What Is The Rhythm?

Paul A. Levine MD, FHRS, FACC, CCDS*

Loma Linda University Medical Center and Olive View UCLA Medical Center, Sylmar, California, USA

Abstract

56 year old male with 3 pack-per-day smoking history presents to the hospital in acute respiratory failure with hypoxemia and hypercapnea. He is intubated and admitted to the ICU. Within hours he goes through a variety of progressively more severe tachyarrhythmias. While he denies a history consistent with ischemic heart disease, his primary care physician told him that his ECG had revealed evidence of a prior myocardial infarction.

Copyright © 2015 Science International Corp.

Key Words

Atrial flutter • Atrial fibrillation • Wolff-Parkinson-White • Fusion • Atrial flutter • Atrial fibrillation • Genetic syndromes

Introduction

56 year old Caucasian male with a long history of chronic obstructive pulmonary disease superimposed on a 3 pack a day smoking habit since the age of 16 presents to the Emergency Room with decompensated lung disease. He was cyanotic, hypoxemic with an oxygen saturation of 66% and retaining CO₂. His baseline vital signs included a pulse rate of 120 bpm and regular, BP 100/60, respiratory rate of 22 with a prolonged expiratory phase. He was afebrile. His chest was hyperinflated and there were diffuse expiratory wheezes on exam. No distinct abnormal cardiac sounds could be appreciated. He was intubated and started on alpha adrenergic agonists, systemic steroids and theophylline. On his history as obtained from his wife, a prior physi-

Slideshow. Click on the presentation above to open a new browser window to view the slideshow. Direct link to presentation: <http://tiny.cc/agr-00003-15-a>.

cian had told him he had evidence of an old myocardial infarction on a routine ECG but he never complained of chest pain at either rest or exertion and his dyspnea was attributed to his smoking and COPD.

In the course of 4 hours from admission, his heart rate progressively increased with progressive alterations in his QRS complexes (Figures 1 through 4 are sequential). Each figure will be a 12 lead ECG. Figure 1 also shows a simultaneously recorded three channel rhythm strip utilizing V1, II and V5.

Before proceeding, it is necessary to answer two questions:

1. What is the rhythm(s)?
2. What should be done about them?

Figure 1. Initial presenting ECG.

Figure 2. After maximum doses of lidocaine.

Figure 3. Halfway through IV procainamide.

Figure 4. Following completion of IV procainamide.

Discussion

The staff caring for this patient made an initial diagnosis of sinus tachycardia with a brief paroxysm of ventricular tachycardia. They attributed his sinus tachycardia to his decompensated lung disease and multiple adrenergic stimulants being given to treat his lung disease. They attributed his salvo of ventricular tachycardia to his purported underlying ischemic heart disease and all the adrenergic stimulants being given so he was given a lidocaine bolus and started on a lidocaine infusion. When this proved ineffective, he was given sequential bolus' of procainamide with a progressive worsening of the rhythm. At that point, a stat consultation from cardiology was requested.

A careful review of his serial ECGs were performed. It was noted that on the first ECG, his rhythm was not sinus tachycardia but atrial flutter. This is best visualized in the V1 rhythm strip as shown in [Figure 5](#) and arrows will be used to identify the flutter waves with 2:1 conduction.

A continued analysis of the rhythm strip shown in [Figure 5](#) reveals progressive aberration of the QRS com-

plex as identified by labels A through E. There are three basic causes of this pattern. One is progressive ventricular fusion as might occur with a dual tachycardia, the second tachycardia being ventricular tachycardia. A second might be progressive aberration but there is not a distinct long-short cycle to favor Ashman's type ventricular aberration even though it has a RBBB pattern nor is this a phase 4 (bradycardia-dependent) aberration. The other phenomenon that might cause progressive aberration is varying degrees of conduction over an accessory pathway.

Despite the IV lidocaine, there were increasing salvos ([Figure 2](#)) of these broad-complex tachycardia episodes. At this point, the staff caring for the patient added incremental bolus' of procainamide to the regimen yet the overall heart rate increased as did the wide-complex salvos ([Figures 3](#) and [4](#)). The BP was falling and a stat consultation with cardiology was requested.

Note that in [Figure 2](#), the flutter waves are no longer readily visible and the rhythm is a little more irregular. This becomes very obvious in [Figures 3](#) and [4](#). The salvos of wide-QRS tachycardia show two phenom-

Figure 5. Last 5 complexes on the v1 rhythm strip.

Figure 6.

ena. They are also irregularly irregular similar to the narrow QRS rhythm but their morphology is basically stable. Ventricular tachycardia is either monomorphic but then it is regular or if irregular, then it is either Torsade de Pointes or polymorphic in morphology and the morphology was unstable. As such a diagnosis at this point of atrial fibrillation with a rapid ventricular response, attributed in part to the antiarrhythmic medications, his alpha adrenergic stimulants and his endogenous adrenergic agents associated with his deteriorating hemodynamic status combined with intermittent conduction over an accessory pathway Wolff-Parkinson-White (WPW).

The treatment at this point was emergent cardioversion which was performed with an immediate hemodynamic improvement and a return of a normal rhythm. This is shown in [Figure 6](#).

[Figure 6](#) shows classic WPW with an initial delta wave best seen in the inferior leads and anterior precordial leads confirming the clinical diagnosis. His initial atrial flutter deteriorating to atrial fibrillation was probably a consequence of his decompensated lung disease combined with the multiple stimulants he was given to treat his acute pulmonary decompensation [1, 2]. In retrospect, a prior baseline ECG had been

misread by the patient's primary care physician as an old myocardial infarction.

Now that he was clinically stable at least with respect to his arrhythmia, his lung disease was more effectively treated; he was extubated in 2 days and discharged after five days. During that time a noninvasive cardiac work-up was performed and did not demonstrate any evidence for overt ischemic heart disease. Still it is known that WPW may also predispose to paroxysmal atrial fibrillation and it would be anticipated that he would have a recurrence of his acute pulmonary problems since he refused to give up smoking. As such, an electrophysiology study was recommended with a plan for catheter ablation of the bypass tract [3]. He declined this. His subsequent course is unknown as he was not routinely followed by the physicians at this hospital.

Conflict of Interest

The author has no conflict of interest relevant to this publication.

Comment on this Article or Ask a Question

References

1. Levine PA, Klein MD. Mechanisms of arrhythmias in chronic obstructive lung disease. *Geriatrics*. 1976;31:47-56. PMID: 10230
2. Shusterman V, Lampert R. Role of stress in cardiac arrhythmias. *J Atrial Fib*. 2013;5:74-84. Available from JAFIB: <http://www.jafib.com/published/webFormat/Shusterman/shusterman.pdf>
3. Pappone C, Vicedomini G, Manguso F, Baldi M, Pappone A, Petretta A, et al. Risk of malignant arrhythmias in initially symptomatic patients with Wolff-Parkinson-White syndrome: Results of a prospective long-term electrophysiologic follow-up study. *Circ* 2012;125:661-668. DOI:

10.1161/CIRCULATIONAHA.111.065722

Cite this article as: Levine PA. What Is The Rhythm? *Arrhythmia Grand Rounds* 2015;1(2):72-75. DOI: <http://dx.doi.org/10.12945/j.agr.2015.003-15>