

C01: Acute Coronary Syndrome

Mike Sugimoto

Updated: April 30, 2021

Reviewed:

Introduction

The acute coronary syndromes represent a spectrum of diseases resulting from insufficient blood flow through the coronary arteries, with a wide range of presentations.

Essentials

- Rapid identification of STEMI to facilitate timely reperfusion strategies is the primary goal of prehospital 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 (i.e., management of nausea, pain, and limiting patient exertion)

Additional Treatment Information

- Acetylsalicylic acid (ASA) is the prehospital 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 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 prior to initiating transport.
- Vascular access: 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 transport 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 as a result of arterial narrowing, or spasm in the arterial wall.
 - Non-ST elevation MI (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; transient ST segment elevation may also be observed.
 - ST elevation MI (STEMI) occurs when a coronary artery is completely occluded by thrombus. The diagnosis is dependent on ST segment elevation in two or more anatomically contiguous leads.
- Classic presentations include chest pain, pressure, 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 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 transport destination selections.
- Contraindications to acetylsalicylic acid therapy include known allergy or 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 ClinicaCall if unsure.

Interventions

First Responder

- Supplemental oxygen as required
 - → [A07: Oxygen and Medication Administration](#)

Emergency Medical Responder – All FR interventions, plus:

- Oxygen as required to maintain SpO₂ ≥ 94%
 - → [A07: Oxygen and Medication Administration](#)
- [Acetylsalicylic acid](#) chew and swallow, if not already done
- Assist patient with [nitroglycerin](#) spray if systolic blood pressure ≥ 110 mmHg and heart rate > 50 and < 150
 - MANDATORY CLINICAL CONSULTATION (1-833-829-4099) IF NO PRIOR NITRO PRESCRIPTION, AND/OR PRIOR TO EXCEEDING 3 DOSES.
- Position of comfort
- Transport with early notification; consider ACP intercept

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.
 - MANDATORY CLINICAL CONSULTATION (1-833-829-4099) IF NO PRIOR NITRO PRESCRIPTION, AND/OR PRIOR TO EXCEEDING 3 DOSES.
- Consider [dimenhydrinate](#) for nausea
 - → [E07: Nausea and Vomiting](#)
- Consider [nitrous oxide](#) as required for pain
 - → [E08: Pain Management](#)
- If trained and authorized:
 - Obtain and transmit 12-lead ECG
 - → [PR16: 12 Lead ECG](#)
- If STEMI criteria are met:
 - Obtain physician consultation prior to transport destination selection to verify treatment plan and selection of reperfusion strategy
 - Attach therapy electrodes (place pads anterior-lateral, wires cephalad)
 - → STEMI Program Manual

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

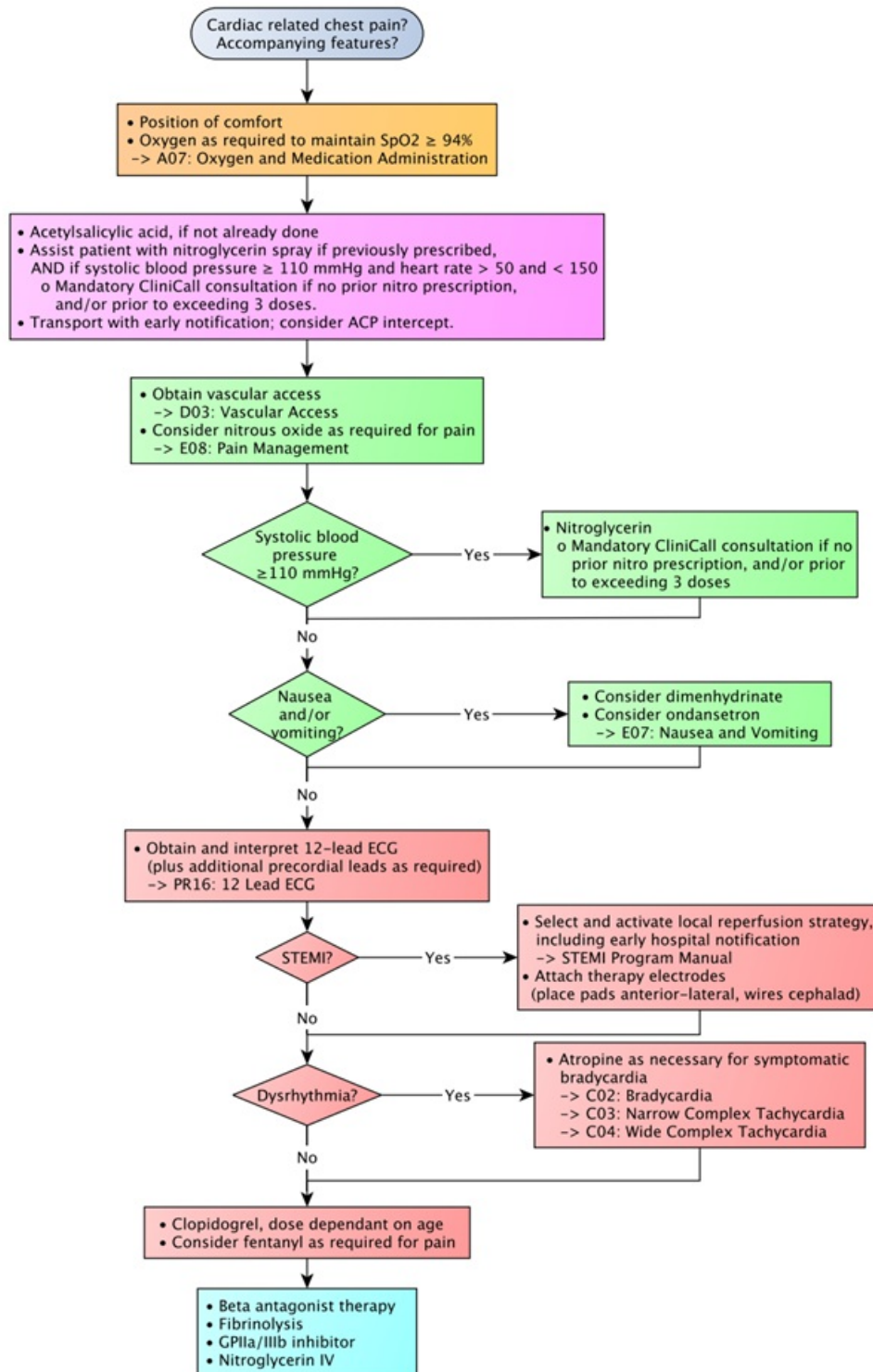
- Obtain and interpret 12-lead ECG (plus additional precordial leads as required)
 - → [PR16: 12 Lead ECG](#)
- In cases of STEMI:
 - Select and activate local reperfusion strategy, including early hospital notification
 - → STEMI Program Manual
 - Consider eligibility for out-of-hospital reperfusion pathways.
- 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](#)

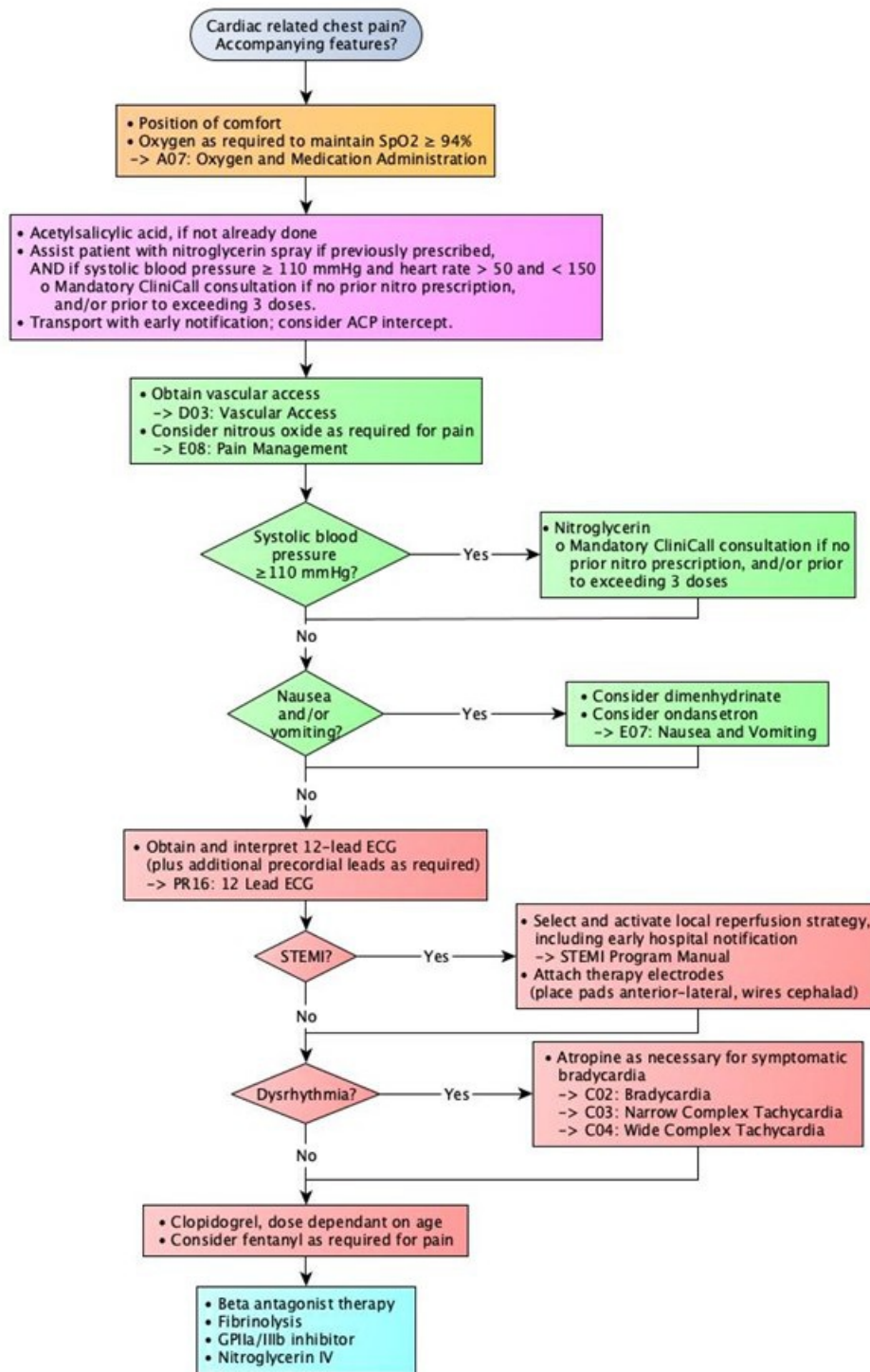
- [→ C03: Narrow Complex Tachycardia](#)
- [→ C04: Wide Complex Tachycardia](#)

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

- [Ticagrelor](#)
- Heparin / low molecular weight heparin
- [Nitroglycerin](#) IV
- Fibrinolysis in consultation with EPOS/cardiologist

Algorithm





Evidence Based Practice

[Chest Pain: ACS/Suspected Cardiac Origin](#)

References

1. Kawakami S, et al. Time to reperfusion in ST-segment elevation myocardial infarction patients with vs. without pre-hospital mobile telemedicine 12-lead electrocardiogram transmission. 2016. [\[Link\]](#)
2. Welsford M, et al. Part 5: Acute coronary syndromes: 2015 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. 2015. [\[Link\]](#)

C02: Bradycardia

Mike Sugimoto

Updated: December 19, 2020

Reviewed:

Introduction

While bradycardia is defined as a heart rate of less than 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 and resolves 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 transport, 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 cases. 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. In long transports, 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 (i.e., 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 treatment option in these cases, but 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 in turn cause a dangerous accumulation of AV node blocking agents (calcium channel 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 dysfunction. Fluid resuscitation and consultation with ClinCall for management of suspected hyperkalemia are recommended.

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 color and temperature, and mentation) are better indicators of the need for intervention than blood pressure (either systolic blood pressure or mean arterial pressure) alone. Paramedics 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

- Supplemental oxygen as required
 - [→ A07: Oxygen and Medication Administration](#)
- Keep patient at rest
- Position of comfort; place patient supine if symptoms of hypotension are present

Emergency Medical Responder – All FR interventions, plus:

- Oxygen as required to maintain $SpO_2 \geq 94\%$
 - [→ A07: Oxygen and Medication Administration](#)
- Transport early
- Consider ACP intercept where available

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

- Consider vascular access
 - [→ D03: Vascular Access](#)
- Consider fluid bolus

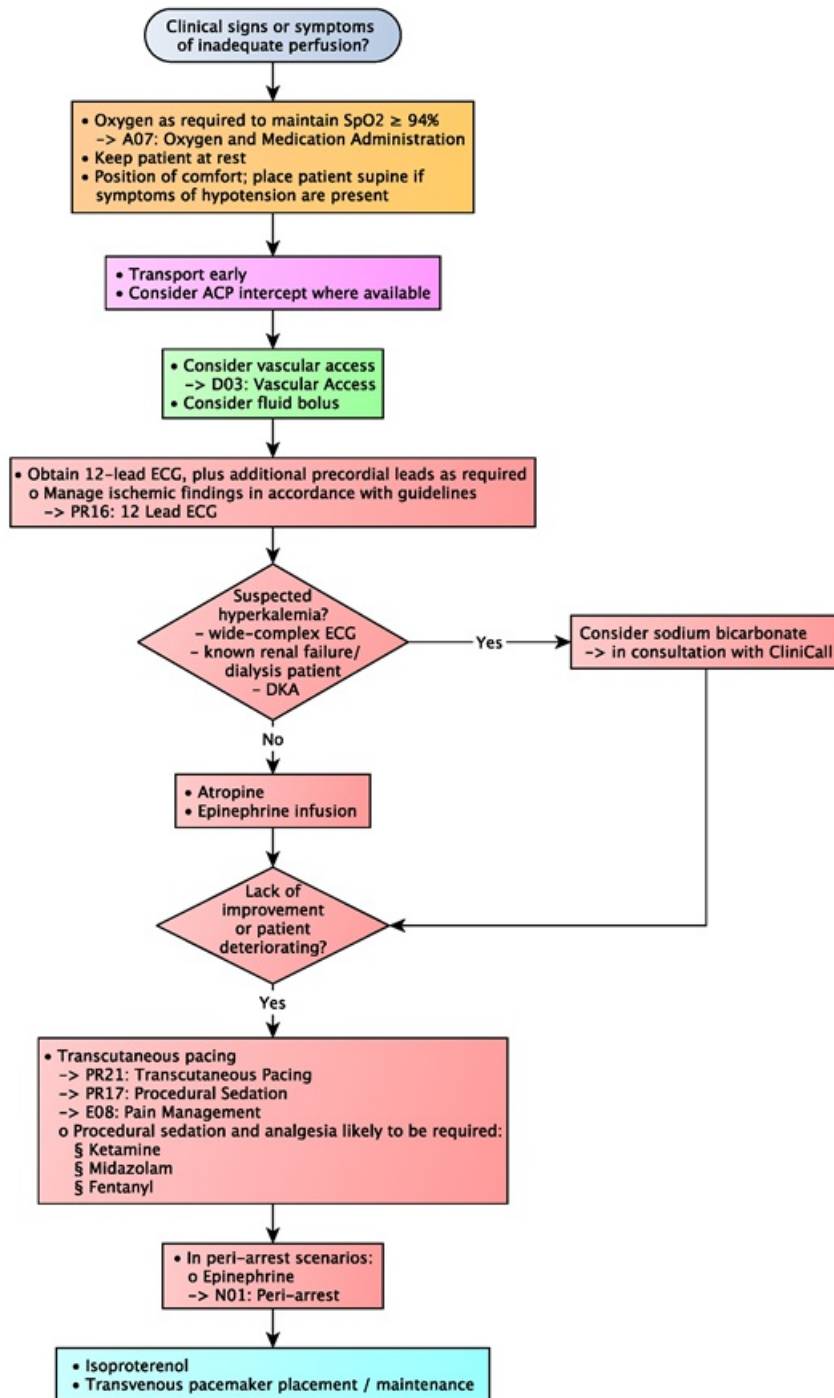
Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- 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), in consultation with CliniciCall.
- [Atropine](#) to effect
- [EPINEPHrine](#) infusion to effect (increase dose q 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
- Transvenous pacemaker placement / maintenance

Algorithm



Evidence Based Practice

[Bradycardia](#)

C03: Narrow Complex Tachycardia

Mike Sugimoto

Updated: December 08, 2020

Reviewed:

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 narrow complex tachycardias originate above the level of the atrioventricular node, and all 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 and their legs are 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, ideally through a proximal IV site. Patients often complain of a flushing feeling, or of a metallic taste in their mouth during adenosine administration – this is normal and to be expected, and indicates 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.
- The use of amiodarone to control atrial fibrillation is reasonably well documented through research but its applicability to prehospital care is unknown. Patients with atrial fibrillation who are hemodynamically unstable should be electrically cardioverted; amiodarone, in consultation with CliniCall, may be considered as a therapeutic option of last resort in the context of a prolonged transport.

Referral Information

Individuals with known re-entry tachycardias may be referred to follow-up care in consultation with CliniCall after termination of their dysrhythmia using adenosine if they are asymptomatic and have no complaints.

General Information

- Atrial fibrillation is the result of electrical activity at ectopic foci in the atria that overwhelm the atrioventricular node, and can produce rapid heart rates. The rhythm in atrial fibrillation is 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.
- Narrow complex tachycardias 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 narrow complex tachycardia 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 narrow complex tachycardia, whether atrial fibrillation, 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 narrow complex tachycardia patients, both for their own clinical purposes but 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 diagnostic for atrial flutter; adenosine should not, however, be used by paramedics solely for the purpose of diagnosis.
- 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 electrically cardioverted; use caution if the onset of the atrial fibrillation is believed to be greater than 48 hours prior to EMS contact as there is a risk of embolization if the patient is not anticoagulated. Consultation with ClinCall is mandatory in these cases.

Interventions

First Responder

- Position of comfort
- Supplemental oxygen as required
 - → [A07: Oxygen and Medication Administration](#)

Emergency Medical Responder – All FR interventions, plus:

- Oxygen as required to maintain SpO₂ ≥ 94%
 - → [A07: Oxygen and Medication Administration](#)
- Transport early
- Consider ACP intercept

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 better) catheter is preferred
 - Consider fluid bolus if hypovolemia is suspected
- For atrial fibrillation with a rapid ventricular response (> 120/minute):
 - If stable, transport and observe

- May consider [amiodarone](#) in consultation with CliniCall
- For suspected atrial flutter:
 - If stable, transport and observe
- For suspected supraventricular tachycardia:
 - Modified Valsalva maneuver
 - [Adenosine](#). CliniCall consult required if conversion fails after two doses.
- **For all rhythms, if unstable:**
 - Synchronized cardioversion, 100-300 J (procedural sedation will be required)
 - → [PR17: Procedural Sedation](#)
 - → [PR20: Synchronized Cardioversion](#)

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

- May consider verapamil
- May consider procainamide
- May consider [metoPROLOL](#)

Evidence Based Practice

[Narrow Complex Tachycardia](#)

[Unstable Tachycardia: Wide or Narrow Complex](#)

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. [\[Link\]](#)

C04: Wide Complex Tachycardia

Mike Sugimoto

Updated: December 08, 2020

Reviewed:

Introduction

Wide complex tachycardias are characterized by QRS widths greater than 0.12 ms on an ECG. They are often, but are not always, synonymous with ventricular tachycardia (VT), which is a period of three or more ventricular origin beats at a rate of 100/minute or greater, and can either be 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 problem, such as ischemia, hypoxia, hyperkalemia, or increased sympathetic tone. A thorough history should be performed prior to formulating a management plan, and these underlying conditions should be considered and addressed concurrently with the tachycardia.
- Consider as unstable any patient with WCT and any of:
 - Ischemic chest pain
 - Systolic blood pressure of less than 80 mmHg
 - Altered or rapidly falling level of consciousness
 - Significant shortness of breath or signs of cardiogenic pulmonary edema

Additional Treatment Information

- Patients with a wide complex tachycardia who are clinically stable can be managed with supportive care only. However, these patients can deteriorate quickly, so preparatory measure should be taken (IV access, therapy electrodes placed and attached). For longer transport times (> 20 minutes), infusion of amiodarone can be considered in consultation with CliniCall.
- Unstable patients should be cardioverted as soon as possible. Sedation will generally be required.
 - Synchronized cardioversion is the preferred choice in monomorphic wide complex tachycardia. Begin at 100J, escalating by 100J to a maximum of 360J. If cardioversion fails, consider switching to the alternate pad placement (i.e., if positioned anterior-lateral, place new pads anterior-posterior). Consultation with CliniCall for refractory VT is recommended. 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 (i.e., defibrillation) is the preferred choice. Begin at 200J and follow the standard energy escalation protocol.
- Stable polymorphic wide complex tachycardia can be managed with magnesium sulfate. Unstable polymorphic WCT should be defibrillated (unsynchronized cardioversion), beginning at 200J.

General Information

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

Interventions

First Responder

- Oxygen as required to maintain $\text{SpO}_2 \geq 94\%$
 - [→ A07: Oxygen and Medication Administration](#)
- Position of comfort
- Monitor patient closely. Consider potential for sudden deterioration. An AED must be available. Be prepared to perform chest compressions.
 - [→ PR06: High-performance CPR](#)

Emergency Medical Responder – All FR interventions, plus:

- Oxygen as required to maintain $\text{SpO}_2 \geq 94\%$
 - [→ A07: Oxygen and Medication Administration](#)
- Transport early
- Consider ACP intercept if available

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 WCT:
 - CliniCall consult
 - [Amiodarone](#)
- For unstable, monomorphic WCT:
 - Sedation and analgesia as required
 - [→ PR17: Procedural Sedation](#)
- Synchronized cardioversion, 100-200-300-360J
 - [→ PR20: Synchronized Cardioversion](#)
 - Consider switching electrical axis if cardioversion fails
- For stable, polymorphic WCT:
 - CliniCall consult
 - [Magnesium sulfate](#)
- For unstable, polymorphic WCT:
 - Sedation and analgesia as required
 - [→ PR17: Procedural Sedation](#)
- Defibrillate 200-300-360J

Evidence Based Practice

[Stable Wide Complex Tachycardia](#)

[Unstable Tachycardia: Wide or Narrow Complex](#)

C05: Acute Aortic Dissection

Richard Armour

Updated: December 08, 2020

Reviewed:

Introduction

The incidence of acute aortic dissections is reported 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 three 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. Paramedic care is focused on early recognition, expedient transport, analgesia, and judicious resuscitation.

Essentials

- Paramedics 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, and radiating 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.
- The 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, paralysis, voice hoarseness, or limb ischemia, paramedics 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. 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 titrated carefully 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, and individuals with Marfan or Ehler-Danlos Syndrome are particularly at 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 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

- **WARNING: DO NOT EXERT THE PATIENT DURING MOVEMENT**
- Supplemental oxygen as required
 - → [A07: Oxygen and Medication Administration](#)
- Position patient supine to optimize blood pressure

Emergency Medical Responder – All FR interventions, plus:

- Provide supplemental oxygen to maintain SpO₂ ≥ 94%.
 - → [A07: Oxygen and Medication Administration](#)
- Transport 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](#)

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
 - [LABETalol](#), propranolol, or esmolol (reduce heart rate to < 60 beats per minute)
 - Nitroprusside can be added if target systolic blood pressure of 100-110 mmHg cannot be reached with beta-blockers alone

References

1. Diercks D, et al. Clinical policy: Critical issues in the evaluation and management of adult patients with suspected acute nontraumatic thoracic aortic dissection. 2015. [\[Link\]](#)
2. CORE Emergency Medicine. Aortic Dissection. 2016. [\[Link\]](#)
3. Imamura H et al. Painless acute aortic dissection - Diagnostic, prognostic and clinical implications. 2011. [\[Link\]](#)
4. Milewicz DM. Stopping a killer: Improving the diagnosis, treatment, and prevention of acute ascending aortic dissections. 2011. [\[Link\]](#)
5. Rosman HS, et al. Quality of history taking in patients with aortic dissection. 1998. [\[Link\]](#)
6. Hiratzka L, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM Guidelines for the Diagnosis and Management of Patients With Thoracic Aortic Disease: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. 2010. [\[Link\]](#)

C06: Acute Pulmonary Edema

Chris Morgan

Updated: December 19, 2020

Reviewed:

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 prehospital 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 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, or if chest pain is present with hypotension, an altered level of consciousness, pale and cool skin, and/or 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 – i.e., those whose respiratory rate and/or tidal volume are 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 and requires a thorough understanding of the pathophysiology of the underlying condition, and an assessment of multiple clinical variables. There are significant risks to the use of nitroglycerin in these cases.
- CPAP is a form of non-invasive device that uses positive pressure to improve oxygenation, and is very effective in cases of pulmonary edema, regardless of 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 and 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 transport. 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 salbutamol use must be considered for each individual patient.
- Diuretics are no longer considered a mainstay of prehospital treatment for pulmonary edema.
- Some patients with pulmonary edema will require bag-valve mask ventilation, particularly after position changes. Paramedics 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

- 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
- Supplemental oxygen as required
 - [→ A07: Oxygen and Medication Administration](#)
- Provide intermittent positive pressure 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 and Medication Administration](#)
- Transport early
- Consider ACP intercept

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

- Consider continuous positive airway pressure (requires CliniCall consult)
 - [→ PR09: Continuous Positive Airway Pressure](#)
- If positive pressure ventilation by bag-valve mask is required, consider use of PEEP valve (5 cmH₂O 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 treatment of hypertension:
 - [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:

- Consider etiology of restrictive lung and correct if possible (e.g., restrictive straps, circumferential burns, pneumo- or hemothorax)
- Improve oxygenation: consider BiPAP, intubation, and mechanical ventilation
 - Consider use of ACV mode, Vt 6-8 mL/kg
 - Increase PEEP/FiO₂ to $SpO_2 \geq 90\%$ and/or $PaO_2 \geq 60$ mmHg

- For persistent hypoxemia, consider (may require neuromuscular blockade):
 - Recruitment maneuver
 - Open lung ventilation strategy
 - Pressure control ventilation (inverse ratio)
 - Permissive hypercapnia
- Arterial and/or venous blood gas analysis may provide guidance for management
- In air transport, consider a reduced cabin altitude where possible

Evidence Based Practice

[Pulmonary Edema \(CHF\)](#)

References

1. Alberta Health Services. AHS Medical Control Protocols. 2020. [\[Link\]](#)
2. Ambulance Victoria. Clinical Practice Guidelines: Ambulance and MICA Paramedics. 2018. [\[Link\]](#)
3. Gray A, et al. Noninvasive ventilation in acute cardiogenic pulmonary edema. 2008. [\[Link\]](#)
4. Purvey M, et al. Managing acute pulmonary oedema. 2017. [\[Link\]](#)
5. Tintinalli JE, et al. Tintinalli's emergency medicine: A comprehensive study guide. 9th. 2019.

