C01: Acute Coronary Syndrome

Mike Sugimoto

Updated: July 15, 2023
Reviewed: March 01, 2021

Introduction

Acute coronary syndrome (ACS) represents a spectrum of diseases resulting from insufficient blood flow through the coronary arteries culminating in a wide range of presentations.

Essentials

- Rapid identification of ST segment elevation myocardial infarction (STEMI) to facilitate timely reperfusion strategies is the primary goal in out-of-hospital management. Consider ACP intercept for ECG acquisition and interpretation if not available at the scene.
- Antiplatelet therapy should be initiated as early as possible in all patients with suspected coronary ischemia.
- Reduction of myocardial oxygen demand should be accomplished wherever and whenever possible (e.g., management of nausea, pain, and limiting patient exertion).

Additional Treatment Information

- Acetylsalicylic acid (ASA) is the out-of-hospital antiplatelet drug of choice. Emergency medical dispatch will instruct patients to chew and swallow ASA 320 mg and patients may have taken their own prior to paramedic or EMR/FR arrival. Unless otherwise contraindicated, ASA should be administered to bring the total dose, for this event, to at least 160 mg orally.
- Nitroglycerin, 0.4 mg sublingually, may be given to alleviate pain in cases of angina. Systolic blood pressure must be monitored prior to and during nitroglycerin therapy. Nitroglycerin has not been demonstrated to change outcomes in ischemic chest pain and may in fact worsen myocardial ischemia under some circumstances. The on-going use of nitroglycerin in patients who have not experienced symptom relief following the first few doses is unlikely to produce any benefit.
- To minimize handover delays in suspected STEMI and to facilitate angiography and fluoroscopy, place therapy electrodes anterolaterally with wires positioned cephalad (toward the head) prior to initiating conveyance.
- All patients with suspected coronary ischemia should have vascular access established with running intravenous fluid. When selecting a site for access, use of the distal third of the right arm is relatively discouraged (particularly in the setting of anticipated percutaneous coronary intervention). Do not delay conveyance to obtain vascular access.

General Information

- ACS exists on a spectrum, from angina through to STEMI:
  - Angina is pain resulting from a temporary increase in myocardial oxygen demand. This may be the result of reduced blood flow in the coronary arteries due to arterial narrowing, or spasm in the arterial wall.
  - Non-ST segment elevation myocardial infarction (NSTEMI) is the result of an incomplete occlusion of a coronary artery, either by a thrombus alone or in concert with vasospasm. ECGs generally show ST segment depression or T wave inversion, though transient ST segment elevation may also be observed.
  - STEMI occurs when a coronary artery is completely occluded by a thrombus. The diagnosis is dependent on ST segment elevation in two or more anatomically contiguous leads.
- Common presentations include chest pain, "heaviness", or discomfort associated with shortness of breath, nausea, and/or diaphoresis. Be aware that although these are common findings, certain populations – in particular, women, the elderly, those with a history of diabetes, and younger individuals – may present differently. Atypical ACS presentations can include weakness or fatigue, syncope/presyncope, abdominal pain, and nausea.
- The presence of palpable chest wall pain does not exclude ischemic origins. Paramedics and EMRs/FRs should maintain a high suspicion of ischemic-origin pain in cases of chest pain without a clear history of trauma.
- Patients presenting with symptoms consistent with ACS should be managed as such, regardless of ECG findings,

up to and including a clinical pathway selection.

- Contraindications to ASA therapy include known hypersensitivity or a recent history of upper or lower gastrointestinal bleeding. Patients on oral anticoagulant therapies are often told by their physician to avoid ASA. In the setting of suspected or known ACS, the antiplatelet activity of ASA is of more importance than the temporary rise in INR. Consult with CliniCall if unsure.

Interventions

First Responder

- Keep the patient warm and protect from further heat loss
- Place the patient in a position of comfort, as permitted by clinical condition; in general, limit patient movement
- Provide supplemental oxygen where indicated
  - → A07: Oxygen Administration
- Conduct ongoing assessment and gather collateral information, such as medications and identification documents
- Establish ingress and egress routes from the patient's location
- Communicate patient deterioration to follow-on responders

Emergency Medical Responder – All FR interventions, plus:

- Oxygen as required to maintain SpO_2 ≥ 94%
  - → A07: Oxygen Administration
- Acetylsalicylic acid chew and swallow, if not already done
- Nitroglycerin spray if systolic blood pressure ≥ 110 mmHg and heart rate within range of 50-150 beats/minute
  - CliniCall consultation required prior to nitroglycerin administration if no prior prescription or if more than 3 doses are required.
- Consider nitrous oxide as required for pain
  - → E08: Pain Management
- Convey with early notification; consider intercept with additional resources

Primary Care Paramedic – All FR and EMR interventions, plus:

- Obtain vascular access with running intravenous fluid
  - → D03: Vascular Access
- Nitroglycerin spray every 4-5 minutes if systolic blood pressure ≥ 110 mmHg and heart rate is between 50-150 beats/minute
  - CliniCall consultation required prior to nitroglycerin administration if no prior prescription or if more than 3 doses are required.
- Consider dimenhyDRINATE for nausea
  - → E07: Nausea and Vomiting
- Obtain and transmit 12-lead ECG.
  - → PR16: 12-Lead ECG
  - The LifePak 15 may be used to acquire a 12-lead ECG if:
    - Paramedics have completed the online and face-to-face training and required EMALB endorsement,
    - The patient is over 18 years of age,
    - The patient presents with active, recent onset (< 12 hours) non-traumatic chest pain that is suspicious for acute coronary syndrome, and
  - Clinical judgment will be required to establish the optimal timing of ECG acquisition. In general, paramedics should minimize scene time wherever possible. Refer to the standard operating procedure for out-of-hospital use of the LifePak 15 for additional information.
- If STEMI criteria are met, and a local PCP STEMI pathway to primary percutaneous coronary intervention has been developed:
  - Request ACP intercept where available.
  - Attach therapy electrodes (place pads anterior-lateral, wires cephalad)
    - Transmit ECG to receiving hospital, and follow the appropriate STEMI pathway provided transport time

is **less than 60 minutes**.

- Kelowna General Hospital Area STEMI Pathway (PCP)
- Sea to Sky STEMI Pathway (PCP)
- South Island STEMI pathway (PCP)

- If STEMI criteria are met, and a local PCP STEMI pathway **has not been developed**:
  - Request ACP intercept where available.
  - Notify receiving hospital as soon as practicable.

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- Obtain and interpret 12-lead ECG (plus additional precordial leads as required)
  - → PR16: 12-Lead ECG
- In cases of STEMI:
  - Select and activate local reperfusion strategy, including early hospital notification
    - Consider eligibility for out-of-hospital reperfusion pathways:
      - → PR51: Prehospital fibrinolysis
- Attach therapy electrodes (place pads anterior-lateral, wires cephalad)
- Consider fentaNYL as required for pain
- Manage dysrhythmias as necessary
  - Atropine as necessary for symptomatic bradycardia
    - → C02: Bradycardia
  - → C03: Narrow Complex Tachycardia
  - → C04: Wide Complex Tachycardia

Critical Care Paramedic – All FR, EMR, PCP, and ACP interventions, plus:

Oxygenation/ Ventilation

(Not routinely used but should be considered for presenting cases of cardiogenic pulmonary edema or cardiogenic shock)

- NIPPV
  - CPAP
  - High-flow oxygen
  - BiPAP
- IPPV
  - Mechanical ventilation

Antiplatelet

- P_2Y_{12}
  - Ticagrelor
  - Clopidogrel
- Consider Gp IIb/IIIa
  - Eptifibatide

Nitrates

- Nitroglycerin IV
- Nitroglycerin Topical

Opioids (recognize the reduction in P_2Y_{12} effectiveness. Routine use should be avoided)

- Morphine
- Fentanyl
- Hydromorphone

Anticoagulants (age and renal function dependant)

- Heparin
- Low molecular weight heparin
  - Enoxaparin
  - Lovenox
  - Fondaparinux

Beta blockade

- Metoprolol
- Atenolol

Consider Calcium channel blocker (patients unable to take Beta blocker)

- Verapamil
- Diltiazem

Statin

- Atorvastatin

ACE inhibitor (administered in the first 24 hours for refractory hypertension)

- Ramipril

ARB

- For patient's intolerant of ACE inhibitors

Consider Fibrinolytic therapy (> 90 minutes to a PCI center)

- Call ETP prior to thrombolytic administration.
- TNK
- tPA
- rPA

Bleeding or anemia

- Consider pRBC's for patients with a Hgb < 100

Hyperglycemia (stress induced in diabetics)

- Consider insulin

Consider correction of electrolytes

- Mg^{+}
- K^{+}

Evidence Based Practice

ACS/Suspected Cardiac Origin

Supportive

- 12-Lead ECG
- Anti-platelet (other)
- ASA/Aspirin
- Bypass/Direct to PCI
- Drip and ship
- Fibrinolysis

- Nitrates
- RIC (remote ischemic conditioning)
- Advanced Notice/Cath Lab Activation by EMS
- Pharmacoinvasive approach
- Ketamine
- PAI-ASA

Neutral

- Beta Blockers
- GIK (Glucose-Insulin-Potassium)
- Heparin
- Lidocaine
- Magnesium
- Nitrous Oxide
- HEMS
- Morphine
- Point of Care Troponin

Against

- Fentanyl
- Oxygen-high flow
- Oxygen-titrated

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Practice Updates

15 July 2023: Updated STEMI Clinical Pathway Links for PCP and ACP

C02: Bradycardia

Mike Sugimoto

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Introduction

While bradycardia is defined as a heart rate of < 50 beats/minute, symptomatic bradycardia refers to weakness, a decreased level of consciousness, shortness of breath, hypotension, or chest pain that is the result of bradycardia. The treatment of bradycardia focuses on optimizing hemodynamics and addressing the underlying cause.

Essentials

- Patients with bradycardia often complain of dizziness, which is frequently exacerbated by positional changes that are resolved when positioned supine, or mild shortness of breath. These patients can be managed with supportive care only, provided they are otherwise asymptomatic.
- Patients with adequate perfusion and a low heart rate may require monitoring and conveyance, but no treatment. Unless the patient requires immediate resuscitation, a conservative approach to management should prevail.
- Clinical end points are defined by the amelioration of symptoms rather than any particular heart rate or blood pressure.
- Management of the prevailing underlying condition is often more critical than correction of the dysrhythmia.

Additional Treatment Information

- Although atropine remains the first-line therapy in adult symptomatic bradycardia, it is unlikely to be effective in 2nd and 3rd degree heart blocks; its use is, however, still recommended in these patients. Atropine is ineffective and potentially harmful in patients who have had a heart transplant.
- Small doses of atropine may produce a transient slowing of the heart rate. In these cases, administer a second dose immediately. For prolonged conveyances, additional atropine may be required to sustain its effect to a maximum total dose of 3 mg.
- Epinephrine infusion should be considered in cases where atropine has failed to produce a meaningful improvement in heart rate or blood pressure.
- Rapid intervention in patients who are peri-arrest (e.g., who have a markedly decreased level of consciousness and signs of profound hypoperfusion) can prevent further deterioration and stave off a progression to cardiac arrest. Epinephrine, rather than atropine, is the preferred pharmacological treatment option in these cases. Note that there is no published data that supports the routine use of epinephrine in preference to atropine for patients not at imminent risk of cardiac arrest.
- Renal failure can precipitate hyperkalemia, which can cause a dangerous accumulation of AV node blocking agents (calcium channel blockers or beta blockers), producing significant bradycardia and hypoperfusion (the so-called 'BRASH syndrome'). This is often triggered by underlying hypovolemia in elderly patients with pre-existing renal dyfunction. Fluid resuscitation and consultation with CliniCall for management of suspected hyperkalemia is required (see ACP interventions below).

General Information

- In all cases of bradycardia, consideration must be given to the overall clinical condition of the patient. Signs of effective perfusion (particularly skin colour, skin temperature, and mentation) are better indicators of the need for intervention than blood pressure (either systolic blood pressure or mean arterial pressure) alone. Paramedics and EMRs/FRs should have a nuanced understanding of the degree to which a patient is symptomatic.
- In all cases of symptomatic bradycardia, search for and address treatable or reversible causes. Such cases may include:
  - Hypoxia (especially in younger patients)
  - Increased parasympathetic (vagal) tone
  - Drug effects or overdoses

- Hyperkalemia, with or without concurrent metabolic acidosis
- Myocardial ischemia, particularly if it involves the SA or AV nodes and conduction system

- In the setting of myocardial infarction, bradycardia is often compensatory and somewhat beneficial. Be cautious of initiating rate-specific therapies as these may increase myocardial oxygen demand and extend the margins of infarct. Therapy should be reserved for those patients who are significantly hypotensive.

Interventions

First Responder

- Keep the patient warm and protect from further heat loss
- Place the patient in a position of comfort, as permitted by clinical condition; in general, limit patient movement
- Provide supplemental oxygen where indicated
  - → A07: Oxygen Administration
- Conduct ongoing assessment and gather collateral information, such as medications and identification documents
- Establish ingress and egress routes from the patient's location
- Communicate patient deterioration to follow-on responders

Emergency Medical Responder – All FR interventions, plus:

- Oxygen as required to maintain SpO_2 ≥ 94%
  - → A07: Oxygen Administration
- Convey early
- Consider intercept with additional resources

Primary Care Paramedic – All FR and EMR interventions, plus:

- Consider vascular access
  - → D03: Vascular Access
- Consider fluid bolus

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- Obtain 12-lead ECG, plus additional precordial leads as required:
  - Manage ischemic findings in accordance with guidelines
    - → PR16: 12-Lead ECG
    - → C01: Acute Coronary Syndrome
- Consider sodium bicarbonate only in cases of suspected hyperkalemia (wide-complex ECG, known renal failure/dialysis patient, diabetic ketoacidosis)
  - → E03: Hyperkalemia
    - CliniCall consultation required prior to treatment for cases of suspected hyperkalemia.
- Atropine to effect
- EPINEPHrine infusion to effect (increase dose every 2-3 minutes)
- Transcutaneous pacing
  - → PR19: Transcutaneous Pacing
  - → PR17: Procedural Sedation
  - → E08: Pain Management
- In peri-arrest scenarios:
  - Consider push-dose EPINEPHrine
  - → N01: Peri-arrest

Critical Care Paramedic – All FR, EMR, PCP, and ACP interventions, plus:

- Isoproterenol

- Dopamine infusion
- Call ETP prior to transvenous pacing
- Transvenous pacemaker placement/maintenance

Evidence Based Practice

Bradycardia

Supportive

Neutral

- Inotrope
- Anticholinergic
- Transcutaneous Pacing

Against

C03: Narrow Complex Tachycardia

Mike Sugimoto

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Introduction

The narrow complex tachycardias (NCT) are a number of clinical conditions that are defined primarily by their ECG findings but differ in their significance. All NCTs originate above the level of the atrioventricular node and use the ventricles' normal conduction pathways.

Essentials

- Do not attempt to control heart rate or rhythm, using either medications or cardioversion, if the tachycardia is believed to be compensatory (e.g., pain, hypovolemia, fever, hypoxia). A thorough history must be obtained prior to initiating therapy. Manage any of these major underlying conditions prior to addressing the tachycardia.
- Adenosine is the preferred treatment option for patients experiencing mild to moderate symptoms believed to be associated with a supraventricular tachycardia and whose dysrhythmias cannot be terminated through a modified Valsalva maneuver.
- Electrical cardioversion should be reserved for those patients with severe symptoms or who show signs of significant hemodynamic instability, regardless of the underlying rhythm.

Additional Treatment Information

- Print rhythm strips during all conversion attempts.
- The modified Valsalva (as described by Appelboam et al) has been demonstrated to be effective at terminating paroxysmal supraventricular tachycardia in some settings. It has very few risks and can be used in stable patients while vascular access is being established. The standard Valsalva maneuver is modified by having the patient attempt to bear down, or blow the plunger out of a 10 cc syringe, for 15 seconds. The patient is then laid supine, their legs raised to maximize venous return to the core, and held in this position for 15 seconds.
- Owing to its extremely short half-life, adenosine must be administered rapidly and ideally through a proximal IV site. Patients often complain of a flushing sensation or of a metallic taste in their mouth during adenosine administration. This is normal and to be expected, indicating that an effective dose has been delivered. The monitor should be printing during adenosine administration to record changes to rhythm.
- Patients should, however, be warned of common adenosine side effects prior to administration. These include facial flushing, shortness of breath, palpitations, chest pain, and light headedness. Paramedics must be prepared for rare complications of adenosine, such as bradycardia or prolonged asystole following administration.

General Information

- Atrial fibrillation is the result of electrical activity at multiple ectopic foci in the atria that overwhelm the atrioventricular node and can produce rapid heart rates. The rhythm in atrial fibrillation is irregularly irregular and there are no discernable P-waves on the ECG.
- Atrial flutter is produced by a re-entry circuit within the atria, coupled with an AV node that fails to consistently conduct impulses to the ventricles. Conduction to the ventricles usually follows a 2:1 or 3:1 ratio, which produces a difference between atrial activity and ventricular activity. The rhythm is generally regular, with characteristic 'sawtooth' P-waves on the ECG. Both atrial fibrillation and atrial flutter are associated with structural heart disease as well as age.
- Paroxysmal supraventricular tachycardia (PSVT or SVT) is the result of the development of an accessory conduction pathway between the atria and the ventricles, separate from the AV node. SVT can develop in any individual, at any age, and can be triggered by caffeine or other stimulants, exertion, or – in many cases, nothing at all.
- NCTs may present with chest pain, palpitations, dizziness, pounding in the chest, shortness of breath, or weakness. A history of previous episodes, with similar symptoms, is highly suggestive of a recurrent disease process. Consider a patient with a NCT to be unstable when presenting with:

  - An altered level of consciousness
  - A systolic blood pressure < 80 mmHg
  - Ischemic-type chest pain
  - Significant shortness of breath and/or evidence of acute cardiogenic pulmonary edema.
- The formal diagnosis of NCT, whether atrial fibrillation, atrial flutter, or SVT, often requires prolonged Holter monitoring (at some significant cost to the health care system as the arrhythmias often do not develop during monitoring). Paramedics should therefore endeavour to acquire a high-quality electrocardiogram on all NCT patients, both for their own clinical purposes and also for the patient's benefit as well, particularly if no formal diagnosis has been made.
- In atrial flutter, adenosine may temporarily suppress ventricular activity allowing the flutter waves to be seen more clearly. This is a diagnostic for atrial flutter; adenosine should not, however, be used by paramedics solely as a diagnostic tool.
- Many patients with atrial fibrillation are only mildly symptomatic and require no care beyond monitoring and reassurance. Patients with atrial fibrillation who are symptomatic can be cardioverted; use caution if the onset of the atrial fibrillation is believed to be > 48 hours prior to EMS contact as there is a risk of embolization if the patient is not anticoagulated. Consultation with CliniCall is mandatory in these cases (see ACP interventions below).

Interventions

First Responder

- Keep the patient warm and protect from further heat loss
- Place the patient in a position of comfort, as permitted by clinical condition; in general, limit patient movement
- Provide supplemental oxygen where indicated
  - → A07: Oxygen Administration
- Conduct ongoing assessment and gather collateral information, such as medications and identification documents
- Establish ingress and egress routes from the patient's location
- Communicate patient deterioration to follow-on responders

Emergency Medical Responder – All FR interventions, plus:

- Oxygen as required to maintain SpO_2 ≥ 94%
  - → A07: Oxygen Administration
- Convey early
- Consider intercept with additional resources

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- Obtain and interpret 12-lead ECG with additional precordial leads as required; if significant ischemia is present, manage according to ACS/STEMI guidelines
  - → PR16: 12-Lead ECG
  - → C01: Acute Coronary Syndrome
- Establish vascular access:
  - → D03: Vascular Access
  - If adenosine administration is anticipated, a proximal large-bore (18 g or larger) catheter is preferred
  - Consider fluid bolus if hypovolemia is suspected
- For atrial fibrillation with a rapid ventricular response (> 120/minute):
  - If stable, convey and observe
- For suspected atrial flutter:
  - If stable, convey and observe
- For suspected supraventricular tachycardia:
  - Modified Valsalva maneuver

- Adenosine - CliniCall consultation required if conversion fails after 2 doses.
  - The use of sedation prior to the administration of adenosine is neither supported by evidence nor recommended by BC Emergency Health Services
- **For all rhythms, if unstable:**
  - Synchronized cardioversion 100-300J (procedural sedation will be required)
    - CliniCall consultation required prior to synchronized cardioversion of atrial fibrillation if onset is believed to be > 48 hours of EMS contact.
  - → PR17: Procedural Sedation
  - → PR20: Synchronized Cardioversion

Critical Care Paramedic – All FR, EMR, PCP, and ACP interventions, plus:

- Consider sodium channel blockade
  - May consider procainamide
- Consider beta blockade
  - May consider metoPROLOL
  - May consider Propranolol
  - May consider Esmolol
- Consider potassium channel blockade
  - Amiodarone
- Consider calcium channel blockade
  - May consider diltiazem

Evidence Based Practice

Stable Narrow Complex Tachycardia

Supportive

- Antiarrhythmic - Class I (Na+ channel blockers)
- Antiarrhythmic - Class IV (Ca+ channel blockers)
- Antiarrhythmic - Class V (other mechanism)
- Electrical Cardioversion
- Modified Valsalva
- Treat and Release-SVT

Neutral

- Antiarrhythmic - Class III (K+ channel blockers)
- Beta Blockers
- Carotid Massage
- Valsalva maneuver
- Vagal Maneuvers

Against

Unstable Tachycardia (Wide or Narrow Complex)

Supportive

- Electrical Cardioversion

Neutral

- Vagal Maneuvers

Against

References

1. Appelboam A, et al. Postural modification to the standard valsalva manoeuvre for emergency treatment of supraventricular tachycardias (REVERT): A randomised controlled trial. 2015. [Link]
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Practice Updates

- 2023-07-05: removed verapamil from critical care interventions

C04: Wide Complex Tachycardia

Shauna Speers

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Reviewed: February 05, 2025

Introduction

Wide complex tachycardias (WCT) are characterized by QRS widths greater than 0.12 s on an ECG. They are often, but not always, synonymous with ventricular tachycardia (VT), which is a period of three or more ventricular originated beats at a rate ≥ 100/minute. VT can be either monomorphic or polymorphic in nature.

Essentials

- The objective of care is the rapid termination of life-threatening ventricular tachycardia. Electrical cardioversion is the safest, most reliable mechanism to convert VT into a stable perfusing rhythm.
- Although WCT can develop primarily, it is often a sign of an underlying clinical condition, such as ischemia, hypoxia, hyperkalemia, or increased sympathetic tone. A thorough history should be performed prior to formulating a management plan and any underlying conditions should be considered and addressed concurrently with the tachycardia.
- Consider as unstable any patient with WCT and any of:
  - Altered or rapidly falling level of consciousness
  - Systolic blood pressure < 90 mmHg
  - Ischemic chest pain
  - Significant shortness of breath or signs of cardiogenic pulmonary edema

Additional Treatment Information

- Patients with a WCT who are clinically stable can be managed with supportive care alone. However, these patients can deteriorate quickly, so preparatory measure should be taken (IV access, therapy electrodes placed and attached). For longer conveyance times (> 20 minutes), infusion of amiodarone can be considered in consultation with CliniCall (required; see ACP interventions below).
- Unstable patients should be cardioverted as soon as possible. Sedation will generally be required.
  - Synchronized cardioversion is the preferred choice in monomorphic WCT. Begin at 100J, escalating by 100J increments to a maximum of 360J. If cardioversion fails, consider switching to the alternate pad placement (e.g., if positioned anterior-lateral, place new pads anterior-posterior). Consultation with CliniCall for refractory VT is recommended (see ACP interventions below). When performing a synchronized cardioversion, ensure that the shock button is pressed and held until the energy is delivered.
  - For unstable polymorphic ventricular tachycardia, unsynchronized cardioversion is the preferred choice. Begin at 200J and follow the standard energy escalation protocol.
- Stable polymorphic WCT can be managed with magnesium sulfate. Unstable polymorphic WCT should be defibrillated (unsynchronized cardioversion) beginning at 200J.

General Information

- WCTs are generally regular. Some irregularity can be normal in ventricular tachycardia, but consistently irregular wide complex rhythms should prompt consideration of a rhythm that is atrial in origin, usually atrial fibrillation, in conjunction with a bundle branch block.
  - Note that this must be distinguished from polymorphic WCT or torsades de pointes (TdP), where the morphology of each QRS complex is different and the R-R interval continues to change
- A small percentage of regular, WCTs are actually supraventricular in origin and result from an aberrantly conducted electrical impulse. However, the vast majority are, and should be managed as, ventricular tachycardia.

Interventions

First Responder

- Keep the patient warm and protect from further heat loss
- Place the patient in a position of comfort, as permitted by clinical condition; in general, limit patient movement
- Provide supplemental oxygen where indicated
  - → A07: Oxygen Administration
- Conduct ongoing assessment and gather collateral information, such as medications and identification documents
- Establish ingress and egress routes from the patient's location
- Communicate patient deterioration to follow-on responders
- Monitor patient closely; consider potential for sudden deterioration
- An AED must be ready and available; be prepared to perform chest compressions
  - → PR06: High-performance CPR

Emergency Medical Responder – All FR interventions, plus:

- Supplemental oxygen as required to maintain SpO_2 ≥ 94%
  - → A07: Oxygen Administration
- Convey early
- Consider intercept with additional resources

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- Obtain and interpret 12-lead ECG
  - → PR16: 12-Lead ECG
- Attach therapy electrodes
- Obtain vascular access
  - → D03: Vascular Access
- For stable, monomorphic Wide Complex Tachycardia (WCT)
  - Amiodarone infusion is indicated for recurrent sustained episodes (>30 secs)
  - Consider cardioversion if patient becomes symptomatic
    - Clinicall/EPOS consult recommended
- For symptomatic sustained runs of monomorphic WCT:
  - Amiodarone infusion
- For stable Torsades de Pointes (TdP)
  - Magnesium Sulfate
- Symptomatic monomorphic WCT or TdP
  - Procedural sedation as required
  - Synchronized cardioversion (100J-200J-300J-360J)
    - R wave flagging for synchronized cardioversion in polymorphic WCT may not be possible; provide unsynchronized defibrillation
  - Consider vector change if cardioversion fails
  - Amiodarone for WCT OR Magnesium Sulfate for TdP if refractory to cardioversion

Critical Care Paramedic – All FR, EMR, PCP, and ACP interventions, plus:

- Consider sodium channel blockade
  - May consider procainamide
  - May consider lidocaine
- May consider potassium blockade
  - Amiodarone

Evidence Based Practice

Stable Wide Complex Tachycardia

Supportive

- Antiarrhythmic - Class III (K+ channel blockers)
- Antiarrhythmic - Class I (Na+ channel blockers)
- Electrical Cardioversion

Neutral

- Adenosine

Against

Unstable Tachycardia (Wide or Narrow Complex)

Supportive

- Electrical Cardioversion

Neutral

- Vagal Maneuvers

Against

References

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C05: Acute Aortic Dissection

Richard Armour

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Reviewed: March 01, 2021

Introduction

The incidence of acute aortic dissection is reported to be as high as 4.6/100,000 and appears to be increasing. Although infrequent, approximately 80% of patients experiencing an acute aortic dissection will arrive in the emergency department by ambulance. Mortality increases by 2% for every hour of delay in diagnosis, and fully half of all patients die within 3 days of the onset of their symptoms.

Despite the severity of the disease, 1 out of every 6 patients will be misdiagnosed. Acute aortic dissection often masquerades as a number of other conditions, including acute coronary syndrome and stroke. Out-of-hospital care is focused on early recognition, expedient conveyance, analgesia, and judicious resuscitation.

Essentials

- Paramedics and EMRs/FRs must consider acute aortic dissection in any patient experiencing a sudden onset of chest, back, or abdominal pain. Patients commonly describe pain as "sharp" or "tearing" with the maximal intensity at onset. The pain tends to radiate into the back, abdomen, or along the path of the aorta. Up to 17% of patients will not experience pain and will instead present with a decreased level of consciousness, transient syncope, or focal neurological deficits.
- A tear in the aorta can interrupt blood supply to any organ. In patients with pain suggestive of an aortic dissection who also have stroke-like symptoms, such as paralysis, voice hoarseness, or limb ischemia, paramedics and EMRs/FRs should consider the possibility that these symptoms are a result of the dissection.
- Differences in blood pressure between arms are not a consistent indicator of an aortic dissection and must not be used to exclude the diagnosis.

Additional Treatment Information

- Tachycardia can significantly worsen the clinical trajectory of acute aortic dissection. Control of the heart rate is not indicated for paramedics or EMRs/FRs. Every effort must be made to avoid patient exertion during movement.
- Patients with acute aortic dissections may initially present with hypertension. In patients who are hypotensive, fluid resuscitation must be undertaken carefully so as to not exacerbate the dissection. A mean arterial pressure (MAP) of 65 mmHg is sufficient.
- Analgesia should be provided to patients but carefully titrated given the patient's hemodynamic status.

General Information

- An acute aortic dissection occurs when the intima of the aorta tears and blood enters the medial layer of the aortic wall, creating a false lumen.
- Risk factors for aortic dissections include a family history of dissections, hypertension, and/or cardiovascular surgery. Dissections are more common in older males. Individuals with Marfan or Ehler-Danlos Syndrome are at higher risk.
- A new aortic regurgitation murmur, and/or a pulse deficit in the setting of pain suggestive of an aortic dissection, is strongly suggestive of the diagnosis.
- Patients with a widening pulse-pressure are in a critical stage of their disease and paramedics and EMRs/FRs should make preparations for an impending cardiac arrest.
- Acute aortic dissections are described using the Stanford Classification:
  - Type A dissections involve the ascending aorta, with or without the involvement of the arch or descending aorta
  - Type B dissections involve the descending thoracic and/or abdominal aorta
- Do not confuse acute aortic dissection with abdominal aortic aneurysms.

Interventions

First Responder

- Keep the patient warm and protect from further heat loss
- Place the patient in a position of comfort, as permitted by clinical condition; consider supine positioning to optimize blood pressure
  - Warning: do not exert the patient
- Provide supplemental oxygen where indicated
  - → A07: Oxygen Administration
- Conduct ongoing assessment and gather collateral information, such as medications and identification documents
- Establish ingress and egress routes from the patient's location
- Communicate patient deterioration to follow-on responders

Emergency Medical Responder – All FR interventions, plus:

- Provide supplemental oxygen to maintain SpO_2 ≥ 94%
  - → A07: Oxygen Administration
- Convey to appropriate facility with early notification
- Consider analgesia
  - → E08: Pain Management
    - Nitrous oxide

Primary Care Paramedic – All FR and EMR interventions, plus:

- Establish vascular access:
  - Consider fluid bolus if hypotensive and without signs of pulmonary edema
  - Caution: target blood pressure to MAP of 65 mmHg; do not over-resuscitate
  - → D03: Vascular Access
- Consider analgesia
  - → E08: Pain Management

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- Consider analgesia
  - → E08: Pain Management
    - FentaNYL

Critical Care Paramedic – All FR, EMR, PCP, and ACP interventions, plus:

- Consider blood pressure lowering agents in cases of confirmed or highly suspected dissection where the patient is hypertensive. Goals are heart rate < 60 beats per minute and a systolic pressure of 100 - 120 mmHg.
  - Consider beta blocker
    - LABETalol
    - Propranolol
    - Esmolol
  - Calcium channel blocker
    - Consider if beta blockers are not tolerated
      - Verapamil
      - Diltiazem
  - Nitrates
    - Beta blockade must be started prior to nitrates to avoid reflex tachycardia.
      - Nitroprusside can be added if target systolic blood pressure cannot be reached with beta-blockers alone.
      - Nitroglycerine

Leaking /ruptured AAA

- This is a surgical emergency. Do not delay transport to a surgical center for any intervention.
- Consider permissive hypotension
- Consider blood product administration
- Avoid intubation due to further decrease in preload when possible.

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C06: Acute Pulmonary Edema

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Introduction

Pulmonary edema is a clinical phenomenon where fluid accumulates in the alveoli in the lungs, resulting in impaired oxygen exchange and shortness of breath. Although pulmonary edema is associated with a number of clinical problems, in the out-of-hospital environment, it is most commonly the result of congestive heart failure (CHF). Impairment of ventricular function causes blood to accumulate in both the pulmonary and systemic circulation. Pulmonary edema as a result of CHF may develop slowly, over days, or very suddenly (also known as 'flash' pulmonary edema). Treatment options for pulmonary edema depend heavily on underlying cause, so careful assessment is required.

Essentials

- To the maximum extent possible, paramedics and EMRs/FRs should attempt to determine the origin of the fluid and differentiate between cardiogenic pulmonary edema, asthma, pneumonia, or chronic obstructive pulmonary disease.
- Consider cardiogenic shock if the patient: has a history of cardiac dysfunction; is experiencing chest pain with hypotension; has an altered level of consciousness, exhibits pale and cool skin, and/or has a decreased urine output.
- Position patients to limit venous return. Be aware that many patients with pulmonary edema will be unable to tolerate a supine or semi-recumbent position. Respiratory arrest may follow if patients are forced to lie down.
- Patients with impending respiratory failure (e.g., those with a respiratory rate and/or tidal volume that is decreasing and whose level of consciousness is falling) must be ventilated with a bag-valve mask (including a PEEP valve, if indicated).

Additional Treatment Information

- Cardiogenic pulmonary edema is often accompanied by significant hypertension. Nitroglycerin decreases systemic vascular resistance through a number of mechanisms. The decision to use nitroglycerin is complex, requires a thorough understanding of the pathophysiology of the underlying condition, and assesses multiple clinical variables. There are significant risks to the use of nitroglycerin in these cases.
- CPAP is a non-invasive device that uses positive pressure to improve oxygenation and is very effective in cases of pulmonary edema, regardless of the underlying cause. The greatest benefits of CPAP accrue from its use early in the disease course; paramedics should consider the use of CPAP as soon as pulmonary edema has been identified.

General Information

- *Pulmonary edema is not solely caused by congestive heart failure*. Exposure to toxic products (including smoke, bleach, or chlorine) can produce primary pulmonary edema due to epithelial damage. Pulmonary edema can also occur as a result of drug ingestion or submersion and drowning. These patients are generally not hypertensive, do not have a history of heart disease, and have a history of exposure. Although the in-hospital treatment of these patients is different from those with cardiogenic pulmonary edema, the principles remain the same: oxygen, supportive ventilation as required, and rapid conveyance. CPAP can be effective in these cases.
- Early stage pulmonary edema may present as wheezing ('cardiac asthma'). Salbutamol may alleviate some of these symptoms, however, the wheezes in these cases are associated with airway edema rather than bronchospasm. Salbutamol has sympathomimetic properties that increase the workload of an already dysfunctional heart. The risks and benefits of salbumatmol use must be considered for each individual patient.
- Diuretics are no longer considered a mainstay of out-of-hospital treatment for pulmonary edema.
- Some patients with pulmonary edema will require bag-valve mask ventilation, particularly after positional changes. Paramedics and EMRs must be prepared to intervene during or immediately after a transfer and should strive to minimize patient exertion during these maneuvers.

- Patients in respiratory failure, or who otherwise do not improve with CPAP, should be ventilated using a bag-valve mask. The use of positive end-expiratory pressure (PEEP) valves may be effective in improving both oxygenation and ventilation in these patients.

Interventions

First Responder

- Caution: Keep the patient at rest and avoid exertion during transfers. Bring equipment to the patient, including lifting and transfer devices.
- Position patient sitting upright with legs dependent.
- Keep the patient warm and protect from further heat loss
- Provide supplemental oxygen where indicated
  - → A07: Oxygen Administration
- Conduct ongoing assessment and gather collateral information, such as medications and identification documents
- Establish ingress and egress routes from the patient's location
- Communicate patient deterioration to follow-on responders
- Provide ventilation by bag-valve mask as required; addition of a high-flow nasal cannula may be necessary

Emergency Medical Responder – All FR interventions, plus:

- Provide supplemental oxygen to keep $SpO_2 \geq 94\%$
  - → A07: Oxygen Administration
- Convey early
- Consider intercept with additional resources

Primary Care Paramedic – All FR and EMR interventions, plus:

- Consider continuous positive airway pressure
  - → PR09: Continuous Positive Airway Pressure
- If positive pressure ventilation by bag-valve mask is required, consider use of PEEP valve (5 cmH_2O to start)
  - → PR10: Positive End Expiratory Pressure

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- Obtain vascular access; limit fluid administration to minimum required for drug administration and procedures
  - → D03: Vascular Access
- Obtain and interpret 12-lead ECG; correct and manage abnormalities, including arrhythmia and/or ischemia
  - → PR16: 12 Lead ECG
  - → C01: Acute Coronary Syndrome
  - → C02: Bradycardia
  - → C03: Narrow Complex Tachycardia
  - → C04: Wide Complex Tachycardia
- Consider preload reduction:
  - Nitroglycerin
- Consider salbutamol for significant bronchospasm
- If unable to maintain oxygenation or ventilation through non-invasive methods, consider intubation:
  - → B01: Airway Management
  - → PR18: Anesthesia Induction
  - → PR23: Awake Intubation

Critical Care Paramedic – All FR, EMR, PCP, and ACP interventions, plus:

- Identify the probable cause of the pulmonary edema

    - Respiratory support is the primary treatment for acute pulmonary edema but this is largely symptom relief. Specific diseases or injuries need to be addressed as the treatments will vary for the presenting clinical picture. (Cardiogenic vs non-cardiogenic)
- → PR27: Mechanical ventilation
    - Consider NPPV
        - High flow
        - BiPAP
        - CPAP
    - Consider Invasive ventilation
        - Consider use of ACV or PCV mode, targeting a Vt 6-8 mL/kg
        - Increase PEEP/FiO2 to SpO_2 ≥90% and/or PaO_2 ≥60 mmHg
        - Pplat < 30 cmH_2O
    - For persistent hypoxemia, consider (may require neuromuscular blockade):
    - Recruitment maneuver
    - Open lung ventilation strategy
    - Arterial and/or venous blood gas analysis may provide guidance for management
- Hemodynamic support (HFpEF vs HFrEF or non-cardiogenic)
    - Preload reduction
        - Furosemide
        - Fluid restriction
    - Afterload reduction
        - ACE inhibitor or ARB
        - MORPHine
    - Vasopressor support
        - NORepinephrine
        - Vasopressin
        - EpiNEPHrine
        - DOPamine
    - Inotrope support
        - Dobutamine
        - Milrinone
    - Consider albumin for hypoalbuminemia
- Treat the presenting disease/illness.

Evidence Based Practice

Pulmonary Edema (CHF)

Supportive

- NiPPV
- Nitroglycerin-IV
- 12-Lead ECG
- Oxymetry Monitoring

Neutral

- Diuretic
- Ultrasound
- Beta Agonist-MDI

- Beta Agonist-Nebulized
- Nitroglycerin-SL

Against

- Narcotic
- Oxygen

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Practice Updates

2022-02-08: updated language around ACP interventions (reduction of preload)