



Review

Initial evaluation and management of wide-complex tachycardia: A simplified and practical approach

Laszlo Littmann, MD, PhD^{a,*}, Elizabeth G. Olson, MD^b, Michael A. Gibbs, MD^b^a Department of Internal Medicine, Carolinas Medical Center, Charlotte, NC, United States^b Department of Emergency Medicine, Carolinas Medical Center, Charlotte, NC, United States

ARTICLE INFO

Article history:

Received 11 February 2019

Received in revised form 10 April 2019

Accepted 12 April 2019

Keywords:

Electrocardiogram

Wide-complex tachycardia

Supraventricular tachycardia

Ventricular tachycardia

Atrial fibrillation

Wolff-Parkinson-White syndrome

ABSTRACT

The evaluation and treatment of wide QRS-complex tachycardia remains a challenge, and mismanagement is quite common. Diagnostic aids such as wide-complex tachycardia algorithms perform poorly in the real-life setting. The purpose of this review is to offer a simple clinical-electrocardiographic approach for the initial evaluation and management of the adult patient with stable wide-complex tachycardia that does not require recollection of complex guidelines or algorithms.

© 2019 Elsevier Inc. All rights reserved.

1. Introduction

Sustained wide-complex tachycardia (WCT), that is, WCT recorded in a 12-lead electrocardiogram (ECG), is a common presentation in the emergency department and in other acute-care settings. The differential diagnosis includes ventricular tachycardia (VT) and supraventricular tachycardia (SVT), including atrial fibrillation, with aberrant intraventricular conduction. Aberrant conduction of SVT comprises right bundle branch block (RBBB), left bundle branch block (LBBB), non-specific intraventricular conduction block and ventricular preexcitation as seen in the Wolff-Parkinson-White (WPW) syndrome [1,2]. Under specific clinical circumstances, metabolic abnormalities such as severe hyperkalemia or sodium-channel blocker toxicity may also manifest as WCT in the ECG [3]. As the treatment of the listed conditions is vastly different, reaching a quick and accurate diagnosis is paramount. Several studies have shown that diagnostic aids such as WCT algorithms perform poorly at the bedside with resultant misdiagnosis, mismanagement and potential harm to the patient [1,4,5]. The purpose of this review is to provide the clinician with a simple, clinically-oriented method that does not require sophisticated knowledge of the ECG or recollection of complex guidelines and algorithms. Fig. 1 summarizes our approach to the diagnosis and management of the adult emergency department patient with stable WCT.

2. Is the tachycardia regular or irregular?

This should be a relatively easy distinction which only requires a brief review of the ECG. Even irregular tachycardias have regular portions but if there are any obvious irregular segments, the WCT should be considered to be irregular. It is important to understand that the faster the heart rate, the less irregularity one can expect. In less than obvious cases, the use of a caliper is encouraged for the assessment of regularity vs. irregularity.

3. Regular wide-complex tachycardia

3.1. Clinical likelihoods

Although estimations vary regarding the percentage of patients with regular WCT having SVT or VT, it is generally believed that approximately 80% of all regular WCTs are VT [2,6-12]. Most studies that established this high likelihood of VT, however, were done at cardiology referral centers. Unfortunately, there are no prospective studies on WCT cases in the emergency department setting where the percentage of patients with VT is probably lower. Nevertheless, most experts believe that in patients with structural heart disease, a history of myocardial infarction, angina or systolic heart failure, the majority of WCT cases are VT (Fig. 1) [1,2,6-12]. It is important to recognize that WCT algorithms are too complex to be easily recalled by the acute care provider [13-15]. In addition, their accuracy in the real-life setting ranges from 60 to 75% [7,16-19]. The use of WCT algorithms, therefore, is less useful than establishing the clinical likelihood. Adult advanced cardiac life

* Corresponding author at: Department of Internal Medicine, Carolinas Medical Center, P.O. Box 32861, Charlotte, NC 28232, United States.

E-mail address: Laszlo.Littmann@atriumhealth.org (L. Littmann).

CLINICALLY STABLE WIDE-COMPLEX TACHYCARDIA		
<p>REGULAR: UP TO 80% VT</p> <ul style="list-style-type: none"> • H/o MI, CHF, valvular HD: 90-95% VT • Dissociated P waves: 100% VT • QRS concordance in V1-V6: 90-95% VT • Negative QRS concordance: near 100% VT • Suspect SVT: give IV adenosine 	<p>IRREGULAR: 90% AF</p> <p>AF with BBB</p> <p>RATE: 120-160</p> <p>MORPHOLOGY: RBBB/LBBB</p> <p>MORE LIKELY IN ELDERLY PT WITH HEART DISEASE</p>	<p>AF with WPW</p> <p>very fast (>180/min)</p> <p>bizarre</p> <p>FBI: FAST, BIZARRE, IRREGULAR</p> <p>MORE LIKELY IN THE YOUNG, HEALTHY</p>
<p>SYNCHRONIZED ELECTRIC CARDIOVERSION ESPECIALLY IF RATE IS VERY FAST OR POOR LV FUNCTION SEDATION WITH ETOMIDATE OR PROPOFOL</p>		
<p>Monophasic or biphasic: 100 J</p>		<p>Monophasic: 200 J Biphasic: 120-200 J</p>
<p>PHARMACOLOGIC THERAPY</p>		
<ul style="list-style-type: none"> • #1: IV procainamide 10 mg/kg/20 min • #2: IV amiodarone 5 mg/kg/20 min 	<ul style="list-style-type: none"> • diltiazem • beta blocker • digoxin • amiodarone 	<ul style="list-style-type: none"> • #1: IV procainamide • #2: IV ibutilide <p>AVOID: verapamil, diltiazem, digoxin, adenosine, amiodarone</p>

Fig. 1. Combined clinical and electrocardiographic approach to the initial evaluation and management of the adult patient with clinically stable wide-complex tachycardia (WCT). Abbreviations: AF = atrial fibrillation; BBB = bundle branch block; CHF = chronic heart failure; HD = heart disease; IV = intravenous; LBBB = left bundle branch block; MI = myocardial infarction; PO = oral route; PT = patient; RBBB = right bundle branch block; SVT = supraventricular tachycardia; VT = ventricular tachycardia; WPW = Wolff-Parkinson-White syndrome.

support (ACLS) guidelines, appropriately, do not recommend the use of WCT algorithms [20].

3.2. Electrocardiographic diagnosis

Based on the listed clinical likelihoods, the default diagnosis of WCT should be “probable VT until proven otherwise [1,9].” Simple ECG observations can further increase the probability of VT (Fig. 1). If an

inconsistent P-QRS relationship is present such as in atrioventricular (AV) dissociation, the WCT is almost certainly VT [1,9,10,21]. In our experience, approximately half of the cases of VT demonstrate visible AV dissociation. It is always useful to spend one or two minutes with the help of a caliper to find sharp “notches” indicative of P waves in one of the rhythm strips as proof of an inconsistent P-QRS relationship (Figs. 2–4). If AV dissociation cannot be ascertained, a simple QRS morphological sign will still make the diagnosis of VT very likely: the presence of

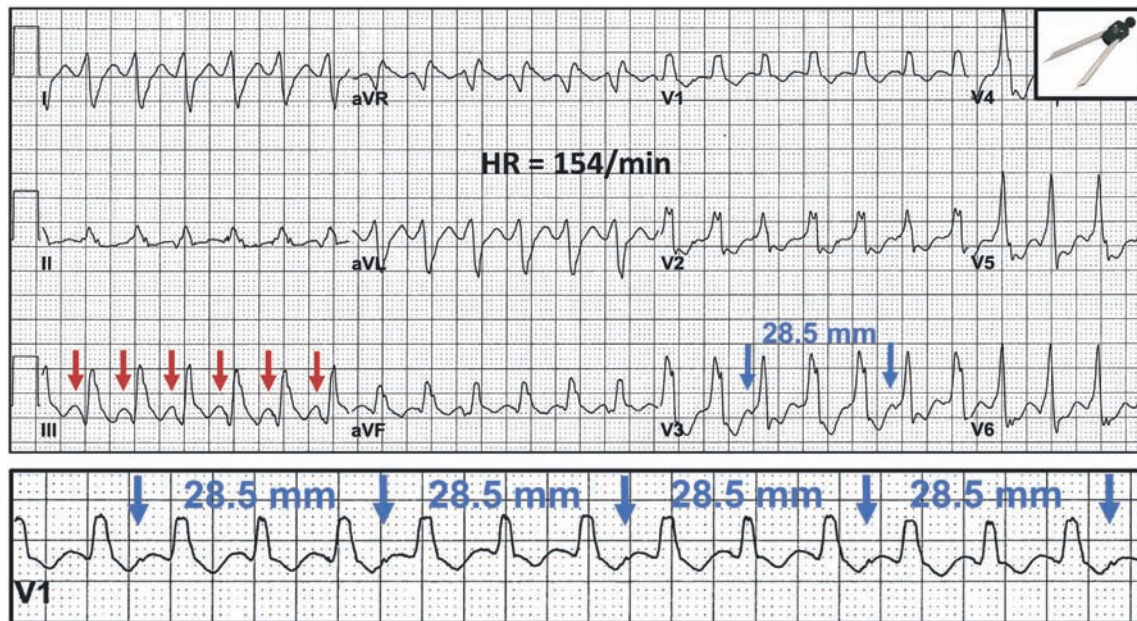


Fig. 2. ECG recorded from a 62-year-old woman with dilated cardiomyopathy who presented with a 2-day history of heart failure exacerbation. By numerous consecutive providers, the ECG was read as sinus tachycardia with bifascicular block. Red arrows indicate what were believed to be sinus P waves. This case highlights several clinical and ECG traps including diagnostic anchoring, the “P-wave trap” and the “QRS morphology trap” (see text). It was ignored that in a patient with heart failure, WCT almost always represents ventricular tachycardia (VT). In an enlarged lead V1 dissociated P waves are clearly visible (blue arrows) firmly establishing the diagnosis of VT. QRS concordance is also present.

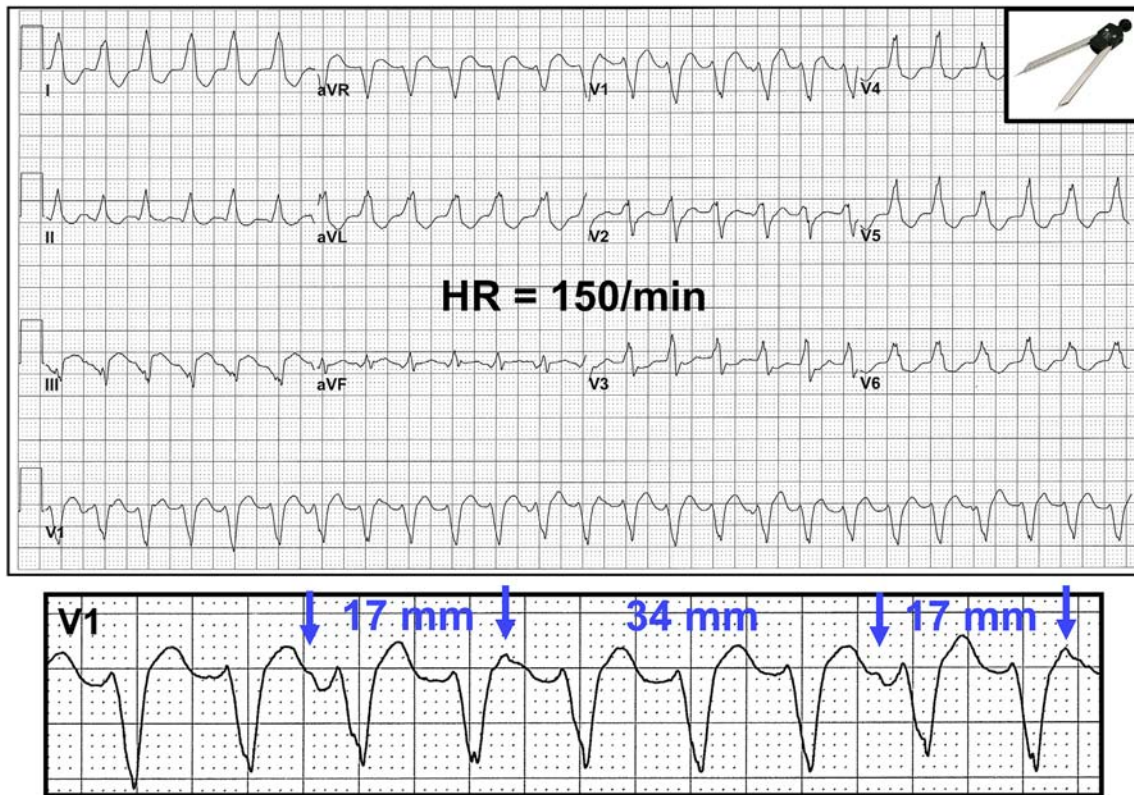


Fig. 3. ECG recorded from a 74-year-old man with remote history of inferior MI who presented with a 4-day history of palpitation and mild shortness of breath. In the emergency department, he was tachycardic but in no distress. By numerous consecutive providers, the ECG was read as tachycardia with left bundle branch block (diagnostic anchoring and the “QRS morphology trap” – see text). In a patient with history of MI, however, 90–95% of WCTs is VT. In the enlarged lead V1 on the bottom dissociated P waves are clearly visible (arrows) firmly establishing the diagnosis of VT.

QRS concordance in the chest leads (i.e., in all six chest leads the QRS complexes go in the same direction, either up or down) (Fig. 2). A negative QRS concordance almost always signifies VT (Fig. 1) [1,10,21,22].

During WCT, one can occasionally detect the presence of QRS complexes that are narrower and have a more normal morphology than that of the wide-QRS beats. These are fusion beats. Fusion beats denote ventricular activation from two separate sites. In regular WCT, fusion beats are essentially diagnostic of VT as they reflect simultaneous

ventricular activation from a ventricular site (the VT) and from conducted atrial impulses (Fig. 4) [23].

3.3. Clinical and ECG traps to avoid

A common clinical trap that frequently results in misdiagnosis is automatically adopting a previous clinician’s false diagnosis (cognitive

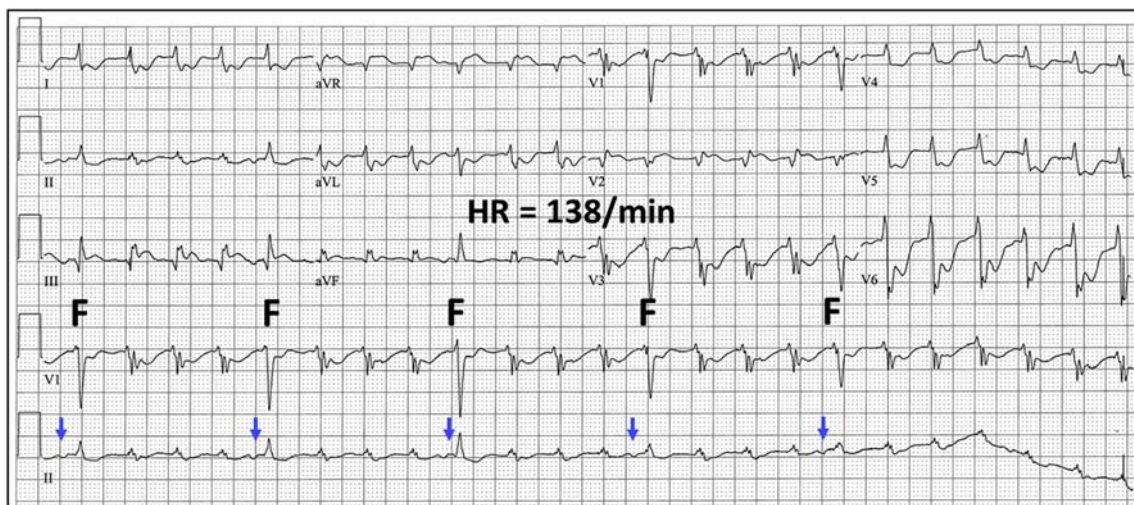


Fig. 4. ECG recorded from a 57-year-old woman with end-stage liver disease and upper GI bleed. Because of suspected supraventricular tachycardia, IV adenosine was administered three times, with no success. The tachycardia finally converted with electric shock. Note the presence of fusion complexes (F) whose morphology is more normal than that of the other beats. Each fusion complex is preceded by a P wave (blue arrows); this finding also established A-V dissociation and confirmed the diagnosis of VT.

transfer or anchoring) (Figs. 2 and 3). When dealing with WCT, each provider should independently assess the clinical and ECG likelihoods of SVT and VT. The second clinical trap is stating that “the patient is too well for the tachycardia to be VT” (Fig. 3). Patients with VT can be clinically stable on presentation; instability is frequently the result of a missed diagnosis and inappropriate treatment [1,4,5,9,10]. One of the ECG traps to avoid is the “P-wave trap,” focusing too much on a possible 1:1 P-QRS relationship (Fig. 2). First, true 1:1 P-QRS relationship can be due to the occasional patient with VT who has 1:1 retrograde conduction. Second, one can never be sure that what appear to be P waves is not part of the ST-T segments (Fig. 2). The second ECG trap is the “QRS morphology trap” when the first responder prematurely focuses on the QRS morphology during WCT (Figs. 2 and 3). As we have seen, the clinical scenario and the finding of AV dissociation should supersede the finding of a resemblance of the QRS complexes to bundle branch blocks or bifascicular blocks. The combination of a younger, healthier patient whose ECG suggests a 1:1 P-QRS relationship and typical bundle branch block morphology does increase the likelihood of SVT, but one still cannot be certain.

3.4. Therapy

If SVT is suspected, as in the scenario discussed above, conversion with intravenous (IV) adenosine strongly supports that assessment (Fig. 1) [24]. If rapid IV push of an appropriate dose of adenosine does not terminate the WCT, it should be considered to be probable VT (Fig. 4) [25]. In questionable cases the use of IV adenosine can be very helpful: if the cause of WCT is uncertain, it can help define the correct diagnosis, and if the diagnosis turns out to be SVT, then adenosine is actually the most appropriate treatment of choice. It should be noted that a small percentage of VTs can also be converted with adenosine, so while this medication can be useful diagnostically, a successful application of adenosine does not completely exclude VT [26]. From the perspective of the acute care provider, however, the most important fact is that the tachycardia terminated. Moreover, the use of IV adenosine in WCT has been shown to be safe, regardless of SVT or VT [26].

In WCT that is presumed to be VT (and this is the vast majority of cases), patients who appear to be unstable should undergo direct current electric cardioversion. Electric cardioversion is also a reasonable first line treatment for stable patients with WCT, especially when the heart rate is very fast or when bedside echocardiography identifies poor left ventricular systolic function (Fig. 1). As we will see, pharmacologic cardioversion takes time, whereas electric cardioversion can be applied almost immediately. Also, the success rate of electrical cardioversion for stable VT is approximately 95%, much higher than the success rate of pharmacological cardioversion [27]. For sedation, etomidate or propofol are the preferred agents (Fig. 1) [28]. The recommended energy for cardioversion in regular WCT is 100 joules, regardless of monophasic or biphasic shock (Fig. 1) [20].

In clinically stable patients with WCT, ACLS allows one attempt at pharmacologic cardioversion. Outside of the acute myocardial infarction setting, lidocaine is one of the least effective agents for conversion of VT. In contrast to widespread beliefs, IV procainamide is both more effective and is safer than IV amiodarone for the acute conversion of VT (Fig. 1) [25,29]. In a recent double-blind randomized trial, IV procainamide at a dose of 10 mg/kg given over 20 min (a dose that is lower than typically used in the United States) converted approximately two thirds of WCTs, whereas IV amiodarone administered as 5 mg/kg over 20 min (a dose higher than typically used in the United States) only converted one third of the tachycardias [25]. Major cardiovascular side effects were much less common with procainamide than with amiodarone [25]. Of note, IV procainamide is the only agent that carries a class IIA indication for the pharmacologic conversion of VT. As such, in the ACLS guidelines IV procainamide is listed ahead of IV amiodarone [20]. On the other hand, IV amiodarone is the most effective agent in preventing recurrences of VT, and therefore, its use in repetitive VT is appropriate [20].

Frequently, IV procainamide is used for the termination of VT, followed by IV amiodarone for the prevention of recurrence. The safety of administering procainamide and amiodarone in sequence, however, has not been clearly established.

4. Irregular wide-complex tachycardia

4.1. Clinical-electrocardiographic diagnosis

In contrast to regular WCTs, sustained irregular WCT is almost never VT (Fig. 1). Here, the default diagnosis should be atrial fibrillation (AF) with bundle branch block, nonspecific intraventricular conduction block or ventricular preexcitation, the Wolff-Parkinson-White syndrome. AF with bundle branch block is the likely diagnosis when the ventricular rate is approximately 120–160 bpm and the QRS morphology is consistent with RBBB or LBBB (Fig. 1). AF with ventricular preexcitation should be considered when the ventricular rate is excessive (e.g., ≥ 180 bpm) and the QRS morphology is bizarre (Figs. 1 and 5). This scenario is frequently called “FBI” for fast, broad-complex and irregular [30]. However, AF with bundle branch block is also fast, broad-complex and irregular. The abbreviation FBI can be kept but it should stand for fast, bizarre and irregular. The term “pseudoventricular tachycardia” is also appropriate because the QRS morphology usually looks like VT but the irregular rhythm suggests that it is not VT (Fig. 5). In irregular WCT, fusion complexes reflect ventricular activation through the AV node and, simultaneously, through an accessory pathway and therefore are diagnostic of AF with WPW (Fig. 5).

With irregular WCTs too, the clinical presentation is important. AF with bundle branch block is more likely in elderly patients and in patients with structural heart disease. AF with ventricular preexcitation is more common in patients without structural heart disease (Fig. 5). With irregular WCT, however, the clinical context is less predictive of the cause of the tachycardia than in regular WCT. Hence, the ECG characteristics are more important in distinguishing AF with bundle branch block vs. AF with ventricular preexcitation (Fig. 1).

Sustained irregular *ventricular tachycardia* is rare. Ventricular tachycardia as the cause of an irregular WCT should be suspected in elderly patients with structural heart disease when the QRS morphology is bizarre but the ventricular rate is relatively slow such as 120 bpm. In such cases, as in regular WCT, it is useful to search for signs of AV dissociation which would nail down the diagnosis of VT.

4.2. Therapy

AF with bundle branch block should be treated according to atrial fibrillation guidelines. The initial step should be ventricular rate control with IV diltiazem or beta blocker (Fig. 1) [20]. If AF with WPW is suspected, the most important decision is to avoid AV nodal blocking agents such as IV adenosine, verapamil, diltiazem, beta blockers and digoxin (Fig. 1). The use of IV amiodarone can also be detrimental [31]. As is the case with regular WCT, here too electric cardioversion is a reasonable initial treatment option, especially if the heart rate is very fast or the patient has poor left ventricular systolic function (Fig. 1). After sedation with etomidate or propofol, monophasic shock should be delivered at 200 joules and biphasic shock at 120–200 joules [20]. If pharmacologic therapy is chosen, the first line agent should be IV procainamide which can suppress conduction through the accessory pathway (Fig. 1). IV ibutilide or, in very stable patients, oral flecainide or propafenone can also be used.

5. Applicability and limitations

This review is only applicable to the adult patient who presents with clinically stable monomorphic WCT. It was not our purpose to elaborate on the evaluation and management of polymorphic VT, pulseless WCT or electrical storm. Our approach is primarily intended for care

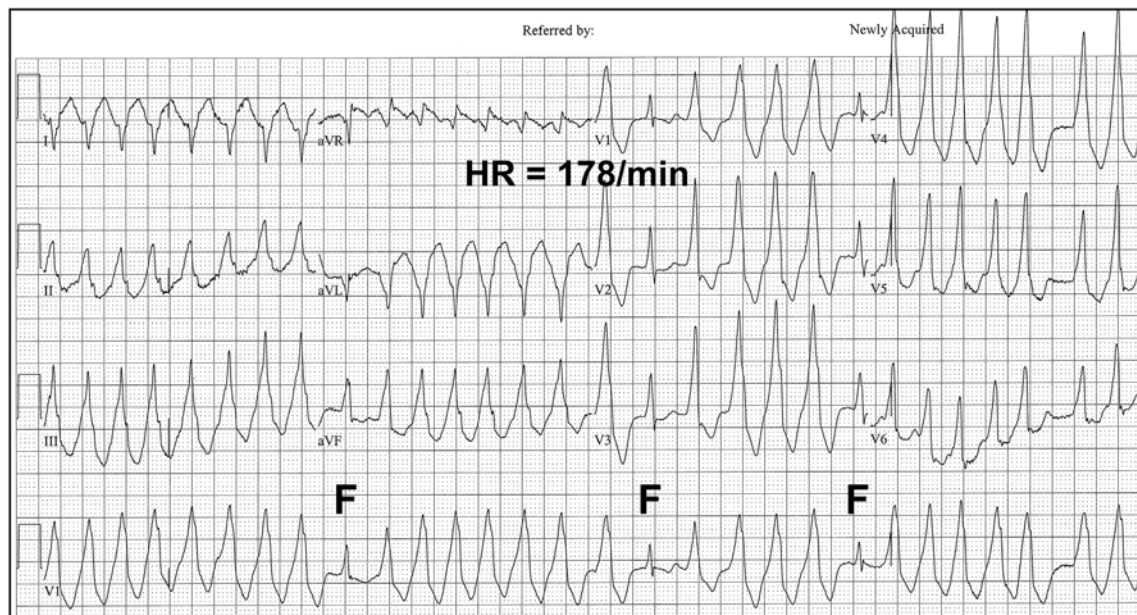


Fig. 5. ECG recorded from a 44-year-old woman with no known heart disease who presented with palpitation and lightheadedness. Note the irregularly irregular ventricular rhythm at a rate of 178 bpm, the bizarre QRS morphology resembling VT (deep negative QRS complexes in lead I; QRS concordance in the chest leads), and the occasional presence of narrower QRS complexes consistent with fusion beats (F). This clinical and ECG scenario was typical for atrial fibrillation and ventricular preexcitation, the Wolff-Parkinson-White syndrome.

delivered in the emergency department by emergency medicine practitioners (EMPs). This approach may be preferable in this setting to ACLS guidelines given that (1) acute management of arrhythmias should be an area of relative expertise for EMPs and (2) by definition, stable arrhythmia will allow for the minimal time required to collect such history as demographics and prior cardiovascular conditions. It is uncertain if our recommendations lend themselves to settings other than the emergency department as, for instance, in an intensive care unit where the proportions outlined here for regular WCT (i.e., 80% of regular WCT is VT) may not apply.

The presented approach does not cover rare forms of WCT such as WPW with antidromic reentrant supraventricular tachycardia or regular pre-excited atrial tachycardia [32–34]. These diagnoses, however, are almost never made at the initial clinical encounter. In addition, such conditions typically present with regular WCT resembling VT, and the use of IV procainamide is appropriate.

Our approach to the initial evaluation and management of stable WCT has not been systematically tested for accuracy and patient outcomes. It would be useful to perform a prospective study in the real-life setting to test what proportion of WCT ECGs will still be ambiguous after evaluation as illustrated in Fig. 1, and to track the outcomes of patients who were managed according to the described approach.

6. Summary

In cases of WCT, the ECG should never be evaluated without consideration of the clinical context. Regular WCT in elderly patients and in patients with structural heart disease is almost always VT. Demonstration of an inconsistent P-QRS relationship essentially confirms the diagnosis of VT. If the rate is very fast or the patient has poor left ventricular systolic function, electric cardioversion should probably be performed. If the rate is not excessive and the tachycardia is tolerated, give IV procainamide. In younger patients with no known heart disease, especially if the QRS morphology resembles bundle branch block, SVT is somewhat more likely; prove it with IV adenosine. Irregular WCT in an elderly patient and in the patient with structural heart disease is almost always AF with bundle branch block; treat it according to AF guidelines. In patients with no known heart disease, especially when the ventricular rate is very fast and the QRS complexes are bizarre (fast, bizarre, irregular –

FBI), AF with WPW is the likely diagnosis. When the heart rate is excessive or the patient has poor left ventricular systolic function, direct current electric cardioversion should be employed. If the ventricular rate is not excessive and the tachycardia is tolerated, treat it with IV procainamide. Patients with any type of WCT that causes clinical or hemodynamic instability should undergo immediate electric cardioversion.

References

- [1] Alzand BSN, Crijns HJGM. Diagnostic criteria of broad complex tachycardia: decades of evolution. *Europace* 2011;13:465–72.
- [2] Brady WJ, Mattu A, Tabas J, Ferguson JD. The differential diagnosis of wide QRS complex tachycardia. *Am J Emerg Med* 2017;35:1525–9.
- [3] Hollowell H, Mattu A, Perron AD, Holstege C, Brady WJ. Wide-complex tachycardia: beyond the traditional differential diagnosis of ventricular tachycardia vs supraventricular tachycardia with aberrant conduction. *Am J Emerg Med* 2005;23:876–89.
- [4] Dancy M, Camm AJ, Ward D. Misdiagnosis of chronic recurrent ventricular tachycardia. *Lancet* 1985;326:320–3.
- [5] Stewart RB, Bardy GH, Greene HL. Wide complex tachycardia: misdiagnosis and outcome after emergent therapy. *Ann Intern Med* 1986;104:766–71.
- [6] Baerman JM, Morady F, DiCarlo Jr LA, de Buitler M. Differentiation of ventricular tachycardia from supraventricular tachycardia with aberration: value of the clinical history. *Ann Emerg Med* 1987;16:40–3.
- [7] Steinman RT, Herrera C, Schugger CD, Lehmann MH. Wide QRS tachycardia in the conscious adult: ventricular tachycardia is the most frequent cause. *JAMA* 1989;261:1013–6.
- [8] Reinelt P, Karth G, Geppert A, Heinz G. Incidence and type of cardiac arrhythmias in critically ill patients: a single center experience in a medical-cardiological ICU. *Intensive Care Med* 2001;27:1466–73.
- [9] Goldberger ZD, Rho RW, Page RL. Approach to the diagnosis and initial management of the stable adult patient with a wide complex tachycardia. *Am J Cardiol* 2008;101:1456–66.
- [10] Garner JB, Miller JM. Wide complex tachycardia – ventricular tachycardia or not ventricular tachycardia, that remains the question. *Arrhythmia Electrophysiol Rev* 2013;2:23–9.
- [11] Tchou P, Young P, Mahmud R, Denker S, Jazayeri M, Akhtar M. Useful clinical criteria for the diagnosis of ventricular tachycardia. *Am J Med* 1988;84:53–6.
- [12] Akhtar M, Shenasa M, Jazayeri M, Caceres J, Tchou PJ. Wide QRS complex tachycardia: reappraisal of a common clinical problem. *Ann Intern Med* 1988;109:905–12.
- [13] Vereckei A. Current algorithms for the diagnosis of wide QRS complex tachycardias. *Curr Cardiol Rev* 2014;10:262–76.
- [14] Jastrzębski M, Kukla P, Czarnecka D. Ventricular tachycardia score: a novel method for wide QRS complex tachycardia differentiation – explained. *J Electrocardiol* 2017;50:704–9.
- [15] El Hajjai I, Becker G, Kus T, Vinet A, Berkovitz A, Sturmer M. Novel criterion for the differential diagnosis of wide QRS complexes and wide complex tachycardia using the initial activation of QRS on leads V1 and V2: differential diagnosis of wide QRS based on V1-V2. *J Electrocardiol* 2018;51:700–6.

- [16] Isenhour JL, Craig S, Gibbs M, Littmann L, Rose G, Risch R. Wide-complex tachycardia: continued evaluation of diagnostic criteria. *Acad Emerg Med* 2000;7:769–73.
- [17] Jastrzebski M, Kukla P, Czarnecka D, et al. Comparison of five electrocardiographic methods for differentiation of wide QRS-complex tachycardias. *Europace* 2012;14:1165–71.
- [18] Baxi RP, Hart KW, Vereckei A, Miller J, Chung S, Chang W, et al. Vereckei criteria as a diagnostic tool amongst emergency medicine residents to distinguish between ventricular tachycardia and supra-ventricular tachycardia with aberrancy. *J Cardiol* 2012;59:307–12.
- [19] May AM, Brenes-Salazar JA, DeSimone CV, Vaidya VR, Ternus BW, Hodge DO, et al. Electrocardiogram algorithms used to differentiate wide complex tachycardias demonstrate diagnostic limitations when applied by non-cardiologists. *J Electrocardiol* 2018;51:1103–9.
- [20] Morrison LJ, Deakin CD, Morley PT, Callaway CW, Kerber RE, Kronick SL, et al. Part 8: advanced life support: 2010. International consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. *Circulation* 2010;122:S345–421.
- [21] Lau EW, Pathamanathan RK, Ng GA, Cooper J, Skehan JD, Griffith MJ. The Bayesian approach improves the electrocardiographic diagnosis of broad complex tachycardia. *Pacing Clin Electrophysiol* 2000;15:19–26.
- [22] Zauner CA, Holshouser JW, Littmann L. Ventricular tachycardia and electrocardiographic ST-segment-elevation myocardial infarction without coronary artery disease. *Circulation* 2018;137:1287–9.
- [23] Brady WJ, Skiles J. Wide QRS complex tachycardia: ECG differential diagnosis. *Am J Emerg Med* 1999;17:376–81.
- [24] Griffith MJ, Ward DE, Linker NJ, Camm AJ. Adenosine in the diagnosis of broad complex tachycardia. *Lancet* 1988;331:672–5.
- [25] Ortiz M, Martín A, Arribas F, Coll-Vinent B, Del Arco C, Peinado R, et al. Randomized comparison of intravenous procainamide vs. intravenous amiodarone for the acute treatment of tolerated wide QRS tachycardia: the PROCAMIO study. *Eur Heart J* 2017;38:1329–35.
- [26] Marill KA, Wolfram S, deSouza IS, Nishijima DK, Kay D, Setnik GS, et al. Adenosine for wide-complex tachycardia: efficacy and safety. *Crit Care Med* 2009;37:2512–8.
- [27] Trohman RG, Parrillo JE. Direct current cardioversion: indications, techniques, and recent advances. *Crit Care Med* 2000;28(10 Suppl):N170–3.
- [28] Choi GJ, Kang H, Baek CW, Jung YH, Ko JS. Etomidate versus propofol sedation for electric cardioversion: a meta-analysis. *Curr Med Res Opin* 2018;34:2023–9.
- [29] deSouza IS, Martindale JL, Sinert R. Antidysrhythmic drug therapy for the termination of stable, monomorphic ventricular tachycardia: a systematic review. *Emerg Med J* 2015;32:161–7.
- [30] Richter S, Brugada P. FBI (fast broad irregular): a case for the secret service? *Circulation* 2006;114:e638–9.
- [31] Nebojša M, Dragan S, Nebojša A, Tamara A. Lethal outcome after intravenous administration of amiodarone in patient with atrial fibrillation and ventricular preexcitation. *J Cardiovasc Electrophysiol* 2011;22:1077–8.
- [32] Megid TBC, Palmegiani E, Lorga-Filho AM. Wide QRS complex tachycardia in patients with Chagas' disease: sustained ventricular tachycardia until proven otherwise. *J Electrocardiol* 2018;51:756–9.
- [33] Nair KKM, Namboodiri N, Abhilash SP, Valaparambil. RBBB tachycardia with north-west axis: what is the mechanism? *J Electrocardiol* 2018;51:121–5.
- [34] Topaloglu S, Ozeke O, Cay S, Ozcan F, Koca S, Aras D. Wide QRS complex supraventricular tachycardia with negative precordial concordance: electrocardiographic clues for Mahaim pathway with Ebstein anomaly. *J Electrocardiol* 2018;51:663–6.