# Adult Care

<table>
<thead>
<tr>
<th>Inside</th>
<th>page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Introduction</td>
<td>2</td>
</tr>
<tr>
<td>Overview of Emergency Cardiovascular Care 2011</td>
<td>3</td>
</tr>
</tbody>
</table>

## 2011 Adult BLS & ACLS Algorithms

- Basic Life Support for Healthcare Providers 5
- Universal Cardiac Arrest Algorithm 7
- Ventricular Fibrillation/VT Arrest 8
- Pulseless Electrical Activity 9
- Asystole 10
- Maternal Arrest 11
- Post-Cardiac Arrest Care 13
- Unstable Bradycardia 15
- Adult Tachycardia 16
- Atrial Fibrillation / Atrial Flutter 17
- Acute Coronary Syndromes 19
- Shock Associated with Acute MI 21
- Stroke 23

## Rapid Reference

- Electrical Therapies 25
- Adult ACLS Medications 27
- Therapeutic Hypothermia Overview 28
- Abbreviation Dictionary 29
- References 31

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On October 18, 2010 the International Liaison Committee On Resuscitation (ILCOR) released a major 5 year update to the CPR and Emergency Cardiovascular Care (ECC) Guidelines. The American Heart Association (AHA) and the European Resuscitation Council (ERC) in turn released similar interpretations of this release.

These guidelines provide core algorithms to outline key actions and decisions for the immediate care of common cardiovascular emergencies:

- cardiac arrest
- post-cardiac arrest
- hemodynamically unstable bradycardia and tachycardia
- acute coronary syndromes
- stroke

These algorithms are central to advanced cardiac life support and pediatric advanced life support courses.

Considerable material included in the major release documents is not included in these core algorithms. This makes sense. Most health providers focus their attention on the more likely core subset of possible cardiovascular emergencies.

For advanced care health professionals in hospital, though, their required skills encompass a broader scope of practice, a full complement of therapeutic interventions and a complex array of morbidities. Support documents and courses specific to this setting harness added content to supplement the core algorithms. This ‘best fit’ approach builds on the ‘one-size-fits-all’ core algorithms to ensure optimal care and improved patient outcomes.

**ACLS & ECC 2011 Essentials** is an education supplement for healthcare professionals tasked with the emergency cardiac care of adults in hospital. This summary of 2010/2011 emergency cardiovascular care guidelines combines recent resuscitation science, suggested procedures and guiding principles into an organized approach to in-hospital emergency cardiovascular care. Canadian Stroke Strategy guidelines and Canadian Cardiovascular Society atrial fibrillation protocols round out this document. We hope that a solid understanding and long term concept adoption of the latest in hospital emergency cardiovascular care science is enhanced with this supplement.

Much thanks go to the advanced care practitioners who reviewed this document. Their significant investments in time and their many suggestions have added to this document. Despite great efforts placed in the creation of these documents, error free results rarely occur despite several reviews and edits. Please direct any suggestions or questions to ecc2011@skillstat.com.

Both the AHA and the ERC are careful to point out that not all recommendations will apply to all rescuers or all situations. The algorithms included here are not intended to replace established AHA | ERC guidelines or sound medical judgement. Resuscitation science is dynamic, with frequent updates.

Find the official ECC 2010 guidelines, executive summaries and updates online. Links are included below for your convenience.

International Liaison Committee for Resuscitation (http://www.ilcor.org/en/home/)
American Heart Association (http://guidelines.ecc.org)
Canadian Cardiovascular Society Atrial Fibrillation Guidelines (http://www.ccs.ca/consensus_conferences/cc_library_e.aspx)
European Resuscitation Council (http://www.cprguidelines.eu/2010/)
A 36 month period of evidence evaluation by 356 resuscitation experts from 29 countries coordinated through the International Liaison Committee on Resuscitation (ILCOR) culminated with a significant 5-year update release of The 2010 International Consensus on CPR Science with Treatment Recommendations (CoSTR) in October 2010. The American Heart Association (AHA) in turn released the 2010 CPR and Emergency Cardiovascular Care (ECC) Guidelines. The European Resuscitation Council published Guidelines for Resuscitation 2010.

The Heart and Stroke Foundation of Canada (HSFC), a founding member of ILCOR, has co-released the 2010 Guidelines for CPR and ECC. The HSFC “sets the Canadian Guidelines for CPR, defibrillation and other aspects of emergency cardiovascular care in Canada.” These guidelines represent the best current understanding of resuscitation science applied to those imminently at risk for a cardiac arrest, in a cardiac arrest and in the first hours post-arrest.

The Canadian Stroke Strategy is a comprehensive initiative designed to optimize stroke care in Canada. The Canadian Cardiovascular Society released the 2010-2011 Atrial Fibrillation Guidelines incorporating the latest science into practical protocols. Included algorithms and support information for atrial fibrillation and stroke care is based on their work.

Resuscitation care strives to preserve life and restore health while limiting disability. In Canada and the US, over 50 000 people were discharged from hospital in 2009 following a cardiac arrest. Recent data show that 75% were discharged with a favorable neurological outcome. Many more averted a cardiac arrest. In the last ten years, mortality from acute coronary syndromes was reduced by 47% and deaths from stroke reduced by 14%. The adoption of the prior 2005 CPR and ECC Guidelines is associated with increased survival to discharge. When used, resuscitation science works.

Key Highlights of the 2010 CPR and ECC Guidelines

- Change in basic life support sequence of steps from ABC (airway, breathing, chest compression) to CAB (chest compressions, airway, breathing) for adults and pediatric patients (not newborns) to reduce the time to start chest compressions
- The reduced importance of pulse checks for pediatrics and adults; healthcare providers often cannot find a pulse quickly or reliably in those who are hemodynamically compromised; limit pulse checks to no longer than 10 seconds
- Together with an absence of pulse, abnormal ‘gasps’ and/or brief seizure activity may also indicate a cardiac arrest
- Continued strong emphasis on high quality CPR with minimum interruptions in chest compressions
- Emphasis to limit interruptions in chest compressions before defibrillations to no longer than 5 seconds (chest compression interruption of even 5-10 seconds before defibrillation is associated with reduced success); chest compressions should continue while monitor-defibrillator is charging
- Use of waveform capnography (end tidal carbon dioxide – $P_{ET}CO_2$) to continuously monitor tracheal tube placement, to assess the quality of CPR, and indicate the return of spontaneous circulation
- Continued emphasis on deferring early tracheal intubation unless done by highly skilled practitioners with interruption of chest compressions not to exceed 10 seconds; alternatives include advanced supraglottic airways (i.e. laryngeal mask airway, King Laryngeal Tube) or the use of an oropharyngeal airway with a bag-valve-mask
- Safety of using cricoid pressure routinely during resuscitation is questioned; do not use cricoid pressure if it impedes ventilation or negatively impacts the speed/ease of intubation
- The delivery of medications via the endotracheal tube is no longer recommended
- Strong emphasis on coordinated post-cardiac arrest care with the inclusion of a comprehensive post resuscitation protocol
Continued emphasis on effective resuscitation team dynamics and team leadership

- There is little evidence to support or refute the routine application of supplemental oxygen for ACS in the absence of hypoxemia; there is evidence that suggests hyperoxemia may lead to harmful effects; encourage the maintenance of oxygen saturation (SpO₂) to 94-98% in all patients not in cardiac arrest; (note that newborns are particularly at risk for harm due to hyperoxaemia; assess a baby’s need for oxygen with pulse oximetry attached to the right upper extremity; for babies born at term, begin resuscitation at room air)

- New recommendations for first line medications in tachycardias and atrial fibrillation/flutter

- Several initiatives outlined to reduce time to effective acute coronary syndromes (ACS) treatment

- Routine use of glycoprotein IIb/IIIa inhibitors is no longer recommended in the treatment of ACS

- IV beta blockers should be only used selectively in the pre-hospital and emergency department settings

- Increased timeline for use of fibrinolytics in stroke from 3 to 4.5 hours for selected patients

The 2010 Guidelines for CPR and ECC reinforce the critical time constraints before, during and after a cardiac arrest. The hemodynamically unstable patient can progress to full cardiac arrest in seconds to minutes. For the arrested patient, seconds determine success. Consider the following:

- For every minute into a cardiac arrest, opportunity for successful resuscitation is reduced by about 10% - 1% for every 6 seconds.

- Brain damage can occur after only 3 minutes into a cardiac arrest

- Coronary perfusion reaches 30% of normal after about 9 seconds of quality CPR and falls to ineffective levels after only a 2-3 second interruption

- Odds for a successful defibrillation diminish after interruptions in compressions of more than 5 seconds.

Time-sensitive interventions are key.

To help ensure a rapid effective response, summary algorithms are provided to highlight relevant concepts and actions of the most likely cardiovascular emergencies facing in-hospital health care providers. Quality of performance of the team leader and the team members in providing timely, effective care is a major determinant in a successful outcome. Remaining current in resuscitation knowledge and skills helps to ensure this level of performance.

This booklet includes essential knowledge presented in algorithms for the resuscitation of adults. Note the adult universal cardiac arrest algorithm of the AHA Guidelines is expanded to three algorithms for clarification and further direction: Ventricular Fibrillation / Ventricular Tachycardia Arrest, Pulseless Electrical Activity (PEA) and Asystole. Direction is also expanded to include emergency atrial fibrillation/flutter management.

Core principles for every algorithm are included to provide quick reference and draw attention to time-sensitive actions that optimize successful outcomes. Rapid reference sheets for electrical therapy, the delivery of ACLS medications, an induced therapeutic hypothermia overview, references and an abbreviation dictionary round out this package.

This document is freely available to be downloaded and copied for learning and teaching. Any changes to this document, alternative packaging or its inclusion into commercial products require the written permission of the authors.

The past six months has seen the release of guidelines that likely represents the best ECC science in 50 years. We hope that this booklet will help hospital-based healthcare professionals learn, adopt and share these guidelines to the ultimate benefit of their patients.
**Quality CPR**
- After assessing no pulse or unsure begin with compressions then open airway and give 2 breaths (CAB)
- Push **Hard** (5-6 cm), **Fast** (100-120/min) & allow for **Full Recoil** on horizontal hard surface
- Compression interruption < 10 sec
- With 2 person CPR but without advanced airway, deliver 30:2 compressions to ventilations – change compressor every 5 cycles
- With 2 person CPR with an advanced airway, one rescuer provides continuous compressions while the second rescuer delivers breaths once every 6-8 seconds; change compressor every 2 minutes

**Core Principles**
- Minimize time to first shock
- Maximize time on chest (CPR)
- Deliver quality CPR
- Do not over ventilate – rate or volume

**Electrical Therapy**
- Automated external defibrillator (AED) should be applied as soon as available
- Adult pads (8-12 cm diameter)
<table>
<thead>
<tr>
<th>Healthcare Provider CPR Skills Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Recognition</strong></td>
</tr>
<tr>
<td>Adult: Adolescent and Older</td>
</tr>
<tr>
<td>Unresponsive</td>
</tr>
<tr>
<td>CPR Sequence</td>
</tr>
<tr>
<td>Compressions Landmark</td>
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<tr>
<td>Compression Rate</td>
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<tr>
<td>Compression Depth</td>
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<tr>
<td>Chest Wall Recoil</td>
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<tr>
<td>Airway</td>
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<tr>
<td>Compression to Ventilation Ratio</td>
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<tr>
<td>(without advanced airway)</td>
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<tr>
<td>Rescue Breaths</td>
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<td>Rescue Breaths with advanced airway</td>
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<td></td>
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<tr>
<td>FBAO - Responsive</td>
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<tr>
<td>FBAO - Unresponsive</td>
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<td>AED</td>
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</tr>
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</table>

Abbreviations: AED, automated external defibrillator; CPR, cardiopulmonary resuscitation; FBAO, foreign body airway obstruction

Note: ERC and Red Cross recommendation for 'FBAO responsive' is 5 back blows alternating with 5 abdominal thrusts.
Universal Cardiac Arrest Algorithm

Cardiac Arrest
Unresponsive
No Pulse (or unsure)
Not breathing (or only gasping)

Activate Emergency Response
Begin Quality CPR
Attach Monitor-Defibrillator

Heart Rate
Too Fast or Too Slow?

YES
Treat Heart Rate
defibrillate or TCP
epinephrine

NO
Quality CPR
Advanced Airway?
Check Rhythm q2 min

NO
Treat Reversible Causes
PEA (4Hs / 4Ts)

YES
ROSC

Post-Cardiac Arrest
Algorithm

Quality CPR

- Push Hard (5-6 cm), Fast (100'/min) & allow for Full Recoil on horizontal hard surface
- Compression interruption < 10 sec
- Without advanced airway, 30:2 compressions to ventilations
- Change compressor every 2 min
- With advanced airway, waveform capnography can assess CPR quality - goal \( P_{ETCO_2} > 10 \text{mmHg} \)

Electrical Therapy

- Biphasic at default energy of 120J-200J; consider escalating energy
- Monophasic or unknown – deliver max Joules
- Transcutaneous pacing (TCP) is an option for severe bradycardias and witnessed asystole

Core Principles

- Minimize time to first shock or to other definitive treatment
- Maximize time on chest (CPR)
- Deliver quality CPR
- Do not over ventilate (rate or volume)
- Too Fast is > 200-age/min i.e. VF/VT
- Too Slow is typically < 40/min

Advanced Airway

- Continuous CPR with supraglottic advanced airway or ETT tube and breaths once every 6-8 seconds
- Waveform capnography to confirm advanced airway placement

Return of Spontaneous Circulation (ROSC)

- Sustained breathing
- Skeletal muscle movement
- Pulse & BP; \( P_{ETCO_2} > 35 \text{mm Hg} \)

Reversible Causes (4 Hs & 4 Ts)

- Hypovolemia
- Hypoxia
- Hyper/Hypo K+ / H+ (acidosis)
- Hypothermia
- Tension pneumothorax
- Tamponade, Cardiac
- Toxins
- Thrombosis – PE / MI

ACLS Medications (IV/O)

- Epinephrine (Epi) 1 mg q 3-5 min - 1st dose given promptly for PEA & for lethal bradycardias or after 2nd shock for lethal tachycardias
- Vasopressin 40 u can replace first or second dose of Epinephrine
- Amiodarone 300mg given for lethal tachycardias; 1st dose after 3rd shock; optional 2nd dose 150 mg

For educational purposes only
Ventricular Fibrillation / Ventricular Tachycardia Arrest

Activate Emergency Response
Begin Quality CPR
Attach Monitor-Defibrillator

VF / VT?

Shock CPR for 2 min

VF / VT?

Yes

No

ROSC

PEA / Asystole Algorithms

Post-Cardiac Arrest Algorithm

VF / VT?

Yes

No

Shock CPR for 2 min
Epinephrine 1mg IV/IO

Amiodarone 300mg IV/IO

Treat Reversible Causes
Consider Advanced Airway
Repeat Epinephrine q3-5 min
Repeat Amiodarone once (150 mg)

Core Principles
- Minimize time to first shock
- Maximize time on chest (CPR)
- Deliver quality CPR
- Do not over ventilate (rate or volume)

Advanced Airway
- Continuous CPR with supraglottic advanced airway or ETT tube and breaths once every 6-8 seconds
- Waveform capnography to confirm advanced airway placement

Return of Spontaneous Circulation (ROSC)
- Sustained breathing
- Skeletal muscle movement
- Pulse & BP
- PETCO2 > 35 mm Hg

Reversible Causes
- Hypovolemia
- Hypoxia
- Hyper/Hypo K+ / H+ (acidosis)
- Hypothermia
- Tension pneumothorax
- Tamponade, Cardiac
- Toxins
- Thrombosis – PE / MI

ACLs Medications (IV/IO)
- In VF/VT, no medications are proven to improve survivability to discharge; consider arrest context pre-admin.
- Epinephrine 1 mg IV push (IVP) q3-5 min - 1st dose after 2nd shock
- Vasopressin 40 u IVP; can replace first or second dose of Epinephrine
- Amiodarone 300mg IVP after 3rd shock; optional 2nd dose 150 mg IVP

Quality CPR
- Push Hard (5-6 cm), Fast (100+/min) & allow for Full Recoil on horizontal hard surface
- Compression interruption < 10 sec
- Without advanced airway, 30:2 compressions to ventilations
- Change compressor every 2 min
- With advanced airway, waveform capnography can assess CPR quality - goal PETCO2 > 10mmHg

Electrical Therapy
- Biphasic at default energy of 120J-200J; consider escalating energy
- Monophasic or unknown – deliver max Joules

For educational purposes only
Activate Emergency Response
Begin Quality CPR x 2 min
Attach Monitor-Defibrillator

IV/IO Access x 2 (large bore)
Consider Epinephrine 1 mg
Advanced Airway

Rapid Identification and Treatment of most likely Cause

- Hypovolemia
- Hypoxia
- Hyper/Hypo K+ / H+ (acidosis)
- Hypothermia
- Tension pneumothorax
- Tamponade, Cardiac
- Toxins
- Thrombosis - PE / MI

Resume CPR
Epi every 3-5 min
(if cardiac rhythm changes to VF/VT or Asystole, proceed to appropriate algorithm)

ROSC

Post-Cardiac Arrest Algorithm

Quality CPR
- Push Hard (5-6 cm), Fast (100+/min)
  & Allow Full Recoil on horizontal hard surface
- Minimal compression interruption < 10 sec
- Without advanced airway, 30:2 compressions to ventilations
- Change compressor every 2 min
- With advanced airway, waveform capnography can assess CPR quality - goal \( P_{ET}CO_2 > 10 \) mmHg
- Do not over ventilate (rate or volume)

ACLS Medications (IV/IO)
- Epinephrine (Epi) 1 mg q 3-5 min
- Vasopressin 40 u can replace 1st or 2nd dose of Epinephrine

Return of Spontaneous Circulation (ROSC)
- Sustained breathing
- Skeletal muscle movement
- Pulse & BP
- \( P_{ET}CO_2 > 35 \) mmHg

Core Principles
- PEA combines an organized ECG rhythm with no cardiac output; treat early and quickly
- Causes of PEA include the 4Hs and 4Ts; other less common causes are possible i.e. anaphylaxis, septic shock, cardiac valve disease, and a cascade of events involving 2 or more causes
- Investigations must provide near immediate results to be of value i.e. FAST echocardiography
- With evidence of heart wall motion and/or narrow QRS complex: exhaust all treatable causes
- A focused head to toe physical exam is crucial. Within context, look for: jugular venous distention, engorged facial vasculature, skin color changes, tracheal deviation, air entry, asymmetrical chest wall motion, abdominal distention, shunts, medical alert items, and needle marks
- Treat any extreme tachy/brady arrhythmias if suspected of contributing to low cardiac output (exception: sinus tachycardia)
- If cause not obvious, treat for best guess i.e. volume challenge, pericardiocentesis, needle decompression...
- Many traditional treatment contraindications do not apply in the unique setting of PEA
**Asystole**

Activate Emergency Response  
Begin CPR for 2 min  
Attach Monitor-Defibrillator

IV/IO Access  
Consider Epinephrine 1 mg  
Consider advanced airway

**Rapid Identification and Treatment of Most Likely Cause**

**History, Physical Exam & Investigations**

- Hypovolemia
- Hypoxia
- Hyper/Hypo K+ / H+ (acidosis)
- Hypothermia
- Tension pneumo
- Tamponade, Cardiac
- Toxins
- Thrombosis - PE / MI

**CPR 2 min**  
Epi every 3-5 min  
(if an organized cardiac rhythm or VF/VT, proceed to appropriate algorithm)

**ROSC**

- No

**Post-Cardiac Arrest Algorithm**

**Quality CPR**

- Push **Hard** (5-6 cm), **Fast** (100+’s/min)  
  & **Allow Full Recoil** on flat hard surface  
- Minimal compression interruption < 10 sec  
- Without advanced airway, 30:2 compression : ventilation ratio  
- Change compressor every 2 min  
- With advanced airway, waveform capnography can assess CPR quality - goal P_{ET}CO2 > 10 mmHg  
- Do not over ventilate (rate or volume)

**ACLS Medications (IV/IO)**

- Epinephrine (Epi) 1 mg every 3-5 min  
- Vasopressin 40 u can replace 1st or 2nd dose of Epinephrine

**Core Principles**

- Asystole: absence of ventricular activity (p waves may still be present) confirmed by ensuring electrode leads are attached correctly  
- Severe vagal reflex is a temporary cause of asystole i.e. with blow to eye or solar plexus  
- If asystole is witnessed - patient was just in a perfusing rhythm - or if P waves are present, consider transcutaneous pacing (TCP)  
- Survival to discharge of in-hospital asystole (11%) is 10 times that of pre-hospital asystole (<1%)  
- If in doubt whether asystole or fine VF, treat as asystole (fine VF unlikely to be successfully shocked)

**Return of Spontaneous Circulation (ROSC)**

- Sustained breathing / skeletal muscle movement  
- Pulse & BP and/or P_{ET}CO2 > 35 mmHg
**Maternal Cardiac Arrest**

- Activate Emergency Response
- Activate Obstetrical/Neonatal team if available

**Begin Quality CPR**
*Place hands slightly higher on sternum than normal*
- Attach Monitor-Defibrillator

**For Gravid Uterus**
- Left lateral (pts left) uterine displacement (LLUD) with keeping the patient supine
- If LLUD unsuccessful may try left lateral tilt using a backboard at angle of 30°
- Remove all fetal monitoring equipment

**Anticipate a difficult airway**
- High risk of regurgitation and aspiration
- Consider early placement of advanced airway by experienced practitioner

**IV/IO access above level of diaphragm**
- Use standard arrest algorithms
- Do not delay defibrillation
- Use standard ACLS drugs and dosages

- Assess for hypovolemia, give fluid boluses
- If IV/IO magnesium used prearrest stop infusion, give calcium chloride or calcium gluconate

**Core Principles**
- Saving life of mother takes priority over fetal survival
- The key to resuscitation of infant is resuscitation of mother
- Viable fetus from 24-25 weeks
- Gestational age > 20-23 weeks, key to resuscitation of mother is removal of fetus from gravid uterus
- Gestational age > 24-25 weeks emergency C-section to save life of mother and fetus
- Organize for stat C-section immediately at beginning of arrest

**Quality CPR**
- Push Hard (5-6 cm), Fast (100'/min) & allow for Full Recoil
- Compression interruption < 10 sec
- Without advanced airway, 30:2 compressions to ventilations
- Change compressor every 2 min
- With advanced airway, waveform capnography can assess CPR quality - goal P_{ETCO2} > 10mmHg
- Do not over ventilate (rate or volume)

**Medications (IV/IO)**
- Calcium Chloride 10 ml of 10% solution
- Calcium Gluconate 30 ml of 10% solution

**Reversible Causes**
- Bleeding/DIC
- Embolism: coronary, pulmonary, amniotic
- Anesthetic complications
- Uterine Atony
- Cardiac disease: MI, Ischemia, aortic dissection, cardiomyopathy
- Hypertension: preeclampsia, eclampsia
- Other: standard H’s and T’s
- Placenta abruptio/previa
- Sepsis

**No ROSC at 4 minutes consider starting emergency cesarean**

**Delivery of fetus at 5 min**

**Continue resuscitation until all interventions have been undertaken**
Adult Immediate Post-Cardiac Arrest Care

Manage Airway and Breathing
- maintain oxygen saturation 94-98%
- chest x-ray
- consider advanced airway & waveform capnography
- ventilate 10-12/minute; titrate to $P_{ET}CO_2$ 35-45 mm Hg

Manage Hemodynamics
- frequent BP monitoring/arterial line;
- continuous ECG monitoring; 12 lead ECG/troponin levels
- avoid prophylactic antiarrhythmics
- treat hypotension; IV/IO bolus; vasopressor infusion; lactate levels
- treat STEMI/suspected AMI with emergent reperfusion

Minimize Neurological Injury
- monitor core temperature
- do not rewarm if temperature > 32°C
- perform serial neurological exams; if unable to follow commands after 10 minutes, induce therapeutic hypothermia if not contraindicated
- continuous EEG monitoring if comatose to detect and treat seizure activity

Optimize Metabolic Status
- monitor and manage serum glucose
- monitor and manage serum electrolytes
- monitor urine output; creatinine levels

Ventilation/Oxygenation
- Avoid excessive ventilation
- Begin at 10-12 breaths/minute – titrate to $P_{ET}CO_2$ 35-40 mm Hg
- Adjust inspired oxygen to minimum required to keep oxygen saturations 94-98%

Hemodynamic Support
- Fluids: normal saline or lactated Ringer’s 1-2 L (4°C fluid if inducing hypothermia)
- Norepinephrine IV Infusion: 2-10 mcg per minute
- Dopamine IV Infusion: 5-10 mcg/kg per minute
- Epinephrine IV Infusion: 2-10 mcg per minute

Core Principles
- Optimize cardiopulmonary function and the perfusion of vital organs
- Transport to advanced critical care unit capable of specialized post-cardiac arrest interventions
- Identify and treat causes of arrest / prevent re-arrest
- Prevent hyperthermia and consider induction of hypothermia to optimize survivability / neurological recovery
- Identify and treat acute coronary syndromes (and suspected acute myocardial infarction (AMI))
- Optimize ventilation and oxygenation without hyperventilation

Treatable Causes
- Hypovolemia
- Hypoxia
- Hyper/Hypo K+ / H+ (acidosis)
- Hypothermia
- Tension pneumothorax
- Tamponade, Cardiac
- Toxins
- Thrombosis – PE / MI

Return of Spontaneous Circulation (ROSC)
- Sustained breathing
- Skeletal muscle movement
- Pulse & BP
- $P_{ET}CO_2 > 35$ mm Hg
For those who survive a cardiac arrest for more than 20 minutes, only a third will ultimately survive to discharge from hospital. Prolonged whole-body ischemia during cardiac arrest triggers a cascade of pathophysiological processes that persist after return of spontaneous circulation. The pathologies of this post-cardiac arrest syndrome (PCAS) include:

1. Post-cardiac arrest brain injury
2. Post-cardiac arrest myocardial dysfunction
3. Systemic ischemia/reperfusion response
4. Persistent precipitating cause of cardiac arrest

A time-sensitive, multiple system approach to post-cardiac arrest care positively impacts survivability to discharge and neurological outcomes.

**Therapeutic Strategies**

- **Transfer to intensive care unit** that specializes in this comprehensive clinical pathway
- General intensive care monitoring, advanced hemodynamic monitoring and cerebral monitoring
- Early hemodynamic and circulatory optimization (fluid bolus, inotropes, vasopressors and blood transusions and possible mechanical circulatory assistance devices if required)
  - Central venous pressure of 8-12 mm Hg
  - Mean arterial pressure of 65-90 mm Hg
  - Hematocrit > 30%
  - Hemoglobin > 80 g/L
  - Urine output at least 0.5ml/kg per hour
  - Lactate levels 2 mmol/l or less
- Oxygenation and Ventilation
  - Immediate adjustment of oxygen delivery post-arrest to produce arterial oxygen saturations of 94-98%
  - Intubation and mechanical ventilation for those requiring therapeutic hypothermia – caution against hyperventilation – titrate to \( P_{\text{ET}} \text{CO}_2 \) of 35-40 mm Hg or \( \text{PaCO}_2 \) of 40-45 mm Hg
- Management of Acute Coronary Syndromes
  - **Early primary percutaneous coronary intervention** (PCI) with ST elevation myocardial infarction (STEMI) or suspected acute MI
  - Use of fibrinolytics if PCI not readily available for STEMI
  - Use of PCI or fibrinolytics can (and should) be concurrent with therapeutic hypothermia efforts if warranted
- Treat the precipitating cause of the cardiac arrest – cardiac, electrolyte, toxicological, pulmonary and neurological
- **Therapeutic Hypothermia**
  - **Therapeutic hypothermia** – induction of core body temperature at 32-34 °C for 12-24 hours beginning minutes to hours after the cardiac arrest - is standard treatment for comatose survivors of a cardiac arrest ;
  - Hypothermia is considered to be neuroprotective; hypothermia decreases: 1) energy utilization; 2) the consumption of oxygen and glucose; 3) cerebral edema; 4) and the release of neurotoxic mediators
- Monitor blood glucose levels and treat blood glucose levels above 8 mmol/L
- Seizure activity is not uncommon post cardiac arrest, causing a 3-fold increase in cerebral metabolic rates

Online Resources:  
1) ilcor.org/data/Post-cardiac_arrest_syndrome.pdf  
2) circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S768
Adult Unstable Bradycardia

If HR is atypical, begin MOVIE & treat underlying cause
- Monitor – continuous ECG, oximetry, blood pressure
- Oxygen - maintain SpO₂ ≥ 94%
- Vital signs - initial full set including glucose
- IV/IO - ensure vascular access
- ECG – 12 lead ECG

Patient Unstable?
- Acute altered level of consciousness
- Hypotension
- Acute heart failure
- Signs of shock
- Ischemic chest discomfort

Give Atropine

Effective?

YES

NO

Consider:
- Transcutaneous Pacing (TCP)
- or Dopamine infusion
- or Epinephrine infusion

Medications (IV/IO)
- Atropine 0.5 mg bolus, repeat q 3-5 min
  Total maximum: 3 mg
- Dopamine infusion: 2-10 mcg/kg per min
- Epinephrine infusion: 2-10 mcg/min
- Isoproterenol infusion: 2-10 mcg/min

Alternatives
- Aminophylline may be effective if the bradycardia is caused by an inferior MI, cardiac transplant or spinal cord injury
- Glucagon if beta-blocker or calcium channel blocker overdose
- Glycopyrrolate can be chosen as an alternative to Atropine

Core Principles
- An atypically slow heart rate (HR) is less than 50/min and markedly slower than usual (may have resting HR less than 50/min)
- In the presence of significant hemodynamic compromise, immediately treat to increase heart rate (HR) while identifying / treating causes
- Bradycardia is caused by several treatable causes: cardiac (i.e. acute coronary syndromes, sick sinus syndrome), and non-cardiac (i.e. hypoxia, vasovagal response, hypothermia, hypoglycemia)

Electrical Therapy
- Transcutaneous Pacing (TCP): initiate immediately if Atropine is ineffective or is unlikely to be effective (Mobitz type II block, complete heart block and cardiac transplant)
- Ensure mechanical capture and SBP>90 before using analgesia and sedation to control pain
Adult Tachycardia (HR>150/minute with a pulse)

If HR is atypical, begin MOVIE & treat underlying cause
- Monitor – continuous ECG, oximetry, blood pressure
- Oxygen - maintain SpO₂ ≥ 94 %, airway, breathe prn
- Vital signs - initial full set including glucose
- IV/IO - ensure vascular access
- ECG – 12 lead ECG

Patient Unstable?
- Hypotension?
- Acute altered level of consciousness
- Signs of shock?
- Ischemic chest discomfort?
- Acute heart failure?

Synchronized Cardioversion
- Consider sedation
- For regular rhythm & narrow QRS complex, consider Adenosine

Regular Rhythm
If stable ventricular tachycardia consider:
- Procainamide
- Amiodarone
- Synchronized Cardioversion

(For regular rhythm and a confirmed SVT with bundle branch block, may consider: Adenosine, Beta Blockers and Calcium Channel Blockers; note that extremely rapid heart rates are increasingly difficult to identify rhythm pattern irregularity; consider expert help)

Irregular Rhythm
- Seek expert help
- If torsades de pointes suspected consider MgSO₄
- May be atrial fibrillation with bundle branch block and WPW (see AF/Fl algorithm)

Narrow QRS? (QRS < 0.12 seconds)

Yes

Regular Rhythm
- Vagal Maneuvers
- Adenosine (watch for atrial flutter – if likely seek expert help)
- Beta-blocker (BB)
- Calcium Channel Blocker (CCB)

Irregular Rhythm
- Probable atrial fibrillation (refer to atrial fibrillation/flutter algorithm)

ACLS Medications (IV/IO)
- Adenosine 6mg IV rapid push; follow with 20 ml NS flush; second dose 12 mg
- Amiodarone 150 mg IV over 10 minutes; repeat if needed; follow with infusion of 1mg/min for 6 hr
- Diltiazem 15-20 mg over 2 min
- Metoprolol 5mg over 1-2 min q5min to max 15mg
- MgSO₄ 1-2 g over 10 min
- Procainamide 20-30 mg/min as an infusion until the rhythm is converted, the QRS is widened by 50%, or total of 17 mg/kg has been given. Do not use if patient has heart failure
- Verapamil 2.5-5 mg IV over 2 minutes; may repeat to a max of 20 mg

Core Principles
- This algorithm does not apply to sinus tachycardia (ST) which is rarely faster than 150/min (for ST treat the cause not the rhythm – i.e. pain, hypovolemia, sepsis, cocaine... )
- In the presence of hemodynamic compromise, immediately treat to slow heart rate while identifying / treating causes
- In general electrical cardioversion is safer than antiarrhythmic drug conversion
- Refer to Electrical Therapies for details on synchronized cardioversion

For educational purposes only
Atrial Fibrillation/Flutter

Begin MOVIE: focused Hx; CHADS₂ score; identify possible causes

- Monitor – continuous ECG, oximetry, blood pressure
- Oxygen - maintain SpO₂ ≥ 94 %
- Vital signs - initial full set including glucose
- IV/IO - ensure vascular access
- ECG – 12 lead ECG

Patient Unstable?

- Hypotension
- Acute altered level of consciousness
- Signs of shock
- Ischemic chest discomfort
- Acute heart failure

Yes → Cardioversion at 120-200J
(if AF is chronic consider rate control to stabilize pt.)

No → Rate control if needed:
- Beta blockers
- Diltiazem
(Digoxin – delayed onset slowing only resting HR)

Avoid conversion
Anticoagulation x 3 wks

Greater than 48 hrs onset?

(≥ 48 hrs with hx of a mechanical valve, rheumatic valve disease, recent TIA/ stroke with non-therapeutic INR)

Yes → Rate control if needed:

No → Rhythm Control

Electrical cardioversion
and/or:
- Procainamide
- Ibutilide
- Propafenone

Rate Control

- Beta blockers
- Diltiazem
(Digoxin – delayed onset slowing only resting HR)

ACLS Medications (IV/IO)

- Procainamide 15-17mg/kg infused over 60 minutes; 60% conversion rate for rapid onset AF/Afl; 5% incidence of hypotension
- Ibutilide: 1-2 mg IV over 10-20 minutes; may pre-treat with MgSO₄ to help reduce incidence of torsades de pointes (2-3% incidence); post conversion monitoring x 4 hrs or until QT interval resolution;
- Propafenone give 450-600 mg PO; monitor for hypotension, bradycardia
- Amiodarone is not recommended for recent onset AF/Afl
- Metoprolol give 2.5-5 mg IV q5min over 2 min max 3 doses; relatively contraindicated in patients with CHF, COPD, asthma and with BP in the low range of normal
- Diltiazem Give 0.25mg/kg IV over 10 min; may repeat with 0.35 mg/kg IV in 15 minutes if first dose is ineffective; decrease dose in elderly patients or those with low BP
- Verapamil 0.075-0.15mg/kg over 2 min; monitor for hypotension and bradycardia

Core Principles

- If doubt as to AF/Afl onset, treat as > 48 hrs.; anticoagulate with Warfarin (INR 2-3) or Dabigatran for 3 weeks before conversion; continue for at least 4 weeks after conversion;
- Risk for ischemic stroke for patients with nonvalvular AF/Afl - persistent or paroxysmal - is 5% annually
- Evidence supports beta blockers as being superior to diltiazem for first line rate control
- If an accessory pathway is suspected (i.e. Wolff-Parkinson-White - irregular wide QRS complexes with HR >240/min), avoid AV nodal drugs (ABCD – adenosine, beta blockers, calcium channel blockers and digoxin); consider electrical cardioversion or antiarrhythmics (Procainamide or Ibutilide)
- Transesophageal echocardiography (TEE) can be used to rule out a embolus in the left atrium
- A CHADS₂ score – CHF, Hypertension, Age, Diabetes, Stroke/TIA - evaluates the risk of stroke


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Acute Coronary Syndromes (ACS)

Signs and Symptoms of ACS³

Assess - Stabilize – MOVIE
- Monitor – continuous ECG, oximetry
- Oxygen – give O₂ if O₂ Saturation < 94 %
- Vital signs - TPR/BP & glucose, blood work, chest x-ray
- IV - ensure vascular access
- ECG – 12 lead ECG

Patient Hx (include symptom duration, allergies, meds) - Physical exam
Aspirin 160-325 mg
Nitroglycerin² if SBP>90 mmHg; Morphine IV if discomfort persists

ECG Interpretation³

ST Elevation MI (STEMI)
ST elevation or new Left Bundle Branch Block
Start Supplemental Therapies⁴
Perform Fibrinolytic Checklist

Symptom Duration?
>12 hr

Candidate for Fibrinolysis & Inappropriate Delay to PCF?
Yes (both conditions)
Fibrinolysis
(Door to needle <30 min)
Consider: Rescue PCI⁶ or delayed PCI

<12 hr

No (to either condition)

Percutaneous Coronary Intervention (PCI)
(Door to Balloon <90 min)

High Risk UA/NSTEMI
Unstable Angina / non-ST Elevation MI
ST depression or dynamic T wave inversion
Consider PCI with:
a. positive troponins or
b. high TIMI risk score or
c. unstable clinical features
(i.e. recurrent or persistent ST deviation, compromised hemodynamic status – poor perfusion, Vtach, heart failure, PCI < 6 months, prior CABG)

Start Supplemental Therapies⁴

Low/Intermediate Risk ACS
Normal or Non-Diagnostic ECG changes
Serial troponin levels & 12 lead ECGs (q6h)
Consider admission &/or non-invasive diagnostics

New UA/NSTEMI sign?
Yes
- unstable clinical features
- dynamic ECG changes
- elevated troponins

Abnormal findings from non-invasive diagnostic test?
Yes
Consider discharge & follow-up

No

Support notes included on reverse page.
1 – **Signs and symptoms of acute coronary syndromes (ACS)** include chest discomfort possibly radiating to the upper body/arms/neck/jaw, shortness of breath, sweating, indigestion, nausea, vomiting and dizziness. Atypical presenters – ACS in the absence of chest discomfort - are more common with women, the elderly and diabetic patients.

2 - **Nitroglycerin**: nitroglycerin (NTG) produces arterial and venous vasodilation. NTG is often effective in the treatment of angina. In contrast, evidence is inconclusive for the routine use of NTG during an acute myocardial infarction (AMI). NTG should be given cautiously for AMI patients if an expected blood pressure drop would prevent the use of proven beneficial medications (i.e. ACE inhibitors and beta-blockers). NTG is contraindicated with:

- patients with systolic BP (SBP) < 90 mm Hg or more than 30 mm Hg below baseline
- bradycardia < 50/minute or tachycardia > 100/minute in the absence of heart failure
- patients with right ventricular infarction (RVI) - caution with inferior MI - approximately 50% of inferior MI are RVI
- recent use of medications for erectile dysfunction - within 24 hours of Sildenafil (Viagra) - 48 hours of Tadalafil (Cialis)

NTG can relieve chest discomfort produced by gastrointestinal causes. The relief of chest discomfort by NTG does not rule out ACS.

3 – **ECG Interpretation**: the first 12 lead ECG may or may not present with immediate findings of a STEMI or NSTEMI. At least two repeat 12 lead ECGs are advised every 15-20 minutes with persistent symptoms typical for cardiac ischemia. Threshold values of ST deviation are provided in the table below for STEMI, UA/NSTEMI, and low to medium risk ACS.

![12 Lead ECG Threshold Values](image)


4 – **Supplemental Therapies**: these include Enoxaparin or heparin, Clopidogrel 75-600 mg according to patient history and strategy chosen (fibrinolytic or PCI), oral beta blockers (IV for select cases), ACE inhibitors or ARBs and statin therapy

5 – **Inappropriate Delay to PCI**: goal for percutaneous coronary intervention balloon inflation is ideally 90 minutes from first medical contact (advanced life support paramedic or emergency department). Additional direction is now available for emergency physicians that potentially shortens or lengthens this timeline based on onset of symptoms, age of patient and whether the MI is anterior or not.

![Acceptable Delay Time for PCI](image)


The figures included are to be interpreted not as an exact science but as general indicators. For example, younger patients presenting with anterior STEMI may well benefit from fibrinolysis if transfer to a PCI is needed – delay is inappropriate. The older patient presenting to the hospital after 2-3 hours of symptoms may well benefit from a transfer to a centre capable of PCI.

6 - **Rescue PCI**: a 12/15 lead ECG is advised 75 minutes after fibrinolytics to evaluate fibrinolytic success. If the ST-elevation deviation has not resolved by 50%, the patient should be transferred for rescue PCI as soon as possible; there is support (possible benefit) for post-fibrinolytic delayed PCI (6 hours or more after fibrinolysis).
Shock Associated with Acute Myocardial Infarction

Arrhythmia Present

If arrhythmia likely to compromise perfusion then tx with appropriate ACLS algorithm

Pulmonary Edema

Yes

Treatment Options:
- Furosemide IV
- Nitroglycerin IV if SBP > 100 mmHg
- Morphine
- O₂ to keep SpO₂ ≥ 94%
- Consider BiPAP or advanced airway
- Dopamine if SBP 70-100 mmHg and s+s of shock
- Dobutamine if SBP 70-100 mmHg and no s+s of shock
- Consider short acting ACE inhibitor such as captopril

Hypovolemia

Administer as needed:
- Fluids
- Blood transfusions
- Cause specific interventions

Cardiogenic

RV Infarct

Yes

- Avoid nitrates, diuretics, and other vasodilators
- Treat hypotension with fluid boluses

No

SBP < 70 mmHg
(s+s of shock)

Norepinephrine

SBP 70 to 100 mmHg
(s+s of shock)

Dopamine

SBP 70-100 mmHg
(no s+s of shock)

Dobutamine

SBP > 100 mmHg

Nitroglycerin

Medications

- Captopril: if SBP > 100 mmHg and not > than 30 mmHg below baseline then consider. Dose 1-6.25 mg
- Dobutamine: 2-20 mcg/kg/min
- Dopamine: 5-15 mcg/kg/min
- Furosemide IV: 0.5-1.0 mg/kg. If no hypovolemia use < 0.5 mg/kg initially
- Oxygen: consider maintaining oxygen saturations between 94-98% as limited evidence that hyperoxaemia is harmful in patients with ACS
- Morphine: 2-4 mg IV prn, do not depress respirations
- Nitroglycerin IV: 10-20 mcg/min
- Norepinephrine: 0.5-30 mcg/min

Core Principles

- It is common practice to start pressors and inotropes through newly placed peripheral IV’s until central line access can be established. Intraosssaeus access can also be used.
- Fibrinolytic therapy if > 90 min to PCI, < 3 hrs post MI onset, and no contraindications.
- When feasible transfer patients at high risk for mortality or severe LV dysfunction with signs of shock, pulmonary congestion, HR >100, and SBP <100 mm Hg to tertiary center capable of percutaneous coronary intervention (PCI), coronary artery bypass grafting (CABG) and available intra-aortic balloon pump (IABP).

Core Principles

- RV Infarct should be suspected in hypotensive patients with inferior STEMI and clear lung fields. Obtain ECG with right sided leads (V4R). RV may be best supported with:
  - Fluid volume loading
  - If ventilated, minimal PEEP; titrate fluid volume loading to minimal respiratory pulse pressure variation
  - Use of pulmonary vasodilators (Milrinone, inspired Nitrous Oxide), inotropes (Dobutamine preferred) and arterial vasoconstrictors such as Norepinephrine, Phenylepinephrine or Vasopressin

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Adult Suspected Stroke

Activate Emergency Response

Assess and Stabilize - Begin MOVIE
- Monitor - airway, blood pressure, ECG if available
- Oxygen - maintain SpO2 94-98%, provide breaths prn
- Vital signs - full set & glucose (treat low glucose stat)
- IV - ensure vascular access
- ECG – 12 lead ECG if available – do not delay therapy

Perform Neurological Screening Assessment
Activate Stroke Team or Emergent Transfer
Order Emergent CT Scan & Baseline Blood Work

Immediate Neurological Assessment by Stroke Team or Designee
- Patient History
- Establish time of symptom onset
- Perform Canadian Neurological Scale
- CT Scan completed

CT Scan shows Hemorrhage?

No Hemorrhage

Probable Acute Ischemic Stroke: Consider Fibrinolytics
- Check for fibrinolytic exclusions
- Repeat neurological exam (if deficits recovering rapidly, avoid fibrinolytics)

Candidate for fibrinolytic therapy?
- Review risks/benefits with patient and Family. If acceptable:
  - Give rt-PA – No antiplatelets/anticoagulants for 24 hours

Begin Post rt-PA Stroke Pathway
- Monitor BP, glucose, temperature, neuro status (treat if indicated)
- Admit to Stroke Unit if available or ICU

Probable Acute Hemorrhagic Stroke: Neuro Consult
(consider transfer if neurologist or neurosurgeon unavailable)
- Begin Hemorrhagic Stroke Pathway
- Admit to stroke unit or ICU

Hemorrhage

Give Aspirin

Not a candidate

Support notes included on reverse page.

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1 - **Adult Suspected Stroke**: Signs and symptoms of stroke include “sudden weakness or numbness of the face, arm or leg, especially on one side of the body; sudden confusion; trouble speaking or understanding; sudden trouble walking, dizziness, loss of balance or coordination; or severe sudden headache with no known cause.” 2010 American Heart Association Guidelines for CPR and ECC. *Circulation*: 2010;122: Supplement p S820.

2 - **Activate Emergency Response**: Time is brain. Alteplase (tPA) must be given within 4.5 hours of symptom onset to eligible patients with ischemic stroke. The earlier the stroke is treated, the greater the benefit. “Pre-hospital stroke assessment and rapid transport to the most appropriate hospital is critical to improving outcomes” BC Stroke Strategy Provincial Stroke Action Plan: November 2010

3 - **Check for fibrinolytics exclusions**: Symptomatic intracranial hemorrhage occurs in about 5% of patients who receive tPA for stroke. Before administering fibrinolytics, the ordering physician must verify that there are no exclusion criteria (additional criteria for 3-4.5 hours from symptom onset) and be prepared to treat any potential complications.

4 - **Stroke Pathway**: When compared to admissions to general medical units, patients who are admitted to dedicated interdisciplinary stroke units with established stroke care pathways are discharged from hospital 20% sooner and are 20% less likely to be discharged into institutional care and are 20% less likely to die while in hospital.

5 – **Aspirin**: Note that aspirin use for the patient having an acute stroke is recommended only for those who are not experiencing a hemorrhagic stroke and who are not candidates for fibrinolytics.

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**Core Principles**

- Survivability to discharge with good neurological function is optimized with time-sensitive stroke care
- Minimize delays to definitive stroke diagnosis and treatment. Major steps in stroke care are also the key delay points (D’s of stroke care):
  - **Pre-Hospital** (less than 3.5 hours from symptom onset – defined as last time patient seen as normal)
    - Detection: rapid recognition of stroke symptoms
    - Dispatch: activate emergency response
    - Delivery: rapid stroke screening assessment, management, and transport to stroke centre
    - Door: triage to appropriate stroke center
  - **Hospital** (60 minutes)
    - Data: rapid triage, evaluation (CT scan), stroke management in the emergency department (ED - first 25 min)
    - Decision: stroke expertise and therapy selection (45 minutes from arrival in ED)
    - Drug: fibrinolytic therapy (rt-PA or Alteplase) or intra-arterial strategies (60 minutes from ED arrival and max 4.5 hours from symptom onset)
    - Disposition: rapid admission to stroke unit or ICU (within 3 hours of arrival to ED)
- Monitor and treat co-morbidities; minimize risks associated with stroke and stroke treatments (i.e. head of bed at 30°; fluctuations in BP, temperature, glucose; airway compromise; oxygenation and ventilation; neurological deterioration; bleeding; NPO until dysphagia assessment);
- For transient ischemic attack (TIA)-a brief reduction of blood flow to the brain typically lasting less than 10 minutes without permanent damage - or minor stroke; as many as 10% will progress to major stroke within the next week in left untreated. With prompt treatment (ideally within 48 hours), long term risk of stroke is reduced by 80%. Management includes brain imaging to rule out hemorrhage, antiplatelet agents, anticoagulation for atrial fibrillation, blood pressure and glycemic control, and possible carotid endarterectomy.

**Additional Online Resources**

- Canadian Neurological Scale (www.neurosurvival.ca/ClinicalAssistant/scales/CNS.html)
- Canadian Best Practice Recommendations for Stroke Care (www.strokebestpractices.ca/)
- 2010 AHA Guidelines for CPR and ECC: Adult Stroke (circ.ahajournals.org/cgi/reprint/122/18_suppl_3/S818)
Defibrillation and Synchronized Cardioversion

Defibrillation is the delivery of a massive amount of electrical current through the heart over a few milliseconds with the goal of taking a critical mass of myocardial cells and depolarizing them into a brief moment of asystole. This asystolic pause allows cells with automaticity to begin dominating the heart in what is hopefully a normal organized pattern. Synchronized cardioversion is similar to defibrillation except that the delivery of the energy is timed to the intrinsic rhythm of the patient to avoid shocking during the relative refractory period of the cardiac cycle. Shocks during this period can produce VF.

- **Waveforms:** Monophasic waveforms deliver the energy of the shock in one direction (one polarity). Very few manufactures worldwide make this type of defibrillator anymore but many are still in use. Most use a monophasic damped sinusoidal waveform. Biphasic waveforms deliver a current that reverses direction during the few milliseconds of the shock as the polarity of the pads/paddles changes. Biphasic waveforms have been shown to be superior to monophasic waveforms in implanted defibrillators and significantly less myocardial current density is required with biphasic waveforms.

- **Defibrillation Energy Selection:**
  - Adult Monophasic: 360 Joules all defibrillations
  - Adult biphasic: Follow manufacturers recommendations (between 120-200 Joules), subsequent shocks can be at the same energy level or escalating energies can be considered. If the recommended starting energy is unknown then using the maximum energy setting can considered.
  - Pediatric: Both waveforms. First defibrillation 2-4 Joules/kg, subsequent shocks should be at least 4 Joules/kg. Higher energies can be considered but do not exceed 10 Joules/kg or the recommended maximum adult energy for the brand of defibrillator.

- **Synchronized Cardioversion Energy Selection: Same for all energy waveform types unless otherwise indicated**
  - Adult Atrial Fibrillation: start at 120-200 Joules escalating with subsequent attempts. Monophasic use 200J initially.
  - Adult Atrial Flutter: start at 50-100 Joules escalating with subsequent attempts.
  - Adult SVT: start at 50-100 Joules escalating with subsequent attempts.
  - VT with pulse: start at 100 Joules escalating with subsequent attempts.
  - Pediatric Cardioversion: start at 0.5-1 Joules/kg escalating with subsequent attempts to 2 Joules/kg

Steps to Defibrillation and Synchronized Cardioversion

1. Turn on monitor/defibrillator
2. Set lead switch to pads/paddles or lead I, II, or III if leads have been connected
3. Choose energy (most brands of defibrillators come on set to charge at the first defibrillation energy for an adult) for defibrillation or synchronized cardioversion.
4. Place defib pads/paddles on patient
   - For patients in cardiac arrest most often a anterior-anterior approach is used as it is quickest for pad application. Follow pictures on pads. Generally one pad is placed on the right upper anterior chest. Try to keep pad off larger bones such as the clavicle and the sternum and off the patient’s areola. Place the second pad in the left axillary position.
   - If the pads are being used in an anterior-posterior position the anterior pad is placed in an apical position and the posterior pad placed beside the spine and below the scapula on the left side. This placement is most common for transcutaneous pacing and is also often used in elective synchronized cardioversion.
   - If paddles are used apply an appropriate conductive medium (gel pads, conductive paste). Apply hard pressure (15-25 lbs.) and ensure that you are not in any electrical contact with the patient.
   - For pediatric patients if pads are too large consider anterior-posterior placement.
   - Attempt to keep paddles/pads 1-3 inches away from implanted devices such as AICD’s and pacemakers.
5. If performing synchronized cardioversion, ensure standard leads are connected; set synch button to on and ensure that the rhythm is being appropriately flagged on the R wave. Give sedation as appropriate for the situation
6. Announce that you are charging. Press the charge button on the machine or if using manual paddles the button on the apex paddle.
7. Warn three times that you are about to shock and visually check that no one is in electrical contact with the patient (direct contact, through liquids, or through metal)
8. Press shock button on machine or two buttons on paddles simultaneously. Note: for Synchronized cardioversion press shock button(s) down until shock occurs. The defibrillator is calculating when to shock and this can be very quick or may take several seconds. Also be sure to re-synch for any subsequent cardioversion attempts as most machines have the synch button turn off after each attempt.
8. For cardiac arrest situations continue CPR if possible as machine is charged and resume with compressions immediately after
the shock to minimize CPR time off chest.

**Transcutaneous Pacing (TCP)**

Transcutaneous pacing (TCP) is a highly effective emergency method of pacing for severe symptomatic bradycardias. Other
methods for increasing heart rate like the use of atropine, dopamine, or epinephrine may also be attempted depending on
situational factors and what rhythm the patient is in. This non-targeted method of pacing is unique in that it will also pace skeletal
muscle, gut muscle and the diaphragm at the currents needed to capture the myocardium electrically. This can mean significant
discomfort for the patient and the need for procedural sedation. The current levels needed to get capture are very high in
comparison to other methods of pacing and the aberrancy of the route of conduction from the pads leads to QRS complexes that
are very wide and bizarre resembling large PVCs. These observations are all normal and are expected.

**Steps to Transcutaneous Pacing**

1. Position pads on patient for pacing as indicated by the manufacturer. This is usually an anterior-posterior position. Look for a
indicator on the pads to show if one pad is specific to the apical position.
2. Ensure standard leads are also connected to the patient
3. Turn pacer on
4. Most TCP will come on default in demand (synchronous) mode, verify not set in non-demand (asynchronous) mode. This
mode is rarely used unless there is a situation where artifact is mistakenly being sensed as intrinsic ECG complexes.
5. Set pacer demand rate to 60-80 per minute
6. Set current by titrating the mA dial upwards until you have consistent electrical capture as indicated by seeing pacer spikes
followed by a new QRS morphology which is very wide with a broad T wave. Buffer the capture current (threshold) by increasing
the mA by approximately 10%.
7. Check for mechanical capture (paced rhythm produces cardiac output) by assessing distal pulses (at sites where skeletal muscles
are not contracting surrounding the vessel), level of consciousness, vital signs and other signs/symptoms of improved infusion.
8. Proceed to give analgesia and sedation as needed to keep patient comfortable.
9. Arrange transvenous or permanent pacemaker placement as needed
<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage (all doses are IV/IO unless otherwise noted)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenosine</td>
<td>6mg rapid push over 1-3 sec followed by a 20 ml syringe flush. If not successful a 12 mg dose can be given also with flush. Use most proximal IV/IO access and most proximal port</td>
</tr>
</tbody>
</table>
| Amiodarone          | VF/Pulseless VT: 300 mg push, a second dose of 150 mg can be given  
Life-Threatening Arrhythmias: Max cumulative dose: 2.2 g over 24 hrs. May be administered as:  
Rapid infusion: 150 mg over 10 min, may repeat every 10 min as needed (15 mg/min)  
Slow infusion: 360 mg over 6 hrs (1 mg/min)  
Maintenance infusion: 540 mg over 18 hrs (0.5 mg/min) |
| Atropine            | 0.5 mg q3-5 min. Max dose: 0.04 mg/kg (total 3 mg). Consider higher doses and shorter dosing interval with severe compromise |
| Calcium Chloride    | 500-1000mg (5-10 ml of the 10% solution) for hyperkalemia or calcium channel blocker overdose. May be repeated as needed |
| Digoxin             | Loading dose 4-6 mcg/kg, followed by 2-3 mcg/kg doses at 4 hr intervals. Maximum dosing total 1 mg |
| Diltiazem           | 15-20 mg (0.25 mg/kg) over 2 min. May repeat in 15 min at 20-25 mg (0.35 mg/kg) over 2 min. Maintenance infusion for rate control 5-15 mg/hr |
| Dobutamine          | 2-20 mcg/kg/min. Titrate so heart rate does not increase > 10% baseline |
| Dopamine            | 2-20 mcg/kg/min |
| Epinephrine         | Cardiac arrest: 1mg q3-5min  
Severe bradycardia: 2-10 mcg/min  
Anaphylaxis: 0.5 mg IM |
| Ibutilide           | 1-2 mg IV over 10-20 minutes |
| Isoproterenol       | 2-10 mcg/min titrated to adequate heart rate |
| Magnesium Sulfate   | Cardiac Arrest: 1-2 g as a bolus  
With pulse: 1-2 g mixed in 50-100 ml D5W over 5-60 min, then infusion of 0.5-1.0 g/hr as needed |
| Metoprolol          | 5 mg IV q5min to a total of 15 mg |
| Norepinephrine      | Start at 0.1-0.5 mcg/kg/min then titrate to response |
| Procainamide        | 20 mg/min infusion to max total dose of 17 mg/kg |
| Propafenone         | 300-600 mg po |
| Sodium Bicarbonate  | 1-2 mEq/kg bolus (44 mEq per preloaded syringe) |
| Sotalol             | 1-1.5 mg/kg over 5 min |
| Vasopressin         | Cardiac arrest: 40 units push  
Vasodilatory shock: 0.02-0.04 units/min |
| Verapamil           | 2.5-5 mg over 2-3 min, repeat dose 5-10 mg every 15-30 min as needed. Max total dose 30 mg |
Induced Therapeutic Hypothermia Overview

Brain injury and hemodynamic instability are the main causes of death for survivors of a cardiac arrest. Induced therapeutic hypothermia (ITH) - active cooling to 32-34°C Celsius for 24 hours - is shown to improve neurological recovery for those who experience a prolonged cardiac arrest.

ITH is recommended for: survivors of out-of-hospital VF arrests who are unable to follow verbal commands.

ITH should be considered for:
- survivors of out-of-hospital cardiac arrest from PEA or Asystole who are unable to follow verbal commands
- survivors of in-hospital cardiac arrest of any initial rhythm who are unable to follow verbal commands 10 minutes after the arrest

Active rewarming is discouraged for unresponsive cardiac arrest survivors who spontaneously become mildly hypothermic (core temperature >32°C Celsius) for the first 48 hours post-arrest.

At the time of writing no universally accepted induced therapeutic hypothermia algorithm has been published by AHA, ERC, or ILCOR. Many regions in Canada have adopted and/or modified the 2005 ITH guidelines of the Canadian Association of Emergency Physicians (CAEP).

Three Phases: Induction, Maintenance and Rewarming

Induction: a combination of external and internal cooling methods are implemented early post-arrest; interventions include rapid infusion (30 ml/kg) of 4°C 0.9% saline, ice packs, cooling blankets, intravascular heat exchangers, ice water gastric/bladder lavage and evaporative cooling techniques

Maintenance: a cooling method is used along with continuous temperature feedback monitoring to keep the core body temperature between 32-34°C

Rewarming: typically after 24 hours of cooling, rewarming should begin and progress slowly (i.e. 0.25-0.5°C/hour); manage fluctuations in electrolyte concentrations, intravascular volume and metabolic rate that occur with changes in body temperature; prevent hyperthermia by maintaining core temperatures at 36-37.5°C

Considerations

- ITH is an important component of a multisystem approach to post-cardiac arrest care; advanced airway management, hemodynamic stabilization, treatment of acute coronary syndromes and metabolic monitoring (serial lactate, electrolyte, glucose testing) are performed simultaneously
- Cooling suppresses several physiologic pathways that cause delayed (post-arrest) cell death; cooling also reduces cerebral metabolic rate by 6-10% for every 1°C reduction in cerebral temperature
- Shivering is an expected physiologic response to mild hypothermia; shivering increases heat production, thus slowing or preventing cooling; shivering should be treated; the threshold temperature for shivering can be decreased with IV sedation; IV boluses of neuromuscular blocking agents (NMBA) may be warranted; note that NMBA can eliminate skeletal muscle movement - the primary indicator of seizures activity (a seizure can triple cerebral metabolic rate); a continuous NBMA infusion requires ongoing EEG monitoring
- Physiologic effects and associated complications of hypothermia include:
  - increased systemic vascular resistance; continuous ECG monitoring for dysrhythmias;
  - diuresis; manage hemodynamic status and electrolyte levels
  - reduced insulin sensitivity and production; monitor for hyperglycemia
  - possible impaired coagulation; monitor for increased bleeding
  - impaired immune response; monitor for an increased incidence of infection
  - impaired medication elimination (one study showed a 30% reduction in medication clearance); re-evaluate medication dosing as temperature is reduced
- Target ITH temperatures are often maintained with simple interventions such as ongoing sedation, keeping the patient uncovered and the occasional use of evaporative cooling (a fan directed over wetted skin)
- Prognosis of post-cardiac arrest patients treated with ITH cannot be reliably predicted for at least 72 hours
Abbreviations Glossary
ACLS and Emergency Cardiovascular Care 2011

abx    antibiotic
ACE    angiotensin converting enzyme
ACLS   advanced cardiac life support
ACS    acute coronary syndrome
AED    automated external defibrillator
AF     atrial fibrillation
AFI    atrial flutter
AHA    American Heart Association
ALS    advanced life support
AMI    acute myocardial infarction
APLS   advanced pediatric life support
ASAP   as soon as possible
BB     beta blocker
BP     blood pressure
°C     degrees Celsius
CAB    chest compressions – airway - breathing
CABG   coronary artery bypass graft
CCB    calcium channel blocker
CCR    cardiocerebral resuscitation
CPAP   continuous positive airway pressure
CPR    cardiopulmonary resuscitation
CVP    central venous pressure
DBP    diastolic blood pressure
DIC    disseminated intravascular coagulation
ECC    emergency cardiovascular care
ED     emergency department
Epi    Epinephrine
ERC    European Resuscitation Council
ETT    endotracheal intubation
FAST   Focused Assessment with Sonography for Trauma
FBAO   foreign body airway obstruction
HR     heart rate
HSFC   Heart and Stroke Foundation of Canada
Hx     history
IABP   intra-aortic balloon pump
ILCOR  International Liaison Committee on Resuscitation
IO     intraosseous
ITH    induced therapeutic hypothermia
IV     intravenous
J      Joules
LLUD   left lateral uterine displacement
LMA    laryngeal mask airway
MAP    mean arterial pressure = (2 DBP + SBP)/3
LLUD   left lateral uterine displacement
MgSO₄  magnesium sulphate
MI     myocardial infarction
mm Hg  millimetres of mercury
MOVIE  Monitor – Oxygen if required – Vital Signs including glucose – IV – 12 lead ECG
MVO₂   mixed venous oxygen saturation
NPO    nothing by mouth
NS     normal 0.9% saline
NSTEMI non-ST elevation myocardial infarction
NTG    nitroglycerin
PALS   pediatric advanced life support
PCI    percutaneous coronary intervention
PE     pulmonary embolus
PEA    pulseless electrical activity
P₆₇CO₂  end-tidal carbon dioxide
PPV    positive pressure ventilations
Pt     patient
ROSC   return of spontaneous circulation
rt-PA  recombinant tissue plasminogen activator
s+s    signs and symptoms
SBP    systolic blood pressure
SIRS   systemic inflammatory response syndrome
SOB    shortness of breath
SpO₂   oxygen saturation as measured by a pulse-oximeter
STEMI  ST-elevation myocardial infarction
SVT    supraventricular tachycardia
TEE    transesophageal echocardiography
TCP    transcutaneous pacing
TIA    transient ischemic attack
TIMI   Thrombolysis in Myocardial Infarction risk score
UA     unstable angina
VF     ventricular tachycardia
VS     vital signs (TPR, BP, SpO₂, glucose)
VT     ventricular tachycardia
WBC    white blood cell
WPW    Wolff Parkinson White pre-excitation syndrome


