RESUSCITATION UPDATES: MAXIMIZINING SURVIVAL OF CARDIAC ARREST

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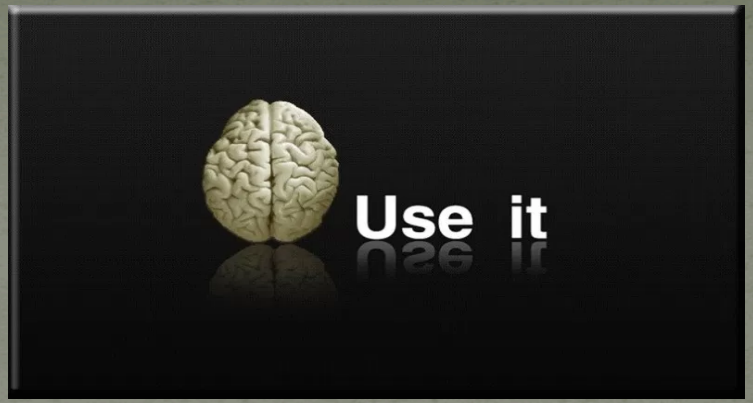
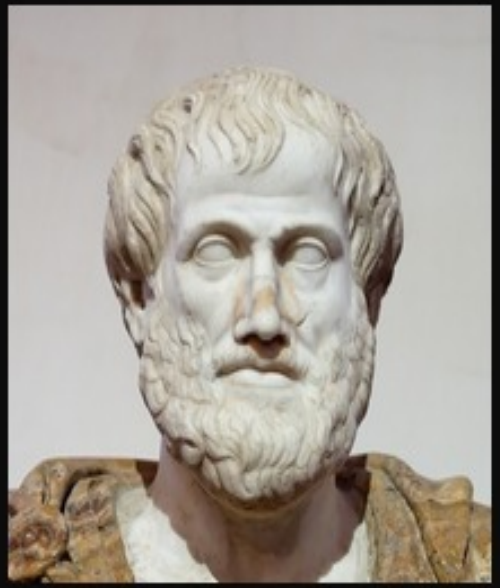
General

- Disclosures
  - NONE




FIRE
DEPARTMENT
Thank you
for your service!


BREAKING NEWS
SCIENTISTS HAVE DISCOVERED THAT AMBULANCES AND TAXIS ARE IN FACT, NOT THE SAME THING



The more you know, the more you know you don't know.

~ Aristotle

AHA

- "Stated succinctly, if ACLS care in the field cannot resuscitate the victim, ED care will not resuscitate the victim."
  - 2005 AHA Guidelines for CPR and ECC
    - Part 7.2: Management of Cardiac Arrest

Fast Facts

- After ROSC what percentage of patients decompensate and refibrillate?
  - 30% all cases
  - 79% OHCA will experience at least one recurrence

- What three actions/medications need to be done on all ROSC patients?
  - Initiate cooling measures
  - $NaHCO_3$
  - An antiarrhythmic
    - Magnesium
    - Amiodarone
    - Lidocaine
      - I use magnesium and lido or amino
      - Start with one and if no response use the other
      - ****THERE IS NO DIFFERENCE BETWEEN AMIODARONE AND LIDOCAINE TO DISHCARGE HOME****

Fast Facts

- What is normal cardiac output?
  - 50 – 75 %
- What is the cardiac output during high quality CPR?
  - 25 – 30 %
- How much ATP is needed to restart the heart?
  - 20 – 25 %
- How fast does the ATP drop to almost zero?
  - 3 – 4 seconds

Fast Facts

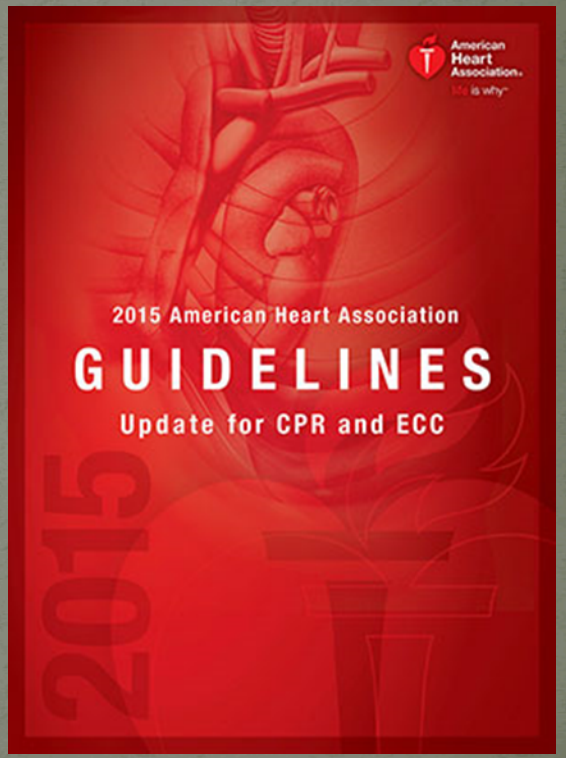
- What does acidosis do to epinephrine?
  - Makes it ineffective
- After ROSC what does the lactic acidosis do?
  - Precipitate arrhythmias
- What is the initial energy setting for defibrillation during cardiac arrest?
  - Maximal allowed by the monitor

Review

- Heart attack occurs how often in the US?
  - Every 43 seconds
    - Every year:
      - **845,000 Americans** have a heart attack.

- Death due to cardiac disease occurs how often in the US?
  - Every 90 seconds

- What percentage of heart attacks are silent?
  - 20%
    - The damage is done, but the person is not aware of it
      - Atypical symptoms, weakness, diabetes, not feeling well



Review

- 350,000 – 400,000 Cardiopulmonary arrests yearly
  - 10% of all total mortality
- Despite all advances in care only a 6.4% survival
  - Range from 3.3% to 40.5%
  - "chain of survival" variability
- Heart attack occurs how often in the US?
  - Every 43 seconds
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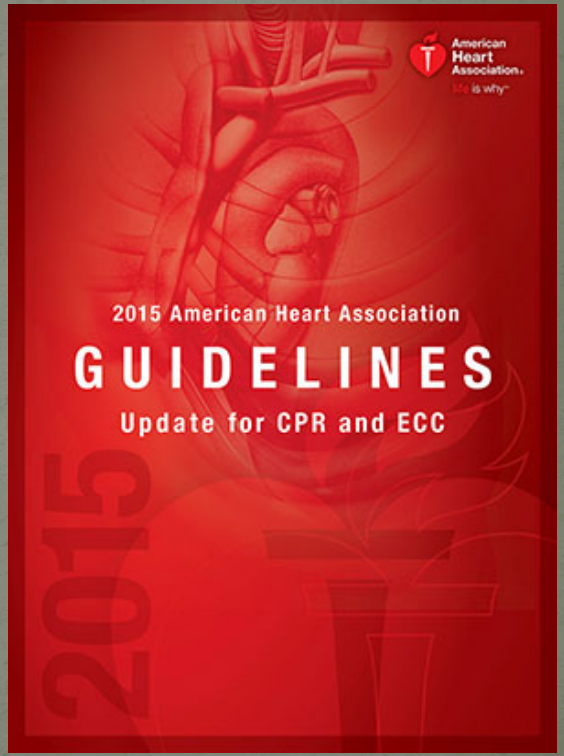
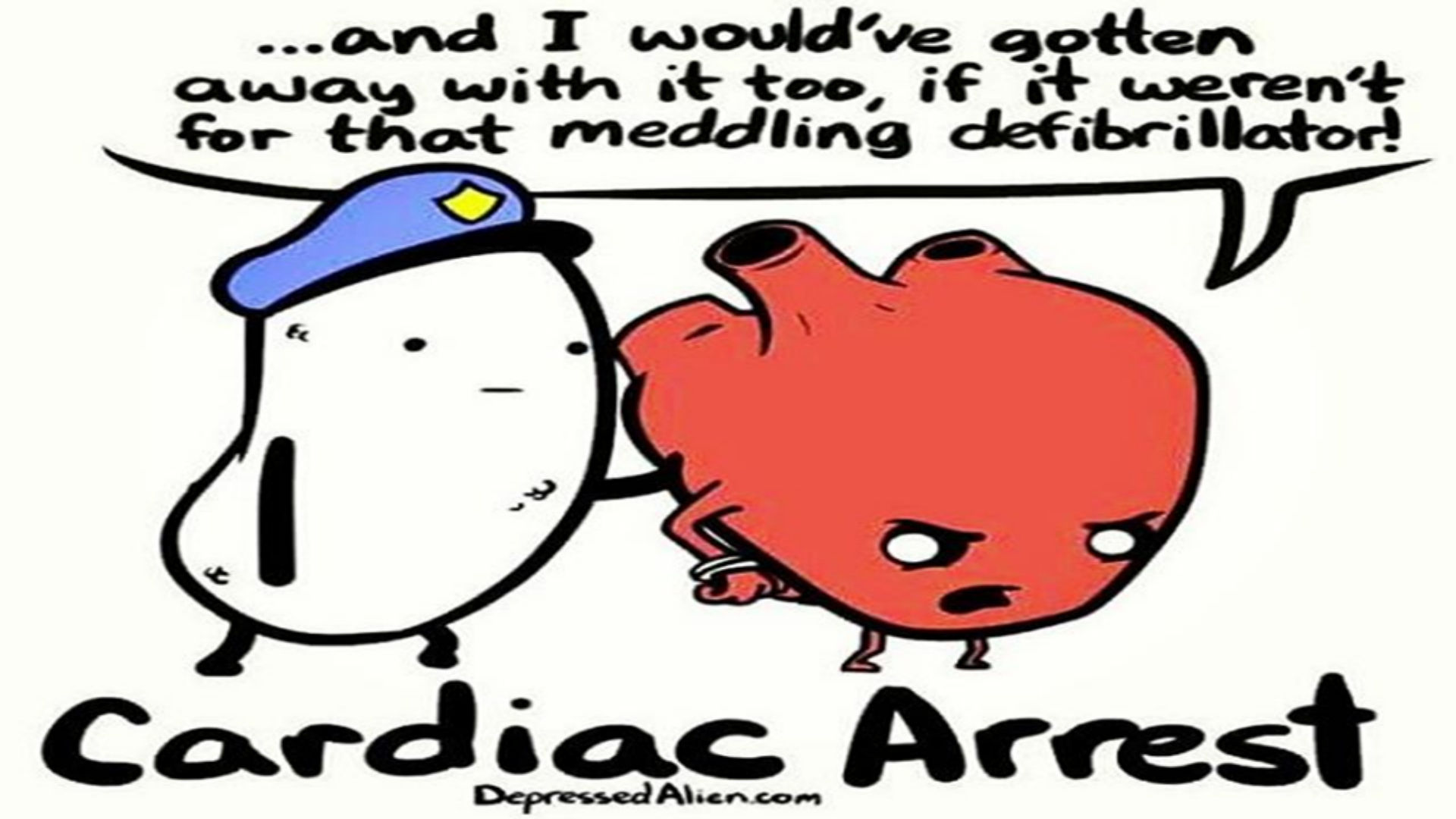



...and I would've gotten away with it too, if it weren't for that meddling defibrillator!
Cardiac Arrest
DepressedAlien.com

Cardiac Arrest Sequence

- Insult Occurs
  - Trauma, sepsis, overdose, hypoxia, etc.
- Diminished Poor Perfusion
  - Hemorrhage, dehydration, compensated shock leading to decompensated shock
- Anaerobic Metabolism
  - Lactic Acidosis
- Increasing Acidosis
  - Cardiac arrhythmias
- Death

Cardiac Arrest Sequence

- Must reverse order
- ABCs
  - Continuous CPR
  - Airway
  - IV
- Reverse Acidosis
  - $NaHCO_3$
  - This is first and foremost
    - If you don't then hard to resuscitate
  - Once this is accomplished look at the etiology of the insult, address it, treat it, and continue resuscitation


Problem
Analysis
Solution

Contributing Causes of Cardiac Arrest

The 7 Hs

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypokalemia
- Hyperkalemia
- Hypothermia
- Hypoglycemia

The 6 Ts

- Toxins
- Tamponade, cardiac
- Tension, pneumothorax
- Thrombosis (Coronary)
- Thrombosis (Pulmonary)
- Trauma

Contributing Causes of Cardiac Arrest

- What do all the contributing causes of cardiac arrest (the 7 Hs and 6 Ts) all have in common?
  - Acidosis
- What does acidosis do to epinephrine that is injected intravenously during cardiac arrest?
  - Makes it ineffective
- Unless the patient collapses immediately in front of you the patient will be acidotic upon your arrival?
  - True

Contributing Causes of Cardiac Arrest

- What medication should be given first to ALL cardiac arrests?
  - $NaHCO_3$
- What medication should be given when there is ROSC?
  - $NaHCO_3$
  - Any anti arrhythmic and/or combination thereof
    - Magnesium, lidocaine, amiodarone

Contributing Causes of Cardiac Arrest

- What is the most important resuscitation medication to be administered first (according to my medical opinion)?
  - $NaHCO_3$

AHA

- "Excessive" $NaHCO_3$ "may" cause arrhythmias

Acidosis Proof

- Hypovolemia
  - Poor perfusion
    - Anaerobic metabolism
      - Lactic acidosis
        - Increased hydrogen ion (acidosis)
  - Multiple etiologies
    - Vomiting
    - Diarrhea
    - Hemorrhage
    - Dehydration

Acidosis Proof

- Hypoxia
  - Poor oxygenation
    - Anaerobic metabolism
      - Lactic acidosis
        - Increased hydrogen ion (acidosis)
  - Multiple etiologies
    - Apnea
    - COPD
    - HF
    - Drug Overdose
    - Pulmonary Embolus

Acidosis Proof

- Hydrogen ion (acidosis)
  - Everything causes acidosis prior to cardiac arrest
    - Multiple etiologies
      - Result of decreased/absent
        - Oxygenation
        - Perfusion
        - Glucose
          - Hyperglycemia
          - Hypoglycemia
    - Increased intravascular hydrogen ion (acidosis)

Acidosis Proof

- Hypokalemia
  - Multiple etiologies
    - Carbonic anhydrase inhibitors
      - Acetazloamide
    - Dialysis
      - Post treatment
    - Renal tubular acidosis
    - Treated DKA with insulin therapy
    - VIPoma
      - A **VIPoma** (also known as **Verner–Morrison syndrome**, after the physicians who first described it) is a rare (1 per 10,000,000 per year) endocrine tumor, usually (about 90%) originating from non-β islet cell of the pancreas, that produce vasoactive intestinal peptide(VIP). It may be associated with multiple endocrine neoplasia type 1.
      - The massive amounts of VIP in turn cause profound and chronic watery diarrhea and resultant dehydration, hypokalemia, achlorhydria (hence *WDHA-syndrome*, or *pancreatic cholera syndrome*), acidosis, vasodilation (flushing and hypotension), hypercalcemia, and hyperglycemia .
  - Increased intravascular hydrogen ion (acidosis)

Acidosis Proof

- Hyperkalemia
  - Potassium shifts intracellularly to maintain electrical neutrality Hydrogen ion shifts extracellularly into the blood stream
    - Increased intravascular hydrogen ion (acidosis)
  - Etiologies
    - Tumor lysis syndrome
    - Rhabdomyolysis
    - Medications

Acidosis Proof

- Hypothermia
  - Part of Trauma Triad of Death
    - Hypothermia, coagulopathy, acidosis
  - Poor perfusion
    - Anaerobic metabolism
      - Lactic acidosis
        - Increased intravascular hydrogen ion (acidosis)

Acidosis Proof

- Hypoglycemia
  - Low glucose
    - No energy substrate
    - Anaerobic metabolism
      - Lactic acidosis
        - Increased hydrogen ion (acidosis)
  - Multiple etiologies
    - Starvation
    - Alcoholism
    - Medications
      - Insulin and oral hypoglycemics
    - Drug Overdose

Acidosis Proof

- Toxins
  - ~ ½ cause anaerobic metabolism and lactic acidosis
  - ~ ½ cause Na+ channel blockage causing QT prolongation
    - Either way $NaHCO_3$ is drug of choice

Acidosis Proof

- Cardiac Tamponade
  - Increased heart rate
  - Decreased blood pressure
  - Muffled heart tones
  - Cardiac constriction
    - Inability to fill heart and diminished preload
    - Decreased cardiac output
    - Decreased blood pressure
  - Poor perfusion
    - Lactic acidosis
      - Increased hydrogen ion (acidosis)

Acidosis Proof

- Tension Pneumothorax
  - Increased heart rate
  - Decreased blood pressure
  - Diminished or absent breath sides on one side
  - Increased intrathoracic pressure
    - Compression of superior and inferior vena cava
    - Decreased blood return to heart
    - Decreased cardiac output
    - Decreased blood pressure
  - Poor perfusion
    - Lactic acidosis
      - Increased hydrogen ion (acidosis)

Acidosis Proof

- Thrombosis, Coronary
  - Acute Myocardial Infarction
  - Impaired heart wall motion
  - Impaired cardiac output
  - Decreased blood pressure
  - Poor perfusion
    - Lactic acidosis
      - Increased hydrogen ion (acidosis)

Acidosis Proof

- Thrombosis, Pulmonary
  - Pulmonary Embolus
  - No perfusion to lungs
  - No oxygenation of blood
  - Hypoxia
  - Poor oxygenation
    - Anaerobic metabolism
      - Lactic acidosis
        - Increased hydrogen ion (acidosis)

Acidosis Proof

- Trauma
  - Crushed tissue
  - Hemorrhage
    - Bleeding
  - Tissue Ischemia and Hypoxia
  - Poor oxygenation
    - Anaerobic metabolism
      - Lactic acidosis
        - Increased hydrogen ion (acidosis)

Acidosis Proof

- Acidosis is the condition universally found immediately prior to cardiopulmonary arrest
- Death ensues if not remediated
- No matter what you do acidosis MUST be addressed and treated before you stand a half way decent chance of obtaining ROSC

Literature

- *Mild metabolic acidosis impairs the β-adrenergic response in isolated human failing myocardium.* Hanna SchotolaEmail author, Karl Toischer, Aron F Popov, André Renner, Jan D Schmitto, Jan Gummert, Michael Quintel, Martin Bauer, Lars S Maier and Samuel Sossalla

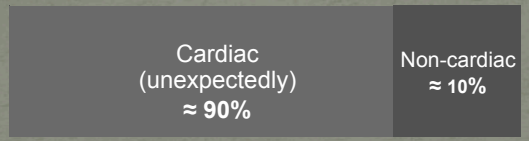
- **Conclusions:**
- Our data show that mild metabolic acidosis reduces cardiac contractility and significantly impairs the β-adrenergic force response in human failing myocardium. Thus, our results could contribute to the still-controversial discussion about the therapy regimen of acidosis in patients with critical heart failure.


Relax buddy, we're just tryin' to save your ass.
CLEAR!!

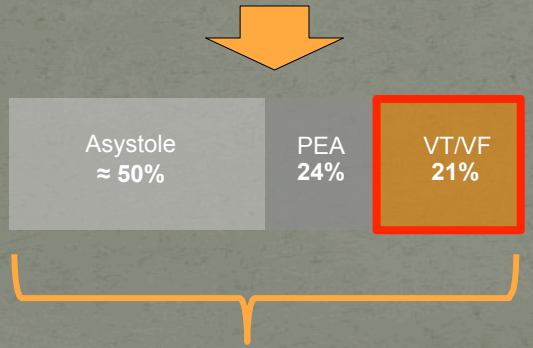
Cardiac Arrest Overview

OHCA

Causes[1,2,3]



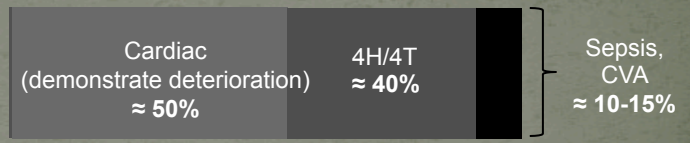
Rhythms[1]

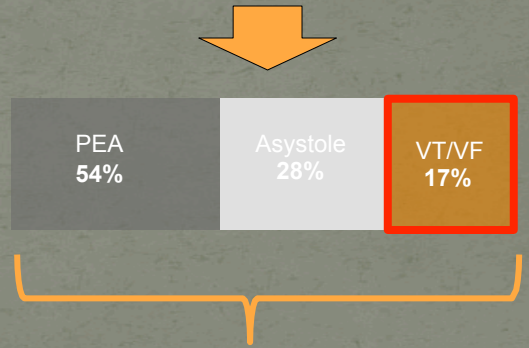


Survival Rates

Avg. survival rate is 10.4% (all rhythms)

IHCA





Avg. survival rate is 18% (all rhythms)

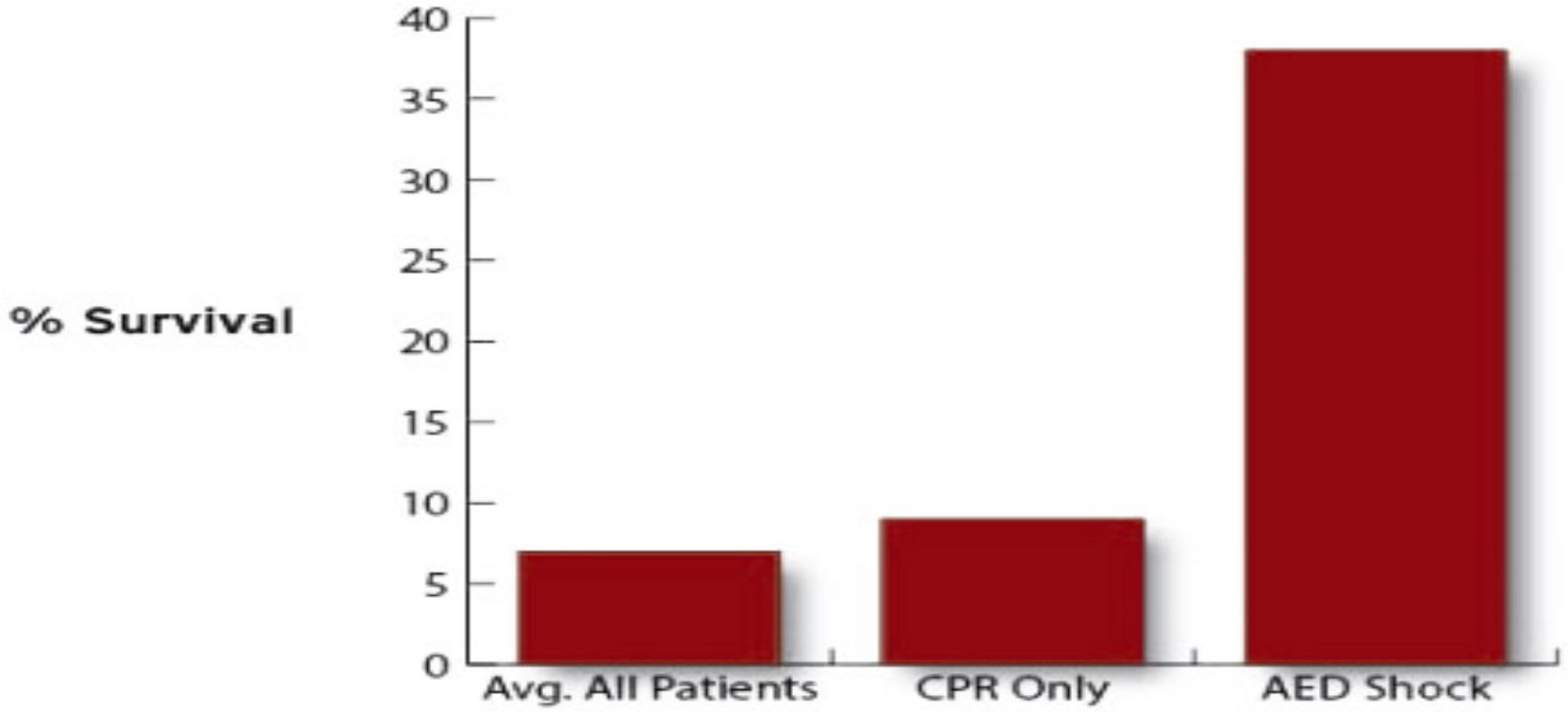
CPR

- Optimize blood flow
- Bystander CPR
  - Increases chance of survival
    - From 1.4% to 4.6%
    - 86% in good neurological outcome
  - Low rates
    - Fear, cause harm, panic, reluctance, diseases, complexity
    - Chest compressions only improved compliance
- Minimally Interrupted Cardiac Resuscitation (MICR)
  - Multiple factors but seems minimal interruptions in chest compressions is responsible for increased outcomes
    - Overall 3.8% vs 9.1%
    - Witnessed arrest 11.9% vs 28.4%

CPR

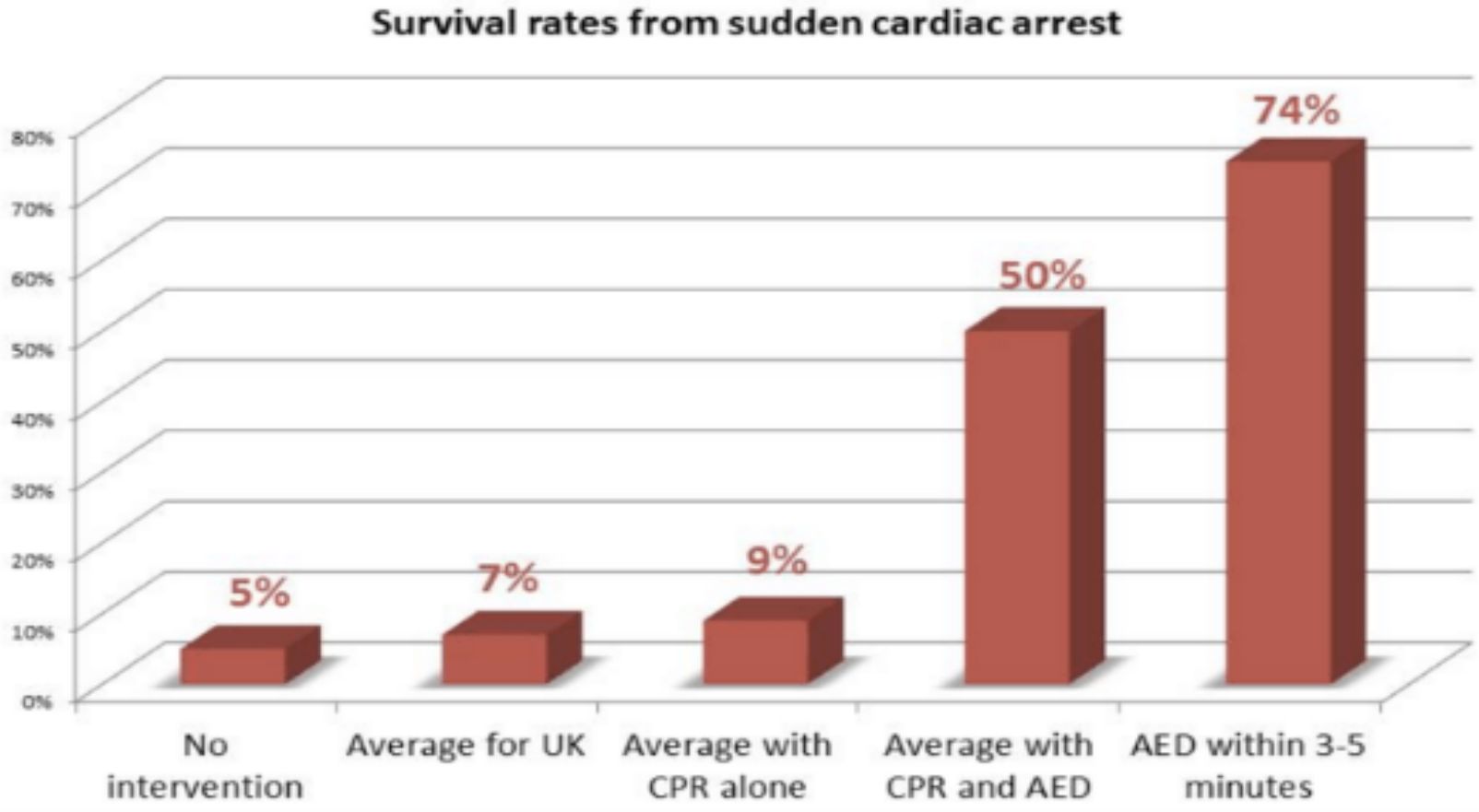
- Rescuer Fatigue
  - Occurs rapidly
    - Within first two minutes
    - Rotate rescuers
    - Mechanical devices
  - Monitor quality during resuscitation
    - Third person objective monitoring
  - Lucas Device
- Recommended that Continuous Cardiac Compression (CCC) fraction be above 90%

Survival Rates of Cardiac Arrest Patients



Source: 2010 Journal of the American College of Cardiology

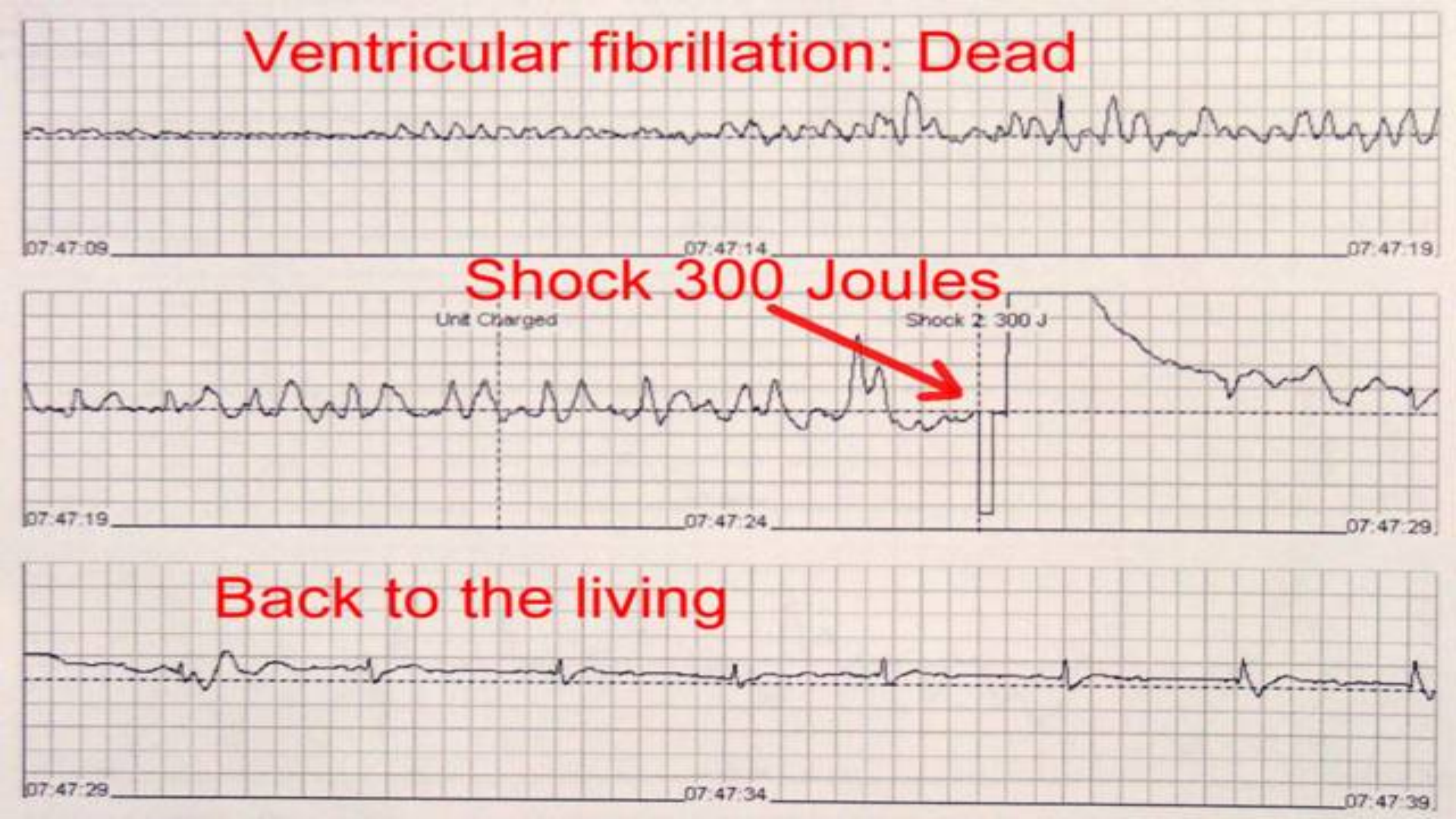
The impact of public-access defibrillators on survival

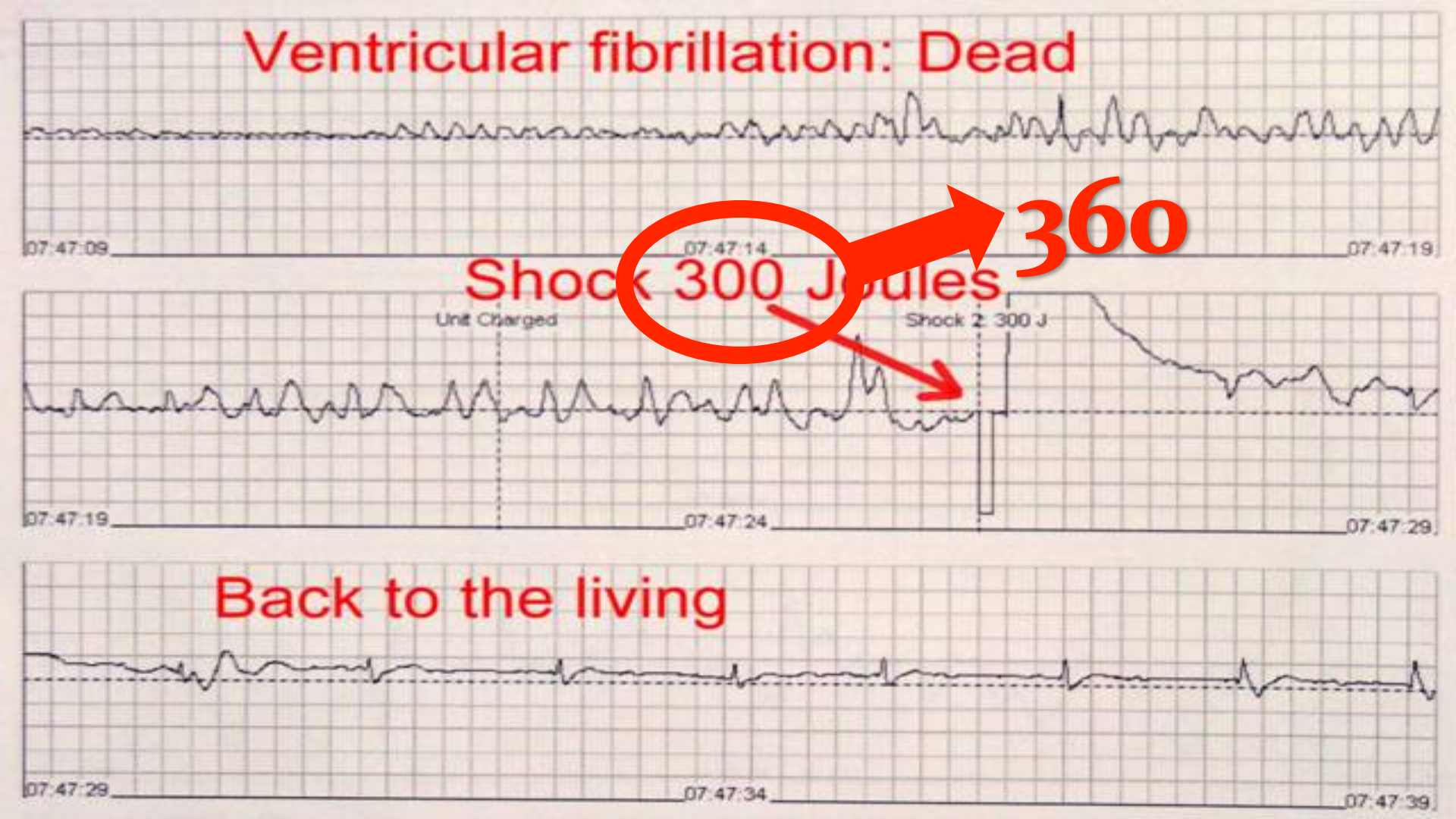


CARDIAC ARREST SURVIVAL

Seattle and King County in recent years have more than doubled their survival rates for bystander-witnessed cardiac arrests caused by ventricular fibrillation.

Source: King County

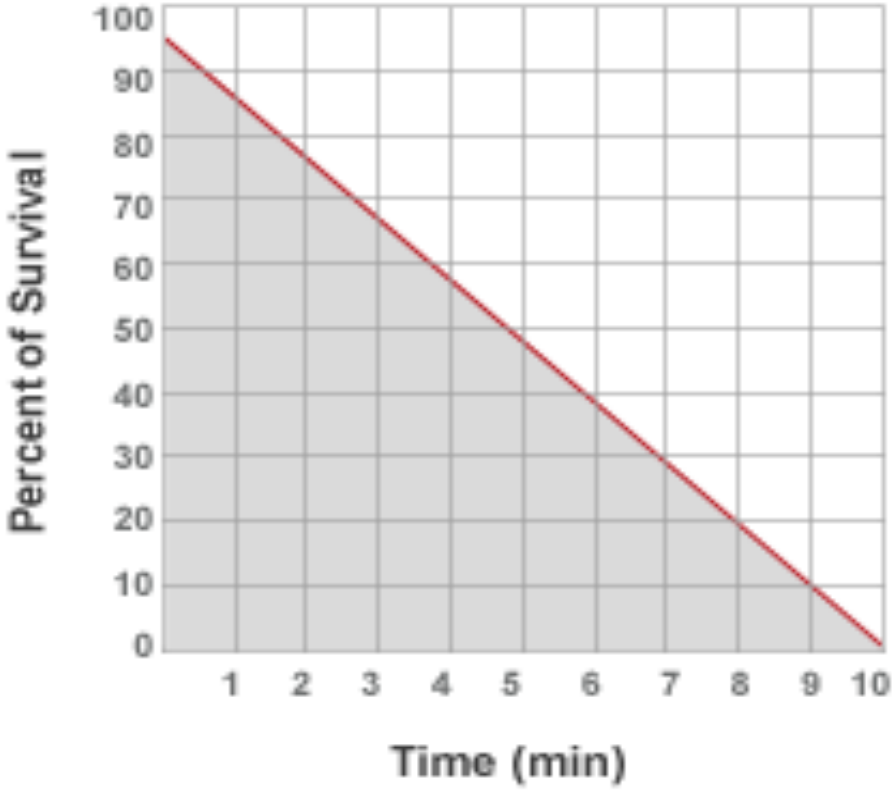

Ventricular fibrillation: Dead
07:47:09
07:47:14
07:47:19
Shock 300 Joules
Unit Charged
Shock 2: 300 J
07:47:19
07:47:24
07:47:29
Back to the living
07:47:29
07:47:34
07:47:39


Ventricular fibrillation: Dead
07:47:09
07:47:14
07:47:19
360
Shock 300 Joules
Unit Charged
Shock 2: 300 J
07:47:19
07:47:24
07:47:29
Back to the living
07:47:29
07:47:34
07:47:39

FACT:

- There is not one study in the medical literature that proves/supports the notion that extra energy causes cardiac damage in the cardiac arrest resuscitation scenario when held to maximum 360 Joules (5 Joules/ kg).
  - Too low an energy does not defibrillate the heart

PERCENT OF SURVIVAL

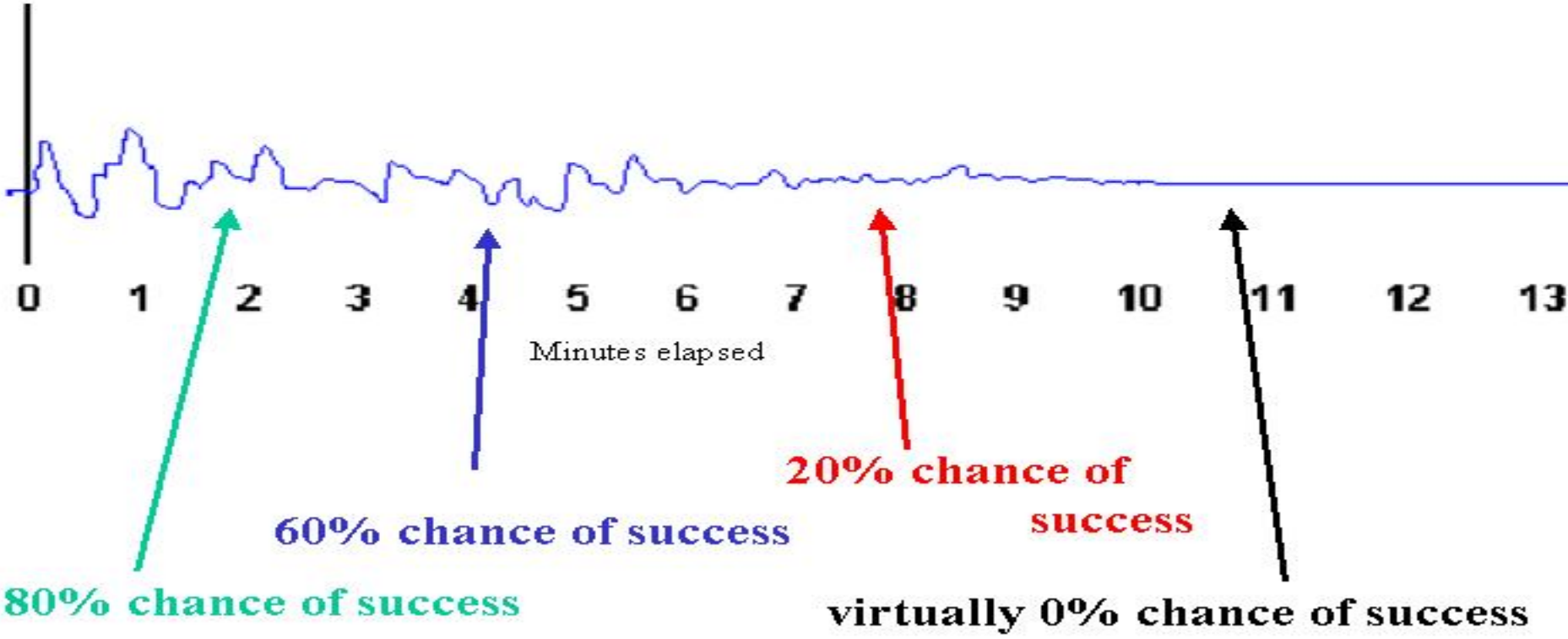


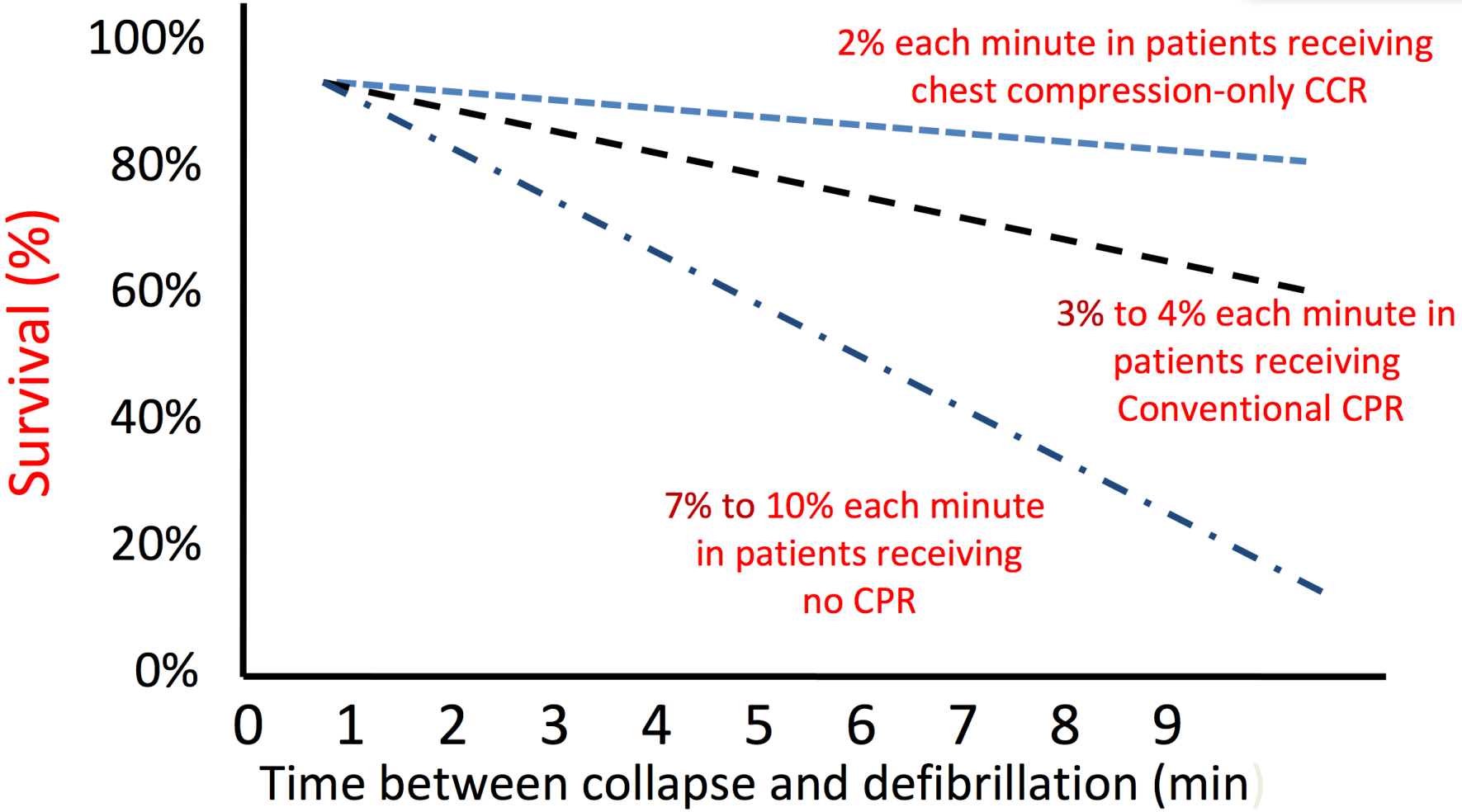
Survival rates after VF cardiac arrest decrease approximately 7% to 10% with every minute that defibrillation is delayed. p 1-61

Guidelines 2000 for Cardiovascular Resuscitation and Emergency Cardiovascular Care. Circulation; 102 (suppl)8. August 22, 2000.

Defibrillation Statistics:

Defibrillation's chances of restoring a pulse decrease rapidly with time



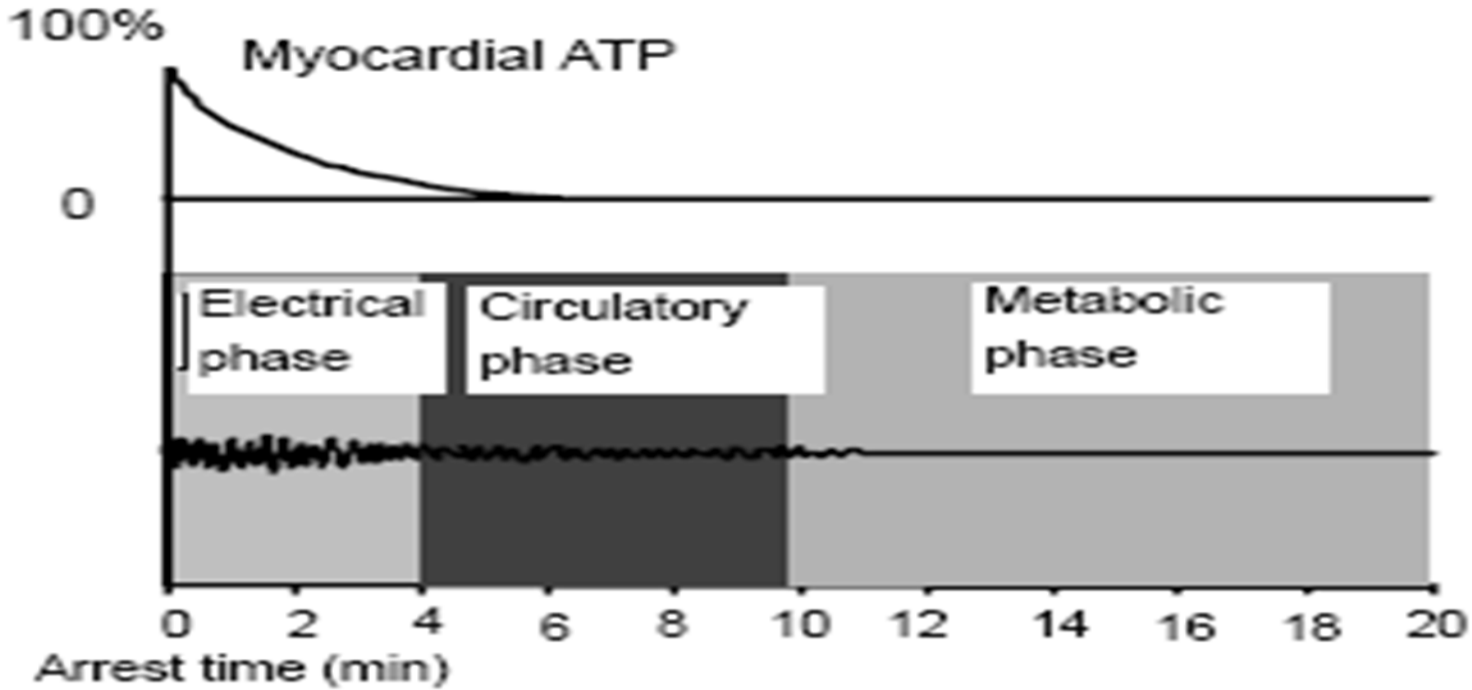


Nagao, K *Current Opinions in Critical Care* 2009

Physiology

- Three Phases
  - Electrical
    - Most important is prompt defibrillation
  - Circulatory
    - Coronary and cerebral perfusion is critical
  - Metabolic
    - Much research needed

Figure 1. Three-Phase Model of VF Cardiac Arrest



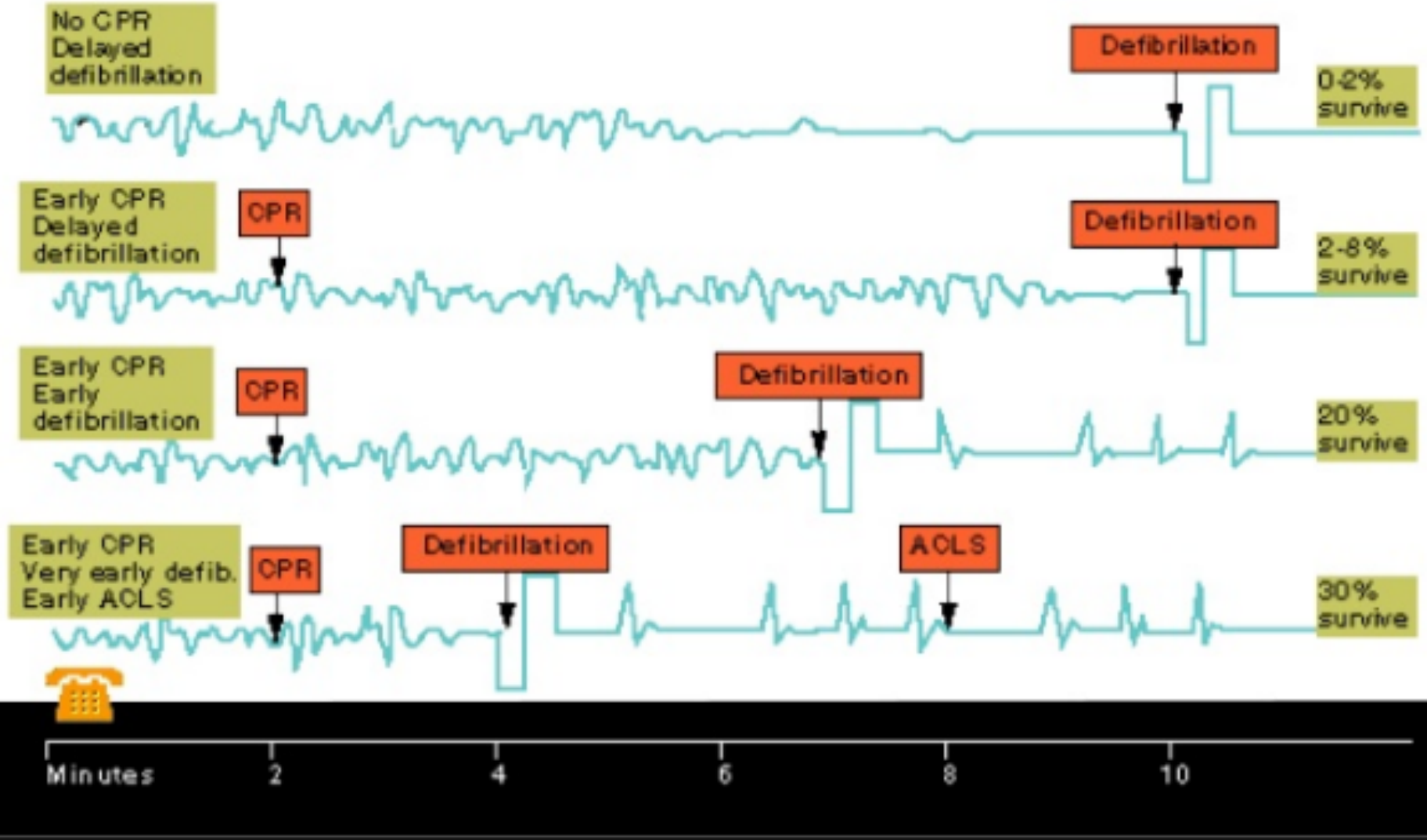

No CPR
Delayed
defibrillation
Defibrillation
0-2%
survive
Early CPR
Delayed
defibrillation
CPR
Defibrillation
2-8%
survive
Early CPR
Early
defibrillation
CPR
Defibrillation
20%
survive
Early CPR
Very early defib.
Early ACLS
CPR
Defibrillation
ACLS
30%
survive
Minutes
2
4
6
8
10

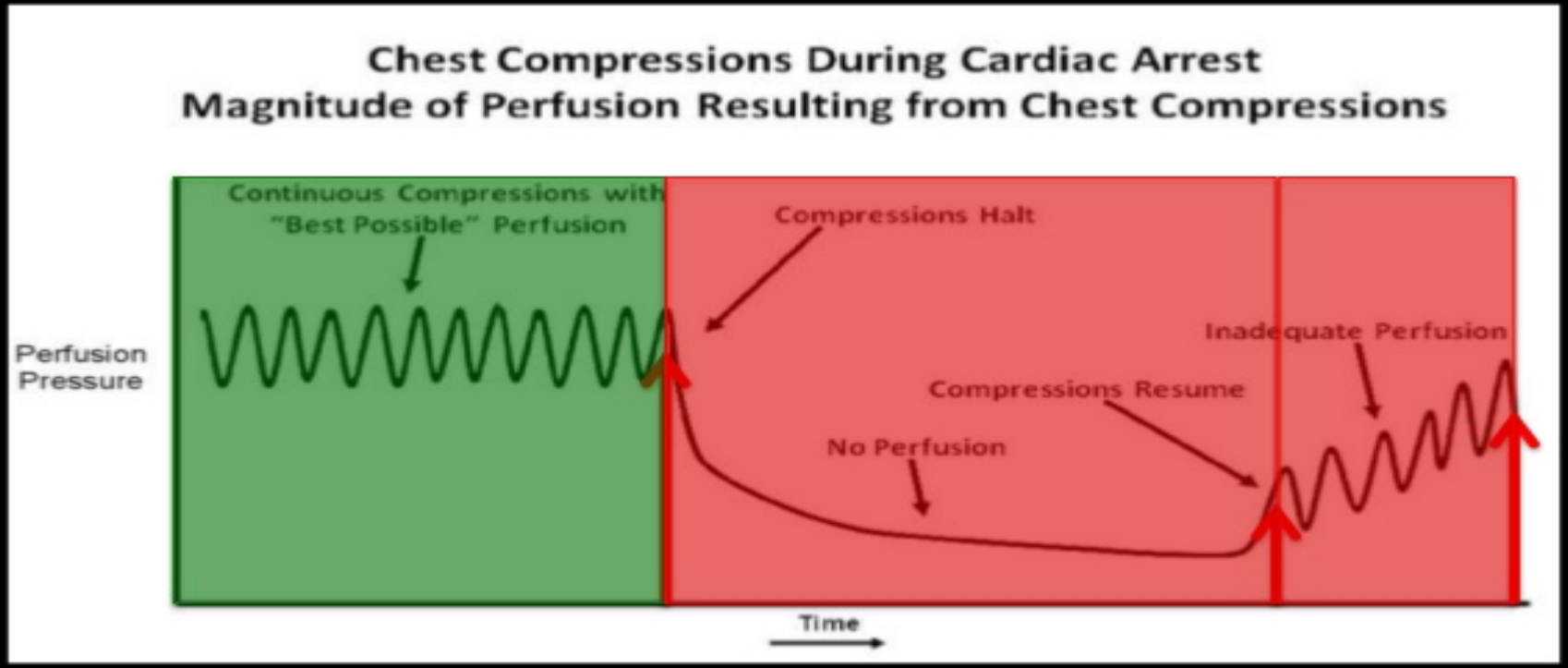
Table 2. Hemodynamically Significant Components of CPR[32]

	OPTIMAL	COMMON ERRORS	EFFECTS OF COMMON ERRORS	KEY POINTS FOR PROVIDERS
Ventilation	• 8-10 breaths/min. • No more than 1 sec/breath • Tidal volume 500-600 ml/breath	• Excessive ventilation rate • Prolonged ventilation duration • Excessive tidal volume	↑ Intrathoracic pressure ↓ Coronary perfusion pressure ↓ Survival	"Don't overventilate"
Chest compression depth	1½ to 2 inches (approx 38-52 mm)	• Compressions too shallow	↓ Coronary perfusion pressure ↓ Cerebral perfusion pressure	"Push hard"
Chest compression rate	100/min.	• Compressions too slow • Compressions too fast	↓ Coronary perfusion pressure ↓ Cerebral perfusion pressure	"Push fast"
Chest recoil	Allow complete chest wall recoil after each compression	• Failure to allow full chest recoil • Leaning on chest • Rescuer fatigue	↑ Intrathoracic pressure ↓ Coronary perfusion pressure ↓ Cerebral perfusion pressure	"Allow complete chest recoil"
"Hands-off time"	• Minimize ALL interruptions to CPR • No pauses > 10 seconds	Prolonged periods of no CPR due to: • Endotracheal intubation • AED voice prompts • Pulse checks • Logistics surrounding defibrillation • Changing rescuers	↓ Success of defibrillation ↓ Return of spontaneous circulation (ROSC) ↓ Survival	"Minimize interruptions to chest compressions"

Other common CPR mistakes:

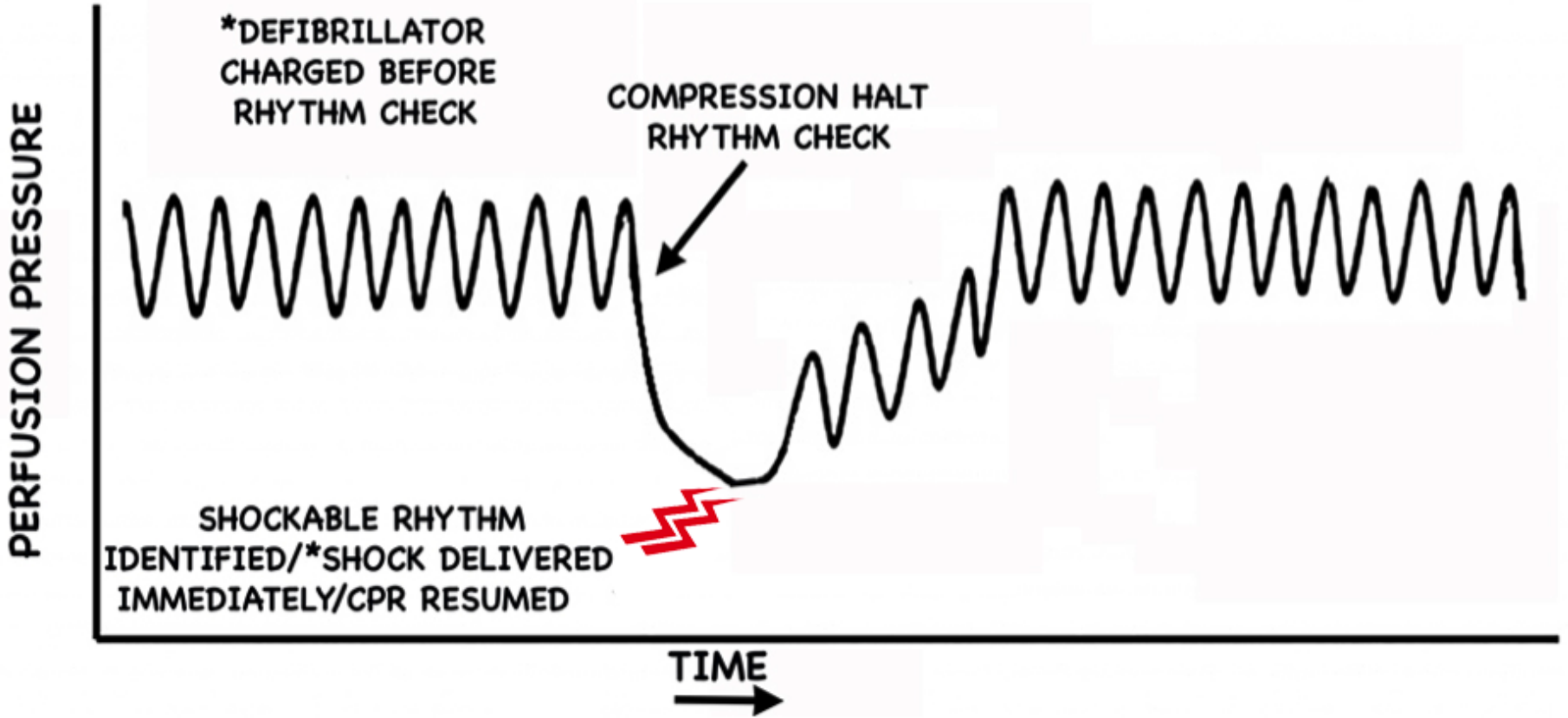
- Not performing CPR on a gasping patient (gasping is common in the first few minutes of VF arrest)
- Not rotating providers, resulting in fatigue and poor CPR performance
- Performing CPR during pre-hospital transport, resulting in poor CPR quality. It is recommended that, if possible, resuscitation occur where the person is found.

Inadequate Perfusion Pressure



Cunningham LM et al. American Journal of Emergency Medicine 2012

Chest Compressions During Cardiac Arrest
Magnitude of Perfusion Resulting from Chest Compressions



Perfusion During Cardiac Arrest with Chest Compressions

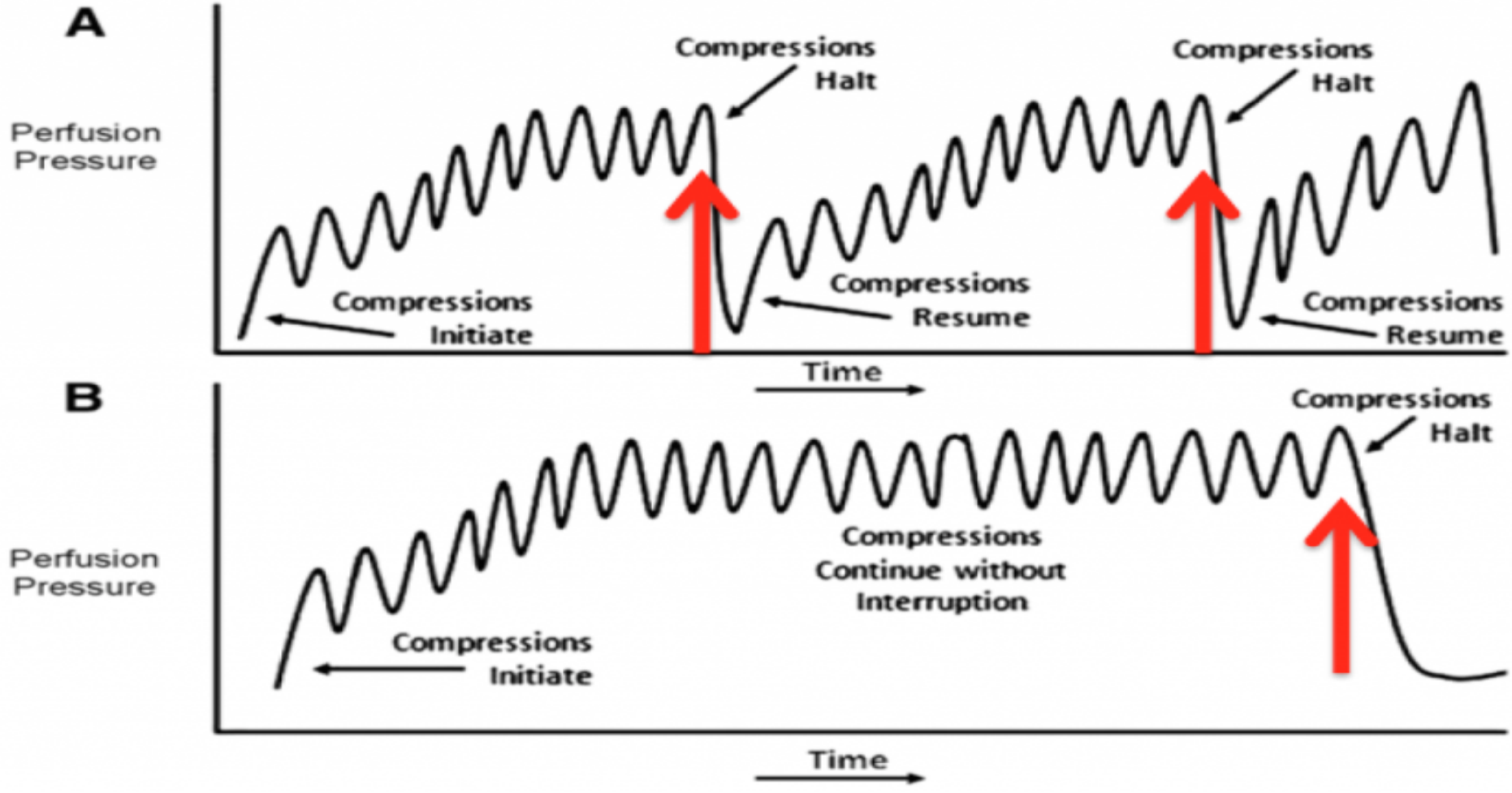
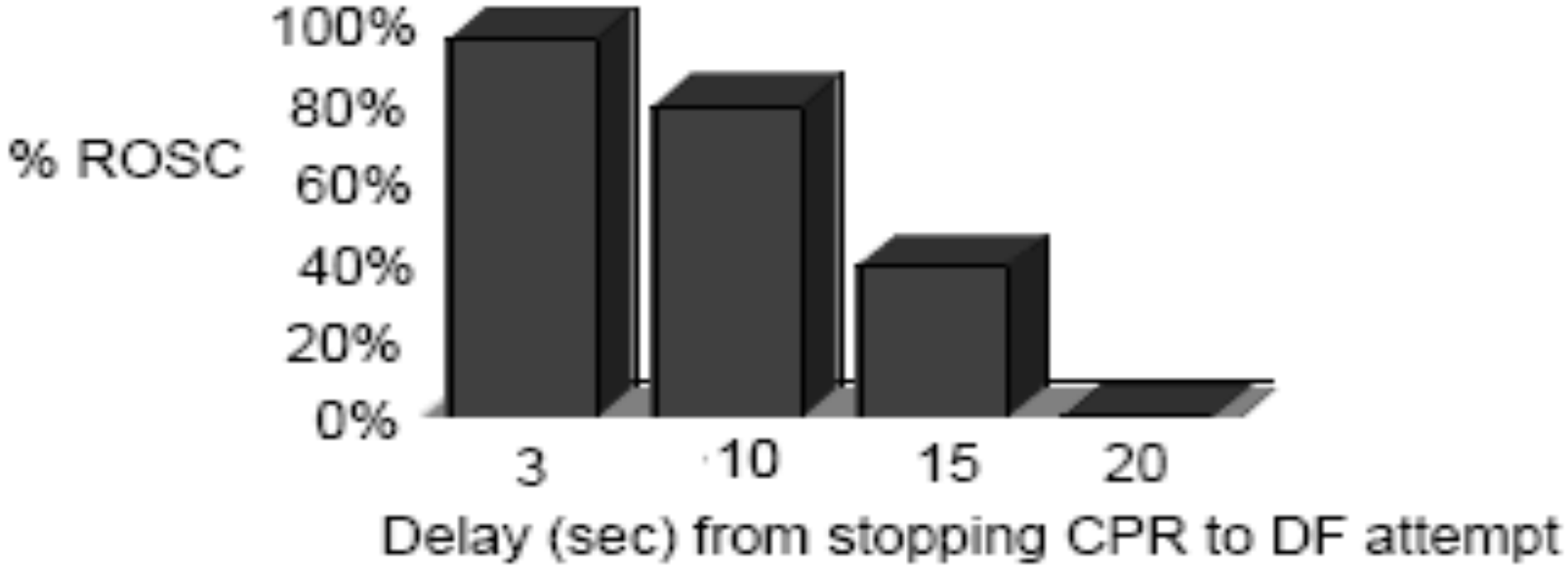


Figure 2. Hands-Off Interval vs. ROSC



Used with permission from: Yu, et al. *Circulation* 2002;106:368-372.

FACTS

- **Published on Oct 21, 2012**
  - Rapid degradation of coronary perfusion pressure occurs when cardiac compressions are interrupted.
  - Excessive delays in CPR commonly occur when compressions are interrupted for defibrillation
  - The continued contact with the patient during defibrillation with 200 J biphasic voltage results in no apparent risk to the operator doing chest compressions.
  - Further evaluation of current recommendations for interrupting CPR during defibrillation should continue.
  - Perhaps it won't be long before the warning, "All Clear, Stand Back" will soon be a thing of the past.

“

GOD HEALS AND THE DOCTOR TAKES THE FEE

BENJAMIN FRANKLIN

Table 2

Effect of charging during compressions on pause times and inappropriate shocks.

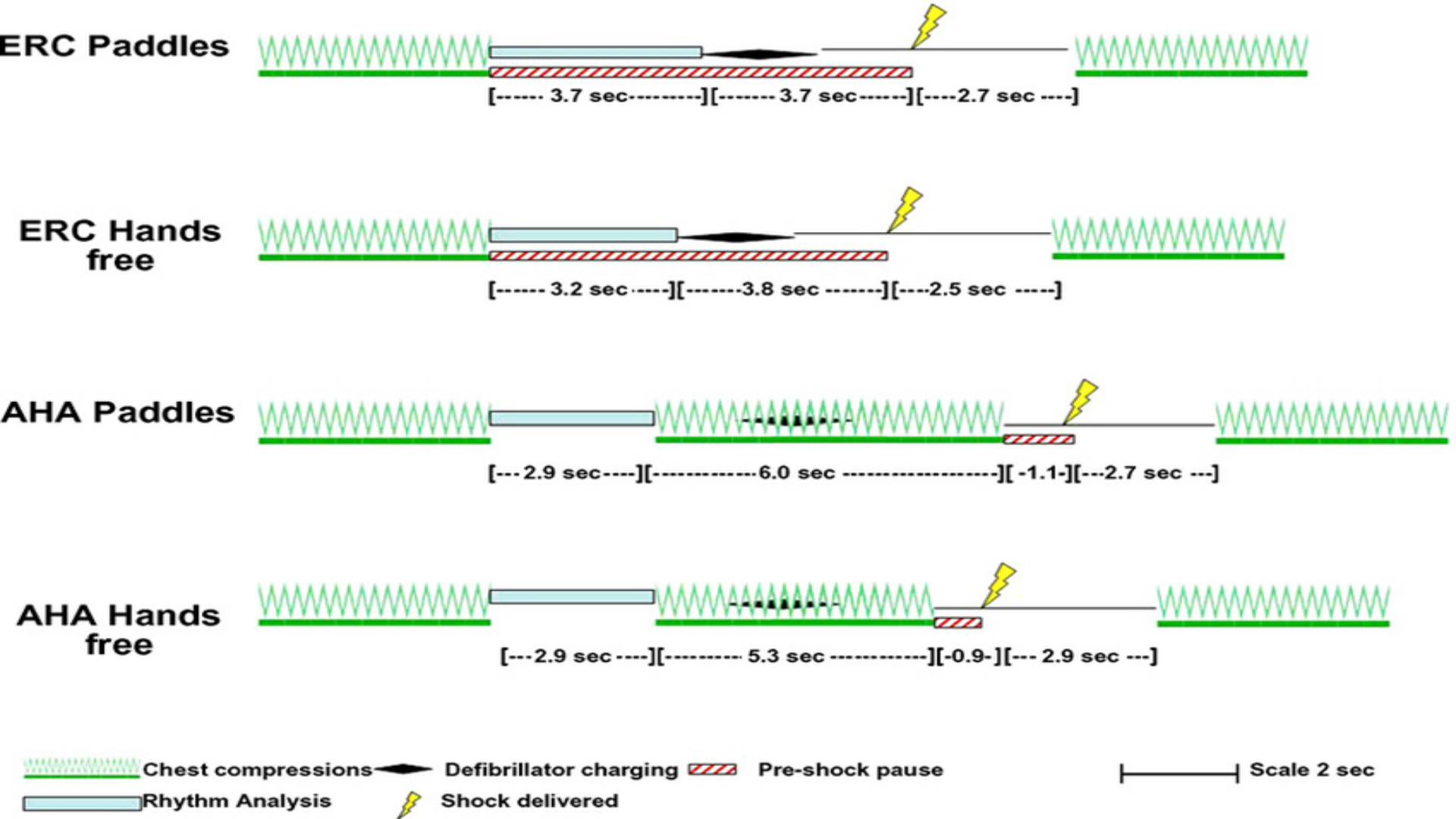
	All [$n = 562$]	Pause to charge [$n = 217$]	Charging during compressions [$n = 345$]	p-Value
Compression depth prior to shock[a], mean (SD), mm	47.3 (10.3)	47.1 (10.6)	47.3 (10.0)	0.81
Compression rate prior to shock[a], mean (SD), min^{-1}	108 (11)	106 (12)	109 (11)	0.002
Pre-shock pause, median (IQR), s	3.8 (2.2–10.0)	13.3 (8.6–19.5)	2.6 (1.9–3.8)	<0.001[b]
Post-shock pause, median (IQR), s	2.0 (1.4–3.0)	2.3 (1.7–3.9)	1.9 (1.3–2.7)	0.01[b]
Hands-off time 30s preceding shock, median (IQR), s	11.7 (8.1–16.3)	14.8 (11–19.6)	10.3 (6.4–13.8)	<0.001[b]
Inappropriate shocks[c], n/total (%)	112/560 (20.0)	43/216 (20.0)	69/344 (20.1)	0.97
Shocks to rescuers, n/total (%)	1/562 (0.2)	0/217 (0.0)	1/345 (0.3)	0.43

IQR, interquartile range.

[a] There was no compression data prior to shock in 17 pause to charge cases. Compression depth could not be analyzed in two additional cases, and compression rate could not be analyzed in one additional case.

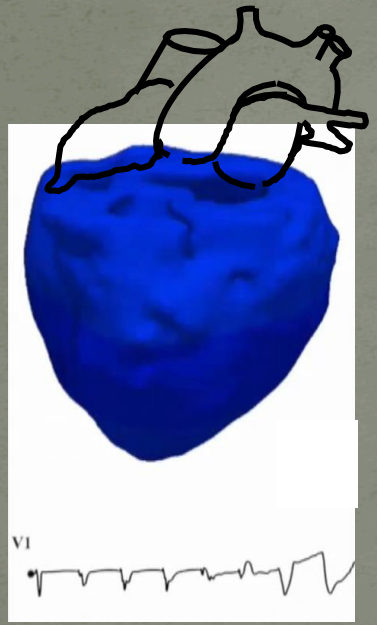
[b] Adjusted for individual resuscitation, hospital, cardiac arrest location, time of day, shock number, and presence of a shockable rhythm.

[c] Rhythm could not be analyzed prior to shock in two cases.

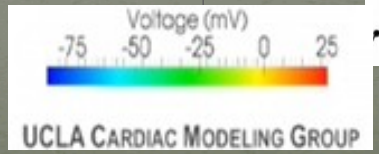

ERC Paddles
[------ 3.7 sec---------][------- 3.7 sec------][----2.7 sec ----]
ERC Hands free
[------ 3.2 sec -----][-------3.8 sec -------][----2.5 sec -----]
AHA Paddles
[--- 2.9 sec----][------------6.0 sec ------------------][-1.1-][---2.7 sec ---]
AHA Hands free
[---2.9 sec ----][--------- 5.3 sec ------------][-0.9-][--- 2.9 sec ---]
Chest compressions
Defibrillator charging
Pre-shock pause
Scale 2 sec
Rhythm Analysis
Shock delivered

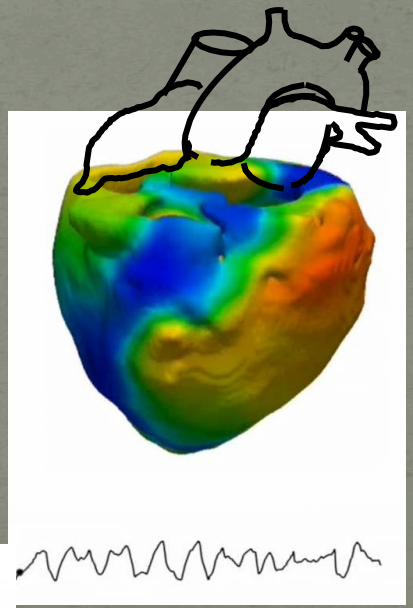
Electrophysiology of VF

Normal electrical activation[18]



Abnormal electrical activation in VF[18]





* VT is a rotary pattern (simultaneous waves of depolarization and repolarization)

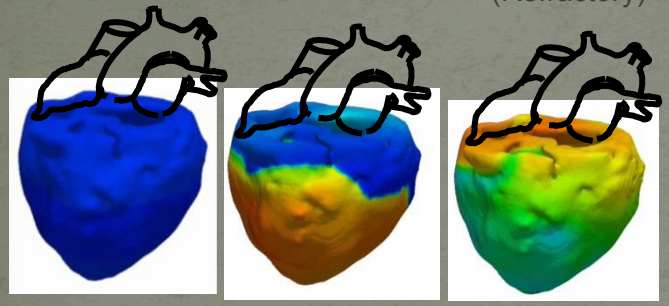
Video UCLA Heart Model via https://drive.google.com . 2016

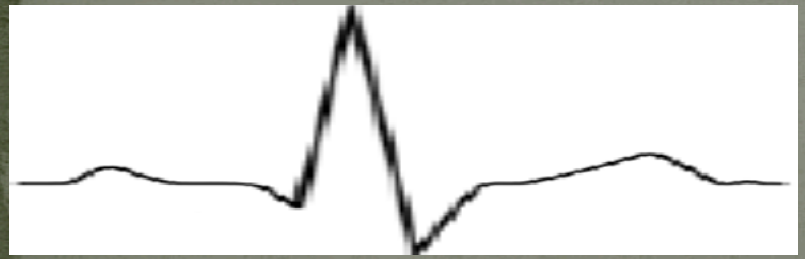
Goal of a shock? Refractory rocks…

Excitable

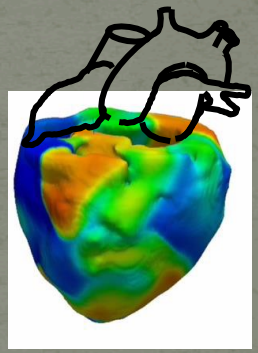
Depolarization

Repolarization (Refractory)





VF/VTp

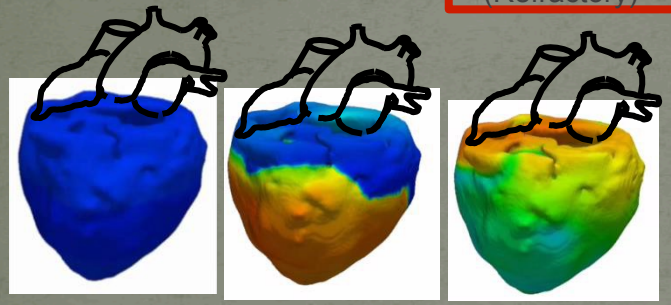


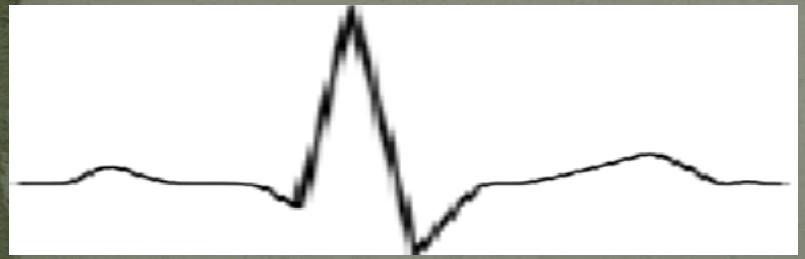
Disorganized mix of excitable, depolarizing & repolarized (refractory) myocardial cells

Goal of a shock? Refractory rocks…

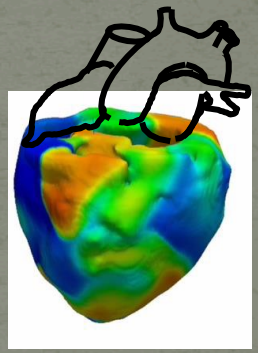
GOAL

Excitable | Depolarization | Repolarization (Refractory)





VF/VTp



Disorganized mix of excitable, depolarizing & repolarized (refractory) myocardial cells

Steen et al. *Resuscitation. 2003; 58: 249-258*


BURGER THEORY
CUSTOM BURGERS & LOCAL CRAFT BEER

Heart Attack Grill®

TASTE WORTH DYING FOR!®

SINGLE BYPASS BURGER®

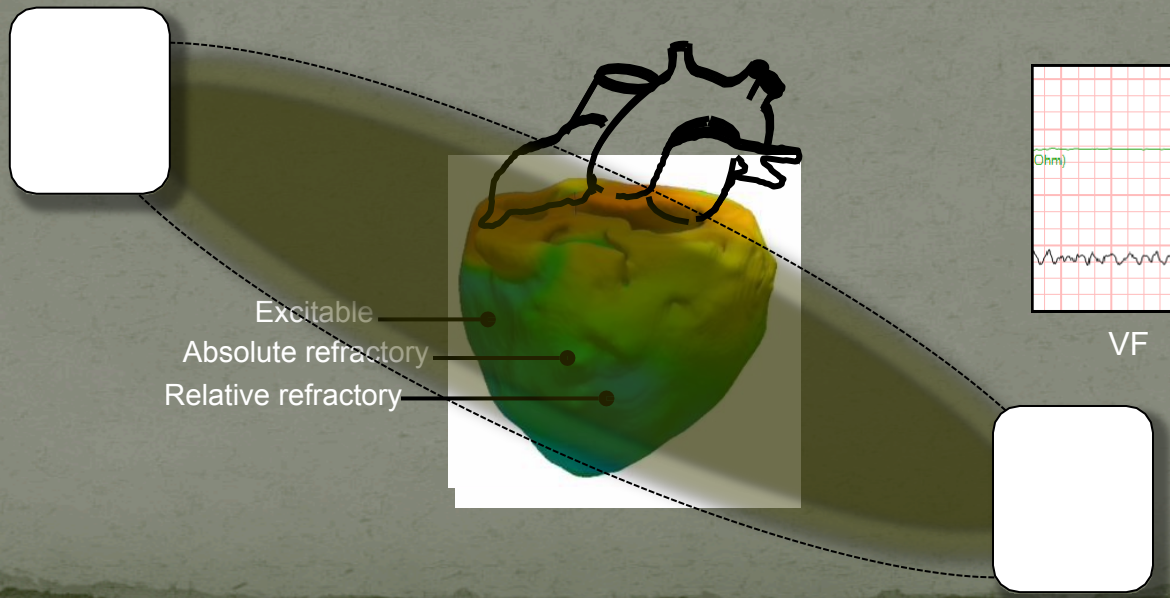
DOUBLE BYPASS BURGER®

TRIPLE BYPASS BURGER®

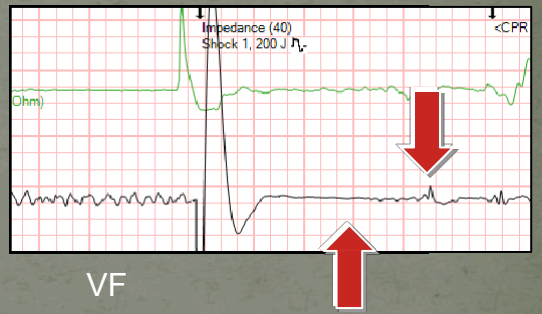
QUADRUPLE BYPASS BURGER®

Why does a shock work?

- Critical Mass Theory- The consensus choice
- Shock success likely occurs by depolarizing the heart
- Shock electrical field must cover the entire heart
- A critical mass of the heart is then in “absolute refractory”



Shock

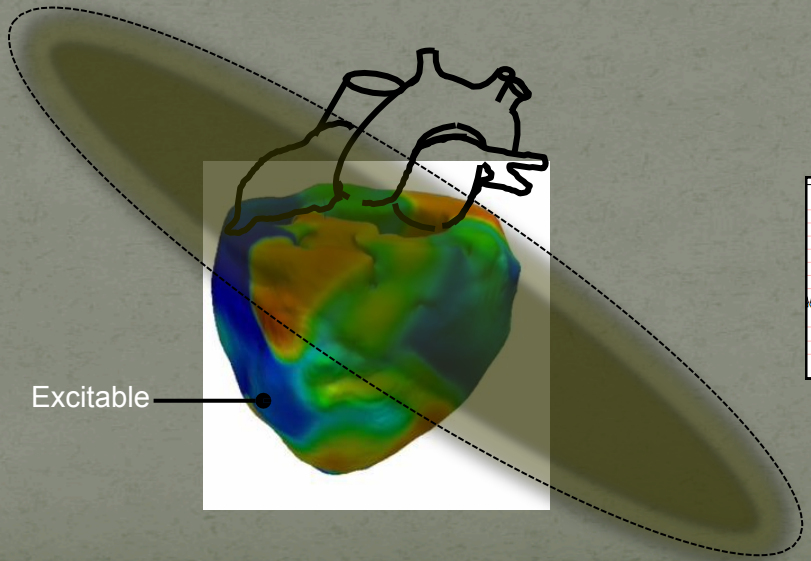


VF

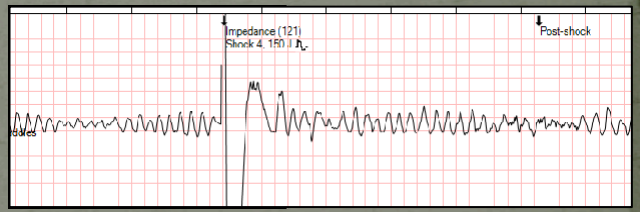
Why does a shock fail?

- Shock failure occurs at the weakest point of the electrical field
- Myocardial cells not depolarized can then restart VF/VT

Shock fails to depolarize ALL the myocardial cells



Shock

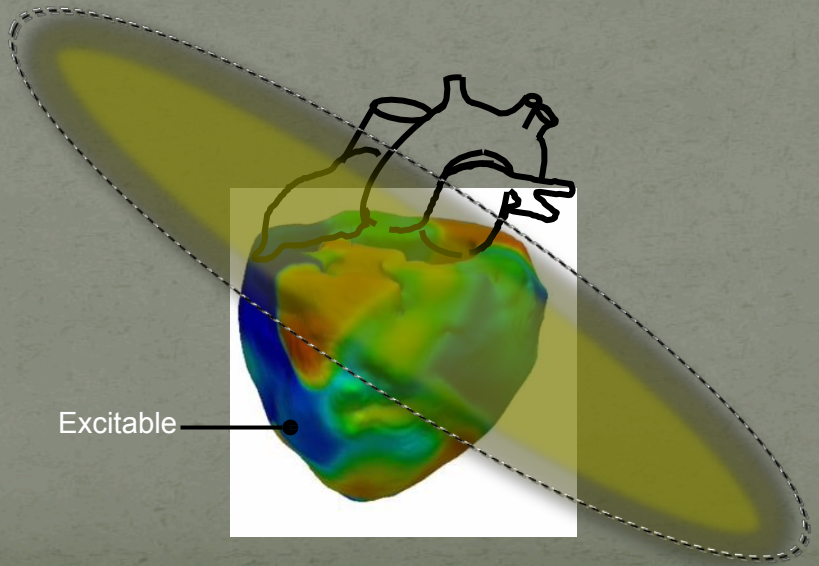


VF

Defibrillation

So, why would a shock look like this?

1. Heart is located outside of the shock's electrical field (axis deviation, LV hypertrophy)
2. Smaller shock electrical field (lower energy shocks)
3. Suboptimal shock vector (i.e. pad placement)



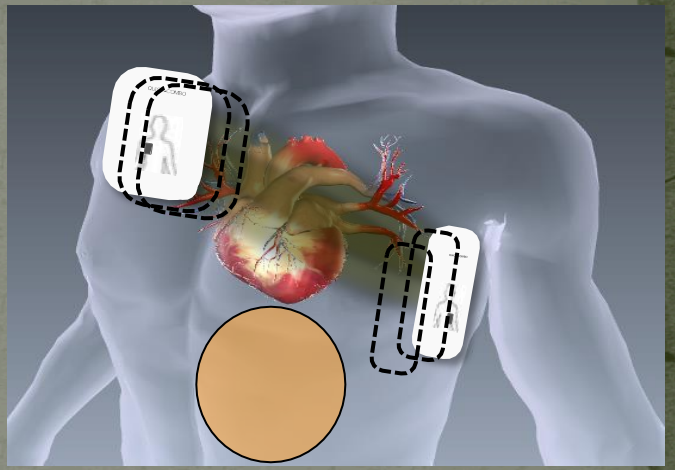
HOW MUCH DOES PAD PLACEMENT REALLY MATTER?

New research

Pad placement study

Hypotheses

- Small changes in pad position will effect defibrillation efficacy
- Higher energy shocks will be more consistently successful than lower energy shocks



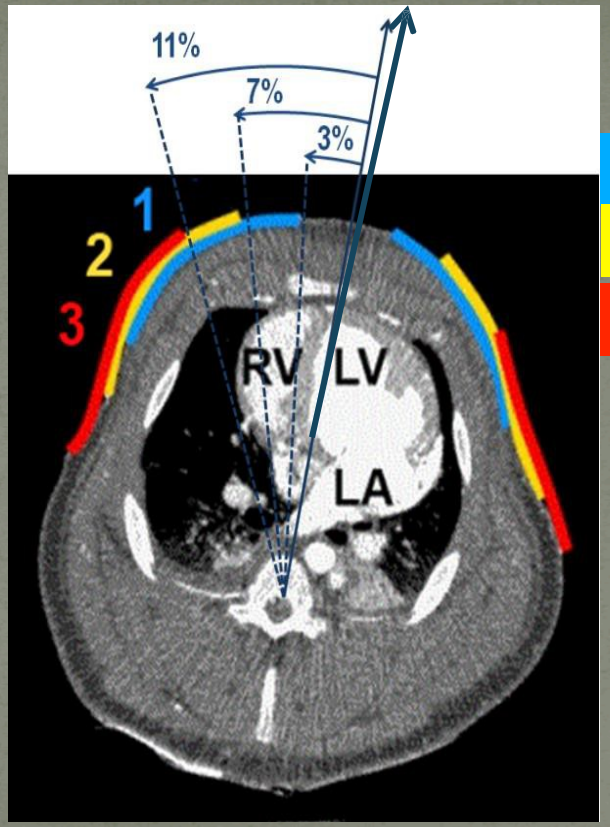
March/April 2016

Minor Variations in Electrode Pad Placement Impact Defibrillation Success

Alexander Esibov, BS, Fred W. Chapman, PhD, Sharon B. Melnick, AAS, Joseph L. Sullivan, MS, MME, Gregory P. Walcott, MD

Study design

- Pads placed at 3 slightly different positions around the chest wall of pigs (anterior-lateral)
- 24 episodes of induced VF were tested at each pad position



Inner pad position (3% off)

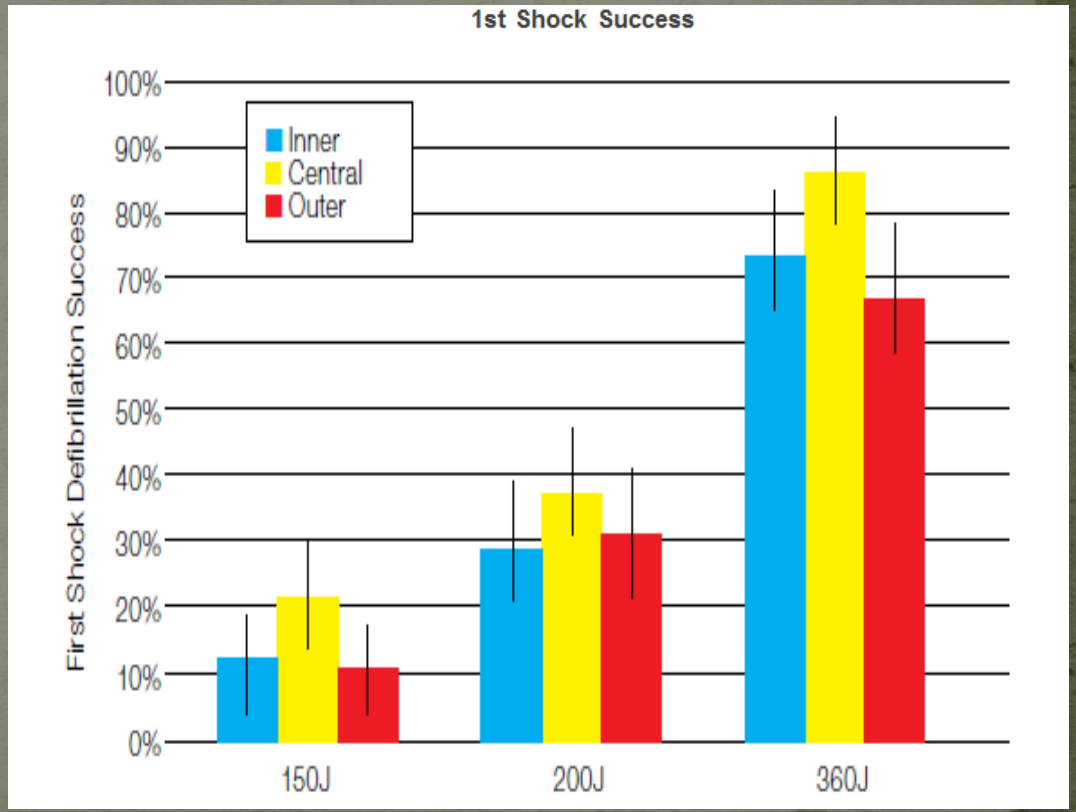
Middle pad position (7% off)

Outer pad position (11% off)

Cross section of animal thorax showing three different pad positions studied

Study results

- Minor differences in pad placement significantly affected shock success ($p<0.02$)
- Lower shock success from suboptimal pad placement was overcome by using a higher shock energy ($p<0.01$)
  - Inner 3% off
  - Middle 7% off
  - Outer 11% off



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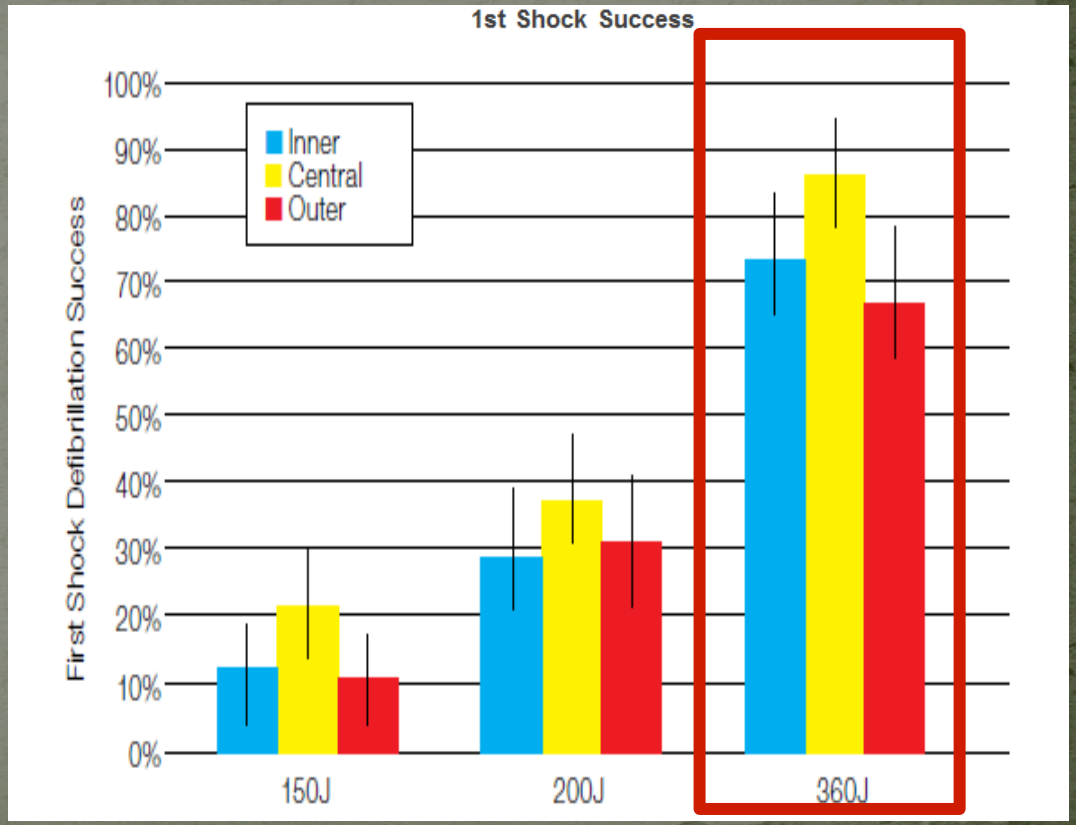
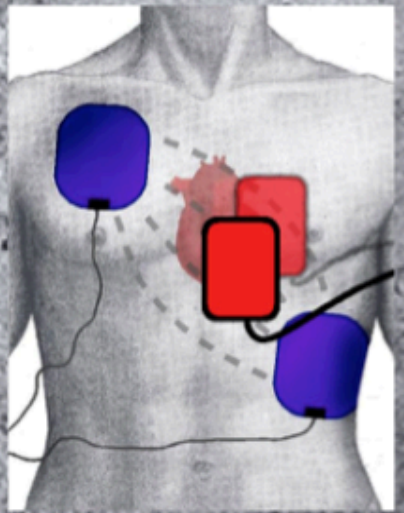
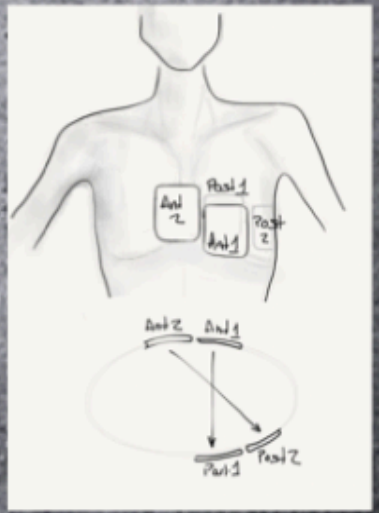


The One-Two Punch

Double Sequential External Defibrillation





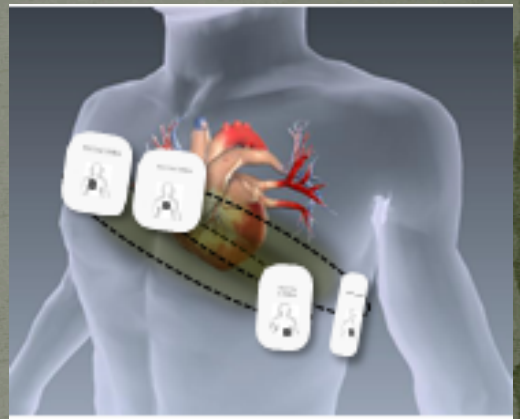
DOUBLE SEQUENTIAL DEFIBRILLATION (DSD)

also known as

DOUBLE SEQUENTIAL EXTERNAL DEFIBRILLATION (DSED)

Double sequential defibrillation (DSD)

- What is it?
  - Not an FDA approved use
  - Using two defibrillators and two sets of pads
  - Shocking the patient at the "same time"
  - Two "myocardial sandwiches" instead of one
- Who might get DSD?
  - Patients in recurrent VF or refractory VF (RVF)
  - Given after 4, 5 or 6 failed max energy shocks
    - Protocol consider after one but definitely after 3
- One mechanistic hypothesis
  - Likely not "doubling" energy (i.e. 360J x 2 doesn't equal 720J)
  - Nearly impossible to hit shock buttons simultaneously
  - Likely delivering two max energy shocks, closely together, to cover more heart



DSD example

Double sequential defibrillation (DSD)

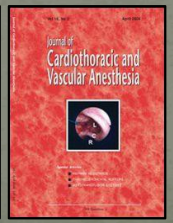
Single IHCA case (2015)

- 66 y/o male, inferior STEMI
- Developed VT/VF, standard single shocks x 15, over 72 min
- DSD (two 200J shocks) x 2 with conversion to NSR with ROSC
- Stented an 80% proximal RCA , very acidotic
- **Did not survive to D/C**

April 2015

Simultaneous Use of Two Defibrillators for the Conversion of Refractory Ventricular Fibrillation

Neal Stuart Gerstein, MD,* Mark Bipin Shah, MD,† and K. Michael Jorgensen, MD†



Double sequential defibrillation (DSD)

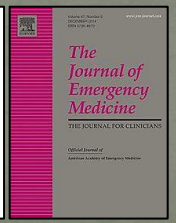
Single IHCA case (2016)

- 56 y/o female, c/o chest pain, VF during admission
- Standard single shocks x 4 with 200J (did not escalate energy to 300J or 360J ?)
- DSD x 1 with two 300J, converted VF to NSR
- 100% mid LAD and 90% distal LAD
- **Survived to D/C, neurologically intact**

In-press. 2016

REFRACTORY VENTRICULAR FIBRILLATION SUCCESSFULLY CARDIOVERTED WITH DUAL SEQUENTIAL DEFIBRILLATION

Rodney C. Sena,* Samuel Eldrich, MD,† Richard M. Pescatore, MD,† Anthony Mazzarelli, MD,† and Richard G. Byrne, MD†



Double sequential defibrillation (DSD)

10 OHCA cases (2015)- Wake County EMS, North Carolina

- All were patients in RVF
- Median resuscitation time 51 min
- Median no. standard single shocks 6.5, median no. of DSD 2
- DSD terminated RVF in 70% of the cases
- **3 patients had ROSC in the field, none survived to hospital D/C**

Jan/March 2015

Double Sequential External Defibrillation in Out-of-Hospital Refractory Ventricular Fibrillation: A Report of Ten Cases

José G. Cabañas MD, MPH, J. Brent Myers MD, MPH, Jefferson G. Williams MD, MPH, Valerie J. De Maio MD, MSc & Michael W Bachman MHS, EMT-P

Double sequential defibrillation (DSD)

Four year OHCA retrospective analysis (2016)- Columbus, OH

- 2428 OHCA cases: 12 total DSD cases (after 5 failed single shocks)
- Shorter median time to DSD than Cabanas study (27 min vs 39 min)
- Shorter median prehospital resuscitation time than Cabanas (32 min vs 51 min)
- 9 pts were converted out of RVF with DSD
- **3 patients survived to D/C, 2 of them with good CPC scores**

In-press. 2016

Use of double sequential external defibrillation for refractory ventricular fibrillation during out-of-hospital cardiac arrest☆

Eric Cortez[a,b], William Krebs[c], James Davis[a], David P. Keseg[a,c], Ashish R. Panchal[c,*]

Double sequential defibrillation vs. single shocks

Two year OHCA retrospective analysis (2016)- San Antonio, TX

- 3,470 OHCA: 279 RVF for inclusion
- 50 DSD (after 3 failed max energy shocks) vs. 229 single shocks- 200J (at least 4 shocks)
- No difference neurologically intact survival: DSD (6%) vs. standard shock (11%) p=0.317
- No difference in ROSC: DSD (28%) vs. standard shock (37%) p=0.255
- **3 DSD patients survived to D/C with good CPC scores**
- **No better than the standard single shock group** (data could be skewed due to higher witnessed arrests in single shocks group)

June 2016

Dual defibrillation in out-of-hospital cardiac arrest:
A retrospective cohort analysis☆

Elliot M. Ross[a,b,c,*], Theodore T. Redman[a,b,c], Stephen A. Harper[a,b,c], Julian G. Mapp[a,b,c], David A. Wampler[a,b], David A. Miramontes[a,b]

Double sequential defibrillation (DSD)

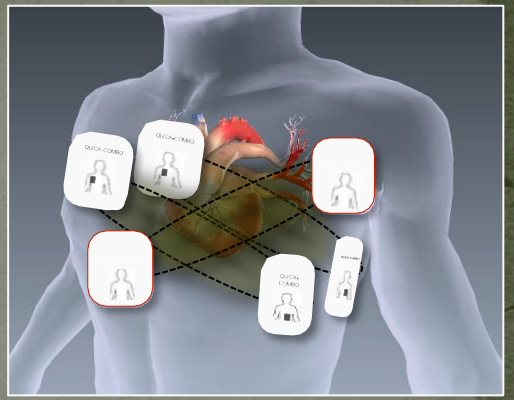
What are the unknowns?

- Optimal pad locations?
- Optimal timing sequence of the two shocks?
- Optimal patient? It may benefit some patients.
- Optimal time to try DSD?
- No prospective clinical trials, only case studies and retrospective reviews?

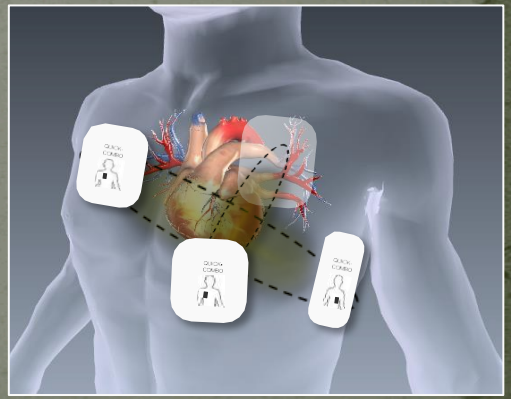
What are the risks?

- Timing of shocks could actually LOWER shock success
- Not FDA approved & is Off-Label use of external defibrillators

anterior-lateral configuration examples



anterior-lateral + anterior-posterior configuration examples



SEQUENTIAL DEFIBRILLATION (DSD)

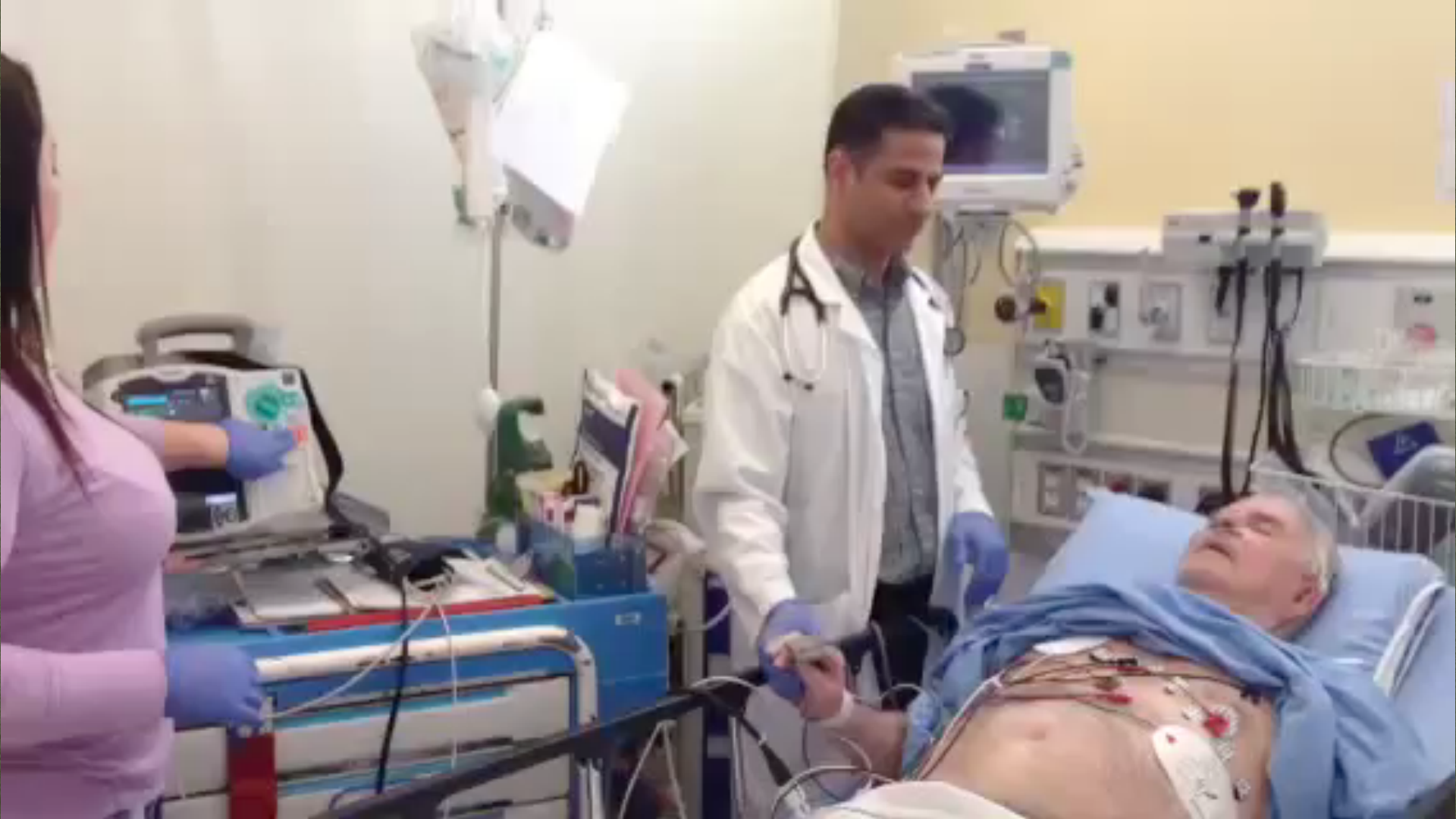
- This involves 2 manual monitor/defibrillators to be present. This treatment modality will not be available with ALL EMS Responses. There is no expectation of Dispatch and/or EMS Service(s) sending 2 ambulances for this possibility alone. In very limited situations, such EMS response may be operationally possible and/or response of the Field Operations Supervisor. OMD does not have to be contacted prior to implementation of protocol. The benefit of this change is to deliver more effective energy during defibrillations, in a shorter period of time. With an ever increasing obesity co-morbidity and refractory VF for other reasons, a physical change in energy strategies will help the effectiveness of defibrillation therapy. It is recommended that after the second attempt, if not on the second attempt, when and if possible, all subsequent defibrillation attempts be double. Notify receiving hospital of situation and utilize their equipment if need be.

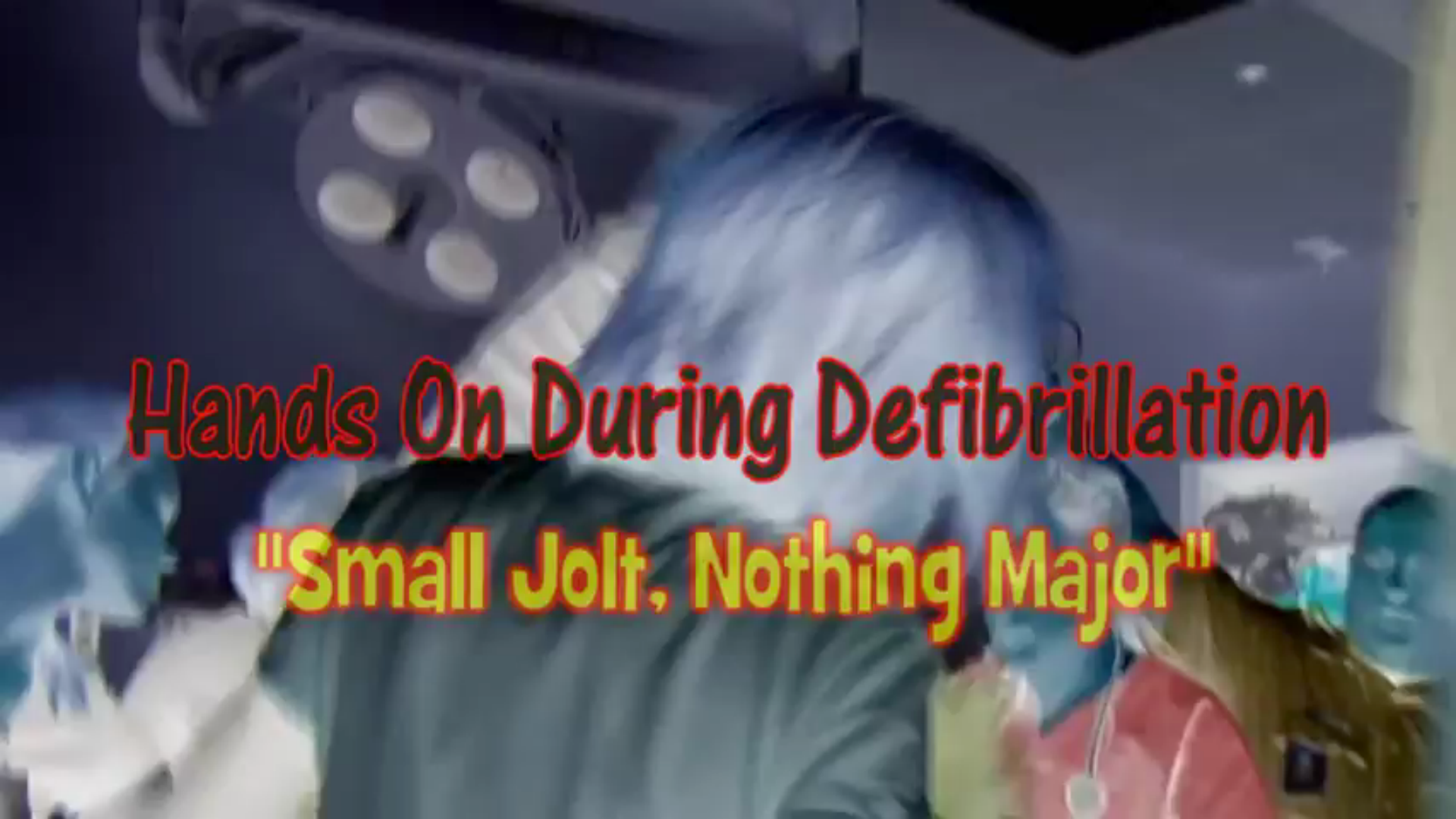
IUH BMH EMS EXPERIENCE

- Since 12/16
  - 7 patients
  - All in refractory V fib
  - Six survived neurologically intact discharged to home
  - One survived 17 days

Shock Through CPR

- Continue compressions while defibrillation occurs
  - Best if LUCAS device is used
  - If manual use gloves and a folded towel
    - NOT MANDATED to do manual




Hands On During Defibrillation
"Small Jolt, Nothing Major"

Hands On Defibrillation!

1. Continuous CPR without interruptions is ideal.
2. Stopping compressions for defibrillation is an interruption.
3. In this video I test to see if I could feel the shock.
4. I keep one hand on a patient during defibrillation.

Hands On Defibrillation!

KEY TAKEAWAYS

1. Where you place the defib pads matters.
    a. Using higher defibrillation energy can overcome changes in pad locations
        i. Monophasic 360 J
        ii. Biphasic 200 J

2. *Double Sequential Defibrillation* is an off-label use of all defibrillators and not FDA approved.
    a. It may give some patients in refractory VF one last chance.
        i. Remember the burger bun concept

3. Antiarrhythmic drugs may help cardiac arrest patients survive to the hospital, but not to leave the hospital
    a. No difference between lidocaine and amiodarone

KEY TAKEAWAYS

3. Limit time between CPR cessation and delivery of shock
   a. Consider uninterrupted CPR
   b. Shock delivery through compressions
   c. Consider gloves (double and folded towel) – NOT MANDATORY
   d. LUCAS Device utilization

4. Continue with CPR after a shock is delivered as seen in video.

5. Narcan has no use if there is no heart beat.

6. In heroin overdoses, CPR, $NaHCO_3$, BVM/ETT, get heart beating, then and ONLY then administer narcan.

KEY TAKEAWAYS

7. NaHCO3 should be administered early if not FIRST in all cardiopulmonary arrests.
8. King Airway is acceptable there is no shame in it.
9. CCR should have a fraction rate > 90%.
10. Do it as if your life depended on it.
11. In effect, someone's life depend on it.
12. Don't be shy, be aggressive, be super aggressive.
13. Dead is not necessarily dead until we say they are dead.
14. Be like NIKE just DO it.

KEY TAKEAWAYS

15. MAKE SURE YOU HAVE YOUR MEDICAL DIRECTORS APPROVAL AND IT IS IN THE PROTOCOLS!!!!

“

THE GOOD PHYSICIAN TREATS THE DISEASE; THE GREAT PHYSICIAN TREATS THE PATIENT WHO HAS THE DISEASE

WILLIAM OSLER

thank you!


THE END

QUESTIONS