article appears as Appendix in the final online version at doi:10.1016/j.resuscitation.2009.09.016.

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Based on the known and hypothesized effects of naloxone, we sought to investigate naloxone's role in cardiac arrest in patients with suspected opioid overdose. This is the first human cohort studied to date. We hypothesized that naloxone administration during cardiac arrest, in suspected opioid overdosed patients, is associated with improvements in cardiac rhythm.

2. Methods

2.1. Design

This was a retrospective cohort study conducted by chart review, with analysis of subgroups. The study was approved by the institutional review board at our institution.

2.2. Setting

The study was conducted at our university-based Level I trauma center in an urban setting surrounded by multiple suburban regions. The emergency medical service (EMS) contains six advanced life support (ALS) units that treat approximately 6500 patients out of 30,000 dispatches per year, including basic life support units. In our setting, naloxone may only be administered by ALS providers.

This system contains 90 paramedics, 140 basic emergency medical technicians, one full time medical director, and two EMS fellows. The system provides 100% on-line medical direction via cellular phone with a board certified or board eligible emergency physician working clinically in the emergency department. The state has multiple standing order protocols whereby the paramedic can initiate treatment; however, naloxone in cardiac arrest must be given under physician medical control. ALS units comply with state protocols and contain two paramedics in ambulances or response units. EMS supervisors respond on all "critical calls" as defined by the medical communicator.

2.3. Selection of subjects

Participants were identified from a database of ALS responses between January 1, 2003 and December 31, 2007. Patients who had received naloxone in cardiac arrest were selected as participants. This query also retrieved records of subjects who received naloxone when they had a pulse, either before or after being in cardiac arrest. These subjects were excluded so only patients who were in cardiac arrest at the time naloxone administration were included.

2.4. Interventions

Patients in cardiac arrest were initially treated by paramedics in accordance with advanced cardiac life support (ACLS) guidelines. As paramedics are not permitted to give naloxone in this circumstance, all administrations were authorized by a physician via online telemetry orders. Subsequently, there were no standard dosages and patients received naloxone at varying steps in the ACLS algorithms. Naloxone was never the first pharmacologic intervention.

2.5. Methods of measurement

On standard patient care reports (PCR), paramedics recorded patients' vital signs before and after each medication administration. Pertinent vital signs recorded included heart rhythm, as interpreted by the paramedics providing patient care. All patient electrocardiogram (EKG) rhythms were verified by two pre-hospital paramedics and the emergency physician upon arrival at the hospital. PCRs were also reviewed after the call by the

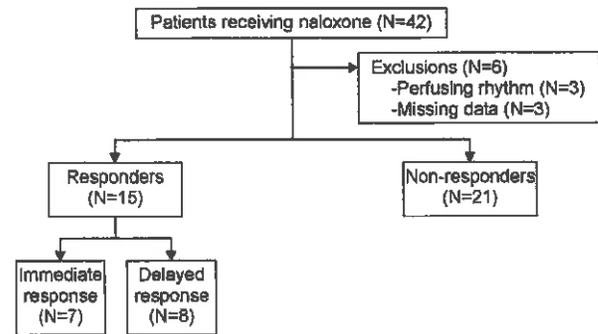


Fig. 1. Study design.

paramedic clinical coordinator and physician medical director during the quality assurance/quality improvement process. Naloxone doses and routes of administration were also recorded on the PCRs. Additional information obtained included time of cardiac arrest, duration of cardiac arrest, and time of pronouncement.

2.6. Data collection and processing

Data was collected by an investigator trained in Microsoft Access, the Emergency Department Information Management (EDIM), and Sunrise Clinical Manager (SCM) databases. From an Access database of EMS responses, a query was performed to retrieve all patients who were in cardiac arrest and received naloxone from January 1, 2003 to December 31, 2007. The original paper PCRs were obtained for each patient. The investigator recorded date, patient age and sex, time of cardiac arrest, duration of anoxia, duration of cardiac arrest, naloxone dosage, destination hospital, emergency department disposition, and time of pronouncement (if applicable). Additionally, the investigator recorded all of the patients' cardiac rhythms as well as pharmacologic interventions documented on the PCRs. Three of the authors reviewed all charts and had 100% agreements on all documented data. After enrolling qualified subjects, records of patients transported to our hospital were cross-referenced with emergency department records in EDIM to obtain information on outcomes. Furthermore, for patients that survived to admission, records were cross-referenced in SCM to determine hospital course and disposition. All data was entered into a standardized abstraction form. Endpoints were reconfirmed twice for each patient by re-inspection of PCRs by the same abstractor. The investigators met monthly to discuss progress.

2.7. Outcome measures

For each participant, cardiac rhythms were compared immediately before and after naloxone administration. The original rhythm was defined as the heart rhythm documented immediately prior to naloxone administration. Participants were then classified based on whether there was a change in EKG rhythm from baseline (respon-

Table 1
Baseline characteristics of patients by confirmation of EKG rhythm change.

	Responders (N = 15)	Non-responders (N = 21)
Age, years (SD)	44 (17)	40 (14)
Gender, % male (no)	53 (8)	81 (17)
Naloxone dose, mg (SD)	2.6 (2.1)	2.0 (0.5)
Initial rhythm, % (no)		
Asystole	53 (8)	71 (15)
PEA	40 (6)	29 (6)
Ventricular fibrillation	7 (1)	0 (0)

Abbreviations: EKG, electrocardiogram; PEA, pulseless electrical activity.

Table 2
Immediate and delayed responders to naloxone therapy.

Age and gender	Rhythm change over treatment interval ^a (Recorded response time)	Narrative of all pharmacologic and external defibrillations interventions ^b	Outcome
73 F	Asystole → ventricular fibrillation ^c 4 min	Found in asystole. Over 22 min received epinephrine 3× and atropine 3× without effect. Subsequently administered naloxone 2 mg followed by epinephrine 1×.	Pronounced in ED
49 F	PEA → sinus tachycardia 1 min	Found in asystole. Over 16 min received epinephrine 1× and atropine 1×. Rhythm changed to PEA (rate unknown). Naloxone 2 mg administered, rhythm changed to sinus tachycardia (118 bpm).	Survived to admission, died on HD #2
37 M	Asystole → accelerated junctional rhythm ^c 1 min	Found in asystole. Received epinephrine 2× and atropine 2× over 8 min. Naloxone 2 mg administered followed immediately by epinephrine at which point EKG converted to accelerated junctional rhythm (60 bpm).	Pronounced in ED
24 M	Asystole → PEA 4 min	Found in ventricular fibrillation. Defibrillated at 200 J and converted to asystole. Administered epinephrine and atropine without rhythm change. Naloxone 2 mg administered, rhythm changed to PEA (rate unknown).	Pronounced in ED
62 M	PEA → ventricular fibrillation ^c 6 min	Found in PEA (rate 40). Over 6 min, received epinephrine 1× and atropine 1×. Remained in PEA (rate 90). Received naloxone 2 mg and epinephrine 1× and rhythm change to ventricular fibrillation.	Survived to admission, died HD #1
47 F	Asystole → PEA ^c 3 min	Arrest witnessed by BLS with <1 min anoxic time. Found in asystole. Received epinephrine 1×, atropine 1×. No changes. Received naloxone 2 mg, epinephrine 1× and atropine 1× and spontaneous non-sustained 20–30 s runs of idioventricular PEA (HR unknown) were noted.	Pronounced in pre-hospital setting
23 M	Asystole → ventricular tachycardia (pulseless) ^c 7 min	Found in asystole. Received epinephrine 2× and atropine 2× over 16 min. Remained in asystole. Received naloxone 2 mg, dextrose 50% 25 g, epinephrine, atropine and sodium bicarbonate 50 mEq over 7 min. Converted to pulseless ventricular tachycardia.	Pronounced in ED
36 F	Asystole → sinus tachycardia 2 min	Found in asystole. Received epinephrine 2× and atropine 2× over 13 min. Remained in asystole. Received naloxone 2 mg. Converted to sinus tachycardia (130 bpm) within 2 min.	Survived to admission, survived to discharge HD#11
72 F	Asystole → PEA 2 min	Arrest witnessed by ALS 2–3 min after arrival with <1 min anoxic time. Fluctuated between PEA, asystole, and ventricular fibrillation. Received epinephrine 8×, atropine 3×, sodium bicarbonate 50 mEq 2×, and calcium chloride 1 g over 32 min. Defibrillated at 360 J 2×. Stayed in asystole. Received naloxone 2 mg. Converted back to PEA (rate unknown).	Pronounced in ED
66 M	PEA → ventricular tachycardia (pulseless) ^c 10 min	Found in PEA (rate 35). Received epinephrine 2× and atropine 1× over 10 min. Remained in PEA. Received naloxone 2 mg, epinephrine and sodium bicarbonate 50 mEq over 6 min. Converted to pulseless ventricular tachycardia (rate 150).	Pronounced in ED
22 M	Asystole → PEA 2 min	Found in asystole. Received epinephrine 2×, atropine 2×, and naloxone 2 mg over 7 min. Remained in asystole. Received 2nd dose of naloxone 2 mg. Converted to PEA (rate unknown).	Survived to admission, further outcome unknown
24 F	PEA → asystole 5 min	Found in PEA (rate unknown). Four minutes after ALS arrival, patient received epinephrine and converted to asystole. Over 9-min interval patient received epinephrine 2× and atropine 2× and rhythm converted to PEA (rate 70). Patient received naloxone 1 mg and rhythm converted back to asystole.	Pronounced in ED
45 M	PEA → asystole ^c 2 min	Found in PEA (rate 10–20). Over 2-min interval, patient received epinephrine 1× and atropine 1×. Remained in PEA. Patient administered naloxone 2 mg, epinephrine 1× and atropine 1×. Converted to asystole.	Pronounced in ED
45 M	Ventricular fibrillation → PEA 2 min	Found in PEA (rate 110) and received epinephrine 1×. Converted to ventricular fibrillation. Patient received naloxone 2 mg and converted back to PEA (rate 60).	Pronounced in pre-hospital setting
43 F	PEA → asystole ^c 8 min	Found in asystole. Over 22 min received epinephrine 3×, atropine 2× and thiamine 100 mg. Converted to PEA. Over 3 min patient received epinephrine 1× and dextrose 50% 75 g without rhythm change. Patient received naloxone 10 mg, epinephrine 2×, and bicarbonate 50 mEq and converted back to asystole.	Unknown outcome

Abbreviations: ALS, advanced life support; BLS, basic life support; ED, emergency department; EKG, electrocardiogram; HD, hospital day; PEA, pulseless electrical activity.

^a The interval between naloxone administration and the first recorded change in EKG rhythm.

^b All pharmacologic and external defibrillations administered starting at the time of ALS arrival and ending at the time of the first recorded EKG change following a naloxone dose. Unless otherwise specified: anoxic times unknown, all medications administered intravenously, epinephrine dose 1 mg of 1:10,000 concentration, and atropine dose 1 mg.

^c Delayed response.

Table 3
Number of patients receiving additional therapy.

	Immediate responders (N=7)	Delayed responders (N=8)
Epinephrine	7	8
Atropine	6	8
Dextrose 50%	0	2
Sodium bicarbonate	1	3
Calcium chloride	1	0

ders) or no change (non-responders). Changes in original rhythm noted immediately following naloxone administration, but before additional pharmacologic interventions, were defined as immediate changes. Delayed changes were defined as cardiac rhythm changes occurring after additional medications were administered but within a 10-min interval following initial naloxone dose. The primary outcome measure was change in cardiac activity from baseline based upon EKG rhythm. Secondary outcome measures examined included return of spontaneous circulation (ROSC), survival to hospital admission, and survival to hospital discharge.

2.8. Primary data analysis

For all patients receiving naloxone during cardiac arrest over the five-year period, the percentage that had any rhythm change following naloxone administration was calculated. Additionally, the percent of participants with changes immediately after receiving naloxone was determined. Finally, the percentage of patients who had a return of spontaneous circulation was obtained.

The participants who were classified as responders were followed through their hospital course. From this information, rates of survival to admission and survival to discharge were computed.

3. Results

3.1. Characteristics of study subjects

From a database of 32,544 advanced life support (ALS) emergency medical calls, 42 patients in non-traumatic cardiac arrest at the time of ALS dispatch received naloxone because of suspicion by a paramedic of acute opioid use. Six patients were excluded from the study, three for having palpable pulses at the time of naloxone administration, and three due to missing data on PCRs. The data points missing were route of administration, dose, or record of EKG rhythm. Of the excluded patients with missing data, one demonstrated a rhythm change following naloxone administration. Thirty-six patients remained for enrollment in the study (Fig. 1).

3.2. Main results

From January 1, 2003 to December 31, 2007, 15 of the 36 (42%) (95% confidence interval [CI]: 26–58) patients in cardiac arrest who received naloxone in the pre-hospital setting had a change in EKG rhythm. Table 1 demonstrates the baseline characteristics of patients with rhythm change (responders: N=15) and without (non-responders: N=21). Prior to naloxone administration, each of the 15 responders received other standard protocol interventions as deemed necessary by ALS personnel and described in Table 2. A summary of these interventions is shown in Table 3.

Of the participants who responded to naloxone, 47% (95% CI: 21–72) (19% [95% CI: 7–32] of all study subjects) demonstrated EKG rhythm changes immediately following the administration of naloxone (Table 4). During treatment by paramedics, 20% (95% CI: 0–40) of all responders converted from cardiac arrest to a perfusing rhythm.

Table 4
Characterization of patients with changes in cardiac rhythm.

	N=15	% (95% CI)
Immediate change	7	47 (21 to 72)
Delayed change	8	53 (28 to 79)
Improved to perfusing rhythm	3	20 (0 to 40)
Survival to admission	4	27 (4 to 49)
Survival to discharge	1	7 (–6 to 19)

Abbreviation: CI, confidence interval.

3.3. Supplemental analysis

A total of four responding patients survived to hospital admission. This includes a patient who regained a perfusing rhythm while in the emergency department. Two of the patients who regained perfusion subsequently died during their hospital course and one patient survived to be discharged from the hospital 11 days after admission. The fourth patient's outcome could not be obtained because of being transported to another hospital. Of note, the single patient who survived to discharge tested positive for opiates in a routine urine toxicology screen performed in the hospital emergency department. All 21 non-responders were pronounced in the pre-hospital setting or in the emergency department.

4. Discussion

Our retrospective chart review demonstrated a possible association between naloxone administration during cardiac arrest and cardiac rhythm changes. Forty-two percent of patients who received naloxone while in cardiac arrest demonstrated some change in their cardiac rhythm. Notably, 19% of all recipients demonstrated changes in cardiac rhythm immediately following naloxone but prior to additional ALS interventions. In addition, 20% of responders (8% of all subjects) had a post-intervention rhythm sustainable with life. However, due to our lack of control group we cannot claim to have established a causal relationship.

Causality must also be determined by a suggested mechanism of naloxone in cardiac arrest, yet this remains elusive. Opioid drug binding is not limited to one receptor type. In addition endogenous opioids exhibit substantial crossover among the different mu, kappa, and delta opioid receptor types, therefore making the mechanism difficult to pinpoint. Opioid induced cardiovascular toxicity manifests as arteriolar and venous dilatation through the release of histamine.^{8,9} Although cardiovascular toxicity results in part from degranulation of histamine containing vesicles, the majority of opioid pharmacologic actions are through the mu, kappa, delta, and nociceptin receptors. For example, respiratory depression is implicated via stimulation of μ_2 receptors that diminish sensitivity to hypercapnea and hypoxia.^{7,10}

The receptor properties that would obviously be most disadvantageous during cardiac arrest are respiratory depression and sedation.^{7,13,17,22,24} The mu receptor exhibits the strongest properties in this regard and is the receptor of most interest in cardiac arrest patients. In the proper doses, naloxone exhibits antagonistic effects at all receptors for endogenous and exogenous opioids.²⁴ The actions of opioid agonists at the mast cell level that cause histamine degranulation are independent of the opioid receptors. As such, they are not blocked by naloxone. The hypotensive effects of histamine are deleterious to the patient in cardiac arrest.²⁴

Alternatively, naloxone may alter cardiac function through involvement with catecholamines and the autonomic nervous system. Naloxone has been shown to stimulate catecholamine release and increase sympathetic nerve activity.^{12,13} This may be a mech-

anism for elevating heart rate and blood pressure as well as raising *in vivo* levels of catecholamines to act synergistically with those administered medically.

Several limitations of our study exist. First, standard ACLS medications were administered to our patients prior to naloxone; true causality between naloxone and its rhythmogenic effects is difficult to establish. However, even minimal improvements in cardiac arrest outcomes may be of meaningful importance. Without a prospective study and patient randomization, there exists the possibility that the results may be due to other interventions, or by chance. The possibility of a prospective randomized study in this population remains difficult as a result of strict pre-hospital consent protocols in our state. Second, time of initial cardiac arrest to naloxone administration could not be determined because most of the patients were found by ALS providers after unknown anoxic times. This interval is a confounding variable, which could not be analyzed in the study. Similarly, we cannot assume that our population mimics the asphyxia induced cardiac arrest model. Though opioid intoxication and subsequent respiratory depression can lead to cardiac arrest, it is not appropriate to assume based upon paramedic suspicion alone that our population was entirely composed of opioid intoxications. Thus, our results could be biased in either direction. Additionally, it was impossible to compare the responders to non-responders in terms of naloxone's adverse effects. Non-responders may have had worsened outcomes as a result of naloxone administration and because so many patients presented in asystole, it is impossible to make judgment about this group. Lastly, we are left with the questions of what should be the endpoint in evaluating the use of naloxone in cardiac arrest and what is the proper dose to observe that effect. We defined responders as those who had cardiac rhythm changes and patients were administered far lower doses than those used in animal models.^{5,6} A transient improvement in rhythm that still results in death does not constitute any real improvement. However, some post-naloxone rhythms may be more likely to lead to a perfusing rhythm, respond to antiarrhythmic medications, or convert to perfusing rhythm following defibrillation. Instead, our results simply represent a potential for improvement that may lead to a clinically significant change in a human model.

5. Conclusion

The utility of naloxone in suspected opioid arrests remains controversial. Based upon our data, we cannot firmly support its use during cardiac arrest involving any suspicion of opioid use. However, with current low rates of survival and low return of spontaneous circulation during cardiac arrest, any potential improvement in rhythm makes this a reasonable modality. With limited success of any medication in cardiac arrest, intervention with naloxone is a reasonable adjunctive treatment that poses little risk with potential benefit.

Conflict of interest statement

Only Dr Mark A Merlin has a potential conflict of interest to disclose. He receives a \$125,000 grant (#7015) from the American Heart Association. This grant is studying the written Advanced Cardiac Life Support (ACLS) exam with skills performance at 0-,

3- and 6-month intervals. The grant is not related to this submitted manuscript. Frank Dos Santos is currently an active duty commander in the US Navy Medical Corps and the views of this article do not necessarily represent the view of the Department of Defense or its components. No financial and personal relationships with other people or organizations that could inappropriately influence (bias) the work exist. Specifically, Scott Alter, Mathew Saybolt, Diane Calello, Kevin Rynn, Daniel Nelson and Frank Dos Santos have no conflict of interest.

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The administration of dextrose during in-hospital cardiac arrest is associated with increased mortality and neurologic morbidity

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Abstract

Introduction: Dextrose may be used during cardiac arrest resuscitation to prevent or reverse hypoglycemia. However, the incidence of dextrose administration during cardiac arrest and the association of dextrose administration with survival and other outcomes are unknown.

Methods: We used the Get With The Guidelines[®]-Resuscitation national registry to identify adult patients with an in-hospital cardiac arrest between the years 2000 and 2010. To assess the adjusted effects of dextrose administration on survival, we used multivariable regression models with adjustment for multiple patient, event, and hospital characteristics. We performed additional analyses to examine the effects of dextrose on neurological outcome and return of spontaneous circulation.

Results: Among the 100,029 patients included in our study, 4,189 (4.2%) received dextrose during cardiac arrest resuscitation. The rate of dextrose administration increased during the study period (odds ratio 1.11, 95% confidence interval (CI) 1.09-1.12 per year, $P < 0.001$). Patients who received dextrose during resuscitation had lower rates of survival compared with patients who did not receive dextrose (relative risk 0.88, 95% CI 0.80-0.98, $P = 0.02$). Administration of dextrose was associated with worse neurological outcome (relative risk 0.88, 95% CI 0.79-0.99, $P = 0.03$) but an increased chance of return of spontaneous circulation (relative risk 1.07, 95% CI 1.04-1.10, $P < 0.001$).

Conclusions: In this dataset, the administration of dextrose during resuscitation in patients with in-hospital cardiac arrest was found to be associated with a significantly decreased chance of survival and a decreased chance of good neurological outcome.

Introduction

In-hospital cardiac arrest (IHCA) is one of the leading causes of death in the United States, with an incidence of over 200,000 patients per year and a mortality rate of more than 75% [1]. Over the past decade, there have been enhancements to cardiac life support interventions, increased quality-improvement efforts, and improved

IHCA survival trends [2]. Nevertheless, the mortality rate for IHCA patients remains extremely high [1,3,4].

In 2005, the American Heart Association guidelines for advanced cardiac life support (ACLS) [5] listed hypoglycemia as a reversible cause of cardiac arrest but removed it upon the publication of the current 2010 ACLS guidelines [6]. Pre-2005 editions of the ACLS guidelines have never included hypoglycemia as a reversible cause of cardiac arrest, and the provision of dextrose during cardiac arrest in the absence of confirmed hypoglycemia is not suggested in the current guidelines [7]. To date, the current 2010 ACLS guidelines recommend the use of dextrose with insulin to treat severe hyperkalemia and suggest that insulin with dextrose can be considered for

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severe beta-blocker overdose, but neither support nor discourage the use of dextrose for any other condition [6,8].

The use of dextrose in cardiac arrest has not been adequately studied in the clinical setting, and the incidence of dextrose administration remains unknown [9]. Experimental evidence has suggested that dextrose administration might be harmful. Animal studies have shown that administering dextrose before, during, or after cardiac arrest leads to higher rates of mortality and worse neurological outcome [10-12]. In a study using pigs, hyperglycemia prior to cardiac arrest was associated with increased ischemic brain injury and increased markers of cerebral injury [13]. Similarly, human studies have shown that higher post-arrest blood glucose levels are associated with increased mortality and poor neurological outcome [14-20]. Hyperglycemia is also an independent predictor of mortality in myocardial infarction and stroke [21,22].

We hypothesized that the administration of dextrose during cardiac arrest resuscitation would be associated with higher post-arrest mortality and worse neurological outcome. To test this hypothesis, we used a large national registry of IHCA to establish the rate of dextrose administration during cardiac arrest. We then compared the survival with discharge of patients who received dextrose with patients who did not receive dextrose during cardiac arrest resuscitation. Secondly, we assessed the association between dextrose administration and return of spontaneous circulation (ROSC) and neurological outcome.

Methods

Data source

The Get With The Guidelines®-Resuscitation (GWTG-R) registry, formerly known as the National Registry of Cardiopulmonary Resuscitation, is a national, prospective, quality-improvement registry of IHCAs and is sponsored by the American Heart Association. The GWTG-R design for data collection and reliability has been described previously in detail [23]. In brief, trained research personnel at participating hospitals collect data on all IHCA patients who do not have prior do-not-resuscitate orders or cardiopulmonary resuscitation events that began outside of the hospital. Cardiac arrest is defined as pulselessness requiring chest compressions or defibrillation or both, with a hospital-wide or unit-based emergency response by acute care facility personnel. Cases are identified and data are extracted from cardiac arrest flow sheets, reviews of hospital paging system logs, routine checks of code carts, pharmacy drug records, and hospital billing charges for resuscitation medication [23].

To facilitate uniform reporting across hospitals, the registry employs Utstein-style templates for cardiac arrest, a set of standardized reporting guidelines used to define patient variables and outcomes [24,25]. Further

integrity of the data is ensured through rigorous certification of data entry personnel and the use of standardized software that checks the data for completeness and accuracy [26]. All participating hospitals are required to comply with local regulatory guidelines. Because data are used primarily at the local site for quality improvement, sites are granted a waiver of informed consent under the common rule. The institutional review board at Beth Israel Deaconess Medical Center (Boston, MA, USA) reviewed the present study and determined that it did not meet the federal definition of human subject research.

Study population

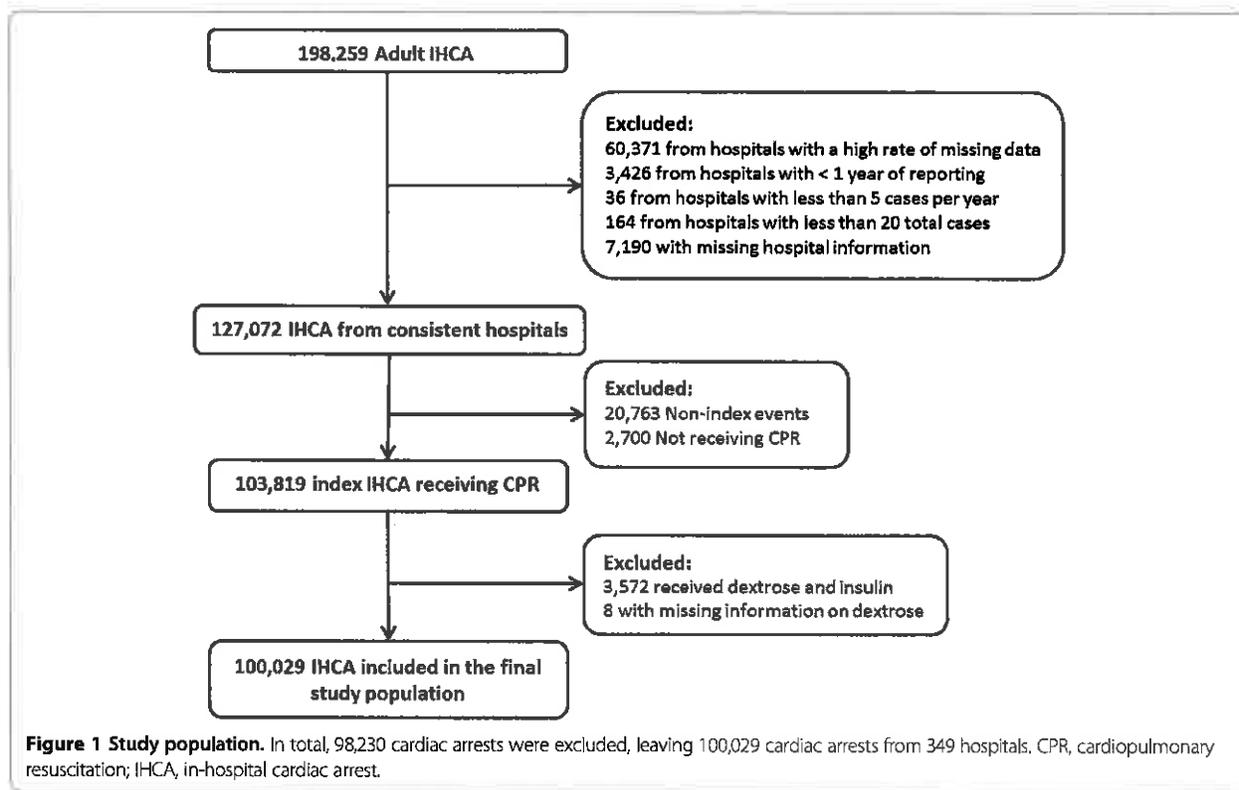
Our cohort study includes data submitted to the GWTG-R registry between January 2000 and September 2010. We included all patients 18 years or older. To secure the accuracy of the data, we excluded cases from hospitals with high rates of missing data, defined as an average rate of missing data for variables in our model of more than 10%. We also excluded cases from hospitals with fewer than five cases per year, fewer than a total of 20 reported cases, less than one year of reporting, and cases with missing hospital data. Non-index events and events without initiation of cardiopulmonary resuscitation were excluded. Patients with missing data on dextrose administration and patients who simultaneously received dextrose and insulin (recommended treatment for presumed hyperkalemia) were also excluded (Figure 1).

Study outcomes

The exposure variable, administration of dextrose, was defined as any administration of a dextrose bolus without concurrent administration of insulin during the cardiac arrest. Dextrose administration before or after the event was not included. The primary outcome of interest was survival to discharge. Secondary outcomes were good neurological outcome at the time of hospital discharge and ROSC, defined as at least 20 minutes with a palpable pulse. Neurological outcome was assessed with the use of the cerebral performance category (CPC) score, in which a CPC score of 1 indicates mild or no neurological deficit, 2 moderate cerebral disability, 3 severe cerebral disability, 4 coma or vegetative state, and 5 brain death [27]. A CPC score of 1 or 2 was considered a good neurological outcome, and a CPC score of 3 to 5 or death was considered a bad neurological outcome.

Statistical analysis

The study population was characterized by using descriptive statistics. Categorical variables are provided in frequencies and continuous variables in means with standard deviation or medians with interquartile range (IQR), depending on the normality of the data. Differences between variables were evaluated by using chi-squared



tests for categorical variables and Wilcoxon rank-sum tests for continuous variables. The change in incidence of dextrose administration over time (treated as a continuous variable for this analysis) was assessed by using unadjusted logistic regression, and the result is presented as an odds ratio (OR) with 95% confidence interval (CI).

To assess the independent association between dextrose administration during cardiac arrest resuscitation and survival to discharge, we used multivariable regression models with generalized estimating equations with an exchangeable (compound symmetry) correlation matrix to account for hospital clustering. Since the outcome was not rare (>10%), we used modified Poisson regression models with robust variance estimates to estimate risk ratios (RRs) as described by Zou [28] and Zou and Donner [29] and previously used in this cohort [2,30]. In our model, we adjusted for age, gender, race, coexisting conditions, arrest characteristics (including presumed cause of the arrest and initial rhythm), interventions during the arrest, and selected hospital characteristics (see Additional file 1: Table S1 for a full list of variables). Year of arrest was entered in the model as a categorical variable with year 2000 as the reference. All variables were chosen *a priori* on the basis of prior work and clinical reasoning [2,26,30-33]. Similar multivariate regression models were used to analyze secondary outcomes. Results from the multivariable regression models are reported as RRs with 95% CIs.

The rate of missing data in the study cohort was low (<1%) except for race (7.0%), initial rhythm (5.7%), downtime (4.7%), time of day (1.3%), and neurological outcome (2.6%). To account for missing data, we imputed the median value for patients of the same gender for all observations with missing covariates. We then did the multivariate logistic regression including the imputed values for those missing covariates. The point estimates for the variables included in the models with and without imputation were similar, and thus we have reported non-imputed models.

To determine the association between dextrose administration and post-resuscitation survival, we conducted a subgroup analysis on all patients who achieved ROSC. We performed a sensitivity analysis in which all patients with a downtime (see Additional file 1: Table S1 for a precise definition) of 5 minutes or less (to avoid confounding by potential survival bias) or with more than 10 minutes of downtime were excluded (to avoid the possibility that administration of glucose was purely a function of longer downtime). To further ensure that the patients who received dextrose were not being treated for hyperkalemia, we conducted a subgroup analysis excluding all patients who received calcium chloride or calcium gluconate. To test for effect modification, we conducted a pre-planned stratified analysis based on coexisting diabetes. We performed a number of *post hoc* analyses to assess other

potential subgroup differences. We assessed interaction terms in the main model between dextrose administration and the following: cardiac cause of the arrest defined as active/evolving myocardial infarction or arrhythmia (yes/no), cardiac reason for admission (yes/no), no coexisting sepsis or hepatic insufficiency (yes/no) (that is, potential reasons for hypoglycemia in a non-diabetic patient), location of the arrest (ICU versus non-ICU), and coexisting metabolic/electrolyte abnormalities within 4 hours of the arrest (inclusive of hypoglycemia), or presumed cause of the arrest as metabolic/electrolyte abnormality (yes/no).

To assess the robustness of our findings, we performed a propensity-matched adjusted analysis to test the association between glucose administration and each outcome. For the propensity-matched analysis, we used the imputed dataset and included all variables that had been included as independent variables in the primary analysis as well as hospital center. Next, we performed a 1:3 propensity score match between patients administered and not administered glucose by using an algorithm match caliper radius of 0.10 around the propensity score. We confirmed that the matched groups were balanced by ensuring that the standardized differences between groups for each covariate were less than 10. There was a small but statistically significant difference between cases and controls for three variables (Additional file 2: Table S2). With the 1:3 propensity-matched dataset, associations between glucose administration and outcomes were assessed with the Cochran-Mantel-Haenszel test to ensure comparison between matched pairs. Using these three variables, we performed an adjusted and unadjusted conditional logistic regression analysis. There was a less than 4% change in the point estimates (adjusted versus unadjusted), and the unadjusted results are presented here. Results from the propensity-matched analysis are reported as OR with 95% CIs.

Statistical analyses were conducted with SAS software version 9.3 (SAS Institute Inc., Cary, NC, USA). All hypothesis tests were two-sided, and a *P* value of less than 0.05 was considered significant.

Results

Characteristics of the study population

In total, 100,029 IHCAs from 349 hospitals were included in the main analysis (Figure 1). The median age was 69 (IQR 57–79), and 42% were female. Additional patient, arrest, and hospital characteristics are shown in Tables 1 and 2. Administration of dextrose occurred in 4,173 (4.2%) cardiac arrests. There was a significant increase in the incidence of dextrose administration from 2000 (2.5%) to 2010 (5.7%) (OR 1.11, 95% CI 1.09–1.12 per year, *P* < 0.001) (Figure 2).

Primary outcome

Eighteen point six percent of patients survived to hospital discharge. Patients who received dextrose during cardiac arrest resuscitation had a lower rate of survival to discharge compared with patients who did not receive dextrose (RR 0.49, 95% CI 0.44–0.54, *P* < 0.001). After multivariable adjustment, dextrose was still associated with a significantly decreased chance of survival to discharge (RR 0.88, 95% CI 0.80–0.98, *P* = 0.02) (Figure 3). See Additional file 3: Table S3 for the full model.

Secondary outcomes

Fifty-eight point two percent of patients achieved ROSC, and 13.7% of patients with full data had a good neurological outcome at hospital discharge (an additional 2.6% survived but had missing data on neurological outcome). In unadjusted analyses, administration of dextrose was associated with decreased chance of ROSC (RR 0.92, 95% CI 0.88–0.96, *P* < 0.001) and decreased chance of good neurological outcome (RR 0.43, 95% CI 0.38–0.49, *P* < 0.001). After multivariable adjustment, dextrose administration was associated with an increased chance of ROSC (RR 1.07, 95% CI 1.04–1.10, *P* < 0.001) and a decreased chance of good neurological outcome (RR 0.88, 95% CI 0.79–0.99, *P* = 0.03, Figure 3).

To further characterize the association between administration of dextrose and post-resuscitation survival, we conducted a subgroup analysis including only patients who obtained ROSC. In this subgroup, our multivariable analysis showed that administration of dextrose was still associated with both decreased chance of survival to discharge (RR 0.84, 95% CI 0.77–0.92, *P* < 0.001) and decreased chance of good neurological outcome (RR 0.84, 95% CI 0.76–0.93, *P* = 0.001). In our sensitivity analysis of patients with a downtime between 5 and 10 minutes, we found that dextrose was associated with an increased chance of ROSC (RR 1.06, 95% CI 1.01–1.10, *P* = 0.008), a strong trend toward decreased chance of survival (RR 0.83, 95% CI 0.68–1.01, *P* = 0.06), and decreased chance of good neurological outcome (RR 0.78, 95% CI 0.62–0.99, *P* = 0.04).

We conducted a pre-planned analysis in order to investigate potential effect modification by coexisting diabetes (type I or type II). Thirty point six percent of the overall population had documented diabetes, and dextrose was more commonly administered in patients with diabetes than in patients without diabetes (5.6% versus 3.5%, *P* < 0.001). There was a significant interaction between dextrose administration and diabetes status with survival as outcome (*P* = 0.02) but not with ROSC (*P* = 0.46) or good neurological outcome (*P* = 0.23) as the outcome measure. In patients with diabetes, administration of dextrose was not associated with survival to discharge (RR 0.94, 95% CI 0.83–1.07, *P* = 0.32), whereas in patients without diabetes, the administration

Table 1 Characteristics of the study population according to dextrose administration

Characteristic	Received dextrose during cardiac arrest		P value
	No (n = 95,856)	Yes (n = 4,173)	
Demographics			
Age in years, median (IQR)	69 (57–79)	65 (53–77)	<0.001
Sex, number (percentage)			0.03
Female	40,306 (42.1)	1,690 (40.3)	
Male	55,550 (58.0)	2,499 (59.7)	
Race, number (percentage)			<0.001
White	68,474 (76.8)	2,527 (64.8)	
Black	17,109 (19.2)	1,208 (31.0)	
Other	3,551 (4.0)	165 (4.2)	
Type of admission, number (percentage)			
Medical-Non-cardiac	41,798 (43.6)	2,267 (54.3)	<0.001
Medical-Cardiac	32,031 (35.4)	1,212 (29.1)	
Surgical-Non-cardiac	10,877 (11.4)	421 (10.1)	
Surgical-Cardiac	6,188 (6.5)	185 (4.4)	
Trauma	2,777 (2.9)	73 (1.8)	
Other	272 (0.3)	14 (0.3)	
Pre-existing conditions, number (percentage)			
Cardiac			
Arrhythmia	31,414 (32.9)	1,188 (28.5)	<0.001
History of MI	15,864 (16.6)	584 (14.0)	<0.001
MI this admission	17,148 (17.9)	472 (11.3)	<0.001
History of heart failure	20,090 (21.0)	911 (21.9)	0.19
Heart failure this admission	17,091 (17.9)	702 (16.8)	0.08
Non-cardiac			
Respiratory insufficiency	40,001 (41.9)	1,672 (40.2)	0.03
Diabetes mellitus	28,759 (30.1)	1,717 (41.2)	<0.001
Renal insufficiency	30,784 (32.2)	1,838 (44.0)	<0.001
Metastatic/Hematologic malignancy	11,801 (12.4)	471 (11.3)	0.05
Hypotension/Hypoperfusion	26,263 (27.5)	1,107 (26.6)	0.21
Pneumonia	13,015 (13.6)	592 (14.2)	0.30
Baseline depression in CNS function	12,273 (12.8)	564 (13.5)	0.18
Metabolic/Electrolyte abnormality	15,477 (16.2)	974 (23.3)	<0.001
Septicemia	14,586 (15.3)	872 (20.9)	<0.001
Acute CNS non-stroke event	6,991 (7.3)	314 (7.5)	0.58
Hepatic insufficiency	6,724 (7.0)	431 (10.4)	<0.001
Acute stroke	3,686 (3.9)	144 (3.5)	0.18
Major trauma	3,587 (3.8)	104 (2.5)	<0.001

CNS, central nervous system; IQR, interquartile range; MI, myocardial infarction.

of dextrose was associated with a decreased chance of survival (RR 0.80, 95% CI 0.69-0.94, $P = 0.005$) (Figure 4). After exclusion of patients who received calcium chloride or calcium gluconate, the association between dextrose

administration and mortality remained (RR 0.88, 95% CI 0.78-0.98, $P = 0.02$). None of the other interaction terms tested (see Methods section) was statistically significant, indicating no subgroup differences.

Table 2 Arrest and hospital characteristics according to dextrose administration

Characteristic	Received dextrose during cardiac arrest		P value
	No (n = 95,856)	Yes (n = 4,173)	
Location and time of the arrest, number (percentage)			
Location			<0.001
Floor without telemetry	15,855 (16.6)	1,025 (24.6)	
Floor with telemetry	16,710 (17.4)	730 (17.5)	
Intensive care unit	45,011 (47.0)	1,597 (38.3)	
Emergency department	10,363 (10.8)	544 (13.0)	
Other	6,974 (7.6)	276 (6.6)	
Time of day			<0.001
Day (7 a.m.-10:59 p.m.)	63,897 (67.6)	2,666 (64.5)	
Night (11 p.m.-6:59 a.m.)	30,673 (32.4)	1,467 (35.5)	
Time of week, number (percentage)			0.30
Weekday (Monday-Friday)	65,269 (68.8)	2,818 (68.0)	
Weekend (Saturday-Sunday)	29,642 (31.2)	1,326 (32.0)	
Hospital-wide response called, number (percentage)	76,720 (80.0)	3,361 (80.5)	0.40
Characteristic of the arrest			
Monitoring, number (percentage)	78,166 (81.6)	3,053 (73.2)	<0.001
Witnessed, number (percentage)	78,262 (81.7)	3,050 (73.1)	<0.001
First rhythm shockable (VT or VF), number (percentage)	18,340 (20.3)	517 (13.0)	<0.001
Mechanical ventilation in place, number (percentage)	27,928 (29.1)	1,103 (26.4)	<0.001
Airway inserted during event, number (percentage)	51,028 (53.3)	2,680 (64.2)	<0.001
Presumed cause(s) of arrest, number (percentage)			
Arrhythmia	56,147 (58.9)	2,215 (53.4)	<0.001
Hypotension/Hypoperfusion	37,571 (39.4)	1,596 (38.5)	0.22
Active/Evolving MI	8,919 (9.4)	270 (6.5)	<0.001
Acute respiratory insufficiency	37,064 (38.9)	1,670 (40.3)	0.08
Metabolic/Electrolyte abnormality	10,809 (11.4)	860 (20.7)	<0.001
Other	7,619 (8.0)	345 (8.3)	0.46
Unknown	10,340 (10.9)	578 (13.9)	<0.001
Downtime in minutes, median (IQR)	12 (6-21)	18 (10-27)	<0.001
Medications given during the event, number (percentage)			
Amiodarone	14,806 (15.5)	703 (16.9)	0.01
Epinephrine	84,336 (88.0)	4,019 (96.3)	<0.001
Atropine	67,947 (70.9)	3,490 (83.6)	<0.001
Magnesium sulfate	7,156 (7.5)	567 (13.6)	<0.001
Lidocaine	10,152 (10.6)	381 (9.1)	0.003
Sodium bicarbonate	43,775 (45.7)	3,111 (74.6)	<0.001
Fluid bolus	28,011 (29.2)	1,452 (34.7)	<0.001
Calcium chloride/gluconate	20,188 (21.1)	1,955 (46.9)	<0.001
Norepinephrine	12,500 (13.0)	675 (16.2)	<0.001
Dopamine	23,078 (24.1)	1,037 (24.1)	0.25

Table 2 Arrest and hospital characteristics according to dextrose administration (Continued)

Hospital characteristics, number (percentage)			
Bed size			<0.001
1-249	23,450 (24.2)	908 (21.7)	
250-499	44,001 (45.9)	1,836 (44.0)	
500+	28,408 (29.6)	1,429 (34.1)	
Teaching status			<0.001
Major	25,246 (26.3)	1,527 (36.6)	
Minor	33,930 (35.3)	1,162 (27.7)	
Non-teaching	36,680 (38.2)	1,484 (35.4)	
Ownership			<0.001
Private	12,247 (12.8)	540 (12.9)	
Government	14,407 (15.0)	899 (21.5)	
Non-profit	69,202 (72.2)	2,734 (65.5)	
Location			<0.001
Rural	6,274 (6.5)	216 (5.2)	
Urban	89,582 (93.5)	3,957 (94.8)	
Geographical location			<0.001
North-East	10,484 (10.9)	491 (11.7)	
South-East	26,019 (27.1)	1,125 (26.9)	
Mid-West	23,539 (21.0)	1,099 (26.9)	
South-West	19,998 (20.9)	864 (21.4)	
West	15,816 (16.5)	564 (13.5)	

IQR, Interquartile range; PEA, pulseless electrical activity; VF, ventricular fibrillation; VT, ventricular tachycardia.

Results from the propensity-matched analysis

The propensity matching resulted in a successful match of 4,171 patients administered dextrose to 12,498 patients who did not receive dextrose. The groups were well matched on covariates (Additional file 2: Table S2).

With our propensity-matched dataset, administration of dextrose was associated with a significantly decreased chance of survival to discharge (OR 0.80, 95% CI 0.71-0.90, $P < 0.001$). Dextrose administration was likewise associated with a decreased chance of good neurological outcome (OR 0.79, 95% CI 0.68-0.91, $P = 0.001$). However, the association between dextrose administration and ROSC was not significant when the propensity-matched analysis was used (OR 1.06, 95% CI 0.99-1.13, $P = 0.12$).

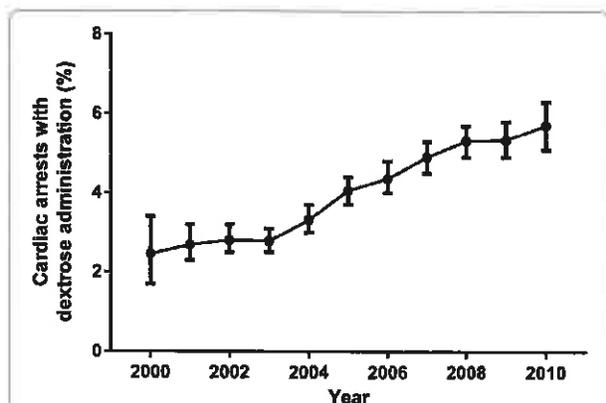
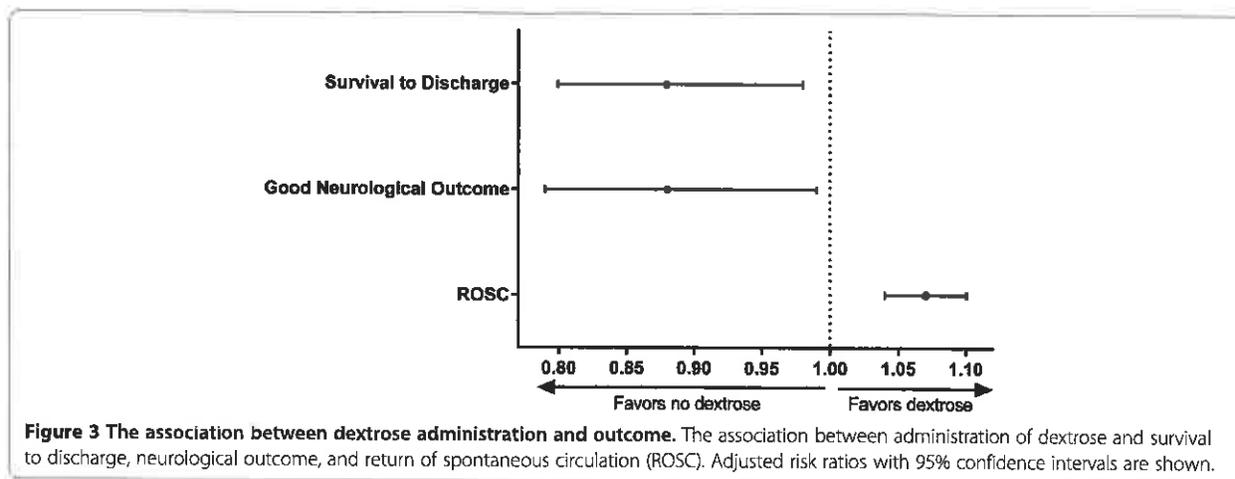


Figure 2 Incidence of dextrose administration over time. Percentage of cardiac arrests with dextrose administration over time. Error bars indicate exact binomial 95% confidence intervals (CIs). There was a steady increase in the incidence of dextrose administration from 2000 (2.5%) to 2010 (5.7%) (odds ratio 1.11, 95% CI 1.09-1.12 per year, $P < 0.001$).

Discussion

In this large cohort study using the GWTG-R national database, we examined the association between dextrose administration and outcomes after cardiac arrest. We found that the use of dextrose during resuscitation is independently associated with lower rates of survival and unfavorable neurological outcomes. These associations remained significant even after multivariable adjustments and sensitivity analyses. The association between dextrose administration and lower rates of survival and unfavorable neurological outcomes furthermore remained in our propensity-matched analyses. In our primary analysis, dextrose administration was associated with slightly higher rates of ROSC; however, this association was no

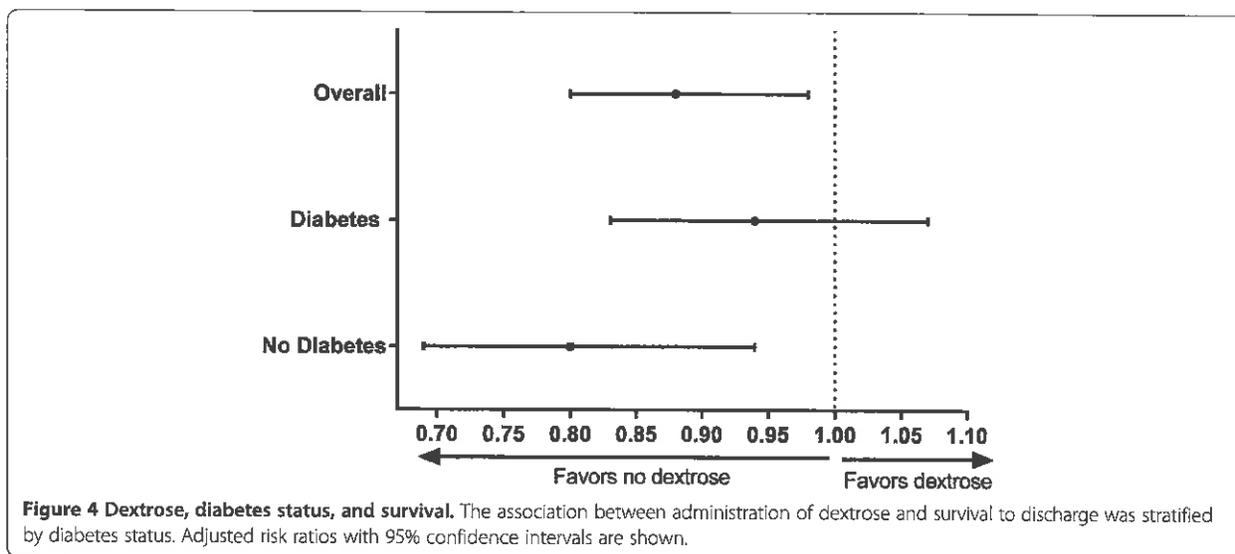


longer significant with propensity matching, making this finding difficult to interpret.

Although we cannot conclude from retrospective data why clinicians were giving dextrose during cardiac arrest, we would hypothesize that concern for hypoglycemia as the cause of arrest was likely a primary reason. Dextrose may also be administered out of concern that hypoglycemia is associated with higher mortality and potentially brain injury [34,35]. In dealing with a high-mortality event like cardiac arrest, clinicians are motivated to find and treat any potential reversible cause. With this in mind, in a truly hypoglycemic patient, the use of dextrose is probably recommended. However, studies have shown that hypoglycemia can be easily misdiagnosed in patients experiencing ischemic injuries. In current medical practice, a sample of capillary blood taken with a fingerstick blood glucose test is the quickest method to assess the possibility of hypoglycemia. However, studies

have shown that fingerstick blood glucose measurements are inaccurate in patients in shock [36,37] or cardiac arrest [38]. However, the use of venous blood with a bedside glucometer is accurate and could be used if deemed necessary.

The administration of dextrose in normoglycemic or hyperglycemic patients can lead to higher blood glucose levels. Patients with hyperglycemia after cardiac arrest [14-18] and other ischemic injuries (stroke [39] and head injury [40,41]) have longer recovery times and worse neurological outcomes. Traditionally, this elevation in blood glucose has been assumed to be part of a systemic stress response. The potential mechanisms behind elevated blood glucose levels and the association with poor outcome are not well understood. Prior studies have suggested that having higher blood glucose levels during periods of ischemia increases anaerobic metabolism, promotes the conversion of pyruvate into lactate, causes



intracellular acidosis, and may decrease cerebral blood flow, exacerbating cerebral ischemic injury [42-44]. Whether hyperglycemia plays a causal role in this process or is simply a marker of a systemic stress response or of dysfunctional metabolism at the cellular level is not clear.

One randomized study examined the relationship between dextrose administration after out-of-hospital cardiac arrest and neurological outcome and found no difference between the patients receiving 5% dextrose and those receiving half normal saline [45]. However, the average dose of dextrose used in this study was low (7 g) compared with the usual bolus dose (50 mL) of 50% dextrose, which contains 25 g of dextrose. To date, no study has explored the relationship between the use of a dextrose bolus during cardiac arrest resuscitation and outcome.

Our subgroup analysis revealed that although the use of dextrose was associated with higher mortality in non-diabetic patients, the association was not significant in patients with diabetes. The resilience of patients with diabetes to the deleterious effects of acute hyperglycemia has been documented [17,46]. Having chronically elevated levels of blood glucose can cause structural and functional modifications, such as a greater buffering capacity and lower cerebral pH levels after induced cardiac arrest [47]. Chronic hyperglycemia has also been found to be partially protective against cerebral hypoxia caused by acute hyperglycemia [42]. The underlying mechanism of the potential protective effects of diabetes on acute hyperglycemia needs to be clarified in future studies. Another potential explanation for our findings is the higher rate of true hypoglycemia in diabetic patients for whom administration of dextrose would likely be beneficial.

The results of our study should be interpreted in the context of the following limitations. Even though the GWTG-R registry has a rigorous training and certification process and employs standardized definitions, the data acquired by the registry may contain data integrity and validity issues. Although the GWTG-R registry is large, participation is still voluntary and this raises the potential for selection bias. Also, GWTG-R is a quality-improvement registry and was not specifically tailored to study the effects of dextrose in cardiac resuscitation. Given the observational nature of the present study, we cannot exclude the possibility that unmeasured or residual confounding remains. We were unable to identify the reason why dextrose was administered, the dosage that was given, and the timing in which it was administered. In addition, information on whether patients were receiving dextrose-containing fluid before or after the event was not available, and glucose levels were not available for any patients. It is also possible that dextrose was more likely to be administered in patients in whom initial resuscitation attempts failed, giving them a poorer prognosis. However, we believe that we addressed this potential issue in multiple ways: downtime was

included in our multivariable model, and our sensitivity analyses of cardiac arrests with downtime of between 5 and 10 minutes gave largely similar results as our primary analyses. Also, potential confounding by intra-arrest variables such as prolonged downtime or non-adherence to recommended protocols cannot explain the increased chance of ROSC seen in our primary analysis. The post-cardiac arrest population is a heterogeneous group. Despite our pre-defined and *post hoc* subgroup analyses, there still could be certain subgroups of patients for whom dextrose administration is beneficial. As always, patient care should be individually tailored to the clinical situation. Finally, given the exploratory nature of our analysis, the results should be verified in a prospective manner or in a dataset with more granular data (that is, reason for dextrose administration, timing of dextrose administration, and intra- and post-cardiac arrest glucose levels) or both before any conclusions regarding clinical practice can be made.

Conclusions

Although a causal relationship cannot be determined, our analysis shows that cardiac arrest patients receiving dextrose during resuscitation have a decreased chance of survival to hospital discharge and a decreased chance of good neurological outcome. This association seems to be driven primarily by an effect in the non-diabetic population.

Key messages

- The association between dextrose administration during cardiac arrest and survival is unknown.
- Dextrose is used in approximately 4.2% of all in-hospital cardiac arrests, with an increasing rate from 2000 to 2010.
- Patients who received dextrose during resuscitation had lower rates of survival compared with patients who did not receive dextrose, although whether this relationship is causal remains unproven.
- This association was maintained when using multivariable regression, sensitivity analyses, and a propensity-matched analysis.
- The association between dextrose administration and poor survival seems to be driven primarily by an effect in the non-diabetic population.

Additional files

Additional file 1: Table S1. Definitions of covariates and outcomes.

Additional file 2: Table S2. Characteristics of propensity-matched groups.

Additional file 3: Table S3. Primary multivariable model: association between multiple variables and survival to discharge.

Abbreviations

ACLS: advanced cardiac life support; CPC: cerebral performance category; GWTG-R: Get With The Guidelines®-Resuscitation; IHCA: in-hospital cardiac arrest; IQR: interquartile range; OR: odds ratio; ROSC: return of spontaneous circulation; RR: risk ratio.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

TJP shared responsibility for study conception and design and helped to draft the manuscript. LWA shared responsibility for study conception and design, helped to perform statistical analyses, and helped to draft the manuscript. MWD shared responsibility for study conception and design. BZS and VN helped to perform statistical analyses. All authors took part in critical revision of the manuscript, interpreted the data, provided intellectual content, shared responsibility for revising the manuscript, and read and approved the final submission and agree to be accountable for all aspects of the work.

Authors' information

TJP and LWA are co-first authors.

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CARDIAC ARREST - ADULT

I. FIELD ASSESSMENT/TREATMENT INDICATORS

Cardiac arrest in a non-traumatic setting.

II. BLS INTERVENTIONS

- Assess patient, begin CPR according to current AHA Guidelines, and maintain appropriate airway.
 - Compression rate shall be 100 per minute utilizing 30:2 compression-to-ventilation ratio for synchronous CPR prior to placement of advanced airway.
 - Ventilatory volumes shall be sufficient to cause adequate chest rise.
- Place patient on AED. CPR is **not** to be interrupted except briefly for rhythm assessment.

III. LIMITED ALS (LALS) INTERVENTIONS

- Initiate CPR while applying the AED.
- Establish advanced airway when resources are available, with minimal interruption to chest compressions. After advanced airway established, compressions would then be continued at 100 per minute without pauses during ventilations.
- Establish peripheral intravenous access and administer a 500 ml bolus of normal saline (NS).
- Refer to ICEMA Reference #12010 - Determination of Death on Scene.
- Obtain blood glucose level, if indicated administer:
 - Dextrose per ICEMA Reference #7040 - Medication - Standard Orders.
 - May repeat blood glucose level. Repeat Dextrose per ICEMA Reference #7040 - Medication - Standard Orders if indicated.

- If suspected narcotic overdose with severely decreased respiratory drive administer:
 - Naloxone per ICEMA Reference #7040 - Medication - Standard Orders.

NOTE: Base hospital contact is required to terminate resuscitative measures.

IV. ALS INTERVENTIONS

- Initiate CPR while applying the cardiac monitor.
- Determine cardiac rhythm and defibrillate if indicated. Begin a two (2) minute cycle of CPR.
- Obtain IV/IO access.
- Establish advanced airway when resources are available, with minimal interruption to chest compressions. After advanced airway established, compressions would then be continued at 100 per minute without pauses during ventilations. Ventilations should be given at a rate of one (1) breath every six (6) to eight (8) seconds.
- Utilize continuous quantitative waveform capnography, for confirmation and monitoring of endotracheal tube placement and for assessment of ROSC and perfusion status. Document the shape of the wave and the capnography number in mmHG.
- Insert NG/OG Tube to relieve gastric distension per ICEMA Reference #10190 - ICEMA Approved Skills.
- Obtain blood glucose level. If indicated administer:
 - Dextrose per ICEMA Reference #7040 - Medication - Standard Orders.
 - May repeat blood glucose level. Repeat Dextrose per ICEMA Reference #7040 - Medication - Standard Orders if indicated.
- If suspected narcotic overdose with severely decreased respiratory drive administer:
 - Naloxone per ICEMA Reference #7040 - Medication - Standard Orders.

- If ROSC is achieved, obtain a 12-lead ECG and contact a STEMI base hospital for destination decision, refer to ICEMA Reference #8130 - Destination Policy.
- Utilize continuous waveform capnography, to identify loss of circulation.
- For continued signs of inadequate tissue perfusion after successful resuscitation, administer:
 - Dopamine per ICEMA Reference #7040 - Medication - Standard Orders to maintain signs of adequate tissue perfusion.
- Base hospital physician may order additional medications or interventions as indicated by patient condition.

Ventricular Fibrillation/Pulseless Ventricular Tachycardia

- Defibrillate at 360 joules for monophasic or biphasic equivalent per manufacture. If biphasic equivalent is unknown use maximum available.
- Perform CPR for two (2) minutes after each defibrillation, without delaying to assess the post-defibrillation rhythm.
- Administer Epinephrine per ICEMA Reference #7040 - Medication - Standard Orders during each two (2) minute cycle of CPR after every defibrillation unless capnography indicates possible ROSC.
- Reassess rhythm after each two (2) minute cycle of CPR. If VF/VT persists, defibrillate as above.
- After two (2) cycles of CPR, consider administering:
 - Lidocaine per ICEMA Reference #7040 - Medication - Standard Orders.
- If patient remains in pulseless VF/VT after five (5) cycles of CPR, consult base hospital.

Pulseless Electrical Activity (PEA) or Asystole

- Assess for reversible causes and initiate treatment.
- Continue CPR with evaluation of rhythm every two (2) minutes.
- Administer fluid bolus of 300 ml NS IV, may repeat.

- Administer Epinephrine per ICEMA Reference #7040 - Medication - Standard Orders during each two (2) minute cycle of CPR after each rhythm evaluation.

Termination of Efforts in the Prehospital Setting

- The decision to terminate efforts in the field should take into consideration, first, the safety of personnel on scene, and then family and cultural considerations.
- Consider terminating resuscitative efforts in the field if ALL of the following criteria are met:
 - No shocks were delivered.
 - No ROSC after a minimum of ten (10) minutes of advance cardiac life support (ACLS).
- Base hospital contact is required to terminate resuscitative measures. A copy of the ECG should be attached to the patient care report for documentation purposes.

V. REFERENCES

<u>Number</u>	<u>Name</u>
7040	Medication - Standard Orders
8130	Destination Policy
10190	ICEMA Approved Skills
12010	Determination of Death on Scene



MEDICATION - STANDARD ORDERS

Adenosine (Adenocard) - Adult (ALS)

Stable narrow-complex SVT or Wide complex tachycardia:

Adenosine, 6 mg rapid IVP followed immediately by 20 cc NS bolus, and
Adenosine, 12 mg rapid IVP followed immediately by 20 cc NS bolus if patient
does not convert. May repeat one (1) time.

Reference #s 7010, 7020, 11050

Albuterol Aerosolized Solution (Proventil) - Adult (LALS, ALS)

Albuterol nebulized, 2.5 mg, may repeat two (2) times.

Reference #s 6090, 7010, 7020, 11010, 11100, 14030

Albuterol Metered-Dose Inhaler (MDI) (Proventil) - Specialty Programs Only Adult (LALS, ALS)

Albuterol MDI, four (4) puffs every ten (10) minutes for continued shortness of
breath and wheezing.

Reference #s 6090, 6110, Sheriff's Search and Rescue

Albuterol - Pediatric (LALS, ALS)

Albuterol nebulized, 2.5 mg, may repeat two (2) times.

Reference #s 7010, 7020, 14010, 14030, and 14070

Aspirin, chewable (LALS, ALS)

Aspirin, 325 mg PO chewed (one (1) adult non-enteric coated aspirin) or four (4)
chewable 81 mg aspirin.

Reference #s 2020, 6090, 6110, 7010, 7020, 11060

Atropine (ALS)

Atropine, 0.5 mg IV/IO. May repeat every five (5) minutes up to a maximum of
3 mg or 0.04 mg/kg.

Organophosphate poisoning:

Atropine, 2 mg IV/IO, repeat at 2 mg increments every five (5) minutes if patient remains symptomatic.

Reference #s 6090, 6110, 7010, 7020, 11040, 12020, 13010

Calcium Chloride (ALS)

Calcium Channel Blocker Poisonings:

Calcium Chloride, 1 gm (10 cc of a 10% solution) IV/IO, base hospital order only.

Reference #s 2020, 7010, 7020, 13010

Dextrose - Adult (LALS, ALS)

Dextrose 10%/250 ml (D10W 25 gm) IV/IO Bolus

Reference #s 2020, 6090, 6110, 7010, 7020, 8010, 11050, 11070, 11080, 13020, 13030

Dextrose - Pediatric (LALS, ALS)

Hypoglycemia - Neonates (0 - 4 weeks) with blood glucose < 35 mg/dL or pediatric patients (greater than 4 weeks) with glucose < 60 mg/dL:

Dextrose 10%/250 ml (D10W 25 gm) 0.5 gm/kg (5 ml/kg) IV/IO

Reference #s 2020, 7010, 7020, 13020, 13030, 14040, 14050, 14060

Diphenhydramine - Adult (ALS)

Diphenhydramine, 25 mg IV/IO

Diphenhydramine, 50 mg IM

Reference #s 6090, 6110, 7010, 7020, 11010, 13010

Diphenhydramine - Pediatric (ALS)

Diphenhydramine, 1 mg/kg slow IV/IO, not to exceed adult dose of 25 mg, **or**

Diphenhydramine, 2 mg/kg IM not to exceed adult dose of 50 mg IM

Reference #s 7010, 7020, 14030

Dopamine - Adult (ALS)

Dopamine, infusion of 400 mg in 250 ml of NS IV/IO, titrated between 5 - 20 mcg/kg/min to maintain signs of adequate tissue perfusion.

Reference #s 7010, 7020, 8010, 8040, 10140, 11070, 11090, 14080

Dopamine - Pediatric (ALS)

Post resuscitation continued signs of inadequate tissue perfusion:

9 to 14 years Dopamine, 400 mg in 250 ml of NS to infuse at 5 - 20 mcg/kg/min IV/IO titrated to maintain signs of adequate tissue perfusion.

Reference #s 7010, 7020, 14040

Epinephrine (1:1000) - Adult (LALS, ALS)

Severe Bronchospasm, Asthma Attack, Pending Respiratory Failure, Anaphylactic Shock/Severe Allergic Reactions:

Epinephrine, 0.3 mg IM

Epinephrine (1:10,000) - Adult (ALS)

For Persistent severe anaphylactic shock:

Epinephrine (1:10,000), 0.1 mg slow IVP/IO. May repeat every five (5) minutes as needed to total dosage of 0.5 mg.

Cardiac Arrest, Asystole, PEA:

Epinephrine, 1 mg IV/IO

Reference #s 2020, 6090, 6110, 7010, 7020, 11010, 11070, 12020

Epinephrine (1:1000) - Pediatric (LALS, ALS)

Severe Bronchospasm, Asthma Attack, Pending Respiratory Failure, Anaphylactic Shock/Severe Allergic Reactions:

Epinephrine, 0.01 mg/kg IM not to exceed adult dosage of 0.3 mg.

Reference #s 2020, 6090, 7010, 7020, 11010, 14010, 14030

Epinephrine (1:10,000) - Pediatric (ALS)

Anaphylactic Shock (no palpable radial pulse and depressed level of consciousness):

Epinephrine (1:10,000), 0.01 mg/kg IV/IO, no more than 0.1 mg per dose. May repeat to a maximum of 0.5 mg.

Cardiac Arrest:

1 day to 8 years Epinephrine (1:10,000), 0.01 mg/kg IV/IO (do not exceed adult dosage)

9 to 14 years Epinephrine (1:10,000), 1.0 mg IV/IO

Newborn Care:

Epinephrine (1: 10,000), 0.01mg/kg IV/IO if heart rate is less than 60 after one (1) minute after evaluating airway for hypoxia and assessing body temperature for hypothermia.

Epinephrine (1:10,000), 0.005 mg/kg IV/IO every ten (10) minutes for persistent hypotension as a base hospital order or in radio communication failure.

Post resuscitation continued signs of inadequate tissue perfusion:

1 day to 8 years Epinephrine (1:10,000), 0.5 mcg/kg/min IV/IO drip

Reference #s 2020, 7010, 7020, 14030, 14040, 14090

Fentanyl - Adult (ALS)

Fentanyl, 50 mcg slow IV/IO over one (1) minute. May repeat every five (5) minutes titrated to pain, not to exceed 200 mcg.

Fentanyl, 100 mcg IM/IN. May repeat 50 mcg every ten (10) minutes titrated to pain, not to exceed 200 mcg.

Isolated Extremity Trauma, Burns:

Fentanyl, 50 mcg slow IV/IO push over one (1) minute. May repeat every five (5) minutes titrated to pain, not to exceed 200 mcg IV/IO, **or**

Fentanyl, 100 mcg IM/IN. May repeat 50 mcg every ten (10) minutes titrated to pain, not to exceed 200 mcg.

Pacing, synchronized cardioversion:

Fentanyl, 50 mcg slow IV/IO over one (1) minute. May repeat in five (5) minutes titrated to pain, not to exceed 200 mcg.

Fentanyl, 100 mcg IN. May repeat 50 mcg every ten (10) minutes titrated to pain, not to exceed 200 mcg.

Reference #s 2020, 6090, 6110, 7010, 7020, 7030, 10190, 11060, 11100, 13030, 15010

Fentanyl - Pediatric (ALS)

Fentanyl, 0.5 mcg/kg slow IV/IO over one (1) minute. May repeat in five minutes titrated to pain, not to exceed 100 mcg.

Fentanyl, 1 mcg/kg IM/IN, may repeat every ten (10) minutes titrated to pain not to exceed 200 mcg.

Reference #s 2020, 6110, 7010, 7020, 7030, 11060, 13030, 14070, 15020

Glucose - Oral - Adult (BLS, LALS, ALS)

Glucose - Oral, one (1) tube for patients with an intact gag reflex and hypoglycemia.

Reference #s 7010, 7020, 11080, 11090, 11110, 13020

Glucose - Oral - Pediatric (BLS, LALS, ALS)

Glucose - Oral, one (1) tube for patients with an intact gag reflex and hypoglycemia.

Reference #s 7010, 7020, 14050, 14060

Glucagon - Adult (LALS, ALS)

Glucagon, 1 mg IM/SC/IN, if unable to establish IV. May administer one (1) time only.

Betablocker Poisoning:

Glucagon, 1 mg IV/IO (base hospital order only)

Reference #s 6090, 6110, 7010, 7020, 11080, 13010, 13030

Glucagon - Pediatric (LALS, ALS)

Glucagon, 0.025 mg/kg IM/IN, if unable to start an IV. May be repeated one (1) time after twenty (20) minutes for a combined maximum dose of 1 mg.

Reference #s 7010, 7020, 13030, 14050, 14060

Ipratropium Bromide Inhalation Solution (Atrovent) - Adult (ALS) use with Albuterol

Atrovent, 0.5 mg nebulized. Administer one (1) dose only.

Reference #s 7010, 7020, 11010, 11100

Ipratropium Bromide Metered-Dose Inhaler (MDI) (Atrovent) - Specialty Programs Only Adult (ALS) use with Albuterol

When used in combination with Albuterol MDI use Albuterol MDI dosing.

Reference #s 6090, 6110, 7010, 7020

Ipratropium Bromide Inhalation Solution (Atrovent) - Pediatric (ALS) use with Albuterol

1 day to 12 months Atrovent nebulized, 0.25 mg. Administer one (1) dose only.

1 year to 14 years Atrovent nebulized, 0.5 mg. Administer one (1) dose only.

Reference #s 7010, 7020, 14010, 14030, 14070

Lidocaine - Adult (ALS)

Intubation, King Airway, NG/OG, for suspected increased intracranial pressure (ICP):

Lidocaine, 1.5 mg/kg IV/IO

VT/VF:

Initial Dose: Lidocaine, 1.5 mg/kg IV/IO

May administer an additional 0.75 mg/kg IV/IO, repeat once in five (5) to ten (10) minutes for refractory VF.

VT/VF Infusion:

Lidocaine, 2 mg/min IV/IO drip

V-Tach, Wide Complex Tachycardia – with Pulses:

Lidocaine, 1.5 mg/kg slow IV/IO

May administer an additional 0.75 mg/kg IV/IO, repeat once in five (5) to ten (10) minutes for refractory VF

Initiate infusion of Lidocaine 2 mg/min IV/IO drip.

Reference #s 2020, 6090, 7010, 7020, 8010, 8040, 10030, 10080, [10190](#), 11050, 11070, 15010

Lidocaine - Pediatric (ALS)

Intubation, King Airway, NG/OG, for suspected increased intracranial pressure (ICP):

Lidocaine, 1.5 mg/kg IV/IO

Cardiac Arrest:

1 day to 8 years Lidocaine, 1.0 mg/kg IV/IO

9 to 14 years Lidocaine, 1.0 mg/kg IV/IO

May repeat Lidocaine at 0.5 mg/kg after five (5) minutes up to total of 3.0 mg/kg.

Reference #s 2020, 7010, 7020, 14040

Lidocaine 2% (Intravenous Solution) - Pediatric and Adult (ALS)

Pain associated with IO infusion:

Lidocaine , 0.5 mg/kg slow IO push over two (2) minutes, not to exceed 40 mg total.

Reference #s 2020, 7010, 7020, 10140, 10190

Magnesium Sulfate (ALS)

Polymorphic Ventricular Tachycardia:

Magnesium Sulfate, 2 gm in 100 ml of NS IV/IO over five (5) minutes for polymorphic VT if prolonged QT is observed during sinus rhythm post-cardioversion.

Eclampsia (Seizure/Tonic/Clonic Activity):

Magnesium Sulfate, 4 gm diluted with 20 ml NS, IV/IO slow IV push over three (3) to four (4) minutes.

Magnesium Sulfate, 2 gm in 100 cc of NS at 30 cc per hour IV/IO to prevent continued seizures.

Reference #s 2020, 7010, 7020, 8010, 14080

Midazolam - Adult (ALS)

Seizure:

Midazolam, 2.5 mg IN/IV/IO. May repeat in five (5) minutes for continued seizure activity, **or**

Midazolam, 5 mg IM. May repeat in ten (10) minutes for continued seizure activity.

Assess patient for medication related reduced respiratory rate or hypotension.

Maximum of three (3) doses using any combination of IM/IN/IV/IO may be administered for continued seizure activity. Contact base hospital for additional orders and to discuss further treatment options.

Pacing, synchronized cardioversion:

Midazolam, 2 mg slow IV/IO push or IN

Reference #s 6090, 6110, 7010, 7020, 10110, 10120, 10190, 11080, 13020, 14080

Midazolam - Pediatric (ALS)*Seizures:*

Midazolam, 0.1 mg/kg IV/IO with maximum dose 2.5 mg. May repeat Midazolam in five (5) minutes, **or**

Midazolam, 0.2 mg/kg IM/IN with maximum dose of 5 mg. May repeat Midazolam in ten (10) minutes for continued seizure. IN dosage of Midazolam is doubled due to decreased surface area of nasal mucosa resulting in decreased absorption of medication.

Assess patient for medication related reduced respiratory rate or hypotension.

Maximum of three (3) doses using any combination of IM/IN/IV/IO may be administered for continued seizure activity. Contact base hospital for additional orders and to discuss further treatment options.

Reference #s 7010, 7020, 14060

Naloxone (Narcan) - Adult (LALS, ALS)*Resolution of respiratory depression related to suspected narcotic overdose:*

Naloxone, 0.5 mg IV/IO/IM/IN, may repeat Naloxone 0.5 mg IV/IO/IM/IN every two (2) to three (3) minutes if needed.

Do not exceed 10 mg of Naloxone total regardless of route administered.

Reference #s 6110, 7010, 7020, 11070, 11080

Naloxone (Narcan) - Pediatric (LALS, ALS)*Resolution of respiratory depression related to suspected narcotic overdose:*

1 day to 8 years	Naloxone, 0.1 mg/kg IV/IO
9 to 14 years	Naloxone, 0.5 mg IV/IO

May repeat every two (2) to three (3) minutes if needed. Do not exceed the adult dosage of 10 mg IV/IO/IM/IN.

Reference #s 7010, 7020, 14040, 14050

Nitroglycerin (LALS, ALS)

Nitroglycerin, 0.4 mg sublingual/transmucosal

One (1) every three (3) minutes as needed. May be repeated as long as patient continues to have signs of adequate tissue perfusion. **If a Right Ventricular Infarction is suspected, the use of nitrates requires base hospital contact.**

Nitroglycerin is contraindicated if there are signs of inadequate tissue perfusion or if sexual enhancement medications have been utilized within the past forty-eight (48) hours.

Reference #s 6090, 6110, 7010, 7020, 11010, 11060

Ondansetron (Zofran) - Patients four (4) years old to Adult (ALS)

Nausea/Vomiting:

Ondansetron, 4 mg slow IV/IO/ODT

All patients four (4) to eight (8) years old: May administer a total of 4 mgs of Ondansetron prior to base hospital contact.

All patients nine (9) and older: May administer Ondansetron 4 mg and may repeat twice, at ten (10) minute intervals, for a total of 12 mgs prior to base hospital contact.

May be used as prophylactic treatment of nausea and vomiting associated with narcotic administration.

Reference #s 6110, 7010, 7020, 9120, 10100, 15010, 15020

Oxygen (non-intubated patient per appropriate delivery device)

Stable:

Mild Hypoxia (SpO₂ 90 - 93%)

- nasal canula at 2 - 4 liters per minute, or
- mask at 8 - 10 liters per minute

Titrate to maintain SpO₂ between 94 - 98%.

Unstable (pending or actual respiratory or cardiopulmonary arrest):

Moderate (SpO₂ 75 - 89%) or Severe Hypoxemia (SpO₂ < 75%) mmHg

- non-rebreather mask at 12 - 15 liters per minute
- BVM with Reservoir - 15 liters per minute

Titrate Oxygen at lowest rate required to maintain SpO₂ at 94%.

Phenylephrine HCL (ALS)

Phenylephrine, 0.5 mg metered dose may be repeated once prior to additional attempt

Reference #s 7010, 7020, 10050, 10190

Procainamide (ALS)

SVT, V-Tach or Wide Complex Tachycardias:

Procainamide, 20 mg/min IV/IO; may repeat until arrhythmia suppressed, symptomatic hypotension, QRS widens by more than 50% or maximum dose of 17 mg/kg administered. If arrhythmia suppressed, begin infusion of 2 mg/min.

Reference #s 7010, 7020, 8010, 8040, 11050

Sodium Bicarbonate (ALS) (base hospital order only)

Tricyclic Poisoning:

Sodium Bicarbonate, 1 mEq/kg IV/IO

Reference #s 2020, 7010, 7020, 13010

Verapamil (ALS)

SVT if adenosine is ineffective:

Verapamil, 5 mg slow IV/IO over three (3) minutes, may repeat every fifteen (15) minutes to a total dose of 20 mg.

Reference #s 7010, 7020, 11050



CARE OF MINORS IN THE FIELD

I. PURPOSE

To provide guidelines for EMS personnel for treatment and/or transport of minors in the field.

AUTHORITY

~~California Welfare and Institutions Code Section 625, Civil Code, sections 25, 34, and 62~~

II. DEFINITIONS

Consent: Except for circumstances specifically prescribed by law, a minor is not legally competent to consent to, or refuse medical care.

Voluntary consent: Treatment and/or transport of a minor shall be with the verbal or written consent of the parent or legal representative.

Involuntary consent: In the absence of a parent or legal representative, emergency treatment and/or transport may be initiated without consent.

Minor: Any person under eighteen (18) years of age.

Minor not requiring parental consent: A person who is decreed by the court as an emancipated minor, has a medical emergency and parent is not available, is married or previously married, is on active duty in the military, is pregnant and requires care related to the pregnancy, is twelve (12) years or older and in need of care for rape and/or sexual assault, is twelve (12) years or older and in need of care for a contagious reportable disease or condition, or for substance abuse.

Legal Representative: A person who is granted custody or conservatorship of another person.

Emergency: An unforeseen condition or situation in which the individual has need for immediate medical attention, or where the potential for immediate medical attention is perceived by EMS personnel or a public safety agency

III. PROCEDURE

Treatment and/or Transport of Minors

- In the absence of a parent or legal representative, minors with an emergency condition shall be treated and transported to the medical facility most appropriate to the needs of the patient.
 - In the absence of a parent or legal representative, minors with a non-emergency condition require EMS field personnel to make reasonable effort to contact a parent or legal representative before initiating treatment and/or transport. If a parent or legal representative cannot be reached and minor is transported, EMS field personnel shall make every effort to inform the parent or legal representative of where the minor has been transported, and request that law enforcement accompany the minor patient to the hospital.
- ~~1. For all ill or injured minors under the age of nine (9) years, Base Station contact is required before leaving scene.~~
 - ~~2. In the absence of a parent or legal representative, minors with an emergency condition shall be treated and transported to the medical facility most appropriate to the needs of the patient.~~
 - ~~3. In the absence of a parent or legal representative, minors with a non-emergency condition require EMS personnel to make reasonable effort to contact a parent or legal representative before initiating treatment and/or transport. If a parent or legal representative cannot be reached and minor is transported, EMS personnel shall make every effort to inform the parent or legal representative of where the minor has been transported, and request that law enforcement accompany the minor patient to the hospital.~~

Minors Not Requiring Immediate Treatment and/or Transport

- A minor evaluated by EMS field personnel and determined not to be injured, to have sustained only minor injuries, or to have an illness or injury not requiring immediate treatment and/or transportation, may be released to:
 - Parent or legal representative.
 - Designated care giver over eighteen (18) years of age.
 - Law enforcement.
 - EMS field personnel shall document on the patient care record to whom the minor was released.

Minor Attempting to Refuse Indicated Care

~~1. Contact Base Station.~~

- Attempt to contact parent or legal representative for permission to treat and/or transport.
- ~~Contact If parent or legal representative cannot be contacted, contact law enforcement and request minor to be taken into temporary custody for treatment and/or transport (only necessary in the event parents or legal representative cannot be contacted).~~

Base Hospital Contact

- ~~Base hospital contact is required, prior to EMS field personnel leaving the scene, for the following situations:~~
 - ~~Minors under the age of nine (9) who are not being transported to the hospital.~~
 - ~~Minors under the age of nine (9) whose parents or guardians are refusing care.~~
 - ~~Minors who in the opinion of EMS field personnel, do not require treatment or transport.~~
- ~~See ICEMA Reference #5040 - Radio Communication Policy.~~

IV. REFERENCE

<u>Number</u>	<u>Name</u>
5040	Radio Communication Policy



ICEMA APPROVED SKILLS PROCEDURE - STANDARD ORDERS

~~I. POLICY~~

~~To provide a list of ICEMA approved skills and affected scope of practice.~~

~~II. AUTHORITY~~

~~California Health and Safety Code, Sections 1797.214~~

~~California Code of Regulations, Title 22, Division 9, Chapters 2, 3, and 4~~

~~III. SKILLS~~

12-lead Electrocardiography (EMT-P)

- ECG should be performed prior to medication administration.
- ECG should be performed on any patient whose medical history and/or presenting symptoms are consistent with an acute coronary syndrome.

Axial Spinal Stabilization (EMT, AEMT and EMT-P)

- Should be placed if patient meets the indicators, per ICEMA Reference #15010 - Trauma - Adult (Neuro Deficits present, Spinal Tenderness present, Altered Mental status, Intoxication, or Distracting Injury).
- An AEMT and/or EMT-P may remove if placed by BLS crew and it does not meet indicators.

Continuous Positive Airway Pressure Device (CPAP) - Adult (EMT, AEMT and EMT-P)

- Start at lowest setting and increase slowly until patient experiences relief or until a maximum of 15 cm H₂O is reached.

External Jugular Vein Access (AEMT and EMT-P)

- Not indicated for patients eight (8) years of age and younger.
- Patient condition requires IV access and other peripheral venous access attempts are unsuccessful.

Intraosseous Infusion (AEMT pediatric patients only and EMT-P)

- EMT-Ps may administer Lidocaine slowly per ICEMA Reference #7040 - Medication - Standard Orders, ~~for to control infusion~~ pain control.
- Approved insertion sites:
 - Eight (8) years of age or younger (LALS and ALS):
 - Proximal Tibia - Anterior medial surface of tibia, 2 cm below tibial tuberosity.
 - Nine (9) years of age and older (ALS only):
 - Proximal Tibia - Anterior medial surface of tibia, 2 cm below tibial tuberosity.
 - Distal Tibia - Lower end of tibia, 2 cm above the medial malleolus.
 - Humeral Head (EZ IO only).
 - Anterior distal femur, 2 cm above the patella - Base Station contact only.
- Leave site visible and monitor for extravasation.

King Airway Device (Perilaryngeal) - Adult (EMT Specialty Program, AEMT, and EMT-P)

- Use of King Airway adjunct may be performed only on those patients who meet **all** of the following criteria:
 - Unresponsive, agonal respirations (less than six (6) breaths per minute) or apneic.
 - Patients 15 years or older.
 - Anyone over four (4) feet in height.
- Additional Considerations:
 - Medications may NOT be given via the King Airway.
 - King Airway adjunct should not be removed unless it becomes ineffective.

King Airway Device (Perilaryngeal) - Pediatric (less than 15 years of age) (EMT Specialty Program, AEMT, and EMT-P)

- Use of King Airway adjunct may be performed only on those patients who meet **all** of the following criteria:

- Unresponsive, agonal respirations (less than six (6) breaths per minute) or apneic.
 - No gag reflex.
 - Pediatric patients meeting the following criteria:
 - 35-45 inches or 12-25 kg: size 2
 - 41-51 inches or 25-35 kg: size 2.5
- ~~Patients less than 15 years of age.~~
- Additional Considerations:
 - Medications may NOT be given via the King Airway.
 - King Airway adjunct should not be removed unless it becomes ineffective.
~~— May initially be contraindicated with suspected ALOC.~~

Nasogastric/Orogastric Tube (EMT-P)

- Use viscous Lidocaine gel per ICEMA Reference #7040 - Medication - Standard Orders, for conscious patients.
- Required for all full arrest patients.

Nasotracheal Intubation (EMT-P)

- Absolute contraindication: Apnea.
- Base hospital contact required: Facial trauma, anticoagulant therapy, airway burns, failed CPAP.
- Prophylactic-Immediately prior to intubation, consider prophylactic Lidocaine per ICEMA Reference #7040 - Medication - Standard Orders, for suspected head/brain injury.
- Administer Phenylephrine per ICEMA Reference #7040 - Medication - Standard Orders.
- Monitor end-tidal CO₂ and wave form capnography.
- Monitor pulse oximetry.
- Contact base hospital if unable to place ET after a maximum of three (3) nasotracheal intubation attempts or in unable to adequately ventilate patient via BVM.

Needle Cricothyrotomy (EMT-P)

- Absolute contraindication: Transection of the distal trachea.
- Monitor end-tidal CO₂ and wave form capnography.
- Monitor pulse oximetry.
- Contact base hospital if unable to ventilate adequately and transport immediately to the closest hospital for airway management.

Needle Thoracostomy (EMT-P)

- In blunt chest trauma consider bilateral tension pneumothorax if pulse oximetry (SpO₂) reading remains low with a patent airway or with poor respiratory compliance.

Oral Endotracheal Intubation - Adult (EMT-P)

- ~~Consider~~ Immediately prior to intubation, consider Lidocaine prophylactically per ICEMA Reference #7040 - Medication - Standard Orders, for head injury.
- Monitor end-tidal CO₂ with capnography and wave form capnography.
- Monitor pulse oximetry.
- After ~~If~~ unable to place ET after a maximum of three (3) unsuccessful intubation attempts (an attempt is considered made when tube passes the gum line) and if all procedures to establish an adequate airway fail, consider Needle Cricothyrotomy.
- Document verification of tube placement (auscultation, visualization, capnography)

Oral Endotracheal Intubation - Pediatric (less than 15 years of age) (EMT-P)

- Uncuffed tubes for patients under eight (8) years old.
- Base hospital contact is required after two (2) failed intubation attempts (an attempt is considered made when tube passes the gum line).
- If all procedures to establish an adequate airway fail, consider Needle Cricothyrotomy.
- Monitor end-tidal CO₂ and wave form ~~with~~ capnography.

- Monitor pulse oximetry.
- Document verification of tube placement. Run a continuous strip of capnography readings during movement of patient to verify tube placement.

Synchronized Cardioversion (EMT-P)

- Consider Midazolam per ICEMA Reference #7040 - Medication - Standard Orders, for anxiety.
- Consider Fentanyl per ICEMA Reference #7040 - Medication - Standard Orders, for pain.
- If rhythm deteriorates to v-fib, turn off the sync button and defibrillate.
- Select initial energy level setting at 100 joules or a clinically equivalent biphasic energy level per manufacture guidelines. Procedure may be repeated at 200, 300 and 360 joules or a clinically equivalent biphasic energy level per manufacture guidelines.
- In Radio Communication Failure or with base hospital order, repeated cardioversion attempts at 360 joules or clinically equivalent biphasic energy level per manufacturer's guidelines may be attempted.

Transcutaneous Cardiac Pacing (EMT-P)

- Start at a rate of sixty (60) and adjust output to the lowest setting to maintain capture. Assess peripheral pulses and confirm correlation with paced rhythm.
- Reassess peripheral pulses. Adjust output to compensate for loss of capture.
- Increase rate (**not to exceed 100**) to maintain adequate tissue perfusion.
- Consider Midazolam per ICEMA Reference #7040 - Medication - Standard Orders, for anxiety
- Consider Fentanyl per ICEMA Reference #7040 - Medication - Standard Orders, for pain.
- Consider medication for pain and anxiety.
- Contact the base hospital if rhythm persists or for continued signs of inadequate tissue perfusion.

Vagal Maneuvers (EMT-P)

- ~~Use with caution for~~ Relative contraindications for patients with hypertension, suspected STEMI, or suspected head/brain injury.
- Reassess cardiac and hemodynamic status. Document rhythm before, during and after procedure.
- If rhythm does not covert within ten (10) seconds, follow ICEMA Reference #11050 -Tachycardias - Adult.

~~IV. REFERENCE~~

Number Name

7040 Medication - Standard Orders

15010 Trauma - Adult (15 years of age or older)