Potential Conflicts of Interest

- **Chairman**, AHA Regional Cardiovascular Care Committee, Greater San Diego Area 2013-2017
- **Current** ACLS National Faculty 2013-2015
- **Current** ACLS, BLS and PALS Regional Faculty
- **Owner**, RCP Advanced Life Support
- **Developer**, RCP Ventilation Timer
- **Director of Advanced Life Support Courses**, Weil Institute of Critical Care Medicine, Palm Springs California

Objectives:

- Review the importance of High Quality CPR as the foundation of saving lives.
- Review the importance of Chest Compressions to Survival
- Review resuscitation science.
- Review current tools to help improve resuscitation skills.
- Review Petco2
'hands-only(compression-only) CPR was better than no attempt at CPR and produced survival equivalent to conventional CPR'

2008 AHA position paper on Compression-only CPR

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"High quality CPR is primary component influencing survival"

"Monitor CPR"

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DON’T LET ACLS GET IN THE WAY OF BLS!
We only circulate a small amount of blood flow :(

10% - 20% of normal blood flow to the heart
20% - 30% of normal blood flow to the brain

Please don't do anything to lower this small amount bit of blood flow.

So how have we been doing?
Many studies showed we did not push hard enough

We didn’t push fast enough
We didn’t allow full chest wall recoil

Incomplete chest wall decompression: a clinical evaluation of CPR performance by trained laypersons and an assessment of alternative manual chest compression-decompression techniques.


Ventricular fibrillation: effects of incomplete chest wall decompression during cardiopulmonary resuscitation on coronary and cerebral perfusion pressures in a porcine model of cardiac arrest.

Resuscitation 2006, 64(3):363-72. [Full text]

We Hyperventilated (and still do)

- Resuscitation
- Volume 86, Issue 2, July 2013, Pages 921-926

- Patients with cardiac arrest are ventilated two times faster than guidelines recommend: An observational prehospital study using tracheal pressure measurement.

- Jack L. McMenamin
- Lieven E.G. De Smedt
- Sabine Lemoyne
- Sofie A.M. Huybrechts
- Kristien Wouters
- Alan F. Kaminer

Many studies showed too much hands off time

An Example:

2 Professional Rescuers, BMV CPR

Assumptions: CCF rate = 100/min, 3 breaths 2 sec breathing, 3 sec audible P3s after 2 breaths, 1 sec required CCF. Breathing time 6 sec, breath 2 sec, resuscitation 0.5 sec.

<table>
<thead>
<tr>
<th>Breath (min)</th>
<th>2 rescuers Breathing</th>
<th>3 rescuers Breathing</th>
</tr>
</thead>
<tbody>
<tr>
<td>15:3</td>
<td>24.0</td>
<td>24.0</td>
</tr>
<tr>
<td>15:1</td>
<td>22.5</td>
<td>22.5</td>
</tr>
<tr>
<td>20:1</td>
<td>40.4</td>
<td>40.4</td>
</tr>
<tr>
<td>30:2</td>
<td>58.5</td>
<td>58.5</td>
</tr>
<tr>
<td>50:2</td>
<td>68.3</td>
<td>68.3</td>
</tr>
</tbody>
</table>

• Stay on the chest every single second they can.
• CCF > 80%
• Measure everything
• Resuscitate the resuscitatable
• Avoid futile resuscitation
• Pit stop approach
• Hot and cold debriefs
• Rapid Response
• System wide training
• Post arrest care

Common Causes of Hands Off Time

- Pausing for procedures (intubation, central line, IV/IO/Central.)
- Pulse checks, rhythm checks
- Pausing after shock to await a post shock rhythm
- Pausing to charge, analyze, clear and shock

So, what do the Hospitals and EMS Systems with the highest Neuro Intact Survival do differently than the rest of us?
“Performing CPR without measuring the effects is like flying an airplane without an altimeter”
Max Harry Weil

“THE AHA DOES NOT ENDORSE ANY PARTICULAR RESUSCITATION PROTOCOL OR STRATEGY”

BLS PHP Instructor Manual, 2014 Pg 9

AHA Guidelines:
- Resource
- Provide guidance
- Lend supportive information
- Substantiate guidelines are based on avail science
- Many use AHA guidelines to devise local protocols
- Protocols and guidelines aren’t always parallel

BLS PHP Rollout Material, 2014 Pg 22

High Quality CPR
UCSD’s CPR Process Data

CPR Process Data
- Chest compression fraction: 91%
- Compression rate: 123/min
- Compression depth: 2.6 inches
- Pre-shock pause: 2.0 sec
- Post-shock pause: 3.6 sec
- Perfusion check: 4.3 sec
- Ventilation rate: 9.7/min
- PetCO2: 15.3 mmHg

UCSD: Pre-ART (2006) to 2012

Arrest Survival

ED Arrest Survival
It is reasonable to consider using quantitative waveform capnography in intubated patients to monitor CPR quality, optimize chest compressions, and detect ROSC (return of spontaneous resuscitation) during chest compressions or when rhythm check reveals an organized rhythm (Class IIb, LOE C).” 2010 AHA Guidelines

<table>
<thead>
<tr>
<th>Traditional</th>
<th>Newer Methods</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monitoring Ventilation</td>
<td>Detecting the quality of CPR</td>
</tr>
<tr>
<td>Intubation</td>
<td>Determining the proper depth of compressions</td>
</tr>
<tr>
<td>Transport</td>
<td>To detect ROSC</td>
</tr>
</tbody>
</table>

Co2 Devices

- Exhaled CO2, Petco2, Waveform Capnography and Quantitative Capnography
- Colorimetric Co2 Detector

Science of Co2

- Under normal conditions etCO2 is in the range of 35 to 40 mm Hg (note, not 35–45 mmHg)
- With no cardiac output or pulmonary circulation, etCO2 will approach zero with continued ventilation. What would happen if we started compressions and produced a small amount of pulmonary blood flow?
- Persistently low etCO2 values (<10 mm Hg) during CPR in intubated patients suggest that ROSC is unlikely
- During CPR, etCO2 is a measure of circulation rather than Ventilation
- During CPR etCO2 is an indirect measurement of cardiac output
- Dependent on a fixed ventilation rate of no more that 10bpm
What does the literature say about ETCO$_2$ during CPR?

**CPR Quality**

- **Physiologic Parameters**
  - Quantitative waveform capnography
    - If PETCO$_2$ < 10 mm Hg attempt to improve CPR quality
  - Intra-arterial pressure
    - If relaxation phase (diastolic) pressure < 20 mm Hg attempt to improve CPR quality

**ETCO2 in adult OHCA**

*Sanders, JAMA 1989*
"96 Minutes Without a Heartbeat", Wall Street Journal, Tuesday, May 17, 2011

"only reason they continued with CPR was due to the capnography readings... which indicated that the metabolic process was working"

"During the resuscitation, Mr. Snitzer was showing visible signs of life, including raising his arms. And we also had data that confirmed blood was flowing through his lungs to his brain."

• In Mr. Snitzer's case, his etCO₂ values remained in the low 20s

• Mayo Clinic doctors and paramedics both credited capnography as an important therapy leading to Mr. Snitzer's survival
Surviving 96 minutes of CPR

- 54 yo male with witnessed sudden cardiac arrest
- Immediate bystander CPR and VF
- Defibrillation & drugs with minimally interrupted CPR
- ~ 20 minutes post-arrest: intubated
- ROSC after 96 minutes of CPR but comatose
- Transported to Mayo Clinic for post-resuscitation care
- Discharged neurologically intact 10 days later
- NPR: Why did you continue so long?
- Roger White: ETCO2 remained high showing good blood flow

ACLS
ETCO2 to detect ROSC

Compressions:
- Increase intrathoracic pressure
- Compresses heart, and lungs
- Squeeze thoracic vessels
- Cause a small amount of forward blood flow

Decompressions:
- Decrease intrathoracic pressure
- Create a negative pressure/suction in the chest
- Enhances venous return/causes blood to return to the heart
Coronary Perfusion Pressure (CPP)

- Simply another word for coronary blood flow. CPP pushes coronary blood flow through the coronaries.

  Remember, if you want the heart to start again, it has to have good coronary circulation.

  The problem is, blood flow does not start immediately with the first compression.

  Many studies demonstrated it took several compressions to really “distend” the system and start moving good forward blood.

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Coronary Perfusion Pressure (and the Consequence of Stopping Compressions)

How well you do CPR is the major factor.
How quickly someone is on the chest.
How deep you push.
How fast you push.
How quickly you defibrillate.
Did you minimize the 20 or so distractions, that cause us to get off the chest??
There is a vast difference between the coronary and cerebral perfusion pressures generated by poor-quality and high-quality CPR.

What affects CPP?

The Benefit of Continuous Chest Compressions on CPP
Don’t Hyperventilate

- Milwaukee:
  - Mean ventilation rate: 37 breaths/min
  - After training for 2 months: 22 breaths/min

- Dallas: 30 breaths/min
- Tucson: 34 breaths/min (24 – 60)
- Chicago: >30 breaths/min

Minimize the Pre and Post Shock Pauses

AHA study: Pause between CPR, defibrillation can be deadly

It’s wise to Analyze and Debrief Every Code Blue
AHA 2010 Guidelines for CPR

- Depth; at least 2 inches
- Rate; at least 100 times per minute, no more than 120
- Full chest wall recoil after each compression
- No more than 10 breaths per minute
- Shorter inspiratory phase of up to 1 second
- Minimize interruptions in chest compressions

AHA 2010 Guidelines for CPR continued...

- Rotate compressors at least every 2 minutes to reduce fatigue
- Use Petco2 Keep > 10mmhg
- Use real time prompting devices.

Some Final thoughts...

- Clinicians must own CPR
- Minimize interruptions in CPR at all times
- Ensure proper depth of compression (at least 2 inches)
- Ensure full recoil/decompression
- Ensure proper chest compression rate (100-120 per minute)
- Do compressions until defibrillator is fully charged
- Rotate compressors every one or two minutes
- Hover hands over chest during shock and be ready to compress
- Intubation or advanced airway with ongoing CPR
- Place IV/IO with ongoing CPR
- Coordination and teamwork between MD’s, RCP’s and RN’s
Conclusion

- Every compression is an important compression
- Stay on the chest every second you can
- Be fanatical, about staying on the chest
- Don’t let ACLS get in the way of BLS
- Don’t just do CPR, do CPR correctly

Any Questions?

Thank you very much.

For My Wife and Kids
Intro to Bedside Ultrasound

sonoroundtable

web: www.sonoroundtable.com
twitter: @sonoroundtable

E-FAST
Technique

Identify free fluid in:
- Pericardium
- Thorax
- Abdomen
- Identify PTX

Free Fluid is:
- Anechoic
- Dependent
Technique

Heart Beating
Pericardial Effusion
LV Systolic Function
RV:LV Ratio
IVC
Findings
Lung Sliding
Present or Absent
"Seashore" Sign
“Stratosphere” Sign
A Lines

A Lines = Air

A Lines

Horizontal repeating artifacts below pleural line

A Lines

Normal pattern
B Lines

B Lines = Alveolar or Interstitial Fluid
B Lines
Vertical artifacts arising from pleural line

B Lines
More than 3 in one rib interspace
Vertical artifacts from pleural line
Obscure A lines
Extend >18 cm
Vertical artifacts from pleural line
Obscure A lines
Extend >18 cm

Consolidation
Fluid filled alveoli
“Tissular”
Air bronchograms
“Tissular”
“Shred Sign”

Effusion

Fluid in thorax
Questions about US findings?

Lung Sliding  A Lines  B Lines
Consolidation  Effusion
Vascular Access

Technique

Ultrasound beam is 1 to 1.5 mm thick
Needle can be difficult to visualize

Visualize needle by

“Ring down” artifact
Soft tissue motion

Ring down artifact
Ring down artifact

Soft tissue motion

Know where the needle tip is

AT ALL TIMES
Artery vs. Vein

Compression
Doppler
Location
Appearance
Long vs. Short Axis

Long Axis
“In Plane”

Short Axis
“Out of Plane”

Long Axis
Short Axis
Long Axis

Allows visualization of needle throughout procedure

Technically more difficult
Difficult to keep needle directly in view

Can see adjacent structures

Needle not under probe
Rocking and Fanning
“Inchworm”

Visualize guide wire in vessel
Peripheral Venous Access
Technique
Peripheral veins run alone

Deep veins run with artery

Long Axis

Short Axis
Peripheral Venous Access Cases
Clinical Toxicology Workshop: Life Saving Tips and Treatments for Care of the Poisoned Patient

Josef G. Thundiyil, MD, MPH, FACEP, FACMT
Associate Professor Emergency Medicine
Orlando Health
April 2015

Objectives

• Provide clinical pearls for evaluation and treatment of the poisoned patient
• Deepen your understanding of the mechanisms
• Avoid pitfalls
• Detail up to date information

Overview

• Interactive, small group, chalkboard, table top and case based sessions
• Unknown acidosis
• Cardiovascular complications
• Update on antidotes
• Common Toxins
Interactive

• Discuss questions
• Management pitfalls that you have encountered
• Learn from each others mistakes

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62 yo male with AMS. He is confused and slightly agitated. Disheveled known alcoholic with no known psychiatric history. Found by EMS when bystandard called about him. History is unclear. EMS states he is drunk, they run on him all the time.

Case 2

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**Comparision**

**Diabetic ketoacidosis**
- Glucose >250 mg/dl
- Hco3 <15 meq/l
- High AG metabolic acidosis
- pH <7.3
- Ketonemia / ketonuria

**Alcoholic ketoacidosis**
- Decreased undetectable ETOH level
- Variable glucose level
- High AG metabolic acidosis
- Ketonemia / ketonuria
30 yo male found down unresponsive and hypothermic. Brought by EMS. P=48, BP 85/40 T= 86

Case 3

54 yo WM admitted to hospital for a small right psoas hematoma. He is a known alcoholic. He does not smell of alcohol on admission. He has been taking homeopathic remedies to alleviate his craving for ethanol. Labs on admission: 138/4.2/24/100/12/07 normal. AST 220; ALT 180 He began showing signs of tremor and shaking with progressive signs of ETOH withdrawal. He was treated for withdrawal and required escalating doses of meds for this.

On HD 7, patient has a seizure BP 80/40, P 50

Case 4
65 yo presents with nausea and vomiting and loose stools for 3 days. Has gotten progressively lethargic today and unable to tolerate po. Has had intermittent fevers. Also complains of foot pain. Feels like his gout. Gout has been flaring recently. Ran out of pain meds, indomethacin, allopurinol, and Percocet. But developed stomach virus, probably from grandson who has had similar symptoms.

Case 5

Salicylate is a Weak Acid (pKₐ 3.8):

<table>
<thead>
<tr>
<th>TISSUES (pH 6.8)</th>
<th>BLOOD (pH 7.4)</th>
<th>URINE (pH variable)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HA</td>
<td>HA</td>
<td>HA</td>
</tr>
<tr>
<td>H⁺ + A⁻</td>
<td>H⁺ + A⁻</td>
<td>H⁺ + A⁻</td>
</tr>
</tbody>
</table>

Acidosis       Alkalosis

48 year old female presents with AMS. She was found by EMS to have accu check of 58. Became alert after one amp of D50 by EMS. Still altered but may be due to language barrier.
PMH: HTN, DM, depression
Meds; KCl, lisinopril, prednisone, doxepin, nifedical, iron, metoprolol, asa, milax, glucophage, simvastatin, cyclobenzprine, lasix
T 93.2, P 98, BP 96/60, R 18

Case 6
29 yo female presents with malaise, HA, nausea with severe vomiting, loose stools, SOB, cough. Her son was diagnosed with the flu and now she and husband are developing symptoms. They are self treating with OTC. But this evening after dinner, she syncopized. Worried about undernutrition and dehydration. Hit her head with fall but no LOC. Husband says she was confused initially but, now seems better.

PMH: none, SVD 4 years ago
Vss: T 100.3, R=20, P=120, BP 140/90, Pox=98% RA
Exam: slightly pale, lethargic, answers simple questions. Soft mild diffuse tenderness

Case 7

Carbon Monoxide

![Graph showing carbon monoxide poisoning and normal oxygen levels.]

Primer on Antidotes
37 year old male rescued from a burning building. He sustained significant smoke inhalation and 9% TBSA burns. He was found with ALOC. He is unable to answer questions about his PMH.
BP 73/30, T 99.8, P 130

Case 1

Paris Fire Study: CO and HCN
Correlation between carbon monoxide and cyanide blood concentrations in 109 fire victims

Correlation between plasma lactate and blood cyanide concentration in 99 fire victims* with no severe burns

*9 fire victims died, 5 of whom had plasma lactate 2.6 mg/L
69 yo male with h/o HIV off his medicines is brought by EMS due to confusion at bus stop. He was at another hospital where he had a lymph node biopsy last week. He was unhappy with the treatment so took some of his BP pills.

ROS: no visual changes, just general weakness.
PMH: HIV
PSH: biopsy
SH: no ETOH/drugs
Meds: does not remember.

Case 2

Intralipid

• IV nutritional solution with lipid emulsion
• Case reports
  – Bupivicaine-related cardiac arrest 20 min (Rosenblatt, 2006)
  – Massive bupropion and lamotrigine OD with 90 minutes vtach/vfib/PEA complete recovery (Sirianni, 2008)
  – Multiple animal studies
  – Reversal of haldol induced torsades
Intralipid SE

- Interferes with laboratory testing
- Pancreatitis
- Pulmonary complications
  - Lipid embolism
  - High doses esp neonates
- Unknown upper limit of dosing
- Must optimize oxygenation/ventilation
- Conflicting studies in OD when compared to epi

Intralipid Mechanism

- Lipid sink (intravascular)
  - Lipid binds toxin and pulls from tissues
  - Works best with lipid soluble drugs
- Energy substrate (intracellular)
- Suppress mitochondrial permeability
- Reduces inhibition of sodium channels
  - Membrane effects

Intralipid

- Bolus 1.5 mL/kg of 20% intralipid therapy
  - Infusion 0.5 mL/kg/min
- Within 10 minutes
  - No Vtach
  - ECG normalized
  - 16 hours: normal GCS
  - Next day extubated dc from ICU
  - Normal at discharge
Intralipid Recommendations

- Establish airway, oxygenate, ventilate, suppress seizure
  - Endorsed by AHA for specific situation—LAST
- 20% intralipid 1.5 ml/kg bolus repeat x1 till ROSC
  - 0.25-0.5 ml/kg/min for 10 min after recovery
- Highly lipid soluble drugs
  - Bupivacaine, ropivacaine, verapamil, propranolol, TCA
- Mod lipid soluble
  - Bupropion, OP insecticides

60 year old presents with AMS. Called by family members. When he woke, he was noted to be acting strange, making incomprehensible sounds.
BG was 30. Given D50 and it resolved.
PMH: DM, HTN
Exam is now normal.

Case 3

Fasano et al. Annals EM 2008

- 40 patients with sulfonylurea induced hypoglycemia randomized to D50/placebo vs. D50/octreotide 75ug SC.

Table 3: Mean difference in serum glucose, octreotide versus placebo.

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Octreotide (n=20)</th>
<th>Placebo (n=20)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>83.7</td>
<td>94.8</td>
<td>0.0001</td>
</tr>
<tr>
<td>15</td>
<td>112.5</td>
<td>102.3</td>
<td>0.005</td>
</tr>
<tr>
<td>30</td>
<td>106.2</td>
<td>93.1</td>
<td>0.0005</td>
</tr>
<tr>
<td>45</td>
<td>83.7</td>
<td>80.7</td>
<td>0.05</td>
</tr>
<tr>
<td>60</td>
<td>98.5</td>
<td>92.3</td>
<td>0.03</td>
</tr>
</tbody>
</table>

No recurrent hypoglycemic events in octreotide group (only single events).
19yo found with AMS. He was found to be incoherent and agitated. He was with friends, but none are currently available. PMH/Meds: unknown. VS: T 99.9, R 18, BP 140/85, P 120

Case 4

- Patient was given 1 mg of physostigmine over 5 minutes and had dramatic improvement of mental status
2 friends 38 and 35 year old males presents with nausea and vomit, diaphoresis, and SOB and WEAKNESS which occurred while on a fishing trip. They stopped for lunch and shortly afterward became ill. Lunch was cold cuts, pretzels and watermelon. P 52 BP 136/90 T 97.5 pulse ox 93% RA
24 year old male presents after a snake bite to his finger. He admits that he was drinking and antagonizing the snake. He thinks it was a venomous snake.

PMH/PSH/All: Unremarkable.

Case 6

Which one would you rather get bitten by?