



Wide-complex tachycardias in the ED: how do we make good care even better?

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Linton et al. [1] describe a valuable snapshot of emergency physicians' approach to wide-complex tachycardias. Primary wide-complex tachycardias include malignant ventricular dysrhythmias that can rapidly deteriorate into cardiac arrest, and also include sinus, atrial and junctional tachycardias in patients with conduction system disease. Wide-complex tachycardias can also be precipitated by high-risk underlying causes, including toxic and metabolic emergencies and acute coronary syndromes. Distinguishing between the types and causes of wide-complex tachycardias, and treating them appropriately, is a fundamental skill for the emergency physician.

The findings of this study suggest emergency physicians are skilled at differentiating between, and appropriately treating, wide-complex tachycardias. Linton reports exceptionally high diagnostic accuracy of whether a primary or secondary etiology is driving the wide-complex tachycardia (98.1% and 96.1%, respectively) [1]. This is arguably the most important determination as it leads to an important branching point in the wide-complex tachycardia management algorithm. Primary wide-complex tachycardia should receive appropriate therapy according to ACLS guidelines, whereas suspicion for a secondary etiology should prompt an extensive search for underlying pathology and directed therapy. Linton provides some insight into features suggestive of primary arrhythmia (vs secondary) including more severe tachycardia (heart rate ≥ 140 : 47.7% vs 26.1%), sudden onset (79.1% vs 44.0%), presenting with palpitations (53.6% vs 5.2%), afebrile (99.3% vs 90.8%) and a Glasgow Coma Score of 15 (92.2% vs 61.1%) [1].

Once primary arrhythmia is recognized, the emergency physician's focus should be on appropriate rhythm or rate control. To select the best strategy requires identification of the wide-complex tachycardia. This cognitive task probably poses the greatest challenge in the acute setting. Linton reports that we are better at diagnosing certain wide-complex tachycardias (Atrial Fibrillation, Ventricular Tachycardia) than others (Supraventricular Tachycardia, Atrial Flutter) but overall have high agreement with cardiologists' ECG interpretation (81.2%) [1]. Specific ECG findings can differentiate aberrant conduction from ventricular tachycardia [2]. Awareness of ECG findings pathognomonic for toxicologic and metabolic causes is also essential [2].

However, determining the exact wide-complex tachycardia is not always required in the acute setting. If the rhythm is regular and wide, it is safer to have a working diagnosis of Ventricular Tachycardia than Supraventricular Tachycardia or Atrial Flutter especially if the patient is older, has a history of cardiac disease or is unwell. If the rhythm is irregular and wide, deciding between Atrial Flutter and Fibrillation matters less than the stability, timing of onset, appropriate choice of rate versus rhythm control, or treatment of an underlying trigger. That said, long-term management after the ED visit is aided by specific identification of the presenting rhythm.

If diagnostic clarification is required, there are tools to assist with this. The presence of conduction system abnormalities with a similar morphology on prior ECGs is most helpful. Linton proposes the use of adenosine (in consultation with cardiology) to help identify underlying atrial rhythms. While this can be useful, other approaches may avoid the unpleasant patient experiences associated with adenosine administration. These include use of the Lewis lead, changing the ECG speed from 25 to 50 mm/s to spread out the distance between QRS complexes, or use of vagal maneuvers.

This study indicates that, in general, emergency physicians manage wide-complex tachycardias well, with 93.5%

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receiving appropriate pharmacological or electrical therapy [1]. Where improvement can be made is by adapting a ‘less is more’ approach for primary arrhythmia patients. Almost 40% of these patients received ancillary therapy, namely fluid boluses, and diuretics [1]. Unless indicated for hemodynamic stability or treatment of concomitant acute heart failure, it is better to avoid these interventions in patients who likely have underlying structural disease and may struggle with sudden fluid shifts.

A discharge heart rate target (less than 100 BPM resting or 110 BPM ambulating) is recommended in primary atrial dysrhythmias to prevent adverse outcomes from prolonged ventricular rate [3]. This is a reasonable goal for any wide-complex tachycardia that is not due to a secondary process, however, was not achieved in 9.1% of patients with a primary wide-complex tachycardia discharged in this study [1]. While rate reduction should not be an objective in patients with tachycardias driven by other causes, persistent tachycardia should alert physicians to a higher risk of adverse outcomes (for example in patients with pulmonary embolism) or of inadequate treatment of the underlying cause (for example septic or hypovolemic shock). Physicians considering discharging patients with persistent tachycardia, should reflect carefully whether discharge is appropriate, or whether a longer period of observation or admission may be more prudent.

Anticoagulation was prescribed in all but 3 (2.0%) cases where it was indicated for stroke prevention [1]. This reflects excellent practice by emergency physicians, and a significant improvement from prior studies. Atzema et al. [4] reported only 18.9% of older adults with atrial fibrillation received a prescription for anticoagulation in the ED between 2009 and 2014.

While anticoagulation was appropriately provided to most patients in this study, it is worth reminding physicians that initiation of anticoagulation in the ED is important to reduce the short-term risk of stroke in patients treated in the ED for atrial fibrillation. The 2021 CAEP Atrial Fibrillation/Flutter Best Practices Checklist provides a detailed discussion on this [3]. Any patient with one or more CHADS-65 criteria (age \geq 65, congestive heart failure, hypertension, diabetes,

stroke/TIA) should be initiated on long-term therapy to reduce stroke risk [3]. A 30-day course of anticoagulation can be considered for patients with a CHADS-65 score of 0. Although cardioembolic events after ED cardioversion are rare, patients will experience an embolic event typically do so in the first few days after cardioversion, meaning that timely initiation of anticoagulation in the ED, rather than deferring to primary care follow-up, will best mitigate the stroke risk [5]. Moreover, prescribing anticoagulation in the ED, rather than deferring to primary care providers, doubles the rate of successful prescription completion in patients for whom anticoagulation is indicated [4].

EM physicians in this study managed wide-complex tachycardias particularly well, but the study gives us opportunities to reflect on optimizing management, particularly with respect to differentiation between primary and secondary wide-complex tachycardias, appropriate anticoagulation of patients at risk for stroke, limiting unnecessary therapeutic interventions, and making prudent disposition decisions.

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