6224.05: Adult Cardiac Arrhythmia

INTRODUCTION

Cardiac arrhythmias are common in the EMS setting and can range from benign to immediately life-threatening. One of the fundamental concepts is the importance of recognizing those patients who are unstable or those who may become unstable during the course of care.

SAFETY

Cardiac arrhythmias can decompensate rapidly and require resuscitation. Clinicians should ensure they have access to appropriate protection if the patient is unstable or at risk of decompensating and upgrade their PPE prior to resuscitation. Observe safety precautions when delivering electrical therapy.

Paramedics must be alert for possible signs of environmental toxins causing the patient's symptoms (e.g. smoke/chemical exposure, enclosed spaces, multiple patients, symptoms inconsistent with the underlying rhythm). Exposure to carbon monoxide, cyanide, hydrogen sulfide, petroleum products or pesticides are known to cause arrhythmias and cardiovascular compromise. Responder safety is the highest priority and patient care should only be performed if it does not place care providers at risk. If safe to do so, the patient should be removed from the environment and be decontaminated. PPE should be worn to prevent secondary exposure and relevant support agencies should be contacted (e.g. Fire/Hazmat).

ASSESSMENT

Clinicians need to rapidly determine whether the patient is 'unstable'. Signs and symptoms of an unstable patient include any of the following:

LOC	Abnormal
Airway	Lacking protection
Breathing	Respiratory distress/ CHF
Circulation	Shock/ Hypotension/ Ischemia

Patients experiencing an arrhythmia may present with a decreased level of consciousness (LOC) if they are experiencing hypotension or shock. They may also present with shortness of breath and ischemic chest pain as earlier findings suggestive of instability.

Hypoxemia is a common cause of both brady and tachy-arrhythmias, therefore initial assessment should include obtaining an SpO_2 level as well as looking for tachypnea, accessory muscle use and other signs of increased work of breathing.

By obtaining a rate (fast, slow or normal) and rhythm (regular vs. irregular) with the first palpated pulse check, the clinician can begin to rule in or rule out certain rhythm diagnoses before the monitor is even applied. Absent pulses (radial and carotid) may indicate severe hypotension or a patient who is in cardiac arrest or peri-arrest.

Both bradycardic and tachycardic patients should undergo cardiac and respiratory monitoring and a rhythm strip should be obtained. Unstable arrhythmias must be addressed immediately. A 12-lead ECG should be obtained as soon as possible.

Symptomatic or Asymptomatic

If a patient is hemodynamically stable, the clinician should assess if the arrhythmia is causing symptoms and determine potential underlying causes. Common symptoms of both brady and tachy-arrhythmia include:

- Altered LOC
- Shortness of breath
- Chest discomfort
- Palpitations
- Pre-syncope/syncope
- Weakness/fatigue
- Light-headedness or dizziness
- Diaphoresis
- Anxiety/panic

An arrhythmia may result from pathology involving the conduction system of the heart, in this context it is the arrhythmia itself causing the clinical presentation (primary arrhythmia). Arrhythmias may also occur as a result of other pathology (a secondary arrhythmia), so it is important to consider alternative etiologies for the clinical presentation as well (e.g. infection, pulmonary embolus, toxicologic causes). The Atlantic Canada Poison Centre may also be contacted when a toxic ingestion is suspected.

When assessing/interpreting any rhythm strip, answering 4 questions will help you to identify the correct rhythm:

- Is the rhythm fast or slow?
- Are there P waves before every QRS and QRS after every P wave?
- Is the QRS wide or narrow?
- Is the rate regular or irregular?

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Bradycardia

Bradycardic rhythms may have multiple causes such as increased vagal tone, hypoxia, ischemia, electrolyte abnormalities or dysfunction of the conduction pathway. If such a cause is identified, treatments should be started to address the underlying etiology concurrently with managing the arrhythmia (e.g. reperfusion therapy for STEMI or management of hyperkalemia).

Some people have a slow heart rate normally due to physical conditioning and others may have a slower rate due to medications such as beta-blockers. Though bradycardia is defined as a heart rate less than 60 bpm, when a bradyarrhythmia is the cause of symptoms, the rhythm is most often less than 50 bpm.

The overall goals of assessing bradycardia:

- Categorize the patient as stable or unstable
- Differentiate between signs and symptoms that are caused by the slow rate vs. those due to an alternative etiology
- Correctly diagnose the presence and type of rhythm

Regular bradycardic rhythms include:

- Sinus bradycardia
- 3rd degree AV block
- Junctional rhythms
- Ventricular escape

Irregular bradycardic rhythms include

- 2nd degree AV block - Type I
- 2nd degree AV block - Type II (in rare cases can be regular)
- Atrial Fibrillation with slow ventricular response

Tachycardia

Tachycardia may occur as an appropriate physiologic response, or be secondary to pathological conditions such as spontaneous depolarization or abnormal conduction. The clinician should consider whether the tachycardia is causing the signs/symptoms and hemodynamic instability, or are the signs/symptoms/hemodynamic changes due to an alternative etiology that is also driving the tachycardia (e.g. pain, fever, hypovolemia, pulmonary embolus)?

When assessing tachycardia on the rhythm strip, certain questions can help identify the rhythm.

1. Are there P waves present before every QRS (e.g. is the rhythm sinus tachycardia)?
   - Sinus tachycardia is generated by the sino-atrial (SA) node, the rate is typically no higher than 180 in an adult. Start considering rhythms other than sinus tachycardia if the rhythm is above 150.
   - Sinus tachycardia has a gradual onset and termination. It is usually caused by external systemic factors (e.g. fever, exercise, blood loss, or anemia), not primary cardiac rhythm disorders.
   - Sinus tachycardia may have a small amount of beat to beat variability in rate (e.g. may vary with respirations).

2. Is the QRS narrow or wide?
   - A narrow QRS indicates the origin of the rhythm is supraventricular. Tachycardic rhythms with narrow QRS complexes include sinus tachycardia, atrial fibrillation, atrial flutter, AV nodal reentry, atrial tachycardia, and junctional tachycardia.
   - A wide QRS may indicate the origin of the rhythm is from the ventricles (e.g. ventricular tachycardia or VT), a pacemaker, or may result from supraventricular tachycardia with either aberrancy or pre-excitation (e.g. Wolff-Parkinson-White syndrome).
   - A wide QRS may also be seen with sinus tachycardia if there is an underlying bundle branch block. In this case, there will always be P waves before every QRS.

3. Is the rhythm regular or irregular?
   - Atrial fibrillation is the most common irregular narrow complex tachycardia.
   - Atrial fibrillation may also occur with a wide QRS in the setting of an underlying bundle branch block, aberrancy, or pre-excitation.

4. Is the QRS monomorphic or polymorphic?
   - Ventricular tachycardia (VT) can be monomorphic or polymorphic. Torsades de pointes is a type of polymorphic VT that is associated with a prolonged QTc interval.

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MANAGEMENT

Bradycardia

If a patient has adequate perfusion, and is not exhibiting signs of hemodynamically unstable bradycardia, do not aggressively treat the heart rate. Conversely, patients who are unstable require immediate treatment.

Treatment of symptomatic bradycardia will depend on the presumed underlying etiology, the type of bradycardia observed and patient stability. This may include pharmacological agents and/or transcutaneous pacing. If it is known that bradycardia is secondary to myocardial infarction, thrombolytics and/or judicious small fluid boluses might be required with extreme caution not to fluid overload the patient. Calcium channel blocker or betablocker overdose will require specific management. Electrolyte abnormalities such as hyperkalemia may occur in a patient with kidney failure and will require significantly different management.

The symptomatic bradycardic patient may present with altered level of consciousness and/or another indication for oxygen therapy and/or airway management.

Atropine increases automaticity and conduction at the SA and atrio-ventricular (AV) nodes and should be the first medication used in treating hemodynamically unstable bradycardia (**PEP 2 neutral**). Conduction blocks beyond the AV node, such as type II - 2nd degree AV blocks or 3rd degree AV blocks with wide QRS (i.e. high degree blocks) are unlikely to improve. If atropine is ineffective and/or the presenting rhythm is a high degree block, either adrenergic agents or transcutaneous pacing are reasonable next steps. Both approaches are effective and the decision of which to start will depend on clinician experience and available resources.

If atropine is ineffective, beta-adrenergic agonists such as dopamine infusion (**PEP 3 neutral**) can be used to treat hemodynamically unstable bradycardia. These agents increase both the heart rate (chronotropy) and contractility (inotropy) and can be used to improve perfusion and the clinical status of the patient.

In severe bradycardia causing shock or if no IV / IO access is available, immediate transcutaneous pacing (**PEP 2 neutral**) should be started while attempts are made to obtain access. Adequate analgesia or anxiolysis should be considered in conscious patients if there are no contraindications.

The goal of therapy for bradycardia is to improve the clinical status of the patient, not necessarily to achieve a particular heart rate. See Figure 1 for the American Heart Association algorithm for bradycardia.

Tachycardia

If the rhythm is sinus tachycardia, management involves treating the underlying condition (e.g. hypovolemia, pain, shock).

Treatment of tachycardia may include vagal maneuvers, pharmacological strategies, and/or synchronized cardioversion. A patient with a tachyarrhythmia is stable if they have no significant signs/symptoms or hemodynamic instability caused by the increased rate.

Stable Narrow Complex Tachycardia

If the rhythm is determined to be sinus tachycardia, look for the cause and treat as appropriate.

If the rhythm is a supraventricular tachycardia with a regular rate, attempt vagal maneuvers (**PEP 1 neutral**). Using a passive leg raise immediately after a sustained Valsalva maneuver increases chances of conversion (**PEP 1 supportive**). If this is unsuccessful, administer adenosine (**PEP 1 supportive**).

Stable rapid atrial fibrillation or flutter does not require intervention in the prehospital setting. Patients who are in atrial fibrillation/flutter for a prolonged or unknown duration have an increased risk of cardioembolic events after converting to normal sinus rhythm. It is only appropriate to cardiovert atrial fibrillation or flutter in the pre-hospital setting if the patient becomes unstable.

Wolff-Parkinson-White (WPW)

WPW is an accessory pathway syndrome, whereby electrical impulses can bypass normal AV node conduction. Any tachycardia at a rate over 200 bpm should raise the suspicion of an accessory pathway like WPW. The patient may give a personal or family history of WPW or sudden cardiac death at a young age. On ECG, WPW will have a short PR interval,

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wide QRS and a slurred upstroke to the QRS, referred to as a delta wave.

Stable or unstable? If the patient is unstable proceed with synchronized cardioversion.

If stable, is QRS narrow? If it is narrow, monitor en route to hospital and consider proceeding with standard SVT treatment.

If stable, is QRS wide? Avoid use of AV nodal blockers such as adenosine or amiodarone. Giving adenosine in this setting may precipitate ventricular fibrillation.

WPW may also present with rapid atrial fibrillation with irregular QRS complexes that may be narrow or wide. In these cases, the rate will be extremely fast. Avoid AV nodal blockers and monitor en route to hospital. Cardiovert if the patient becomes unstable.

Stable Wide Complex Tachycardia

If the patient is stable and has a wide complex tachyarrhythmia, administration of IV amiodarone (**PEP 1 supportive**) or lidocaine (**PEP 2 supportive**) should be considered.

Torsades de Pointes and Polymorphic VT

Torsades usually occurs as brief, recurring runs of unstable polymorphic ventricular tachycardia due to a prolonged QTc interval and often accompanied by bradycardia. Prolonged QTc can be caused by genetic syndromes, medications or electrolyte abnormalities such as hypomagnesemia, as occurs in a patient with alcohol use disorder. Cardioversion is often successful in terminating the arrhythmia but does not prevent recurrences. In patients with sustained or recurrent VT and a long QTc (greater than 500 msec), magnesium sulfate should be administered (**PEP White)**. Medications that prolong the QTc-interval (e.g. amiodarone) should be avoided in these cases. There is no role for magnesium for polymorphic VT with a normal QTc.

Synchronized cardioversion may not always be possible with polymorphic VT as synchronization is difficult due to the changes in QRS amplitude. An unstable patient in polymorphic VT, with a pulse, may require unsynchronized defibrillation if synchronization is not possible.

Unstable Wide or Narrow Tachycardia (Excluding Sinus Tachycardia)

If a patient has significant signs/symptoms or hemodynamic instability due to non-sinus tachycardia, they are considered to be unstable and require synchronized cardioversion (**PEP 3 supportive**). The energy level will depend on the underlying rhythm. Remember to consider sedation when using electrical cardioversion if there are no contraindications.

A patient in ventricular tachycardia without a pulse, any patient in polymorphic ventricular tachycardia, or those who are pre-arrest, will require a high-energy unsynchronized shock at defibrillation energy. Synchronized shocks can be delivered for unstable supraventricular tachycardia, atrial fibrillation, atrial flutter, or regular monomorphic ventricular tachycardia with a pulse. Further details can be found in the table below (Table 1).

Rhythm	Type of Shock	Initial (biphasic) Energy Setting*
Monomorphic ventricular tachycardia with a pulse	Synchronized	100J
Atrial flutter	Synchronized	50-100J
Supraventricular tachycardia	Synchronized	50-100J
Atrial fibrillation	Synchronized	120J
Monomorphic ventricular tachycardia without a pulse	Unsynchronized	120J
Polymorphic ventricular tachycardia	Unsynchronized	120J
Any patient who is pre-arrest	Unsynchronized	120J

*Energy levels can be escalated for subsequent shocks if the initial shock does not convert the rhythm.

Table 1. Electrical therapy for tachycardic rhythms

See Figure 2 for the American Heart Association algorithm for tachycardia.

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TRANSFER OF CARE

[1] Ensure clear communication of the symptom onset metrics to receiving healthcare team.
[2] Provide copies of the rhythm strips and 12 lead ECGs. It is critical for the attending cardiology team to see the initial underlying rhythm as they use this information to determine the treatment plan (e.g. implantable defibrillator, pacemaker etc.)
[3] Provide summary of medications and/or electrical therapy provided prior to arrival at the hospital.

CHARTING

Aside from all mandatory documentation components, ensure to attach relevant 12 lead ECGs to the ePCR and upload all monitor data to the server.

KNOWLEDGE GAPS

It is not always possible to update all local documents regarding brady and tachy-arrhythmias at the same rate at which international guidelines are changed. Please refer to current AHA guidelines for the most up-to-date information on best practices.

EDUCATION

Formal certification in ACLS and PALS will enable improved standardized care in the treatment of patients with brady- and tachy-arrhythmias. Clinicians are encouraged to maintain certification in these courses. Ongoing practice in scenario management can improve the care provided in these cases as well.

QUALITY IMPROVEMENT

It is critical that the attending crew clearly documents the stability of the patient, the identified rhythm, the corresponding treatment provided, and the patient's response to treatment.

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GUIDELINE CONTRIBUTORS

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Figure 1.
Adult Bradycardia Algorithm

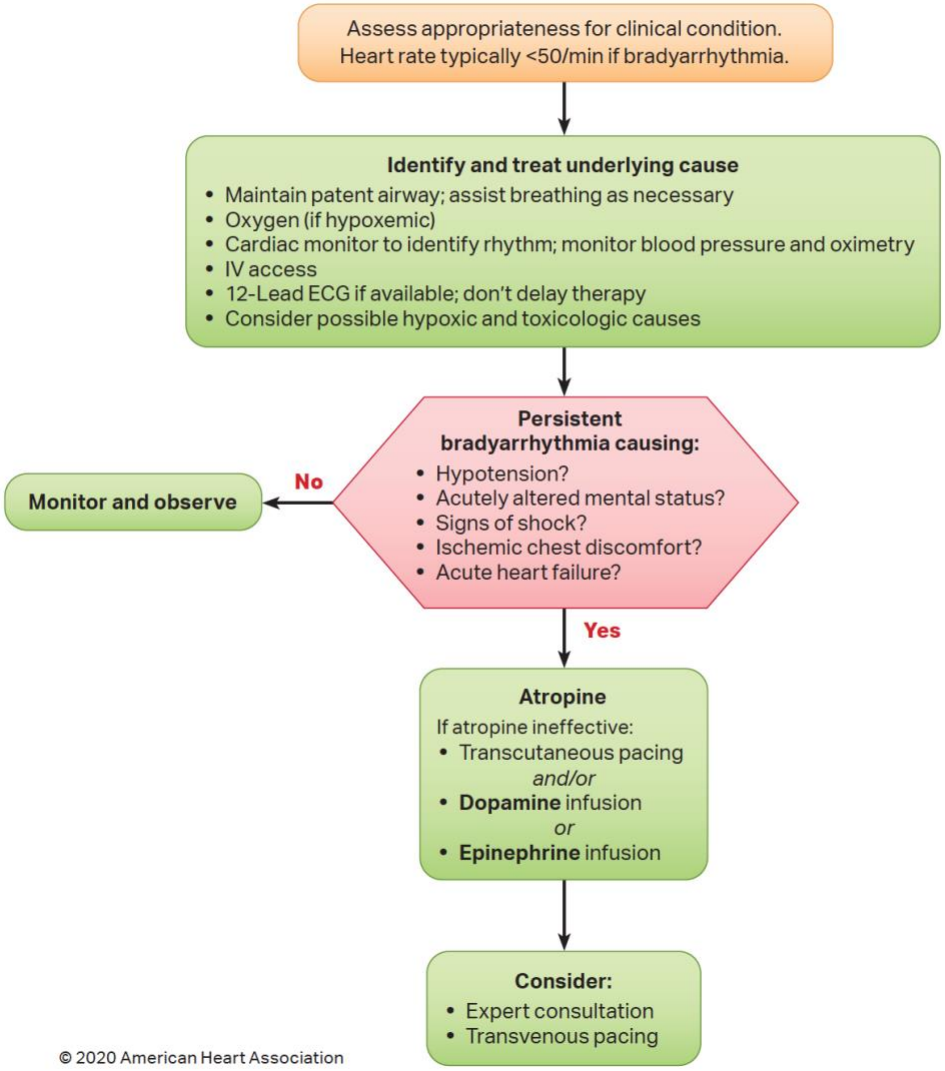

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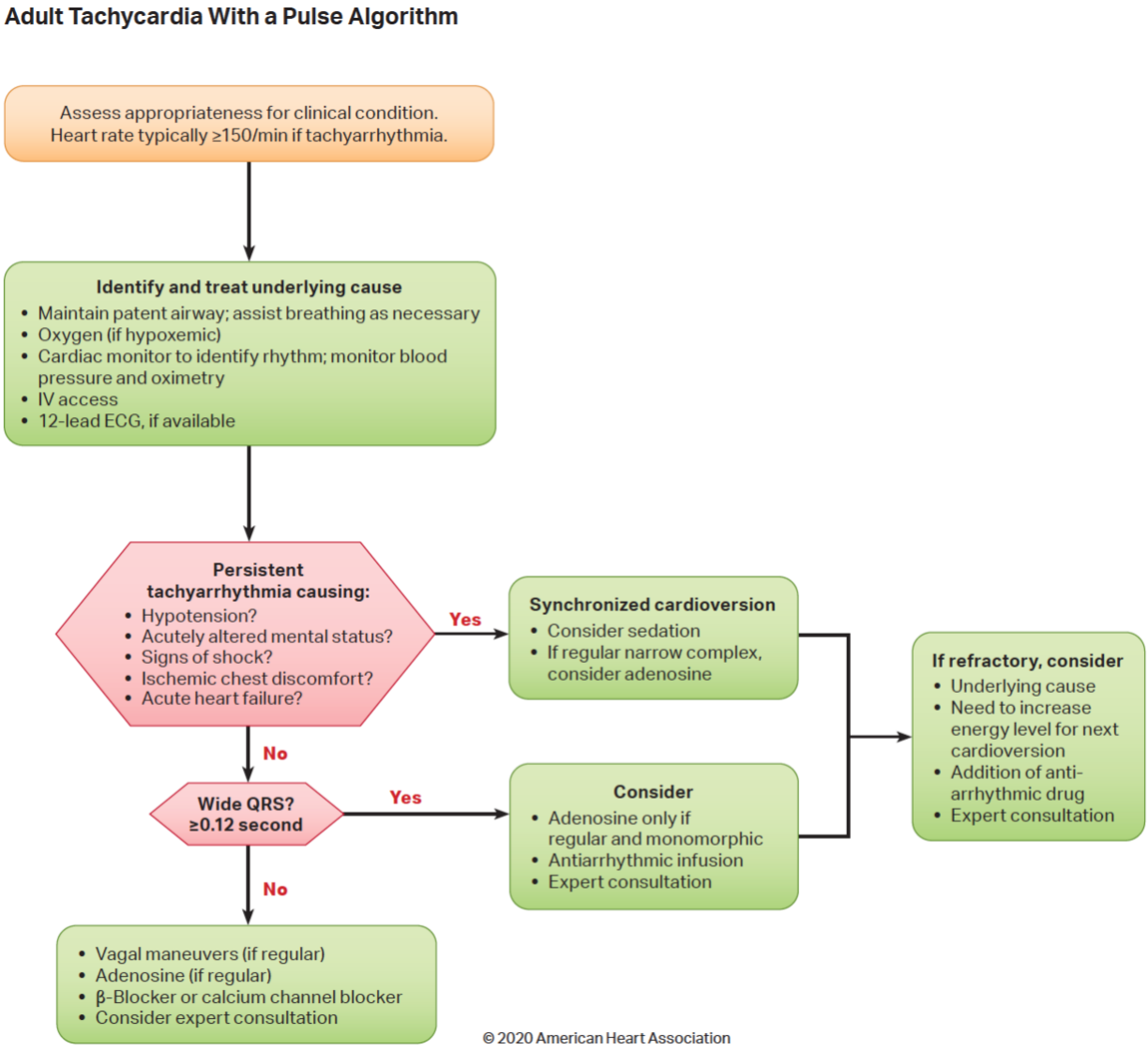
Figure 2.



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PEP 3x3 TABLES for ADULT CARDIAC ARRHYTHMIA

Throughout the EHS Guidelines, you will see notations after clinical interventions (e.g.: **PEP 2 neutral**).PEP stands for: the Canadian **P**rehospital **E**vidence-based **P**rotocols Project.

The number indicates the Strength of cumulative evidence for the intervention:
1 = strong evidence exists, usually from randomized controlled trials;
2 = fair evidence exists, usually from non-randomized studies with a comparison group; and
3 = weak evidence exists, usually from studies without a comparison group, or from simulation or animal studies.

The coloured word indicates the direction of the evidence for the intervention:
Green = the evidence is supportive for the use of the intervention;
Yellow = the evidence is neutral;
Red = the evidence opposes use of the intervention;
White = there is no evidence available for the intervention, or located evidence is currently under review.

PEP Recommendations for Adult Cardiac Arrhythmia Interventions, as of 2022/10/24. PEP is continuously updated. See: https://emspep.cdha.nshealth.ca/ for latest recommendations, and for individual appraised articles.

Bradycardia

Recommendation		RECOMMENDATION FOR INTERVENTION			
		SUPPORTIVE (Green)	NEUTRAL (Yellow)	AGAINST (Red)	NOT YET GRADED (White)
STRENGTH OF EVIDENCE FOR INTERVENTION	1 (strong evidence exists)				
	2 (fair evidence exists)		• Anticholinergic • Transcutaneous Pacing		
	3 (weak evidence exists)		• Inotrope		

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Stable Narrow Complex Tachycardia

Recommendation		RECOMMENDATION FOR INTERVENTION			
		SUPPORTIVE (Green)	NEUTRAL (Yellow)	AGAINST (Red)	NOT YET GRADED (White)
STRENGTH OF EVIDENCE FOR INTERVENTION	1 (strong evidence exists)	• Antiarrhythmic - Class I (Na+ channel blockers) • Antiarrhythmic - Class IV (Ca+ channel blockers) • Antiarrhythmic - Class V (other mechanism) • Electrical Cardioversion • Modified Valsalva	• Antiarrhythmic - Class III (K+ channel blockers) • Beta Blockers • Carotid Massage • Valsalva maneuver		• Fluid Bolus
	2 (fair evidence exists)				
	3 (weak evidence exists)	• Treat and Release-SVT	• Vagal Maneuvers		

Stable Wide Complex Tachycardia

Recommendation		RECOMMENDATION FOR INTERVENTION			
		SUPPORTIVE (Green)	NEUTRAL (Yellow)	AGAINST (Red)	NOT YET GRADED (White)
STRENGTH OF EVIDENCE FOR INTERVENTION	1 (strong evidence exists)	• Antiarrhythmic - Class III (K+ channel blockers)			• Magnesium/Polymorphic VT
	2 (fair evidence exists)	• Antiarrhythmic - Class I (Na+ channel blockers)	• Adenosine		
	3 (weak evidence exists)	• Electrical Cardioversion			

Unstable Tachycardia (Wide or Narrow Complex)

Recommendation		RECOMMENDATION FOR INTERVENTION			
		SUPPORTIVE (Green)	NEUTRAL (Yellow)	AGAINST (Red)	NOT YET GRADED (White)
STRENGTH OF EVIDENCE FOR INTERVENTION	1 (strong evidence exists)				
	2 (fair evidence exists)				
	3 (weak evidence exists)	• Electrical Cardioversion	• Vagal Maneuvers		

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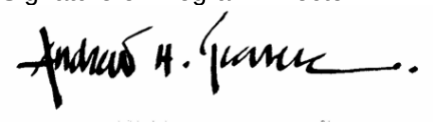
Signature of Program Director 	Signature of Program Document Coordinator *Electronically Signed* *Tanya Fraser*	

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Effective Date: April 2 2013	Revision Date:	
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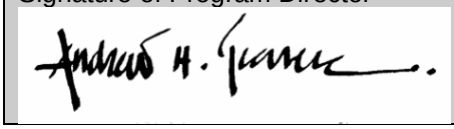
Review Date: April 1, 2013	Revision Date:	
Replaces: 6224.03	Revision Date:	
Signature of Program Director 	Signature of program Document Coordinator *Electronically Signed* *Tanya Fraser*	

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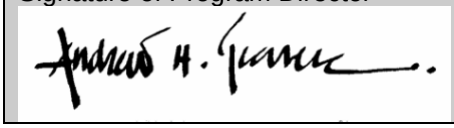
Review Date: April 1, 2013	Revision Date:	
Replaces: 6225.02	Revision Date:	
Signature of Program Director 	Signature of program Document Coordinator *Electronically Signed* *Tanya Fraser*	

EHS has made every effort to ensure that the information, tables, drawings and diagrams contained in the Clinical Practice Guidelines issued Q4 DHW 2022 is accurate at the time of publication. However, the EHS guidance is advisory and has been developed to assist healthcare professionals, together with patients, to make decisions about the management of the patient's health, including treatments. It is intended to support the decision making process and is not a substitute for sound clinical judgment. Guidelines cannot always contain all the information necessary for determining appropriate care and cannot address all individual situations; therefore individuals using these guidelines must ensure they have the appropriate knowledge and skills to enable appropriate interpretation.

PEP is the Canadian Prehospital Evidence-based Protocols Project. Every clinical intervention is given a recommendation based on the strength of available research evidence (1 = randomized controlled trials and systematic reviews of RCTs; 2 = studies with a comparison group; 3 studies without a comparison group or simulation) and direction of the compiled evidence: supportive of intervention; neutral evidence for intervention; or opposing evidence for intervention). See: https://emspep.cdha.nshealth.ca/