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Case Review

An Apple a Day: Modern Atrial Fibrillation Detection

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Case Report Editor Note: This column typically reviews cases brought forward by critical care transport clinicians from around the country. However, this month's case is a little different. It chronicles the experience of one of our providers as he identifies his own cardiac condition. The relevance of this case lies in the fact that occasionally our own staff encounter their own medical issue, and it is educational for out-of-hospital providers to not only review cases of patients for whom we provide care but also, just as importantly, how we work with medical care when critical care transport specialists are the patients themselves.

A typical nighttime routine for me is to get in bed around 10:00 PM with an alarm set for 12:30 AM to monitor my young diabetic son's glucose level. Depending on the glucose level, I either am forced to set another alarm or shut off the alarm completely until morning. During the early morning hours of January 21, 2019, his finger-stick blood glucose was elevated. I provided him with a dose of insulin based on his sliding scale and set a follow-up alarm for 3:00 AM to complete a repeat glucose check. In an effort to avoid waking my wife or my organic alarm, Indigo the dog, I use the silent alarm feature of my Apple Watch (Apple, Cupertino, CA). My alarm woke me up just after 3:00 AM. After checking on my son, I made the mistake of glancing at my e-mails, which depleted more of my sleep time. It was now 3:20 AM. I took my watch off and placed it on the charger attempting to go back to sleep. I suddenly felt a knock in my chest. This was typical of the random premature ventricular contractions (PVCs) that I have experienced for years. However, I felt another and a few seconds later another.

I have been a nurse for over 20 years with the majority of my experience in the air medical transport field. When faced with an illness or injury, I go through a self-diagnostic clinical decision tree with a goal of rapidly identifying a working diagnosis. Most clinicians likely follow a similar process as we assess and diagnose the patients we treat. The difference here is that I was the patient. Do I have pain? Do I have difficulty breathing? Diaphoresis? Nausea? Fortunately, no was the answer to my first round of assessment.

In December, Apple added a feature to their Apple Watch Series 4 that allows an electrocardiogram (ECG) to be performed, a rough equivalent of lead I. Wearing the Apple watch and placing your finger on the crown is all that is required to obtain an ECG. Stanford Medicine, Stanford, CA, and Apple are currently conducting a study to determine the ability of an Apple Watch to analyze pulse rate data to identify periods of atrial fibrillation.[1] It is an impressive feature and one that has not been readily available publicly before the rollout of this version of software and hardware. It does not "read" the rhythm necessarily as much as it determines the irregularity in the heart rate pattern. One advantage to this availability on your wrist is that we often wear a watch for much of our day versus a spot in time analysis such as a formal ECG. This feature intrigued me for a few reasons. I was curious about the random PVCs that we all encounter from time to time. In addition, I am an early adopter of technology and was curious as to its accuracy. On this particular early morning, this new feature more than paid for itself.

The palpations in my chest continued. My resting heart rate is typically low, ranging between the high 40s to the low 50s. At this point, my pulse felt very fast, with a rate between 90 to 100. I finally decided that maybe I should take a peek and verify that I was just having occasional PVCs so I could go back to bed. With the watch strapped on my wrist and my, finger on the digital crown, I performed an ECG. Thirty seconds later I was in disbelief (Fig. 1).

The ECG showed atrial fibrillation with a rate of 101. Despite my years of training, I stared in disbelief at the fact that my watch was reporting that I was experiencing "A fib with RVR [atrial fibrillation with a rapid ventricular rate]." I did what all clinicians do in an emergent situation; I performed another ECG and actually a third. By the time I finished the third ECG, I noted that not only did my rhythm remain irregularly irregular, but also the rate began to accelerate, and I had an intrinsic rate of 121 beats/min. Now I was worried and did a succession of ECGs; each time it was done, the rate was increasing (Fig. 2).

The cardiac anarchy continued with my rate reaching 153 beats/min. It was now 3:52 AM, and 30 minutes had passed. At this point, I gave in, realizing that the atrial fibrillation was not going to self-terminate. I woke up my wife and informed her that we had to head to the emergency department because I was in atrial fibrillation. My wife, who is also a nurse, was appropriately alarmed, and I spent the drive in the car explaining the events of the last hour.

Our local hospital was 4 miles away, and I arrived at an empty waiting room. After a bit of triage skepticism regarding my rhythm, I was placed into a monitored room. After the placement of an intravenous (IV) line, the acquisition of laboratory values, and an ECG, the physician arrived at my bedside. He discussed slowing my rate so that I would feel better. He expressed concern that this may be Wolff-Parkinson-White syndrome because there was a slight QRS upslope noted on my ECG. That coupled

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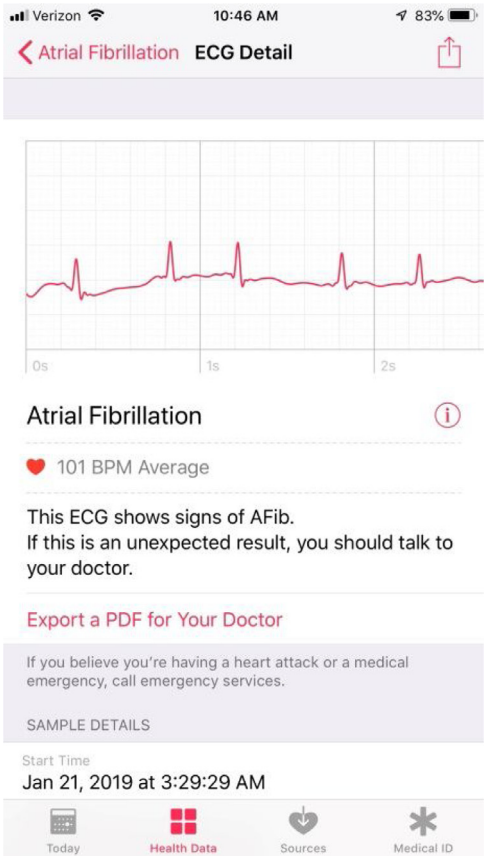

Figure 1. The ECG on the Apple Watch Series 4 showing atrial fibrillation with a rapid ventricular response.

with a natively low heart rate meant that the options were limited because a beta blocker was not optimal, and other medications were slower to act and not indicated in Wolff-Parkinson-White syndrome. He made some calls and was in contact with an electrophysiologist at a cardiac center located approximately 30 minutes away. I was heparinized and the proud owner of a second IV line along with a dose of IV and oral potassium because of a diagnosed new hypokalemia. The ambulance arrived to take me down the road, and I had the good fortune of being very familiar with the service from my time as a flight nurse in the area. The transport was uneventful. The physician arrived at my bedside, and we began discussing the events of the evening.

"When did you go into atrial fibrillation?"
"It was 3:29 AM," I replied. This was followed by a confused and skeptical look.
He responded "3:29? Exactly?"
"Yes, exactly," I said as I shared my ECG from my phone. I said, "I thought it was PVCs initially but then realized the irregularity first and then noticed the lack of P waves. It was clearly atrial fibrillation."
He appeared stunned or amazed, maybe both. He then asked, "Why are you telling me about P waves?"
I briefly explained my last 20 years as a critical care nurse with air ambulance experience.
"What was your heart rate before?" he asked.
I replied, "Ten minutes before this started, it was 52."
He responded, "At this point you are 6 $^{1}/_{2}$ hours into this rhythm. You do not need a TEE [transesophageal echocardiogram] given that time frame; let's get you out of this and get you feeling better right now."

Apparently, "right now" in electrophysiologist terms is translated into immediate action. The nasal cannula and defibrillation pads were placed. I was sedated and was cardioverted immediately. The sedation was adequate because I have no recollection of the events summarized in Figure 3.

I woke up a few minutes later and immediately noticed that I no longer felt like I was sprinting, and the previous sensation in my chest was gone. I had no further feelings of tachycardia with a rate at 170. I looked over and saw a rate of 72 on the monitor and couldn't look away for a few seconds. I never realized how much I appreciated P waves.

Although this story plays out daily for many patients, the difference with my version is in the speed of detection. The electrocardiographic feature on the Apple Watch Series 4 is a significant advancement in technology and field capabilities. My clinical background certainly assisted in understanding that my rate was fast and getting faster and that it was irregular. I definitely suspected atrial fibrillation with a rapid ventricular response, which proved to be the diagnosis. The Apple Watch allowed me to confirm my suspicions and put my mind at ease. There are certainly other rhythms that present a much greater concern, at least initially. The impact on people's lives and health with the availability of this medical technology in the field will no doubt be significant and only continue to evolve. It certainly had a positive impact on mine.

Discussion

Atrial fibrillation is the most common type of heart dysrhythmia, affecting an estimated 2.7 to 6.1 million people in the United States with those numbers expected to increase.[2] Currently, atrial fibrillation affects approximately 1% of the population aged 60 to 65 years, but its frequency increases as patients age. For patients older than 80 years, it can occur in up to 8% to 10% of the population.[3] Its prevalence is higher in whites than other ethnicities. It typically occurs not only as a result of the typical age process but also as a result of underlying cardiac disease including congestive heart failure and valvular disorders (mitral stenosis or regurgitation). Eighty percent of patients with atrial fibrillation and other precipitants including hypertension, thyroid disorders, and sleep apnea can lead to the onset of this condition.[3,4]

This chaotic dysrhythmia is a result of an abnormal rate of atrial electric discharges at a rate of greater than 350 to 600 per minute. Because of the rapidity of electric activity, discrete atrial P waves are not identifiable on the electrocardiogram. Because of the refractory nature of the atrioventricular node, only a portion of the electric discharges is able to pass through this portion of the myocardial conduction system, resulting in the "irregularly irregular" rhythm that clinicians identify. In the average patient with this untreated condition, the ventricular rate is typically elevated at approximately 160 beats/min. Patients with atrial fibrillation also have typically dilated right and left atria that are easily identifiable on the ECG or by echocardiography (left or right atrial enlargement).

There are 2 major categories of complications from this dysrhythmia, including compromised cardiac output and thromboembolic disease. With the high ventricular rates associated with atrial fibrillation, there is an inappropriate time to allow the ventricles to adequately fill, resulting in hypotension and eventually pulmonary congestion from the

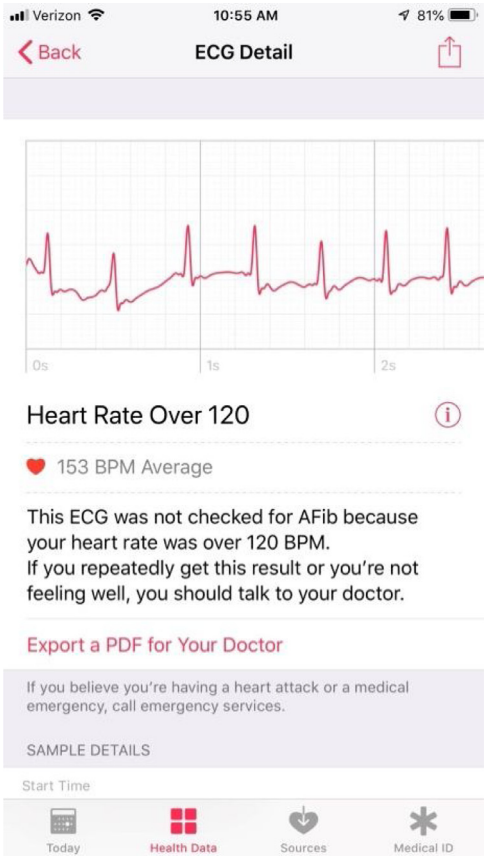

Figure 2. The ECG on the Apple Watch Series 4 showing atrial fibrillation with an increasing rapid ventricular response at 153 beats/min.

decreased cardiac output. The atrial contribution or "atrial kick" also leads to inadequate ventricular filling that can contribute to the aforementioned hypotension. However, the more serious complication is that of thromboembolic disease. Acute ischemic cerebrovascular accidents are the most common form of thromboembolism associated with atrial fibrillation. The annual risk of thromboembolism with atrial fibrillation is approximately 5% in those patients greater than 75 years of age.[3] Most acute cerebrovascular accidents arise from thrombi located in the left atrial appendage.

Treatments for atrial fibrillation are focused primarily on rate/rhythm control and anticoagulation to reduce the possibility of acute clot formation. Traditionally, most clinicians had preferred rhythm control to rate control in patients with atrial fibrillation in the past. However, recent studies have shown that rhythm control does not improve mortality, frequency of thromboembolic disease, or quality of life.[3,5] Rhythm control (ie, cardioversion) can be used in selected symptomatic patients in the acute setting without structural heart disease. Cardioversion can be considered in patients with an acute onset of atrial fibrillation within the first 48 hours.[3,5] If the atrial fibrillation has lasted longer than this period, anticoagulation is recommended with echocardiographic confirmation to insure the lack of thrombus formation before any rhythm control.

In many patients with atrial fibrillation, rate control is the preferred method of treatment. In contemporary medicine, the baseline target for heart rate control is between 60 and 80 beats/min while at rest and 90 to 115 beats/min with moderate exercise.[3,5] The recommended first-line therapy to decrease the ventricular rate in patients with atrial fibrillation includes beta blockers (metoprolol, carvedilol, and so on) and non-dihydropyridine calcium channel antagonists (diltiazem and verapamil).[3,5,6] Amiodarone and digitalis have been now relegated as second-line therapies because of their associated complications. Digoxin can slow conduction through the atrioventricular node but has less efficacy in patients with heart failure and high sympathetic activity. The long-term use of amiodarone is associated with significant toxicities including causing liver damage and pulmonary fibrosis.

Lastly, the use of anticoagulation is indicated in patients with paroxysmal, persistent, and permanent atrial fibrillation. Anticoagulation is indicated in this population when the reasonable risk of thromboembolism outweighs the side effects of bleeding with the use of this class of medications. In recent years, researchers have developed several algorithms to help identify categories of patients who would benefit from the use of anticoagulation. The most common of which is the CHA_2DS_2-VASc score. Based on comorbidities, this guide aids the clinician in determining the risk/

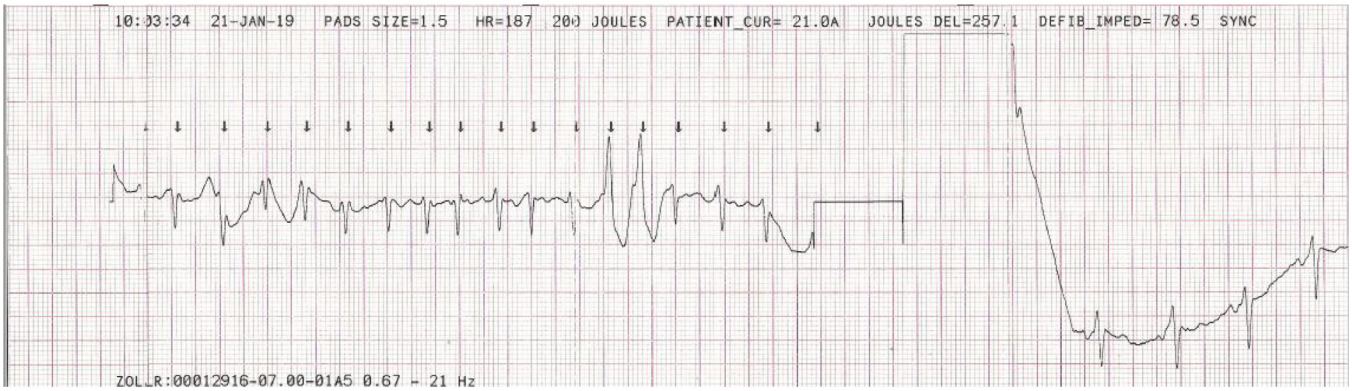

Figure 3. An ECG revealing atrial fibrillation with cardioversion.

Table 1
The CHA_2DS_2-VASc Score

Risk Factors	Score
Congestive heart failure	1
Hypertension	1
Age > 75 years	2
Diabetes mellitus	1
Stroke/transient ischemic attack/systemic embolism	2
Vascular disease	1
Age 65-74 years	1
Sex (female)	1

benefit for the use of anticoagulation. The scoring system is outlined in Table 1. Based on the score, the clinician will then decide the type of anticoagulation therapy and the duration. As the score increases, so does the indication for anticoagulation use.[3,7] The nuances of choice of anticoagulant and duration of treatment are beyond the scope of this overview, but it is essential to risk stratify patients for the appropriate type of medication. In the last decade, pharmaceutical companies have developed medications that compliment the standard warfarin therapy that has been in place long-term. These novel non–vitamin K oral anticoagulants are able to provide protection from thromboembolic disease but have similar risk profiles to the long-standing vitamin K antagonists (ie, warfarin). Factor IIa (dabigatran) and Xa inhibitors (rivaroxaban or apixaban) are gaining wide traction and have been increasing in use as emergent antidotes have been developed to mitigate the catastrophic bleeding effects if they do occur.[3,7]

In the transport environment, a diagnosis of acute atrial fibrillation brings a number of concerns with the initial questions centered on the time of onset, hemodynamic instability, and patient mentation with the treatment during patient transit likely centered on those answers. In a patient who is hemodynamically stable and with normal mentation, it is likely that an attempt at rhythm conversion is best managed at the receiving cardiac center versus attempts during transport. In the patient with a rapid ventricular rate who is either hemodynamically unstable or has evidence of acute myocardial ischemia or heart failure, immediate electrical cardioversion can be entertained if the patient does not promptly respond to pharmacologic measures.[8]

Conclusion

The key difference in my case is that I was able to make a diagnosis in 30 seconds from my home. The ability to rapidly recognize and treat atrial fibrillation is often focused on the time of diagnosis and the need for anticoagulation. With this technology now readily available, that ability has only expanded. Both the emergency room physician and electrophysiologist I encountered were unfamiliar with this ability but equally impressed on the level of precision and accuracy that aided a quick diagnosis. Wrist-worn technology continues to see growing adoption rates and increased capabilities. As these technologies evolve, the insight that they provide day to day can be critical to early recognition of health issues, particularly in problems that may be otherwise asymptomatic. As the Apple Watch 4 continues to increase market share, stories like mine will only increase. Newer versions will likely gain new abilities, and our capacity to analyze our health will expand in ways that many of us have not considered before.

References

1. White T. Stanford, Apple describes heart study with over 400,000 participants. November 1, 2018. Available at: https://med.stanford.edu/news/all-news/2018/11/stanford-apple-describe-heart-study-with-over-400000-participants.html. Accessed June 25, 2019.
2. Atrial fibrillation fact sheet. August 22, 2017. Available at: https://www.cdc.gov/dhdsp/data_statistics/fact_sheets/fs_atrial_fibrillation.htm. Accessed June 25, 2019.
3. Zimetbaum P. Atrial fibrillation [erratum in *Ann Intern Med*. 2017;166:920]. *Ann Intern Med*. 2017;166:ITC33–ITC48.
4. Lilly L. *Pathophysiology of Heart Disease*. 6th ed. Philadelphia, PA: Wolters Kluwer; 2016.
5. Van Gelder IC, Rienstra M, Crijns HJ, Olshansky B. Rate control in atrial fibrillation. *Lancet*. 2016;388:818–828.
6. Kirchhof P. The future of atrial fibrillation management: integrated care and stratified therapy [erratum in *Lancet*. 2017;390:1832]. *Lancet*. 2017;390:1873–1887.
7. Verheugt FW, Granger CB. Oral anticoagulants for stroke prevention in atrial fibrillation: current status, special situations, and unmet needs [erratum in *Lancet*. 2015;386:248]. *Lancet*. 2015;386:303–310.
8. Bajpai A, Rowland E. Atrial fibrillation. *Contin Educ Anaesth Crit Care Pain*. 2006;6:219–224.