The Beat Must go on!

Sudden Cardiac Death, ICD, Ablation, & Cardiac Dysrhythmias

Cheryl Herrmann, APN, CCRN, CCNS-CSC-CMC

CMC Therapeutic Interventions (16%)

A. Cardiovascular
   - Cardiovascular pharmacology
   - Assist devices (IABP)
   - **Electrophysiological Interventions**
     - **ICD**
     - **Ablation**

B. Pulmonary
C. Renal
D. Multisystem
   - **Therapeutic hypothermia post cardiac arrest**

CMC
Cardiac Patient Care Problems (47%)

A. Acute Coronary Syndrome
B. **Dysrhythmias**
C. Heart Failure
D. **Other Cardiac Issues**
   - **Sudden Cardiac Death**

E. Vascular Issues

CSC

CARDIAC SURGERY CERTIFICATION

III. INTERVENTIONS (33%)

A. Cardiovascular
1. Antidysrhythmics
2. Defibrillation/cardioversion
3. Emergent re-opening of the chest
4. Epicardial pacing
5. Fluid volume management specific to cardiac surgery
6. Inotropes
7. Intra-aortic balloon pump
8. Rewarming from hypothermia
9. Vasodilators/vasopressors
10. Ventricular assist devices

IV.

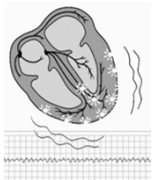
Sudden Cardiac Death (SCD) or Sudden Cardiac Arrest

- Unexpected death from a cardiac cause that occurs in a person who may or may not have preexisting cardiac disease.
- Occurs without warning
- Disruption of cardiovascular function
- Blood flow cannot be maintained → inadequate perfusion→ unconsciousness→death

Sudden Cardiac Death (SCD)

Causes

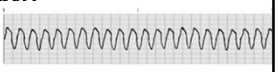
- Electrical instability leading to dysrhythmias
- Most common arrhythmias
  - Ventricular Tachycardia
  - Ventricular Fibrillation
- Other arrhythmias
  - Bradycardia
  - Asystole
  - PEA (pulseless electrical activity)



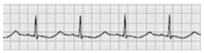
Know ACLS algorithms!

Sudden Cardiac Death

Risk Factors



- No specific tests can predict a person's risk or identify the time an event will occur
- 75% have underlying CAD
- 15% have cardiomyopathies
- EF < 30%
- Inherited electrical abnormalities
  - LQTS (long QT syndrome)
- Drug induced dysrhythmias
  - Prodysrhythmic drugs
  - Acquired LQTS

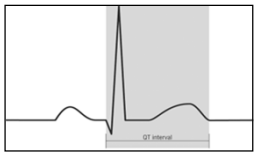


EF < 30%, Long QT interval

Q-T Measurement

Q – T Interval

- Represents the time from when the electrical impulse leaves the AV node – travels through the Bundle of HIS, down the bundle branches, throughout the ventricles and ventricular REPOLARIZATION is complete.
- From the beginning of the "Q" to the end of the "T"

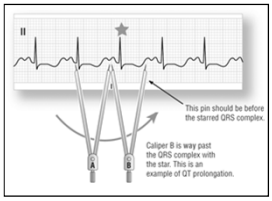


Upper limits of Q-T interval – Rate Based

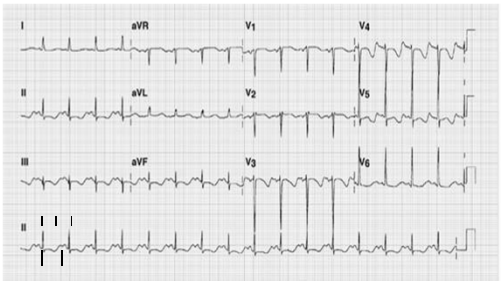
Rate	QT interval
40	0.49 – 0.50 sec.
50	0.45 – 0.46 sec.
60	0.42 – 0.43 sec.
70	0.39 – 0.40 sec.
80	0.37 – 0.38 sec.
90	0.35 – 0.36 sec.
100	0.33 – 0.34 sec.
110	0.32 – 0.33 sec.
120	0.31 – 0.32 sec.

Prolonged QT

- Rate-related value
- QTc represents the QT *corrected* for the rate.
  - Prolonged if over 0.419 sec
  - Markedly prolonged if over 0.440 sec
- Good rule of thumb:
  - If the patient is not tachycardic, the QT interval should not be more than half the R-R interval.



Prolonged QT



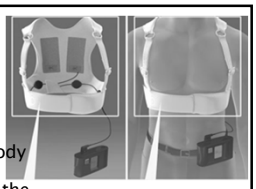
- Normal QT interval rate dependent
- QT should be < half R to R interval
- www.qtdrugs.org

Sudden Cardiac Death (SCD)

Clinical Management

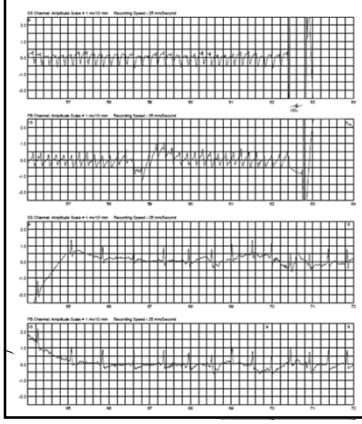
- ACLS protocols
- If unresponsive after ROSC, institute therapeutic hypothermia
- If responsive, followup management of underlying diseases/arrhythmias
- ICD implantation
- Ablation

LifeVest



- The LifeVest is worn outside the body rather than implanted in the chest.
- This device continuously monitors the patient's heart with dry, non-adhesive sensing electrodes to detect life-threatening abnormal heart rhythms.
- If a life-threatening rhythm is detected, the device alerts the patient prior to delivering a treatment shock, and thus allows a conscious patient to delay the treatment shock.
- If the patient becomes unconscious, the device releases a Blue™ gel over the therapy electrodes and delivers an electrical shock to restore normal rhythm.

Source: http://lifevest.zoll.com



- 32 seconds to first shock
- 150 joules

- Delivers shock within 60 seconds
- Time between shocks is 60 seconds
- Can deliver at least 5 shocks of 150 joules
- Leave vest on in ambulance and ED until cardiologist sees the patient

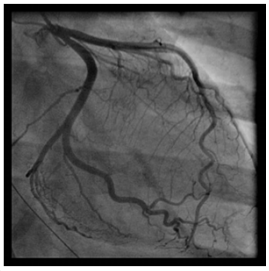
Case Study

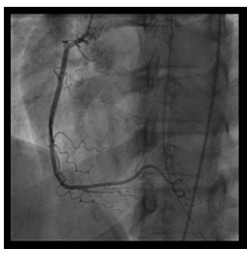
45 y/o white female sudden syncope while teaching

- CPR started, AED applied and delivered one shock

Admission EKG 12:26

EF 40- 45%





45 y/o post cardiac arrest pre ICD

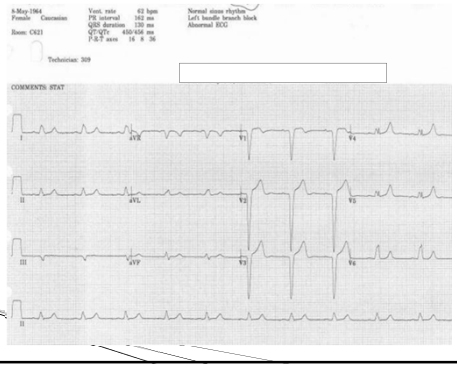
- PMH: Father fatal MI at age 57
- No smoking, alcohol, or illicit drug use
- Wt: 60 kg
- D-Dimer 2443, Potassium 3.3
- DX- Aborted sudden cardiac death etiology secondary to cardiomyopathy along with hypokalemia

- **Thyroid Stimulating Hormone**
  - **TSH = 0.15 µIU/mL (0.4-4.0) ↓**
- **T3-FREE**
  - **FREE T-3 2.2 pg/ml (2.2-4.0)**
- **T4 FREE**
  - **FREE T4 1.16 ng/dl (0.80-1.50)**
- **THYROID PEROXIDASE AB**
  - **TPO AB <0.3 IU/mL (0.0-3.9)**

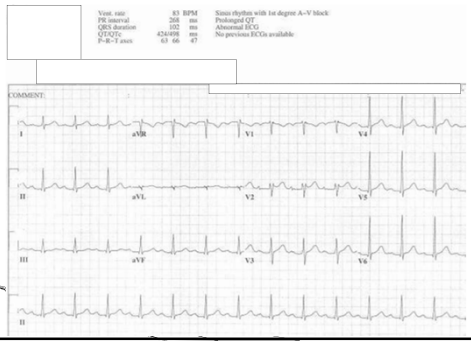
Discharge diagnosis

- Cardiomyopathy with EF 40 – 45% presumably related to thyrotoxicosis
- ICD implanted

EKG 3 days later

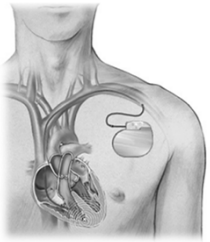


EKG 2 years prior to event

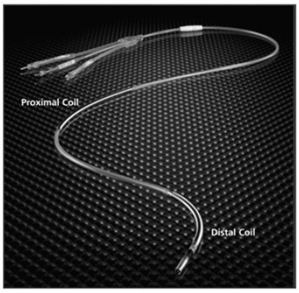


The ICD Today

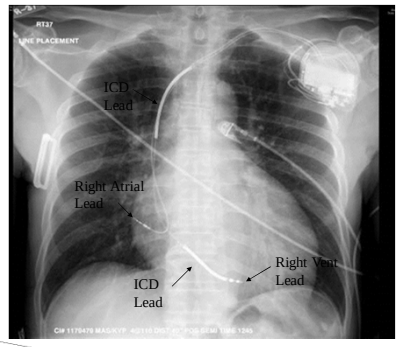
- Longevity approximately 5 years
- Implant similar to pacemaker procedure
- Implant: conscious sedation with additional testing



Defibrillation Leads



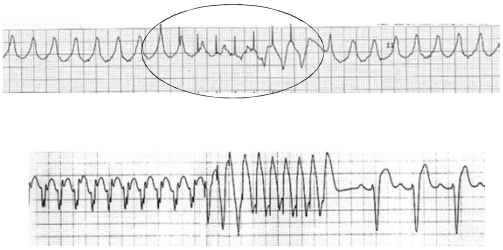
DDD pacer and ICD



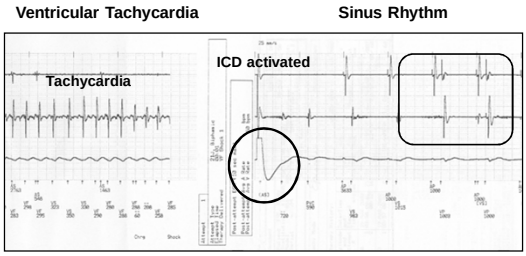
Tiered therapy & ICD's

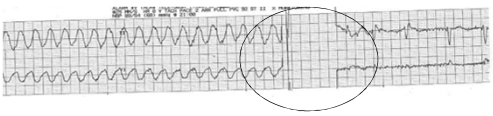
- One of the most important concepts of current ICD's is *tiered therapy*…..where treatment alternatives are programmed into the ICD to specifically meet the needs of the patient.

Antitachycardia pacing (ATP)

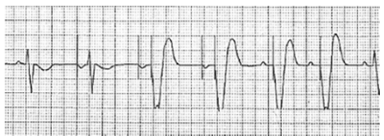


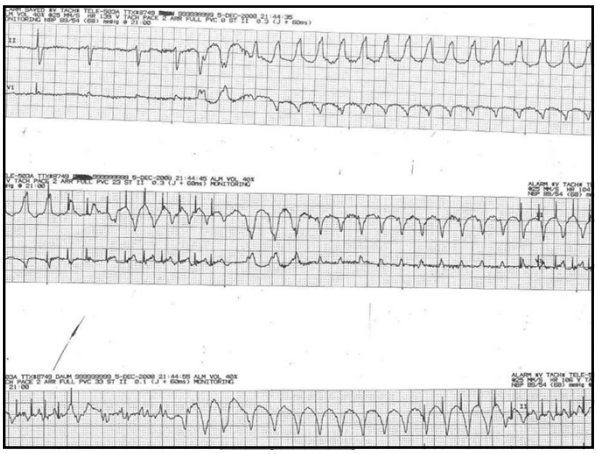
Low energy cardioversion

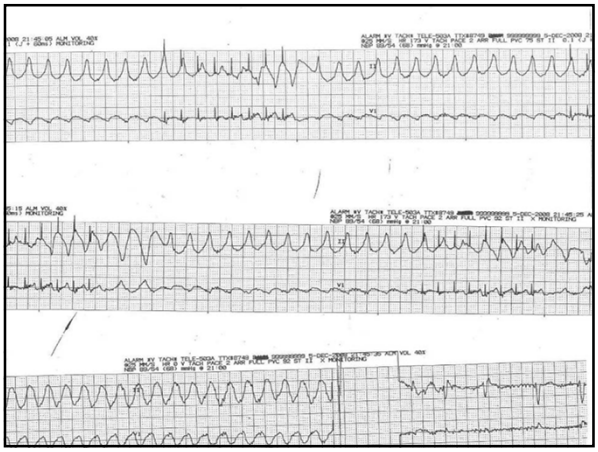




Brady or back-up pacing







Magnet Use

ICD Magnet Use

- Magnet application will NOT affect the Brady pacing function- it will continue as programmed
- ICD magnet placement will disable the sensing function of the device and therefore- no therapy will be delivered
- Once the magnet is removed, Tachy function will be enabled (restored)

Magnet Use for SJM ICDs

- Magnet applied over the ICD
  - Magnet application suspends Tachycardia detection
  - Tachycardia therapy (shocks and ATP) are thereby inhibited
  - No affect on Bradycardia pacing function
  - Removal of the magnet restores Tachycardia therapy

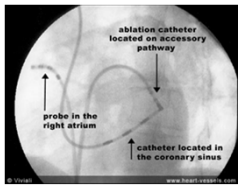
When to use a magnet with ICD's?

- Inappropriate Shocks
  - Use until the company representative can interrogate the device and determine the cause for the inappropriate shocks
- Surgery
  - Patient must be *on monitor* in the OR Suite and Turned back on in Recovery Area
- End stage disease states/hospice care (Physician ordered)

- Cremation – must remove pacer
- Other deaths --- no need to turn off or remove
- Magnet will not turn off– puts in asynchronous mode. For Comfort measures only, call rep to reprogram.

RF Ablation

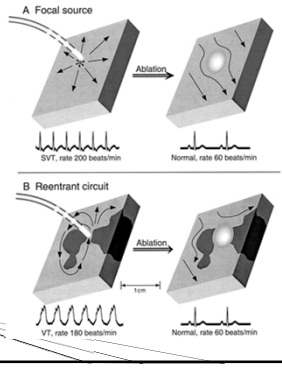
- Controlled lesion created mainly through the effect of local heating.
- Catheter is placed against cardiac tissue and radiofrequency current is applied.
- Within 10 – 30 seconds, a 3– to 5–mm circular area of localized cardiac necrosis is created.
- Thus destroys the arrhythmogenic focus or part of the pathway required for re–entry circuit



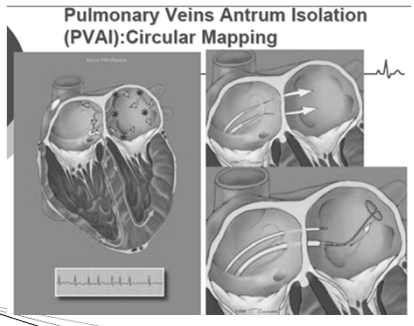
Ablation

Indications	Access
Atrial flutter AVNRT SVT Wolff Parkinson White Ventricular Tachycardia Atrial Fibrillation	Right sided ablations → femoral vein Left sided ablations → femoral artery to aorta & retrograde across the aortic valve to reach site Trans–septal puncture → catheter advanced from right atrium to left atrium via puncture in atrial septum

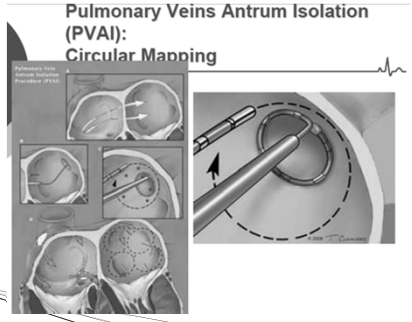
Single foci ablation



Atrial Fibrillation Ablation

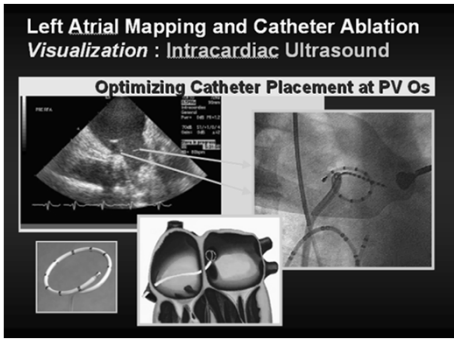


Atrial Aibrillation Ablation



Left Atrial Mapping and Catheter Ablation

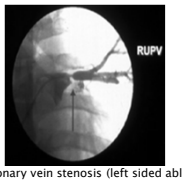
Visualization : Intracardiac Ultrasound



Ablation

Complications

- Cardiac perforation
- Tamponade
- Pneumothorax
- Pulmonary vein stenosis
  - Dyspnea
- Stroke
- Esophageal complications

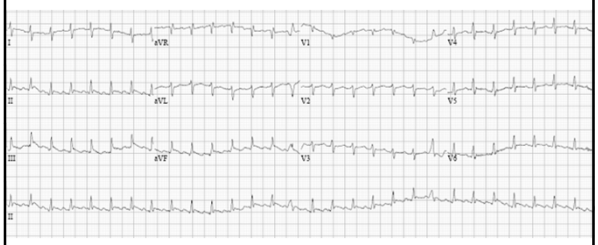


Pulmonary vein stenosis (left sided ablations)

Case Study

- Mr Tachy a 48 y/o is being admitted to progressive care unit from cardiology office after "abnormal echo" per patient
- BP 149/108, HR 170, RR 22, T 97.8 SpO_2 98 on 3 liters/np
- Ht: 6' 3", Wt 182 kg

Interpret the rhythm of this EKG



More History

- Referred out of town for atrial fibrillation ablation. Extensive lengthy procedure.
- EF normal, Dilated Left atrium
- It is now three days since discharge and he is being seen in local cardiology office because he has become progressively SOB

- Denies chest pain
- Winded on minimal exertion
- Had an episode of PND
- Was on amiodarone but dc due to liver enzyme elevation. Now on propafenone
- JVP is elevated
- Some wheezes

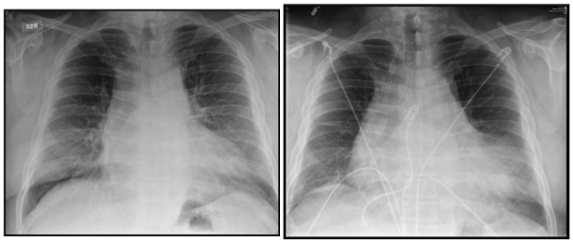
What are some potential causes of his SOB?

What further assessments/ diagnostics would you like?

Medications

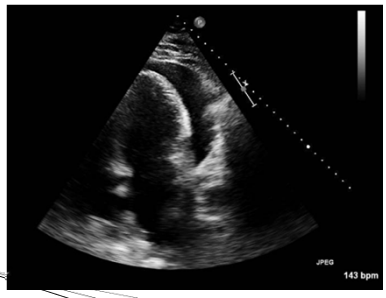
- Prilosec
- Propafenone
- Coumadin
- Atenolol
- ASA
- Lasix
- Tricor
- Potassium

- Three months
- Today



What are your concerns?

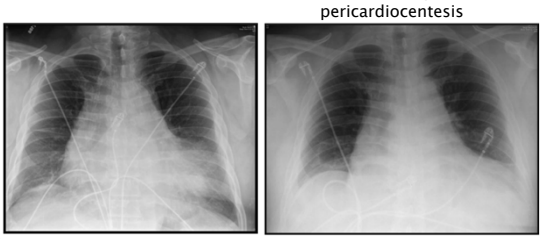
What are your concerns?
What treatment should occur?



- Emergency pericardiocentensis performed
- 850 mg of fluid drained and symptoms much improved.
- Echo two days later showed a large pericardial effusion loculated posteriorly.
- Drain is considered occluded (? Drained 4000ml)
- Patient condition is not improving

BP 138/78 HR 166, RR 30 SpO2 95 on 100% nonrebreather. Pt becoming more SOB. What does he need?

- CXR on admission
- CXR three days after pericardiocentesis



- Pericardial Window surgery done
- 600 ml fluid removed
- Drain (CT) left in
- Sent home 4 days later in stable/good condition.

Case Study

Mr Knot M Day

54 y/o at home 1 week post drug eluting stent for STEMI (total occlusion of LAD)

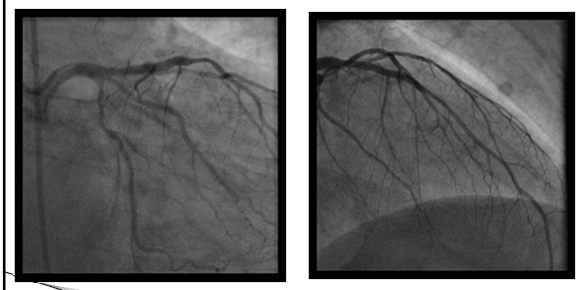
- Home meds
  - ASA, Effient, Beta Blocker, Statin, ACE I
- EF 35%
- Was doing well… walked 2 ½ blocks to gas station to get newspaper
- After dinner, went to recliner.
- Wife heard him sneeze followed by gasping breaths
- Passed out, unresponsive, slumped in chair
- Wife calls 911 and attempts compressions
  - Mr Day is 6'2'' 240#, Mrs Day is 5'4'' 135# and Mr Day is slumped in the chair

Local Fire Department arrives within 5 – 10 minutes

- CPR initiated for two minutes
- AED applied, analyzed
- Shock x 1
- ROSC after one shock
- Unresponsive
- Intubated

EKG in ED
Would you go to Cath Lab?

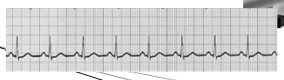
LAD & circumflex: No occlusive disease
LAD stent patent



RCA -- no occlusive disease

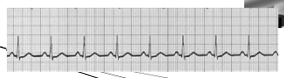
Now what?

- 54 year old
- Back in Sinus Rhythm
- *Unresponsive*



Let's Cool
Down time < 10 minutes

- 54 year old
- Back in Sinus Rhythm
- *Unresponsive*



How Cool!
Therapeutic Hypothermia After Cardiac Arrest

Chery Herrmann
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APN, CCRN, CCNS-CSC-CMC

Therapeutic hypothermia post cardiac arrest

Pathophysiology

- Brain loses oxygen stores within 20 seconds
- Damage starts 4-6 minutes after the heart stops
  - Glucose and adenosine triphosphate stores deplete (brain energy)
  - Membrane depolarization
  - Calcium influxes
  - Glutamine is released
  - Acidosis and edema develop
- Ischemia may persist for several hours after resuscitation (re-perfusion injury)

Anoxic Encephalopathy: Sudden Cardiac Arrest

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Anoxic Encephalopathy

- Global ischemia: no perfusion to brain
  - Sudden Cardiac Arrest (SCA) is an example
- Focal ischemia: lack of perfusion to one area of brain
  - Stroke is example

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Reperfusion Injury

- Occurs after blood flow is restored
- Secondary wave of excitotoxicity and free radical formation
- May exacerbate initial effects of blood deprivation
- Leads to blood brain barrier breakdown, cerebral edema and hemorrhage

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Pathophysiology of Fever

- Rise in temperature due to regulated increase in patient's hypothalamic set point
  - ↑ oxygen consumption
  - ↑ metabolic rate
  - ↑ heart rate
  - ↑ cardiac output
  - ↑ leukocyte count
  - ↑ level of C-Reactive Protein
- May be detrimental to a critically ill patient

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Neuronal Damage from Ischemia

- Negative cascade of reactions occur at cellular level
  - May continue for hours to days after initial insult
- Temperature dependent
  - Increased by fever
  - Inhibited by mild hypothermia
- Terminology:
  - "Secondary injury" in TBI patients
  - "Reperfusion injury" or "Post-resuscitation disease" in SCA or Stroke patients

Polderman, Lancet, 2009

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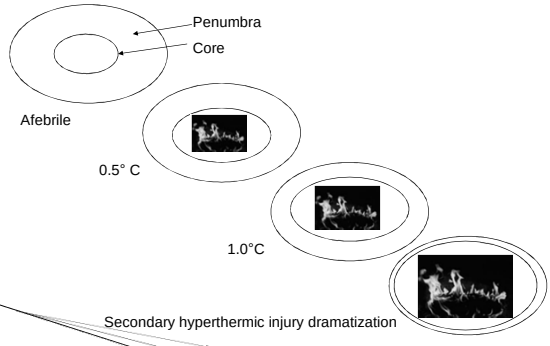
Temp elevations aggravate ischemic neuronal injury and exacerbate brain edema



Adapted from Ginsberg, 2002

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Hypothermia

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2015 AHA Guidelines Post cardiac arrest care

- Recommend induced hypothermia for comatose (lack of meaningful response to verbal commands) adult patients with ROSC after cardiac arrest
  - Should be cooled 32^0 – 36^0 C for at least 24 hour

Inclusion Criteria

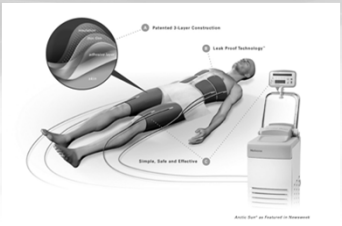
Inclusion Criteria: (all must apply)
___ Cardiac arrest with return of spontaneous circulation (initial rhythm: VF, VT, PEA, or aystole)
___ Men and women age 18 years or older. Women of childbearing age must have a negative pregnancy test (must be documented on the chart).
Unresponsive after return of spontaneous circulation (ROSC) and
___ <6 hours since ROSC
___ ROSC within 60 minutes of collapse
___ GCS ≤5
___ Coma after ROSC (Not following commands and no purposeful movement to noxious stimuli)
___ Patient is a full code
___ Blood pressure can be maintained at least 90 mm Hg systolic either spontaneously

Exclusion Criteria

Exclusion Criteria: (if any of the following is checked, cooling is contraindicated)
___Another reason to be comatose (e.g. drug overdose, head trauma, stroke, overt status epilepticus)
___ Temperature of <30° after cardiac arrest
___ Known, pre-existing coagulopathy or bleeding
*anticoagulation/TPA are not contraindicated
___ Patient is listed as do not resuscitate (DNR) or do not intubate (DNI) code status and patient not intubated as part of resuscitation efforts
___ Refractory shock: SBP <90 mm Hg despite fluids and pressors.
___ Refractory ventricular arrhythmia: VF, VT, Torsades
___ End stage terminal illness (pre-arrest life expectancy <6 months).
___ No limit on duration of resuscitation effort; however, "downtime" of less than 30 minutes most desirable.
___ GCS >5 and following commands.
___ Known respiratory arrest versus cardiac arrest

Medivance

ArcticGel™ Pads
Cover 40% of Body Surface Area (BSA)



Committed to Restoring Life

The goal of Targeted Temperature Management™ in the neurologically impaired patient is neuroprotection

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Hypothermia

- Definitions of hypothermia:
  - Mild: 33–36°C (91.4– 96.8°F)
  - Moderate: 26°–32°C
  - Deep: 20°–25°C
  - Profound: < 20°C

Hammer, *Clev Clinic J Med*, 2002

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Effects of Hypothermia: Neuroprotection

- Inhibit neurotransmitter release
- Inhibit free radical production
- Reduces tissue oxygen demand
- Decrease cerebral metabolic rate (8% for every 1°C)
- Suppress inflammatory cells and factors
- Preserve blood brain barrier integrity
- ICP is decreased

Sessler, *CCM*, 2009

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Physiological Effects of Therapeutic Hypothermia (TH) 32–35°C

- ***Hematological:***
  - Thrombocytopenia
  - Impaired clotting cascade
  - Impaired platelet function
  - Decreased WBC Count
- ***Gastrointestinal:***
  - Impaired bowel function/motility
  - Mild pancreatitis
  - Increased liver enzymes
- ***Cardiovascular:***
  - Initially HR, CO & BP
    - Then ↓ HR, CO, BP
  - EKG Changes
    - Prolonged PR interval
    - Widening QRS complex
    - Increased QT interval
    - Osborne wave

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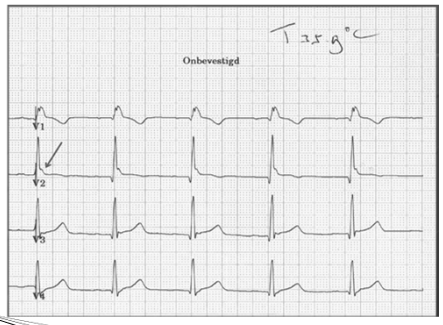
Osborne Wave



Lancet, 2009

Physiological Effects of TH 32-35°C

- ***Systemic:***
  - —↓ O2 Consumption
  - —Left shift on the oxyhemoglobin curve
  - —↓ CO2 production
  - —↑ Lactate levels
- ***Endocrine***
  - —↓ Insulin secretion
- ***Immune suppression:***
  - — ↓Neutrophil and macrophage function
  - —↑Infection (wound infections and pneumonia)
- ***Renal:***
  - —Diuresis
  - —Electrolyte Loss

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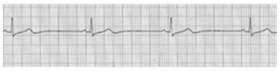
Bradycardia



- Expected – even at rates of 25 – 30 bpm
- No need to treat unless symptomatic
- Atropine probably won't work
- If symptomatic, will need pacer (rare)

Back to Knot M Day

- 10 minutes until good CPR, One shock →ROSC 2/6 1658
- Ice bags 1730 →Cath lab
- Arctic sun 2150 0.5 C/hr; 3 ½ hours later at 0118 on 2/7 reaches target 33^0 C.

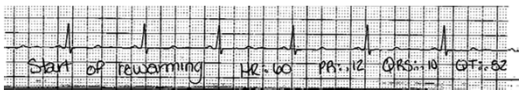
HR 88 bpm at 1900

HR 43 bpm 3 hours after cooled

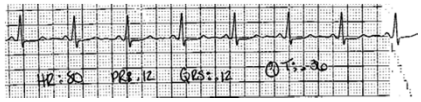
HR 35 bpm 6 hours after cooled

HR 54 bpm 18 hours after cooled

HR 60 bpm Rewarming started



HR 80 bpm 3 hours after rewarming started



HR 93 bpm when rewarmed complete

Potential Complications

- Arrhythmias usually below 30 C
- Electrolyte Abnormalities K+, Mg+, P+, Ca+
- Brady 40-60's No treatment unless evidence of poor perfusion hypotensive or diminished urine output
- Osborne Waves end of QRS complex
- Stop K+ before re-warming
- Insulin Resistance, Increase Serum Amylase

Tips

- Place defibrillator and emergency medications at bedside. Place external defibrillation pads on patient under cooling pads. Keep patient's room cold. Cold room = cold patient
- No warmer on ventilator or dialysis
- Keep ventilator away from head so the temperature from the vent does not warm the patient.
- Skin Care
- Edematous patient
  - Avoid applying pads too tightly
  - Reposition pads as patient swells to avoid irritation at the edges and to provide some "give"
  - Skin integrity may be compromised and more vulnerable to mechanical injury

Other Considerations

Electrolytes

- Hypokalemia: due to intracellular shift and cold diuresis. This will partially correct during re-warming. Do not replace potassium during re-warming.
- Check calcium and magnesium levels.
- Replace potassium per hypothermia protocol, replace magnesium and calcium per standard replacement protocols
- Hyperglycemia occurs during hypothermia

Neurological

- Risk for seizure secondary to medically induced hypothermia or initial arrest risk of brain anoxia.
- Confusion and decreased level of consciousness.
- Depressed reflexes and muscular tone.
- Coma
-

Cardiovascular
- Bradycardia and hypertension during cooling.
- Tachycardia and hypotension during re-warming.
- Both phases may cause arrhythmias.
- Consider discontinuing hypothermia if severe shock, bleeding, or hypoxemia develop.

Gastrointestinal
- Decreased gut motility including ileus, liver function, and insulin release.
- Stress ulcers

Renal
- Decreased urine output, renal plasma flow.
- Decreased ADH.
- Increased specific gravity
-

Hematologic
- Increased blood viscosity.
- Platelet dysfunction
- PTT results are altered by hypothermia. Inform lab patient is cold.

Respiratory
- Pneumonia
- Atelectasis
- Cough suppression with increased oral secretions.
- Decreased tissue perfusion.
- ABG results are altered by hypothermia. Inform lab patient is cold.

Medication
- Do not administer medications labeled as 'DO NOT REFRIGERATE".
- Decreased drug biotransformation.

SHIVERING

Cardiac Surgery Patients can shiver also!

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Shivering

- Involuntary Sympathetic Response
  - Vasoconstriction
  - Muscle contraction and twitching
  - Shiver to produce heat
- Increased in younger patients and increased muscle mass
- Inhibited by neuromuscular disease, muscle relaxants and decrease in muscle tone
- May increase oxygen consumption by 40-100%

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Bedside Shivering Assessment Scale BSAS

0. – No Shivering

1. – Mild Shivering, localized to neck and/or Chest.

2. – Shivering, neck and /or chest and <2 extremities.

3. – Intermittent generalized shivering involving more than 2 extremities.

Badjatia, *Stroke*, 2008

95

Shivering Management

- ***Demerol***: will decrease shivering threshold
- ***Buspirone***: will potentiate Demerol and Fentanyl
- ***Sedatives*** (i.e. Fentanyl, Versed): may sedate enough to decrease shivering, but will not decrease shivering threshold
- ***Anesthetics*** (i.e. Propofol, Precedex, Pento-barbital): may decrease shivering threshold and sedate

Sessler, TH, 2005

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Shivering Management…cont.

- ***Magnesium***: vasodilitation
- ***Neuromuscular Blockade***: alleviate muscle movement and shivering
- ***Non-Pharmacological***: hand, foot and face warming provides "feeling" of warmth

Sessler, TH, 2005

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Shivering Management

Badjatia et al; *Critical Care Med* 2006

Found that surface counter warming provides beneficial control of shivering and improves metabolic profile in patients undergoing therapeutic temperature modulation.

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Steps for Success: Phases of TH Treatment

- ***Induction Phase***:
  - Initiate quickly
  - Careful monitoring of fluid balance
  - Prevention of hypovolemia and hypotension
  - Tight glucose control
  - Electrolyte management
  - Prevention of infectious complications
  - Adjustment of various medications
  - Adjustment of ventilator settings
  - Prevention of shivering

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Steps for Success: Phases of TH Treatment

- ***Maintenance Phase:***
  - Tightly controlled core temperature with minor fluctuations
  - Prevent and manage potential side effects:
    - Bradycardia
    - Glucose management
    - Wound infections
    - Pulmonary infections (especially pneumonia)
    - Skin breakdown
    - Electrolyte imbalance

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Steps for Success: Phases of TH Treatment

- ***Re-warming Phase:***
  - Slow and controlled re-warming
    - Cardiac Arrest: 0.2-0.5˚C/h
    - TBI: 0.1°C per hour or ICP guided
  - Slower re-warming preserves the benefits of TH
  - Rapid re-warming may lead to:
    - Rapid electrolyte shifts (hyperkalemia)
    - Increased ICP
    - Sudden vasodilitation
    - Transient regional or general imbalances between cerebral blood flow (CBF) and oxygen consumption

Polderman, *ICM*, 2002

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They're Warm…Now What?

- ***Controlled Normothermia (4th Phase of Cooling):***
  - Fever within the first 72 hours after Cardiac Arrest is independently associated with poor outcomes. Badjatia, CCM, 2009
  - "It may be reasonable to conclude that Controlled Normothermia is beneficial for at least 48 hours after 24 hours of therapeutic hypothermia."
  - Badjatia, CCM, 2009
- ***Patience is a Virtue:***
  - Reports of patients awakening up to 72 hours after re-warming is complete.

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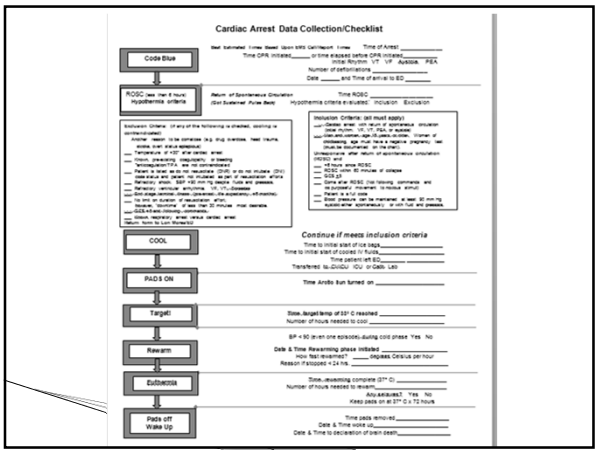
Cardiac Arrest Data Collection/Checklist

Code Blue

ROSC (less than 6 hours: Hypothermia criteria

COOL

PADS ON

Target!

Rewarm

Euthermia

Pads off Wake Up

Continue if meets inclusion criteria

Back to Knot M Day

First 24 hours after waking up (Friday)

- Note on bedside table
- "Your heart stopped beating on Wednesday. Your wife called 911. They shocked your heart. This is the hospital. Today is Friday"
- Walking around the unit 400 feet at a time

Monday morning

- CNS walked by the room and stopped. Pt motioned her in the room and asked what the poster was that she was carrying.

Wednesday Morning (1 week post arrest)

- CNS out of hospital on Tuesday
- Wedneday patient is on the progressive unit.
- Sees the CNS and recognizes her
- Talks about family and his hobby. Shows photos of his hobby.
- ICD inserted on Tuesday. Plan to go home today. Some short term memory loss --- forgets to keep arm in sling.

Week later

- Brings two plaques to the hospital

Other Dysrhythmias

Atrial Fibrillation

- Leading risk factor for stroke
  - direct cause of 1 in 4 strokes.
- Up to 80 percent of strokes in people with Afib can be prevented.

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Assessment of Thromboembolic Risk Factors

CHA2DS2-VASC Score		
Letter	**Clinical Characteristic**	**Points**
C	Cardiac Failure	1
H	Hypertension	1
A2	Age ≥ 75	2
D	Diabetes Mellitus	1
S2	Stroke/TIA/Thromboembolism	2
V	Vascular Disease (MI, PAD)	1
A	Age 65-74	1
Sex	Category (female sex)	1

0 Points = low risk
1 Point = intermediate risk
2 or more points = high risk and needs anticoagulation

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Treatment Atrial Fibrillation

MEDS
Anticoagulation
Anti-arrhythmics

Cardizem (Ca+ Channel Blocker)
Amiodarone

*****Synchronized Cardioversion 120-200 J**