Research Article

ISSN 2771-9057

Recent Advances in Clinical Trials

Electrical Cardioversion in a Patient with Persistent Atrial Fibrillation- A Case Report and Literature Review

Yaw Adu-Boakye[1,2], Lambert Tetteh Appiah[1,2], Solomon Gyabaah[2*], Chris Oppong[1,2], Freda Aidoo Manu[2] and Adwoa Agyemang Adu-Gyamfi[2]

[1]School of medicine and Dentistry, Kwame Nkrumah University of Science and Technology, Kumasi, Ghana.

[2]Komfo Anokye Teaching hospital, Kumasi, Ghana.

[*]Correspondence:

Dr. Solomon Gyabaah, Komfo Anokye Teaching Hospital, Kumasi, Ghana, P. O. BOX 1934, Kumasi, Ghana, Tel: +233265763129

Received: 11 Sep 2022; **Accepted:** 13 Oct 2022; **Published:** 18 Oct 2022

Citation: Yaw Adu-Boakye, Lambert Tetteh Appiah, Solomon Gyabaah, et al. Electrical Cardioversion in a Patient with Persistent Atrial Fibrillation- A Case Report and Literature Review. Recent Adv Clin Trials. 2022; 1(2): 1-5.

ABSTRACT

Atrial fibrillation is the most common sustained cardiac arrhythmia in adults with an estimated prevalence of between 2-4% of the general population with a significant effect on morbidity and mortality. It is an important contributor to stroke, myocardial infarction, thromboembolism, and heart failure. Management of atrial fibrillation involves rate control, rhythm control, and anticoagulation therapy. We present the case of a 30-year-old male who presented with complaints of persistent palpitations and intermittent lightheadedness for over 6 months. No risk factors were readily identifiable and the transthoracic echocardiogram was essentially normal. He was started on anticoagulation. Flecainide initiated after 4 weeks of anticoagulation and continued for 4 months with anticoagulants and the beta-blocker. There was an improvement in the heart rate but the sensation of palpitations continued. The patient underwent elective electrical cardioversion with 100J of direct current and his rhythm was successfully converted to a sinus rhythm, which was confirmed by a 12-lead ECG. Electrical cardioversion terminates AF in over 90% of cases.

Keywords

Atrial fibrillation, Cardiac arrhythmia, Heart failure.

Introduction

Atrial fibrillation (AF) can simply be defined as a supraventricular tachyarrhythmia with uncoordinated atrial electrical activation and consequently ineffective atrial contraction [1]. It is the most common sustained cardiac arrhythmia in adults with an estimated prevalence of between 2-4% of the general population with a significant effect on morbidity and mortality [2]. It is an important contributor to stroke, myocardial infarction, thromboembolism, and heart failure. The estimated lifetime risk of AF was 1 in 4 in white men and women older than 40 years in 2004 [3]. Currently the lifetime risk is estimated to be about 1 in 3 in white individuals and 1 in 5 for black individuals [4]. Risk factors for atrial fibrillation include; aging, genetics, male sex, ethnicity, hypertension, diabetes, smoking, obesity, inflammatory diseases, vascular and valvular diseases, obstructive sleep apnea, alcohol consumption, high lipid profile, and hyperthyroidism [1,5-7].

Atrial fibrillation can be broadly classified according to presentation, duration, and spontaneous abolishing of episodes. It is classified as first diagnosed AF, paroxysmal, persistent, longstanding persistent, and permanent. It may be termed idiopathic or "lone" in circumstances where there are no identifiable underlying cause or risk factors. Management of atrial fibrillation involves rate control, rhythm control, and anticoagulation therapy [1,8]. The approach depends on whether the patient is hemodynamically stable or not [1,8]. In hemodynamically unstable patients, immediate evaluation and prompt treatment are paramount including emergency cardioversion followed by anticoagulation to reduce the risk of a thromboembolic event [8,9]. There are two ways of cardioversion; 1. Electrical cardioversion and 2. Pharmacologic cardioversion. Pharmacological cardioversion mainly converts recent-onset or

paroxysmal (i.e. in principle self-terminating) AF to sinus rhythm in 50–70% of cases within a few hours [9,10].

Electrical cardioversion is a standard procedure for cardioverting atrial fibrillation both in the acute setting and the chronic setting to a sinus rhythm. It is the preferred option in emergencies, especially in patients who are not hemodynamically stable [1,8,9]. Direct electrical current (shock) is delivered through the chest wall to the heart through special electrodes or paddles that are applied to the chest. The shock wave interrupts and terminates the abnormal electrical rhythm by causing the heart cells to contract simultaneously without damaging the heart cells [11]. The electrode could be positioned anteroposteriorly or anterolaterally to deliver a specified amount of energy to the heart cells. Electrical cardioversion is performed under sedation and patients are usually treated with intravenous midazolam and/or propofol or etomidate or fentanyl [1,12]. In this case study, we present the premier case of elective electric cardioversion at the Komfo Anokye Teaching Hospital, Kumasi, Ghana.

Case Presentation

We report the case of a 30-year-old man who presented with complaints of persistent palpitations and intermittent lightheadedness for over 6 months. There is no history of hypertension, diabetes, COPD, heart disease, thyroid disease, or alcoholism. Examination revealed an irregularly irregular pulse with variable volume at a rate of 89bpm. An electrocardiogram (ECG) showed AF with a ventricular response rate of 98bpm. An echocardiogram concluded on a structurally normal heart; TSH and serum electrolytes were also normal. He was started on anticoagulation - Rivaroxaban 20mg daily for 4 weeks and Bisoprolol 2.5mg daily. Flecainide 50mg bd was initiated after 4 weeks of anticoagulation and continued for 4 months with anticoagulants and the beta blocker. Heart rate control was achieved: 68 beats per minute averagely on Holter ECG yet the sensation of palpitations continued which was a bother to the patient and the patient also noted intermittent dizziness on the medication which prevented escalation of dosage. The patient was then planned for electrical cardioversion.

Procedure for the Electrical Cardioversion

The procedure was explained to the patient and he gave his consent.
The patient was put on a cardiac monitor and pulse oximetry was applied.
The resuscitation trolley was put on standby.
The Patient was sedated with 1 mg/kg of propofol.
The paddles were applied anterolaterally.
100 J of direct current energy was selected and a biphasic Defibrillator was "synced" before discharge.
The patient was given a single shock.
His rhythm was successfully converted to a sinus rhythm, which was confirmed by a 12-lead ECG. The patient was monitored until full recovery from sedation and was subsequently discharged.
He is being followed for the past 5 months with intermittent ECG monitoring. The patient is doing well with the resolution of his symptoms.

Discussion

The general prevalence of atrial fibrillation increases with increasing age [1,7]. However Idiopathic atrial fibrillation, also called lone atrial fibrillation is more common in ages less than 60 years and it is usually diagnosed when there is no clinical history

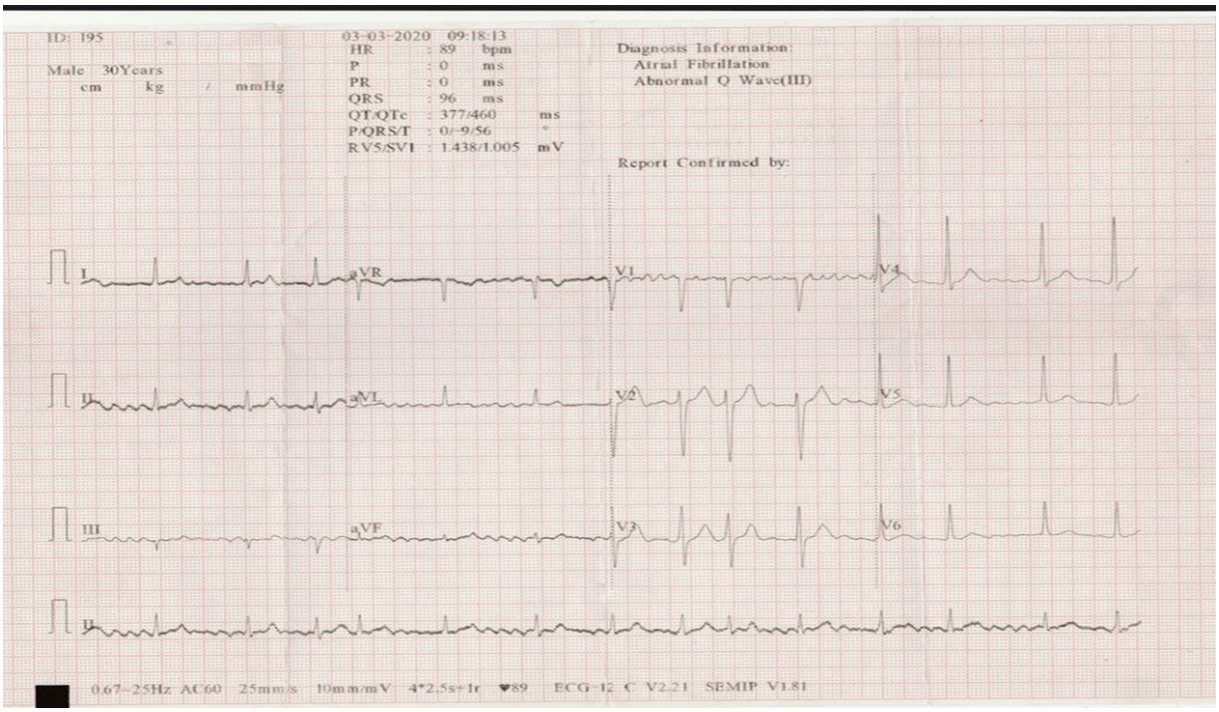

Figure 1: shows the initial ECG findings indicating the absence of discernable P waves with chaotic F waves and irregular R-R intervals or rhythm.

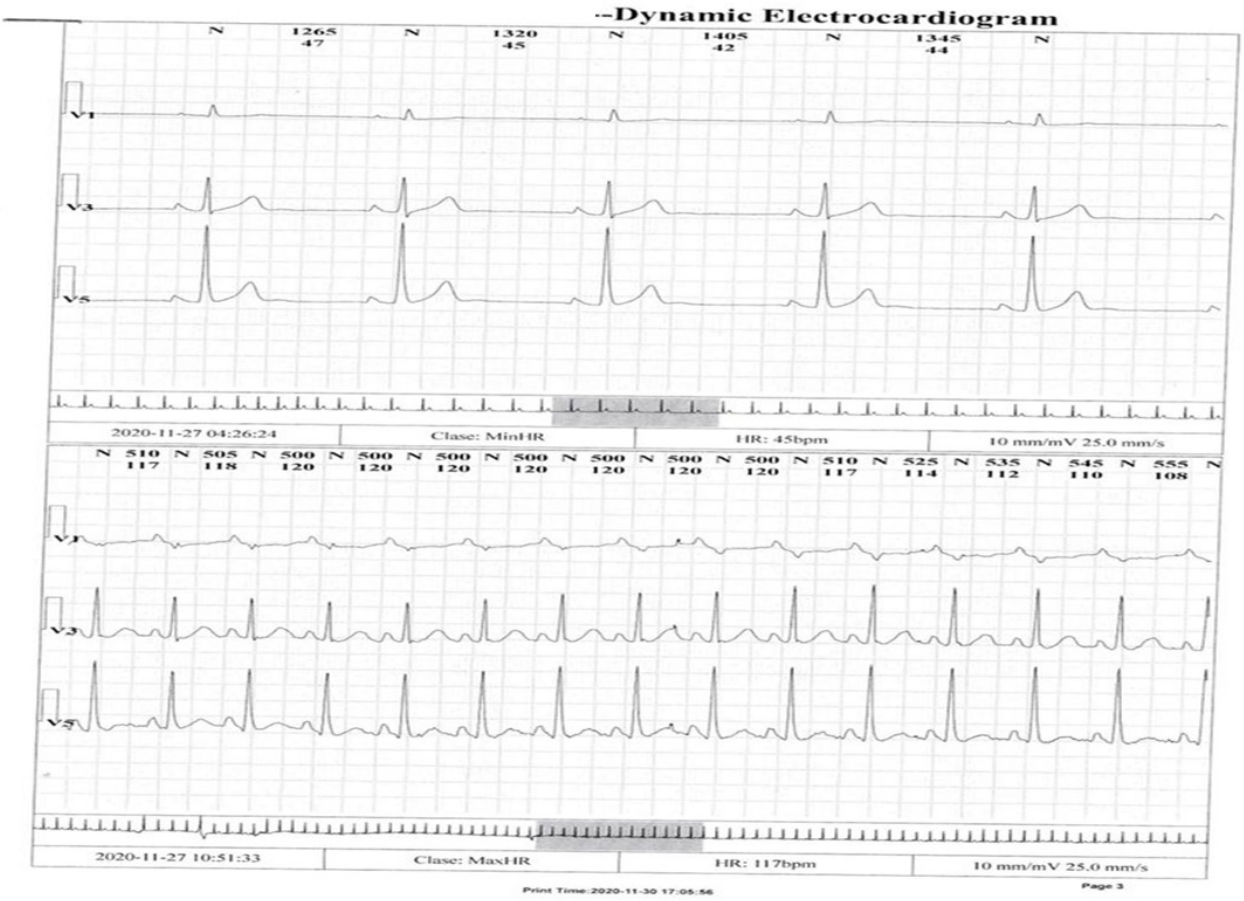

Figure 2: Shows the patient in sinus rhythm after elective electrical cardioversion.

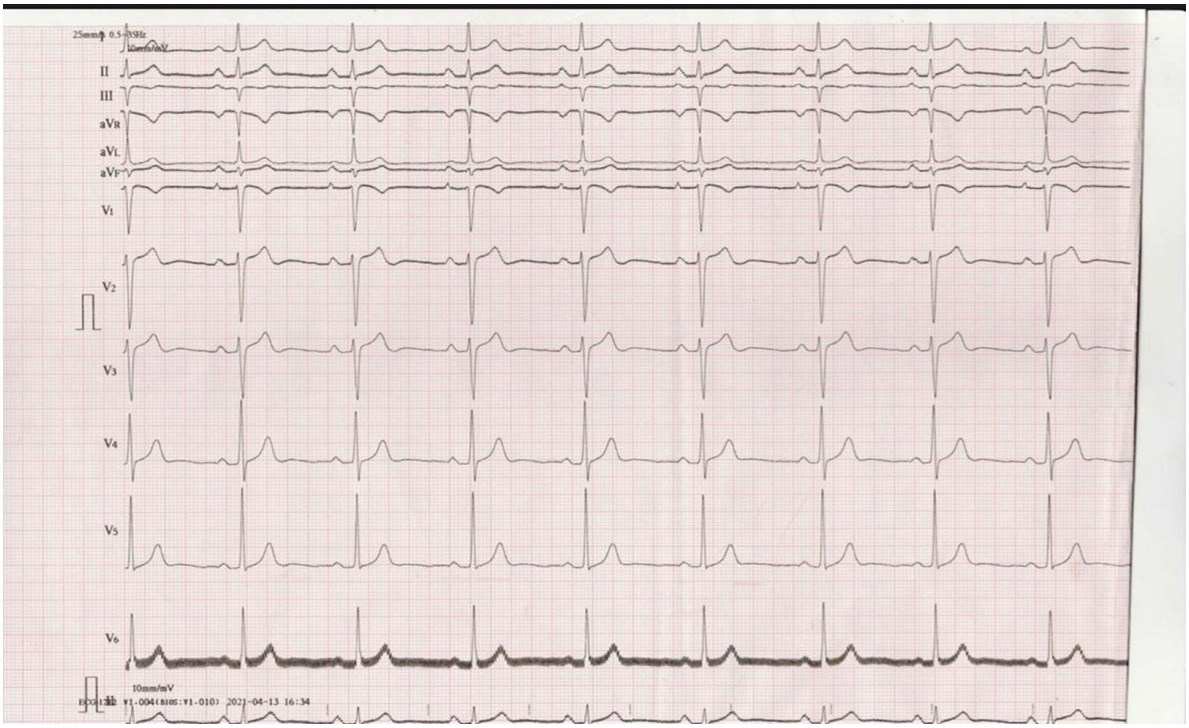

Figure 3: The ECG of the patient shows sinus rhythm after about 5 months of electrical cardioversion.

or echocardiographic evidence of concomitant cardiovascular, or pulmonary conditions or an acute trigger [13,14]. However recent studies and extensive work into the pathophysiology of the condition have revealed that in every AF patient a cause may be identified and therefore the use of the term "lone" or "idiopathic" has been strongly discouraged [1,15,16]. We present in this case report a 30-year-old with first diagnosed and persistent atrial fibrillation of 6 months duration before the presentation. No risk factor was identified from the history and a series of basic and diagnostic investigations done did not reveal any pathology. However, our investigations may not be exhaustive since imaging such as chest CT scan and MRI could be revealing. Undoubtedly, Magnetic resonance imaging (MRI) is considered the 'gold standard for assessment of left atrial volume [15]. Familial transmission of AF has also been established. Genetically the condition is found on loci mapped to chromosomes 10q22-24 and 6q14-16 and gain of function mutation in the *KCNQ1*gene [17-21]. Some genetic studies may also be rewarding in this regard.

The patient was started on anticoagulation using Rivaroxaban 20mg daily after his risk of bleeding was assessed and found to be low using the HAS-BLED scoring. This was aimed at reducing his risk of thromboembolism events such as cardioembolic stroke. AF is known to affect the three components of Virchow's triad. It causes stasis of blood in the left atrium, endocardial damage, and abnormalities of blood constituents and thus leads to a hypercoagulable state [22,23]. Stroke is one of the major complications of AF because of the tendency of cerebral thromboembolism. Efforts therefore should be made at reducing the risk of stroke and embolic phenomenon. Anticoagulation before cardioversion is important to reduce the risk and rate of thromboembolism and stroke, especially in cases of chronic AF [1,12,24,25]. In cases of new-onset AF less than 48hrs, cardioversion may be done before initiation of anticoagulation [8,26].

This patient presented with hemodynamically stable chronic AF and was thus anticoagulated for 4 weeks before pharmacologic cardioversion was attempted with 50mg of flecainide twice daily. The anticoagulation was continued with the flecainide and beta blocker, bisoprolol 2.5mg daily, for a period of 4 months. Heart rate control was achieved but the patient developed intermittent episodes of drowsiness on medication and also had a bothering sensation of palpitations despite a stable controlled heart rate on Holter ECG. The use of Pharmacologic cardioversion is more effective in a recent onset AF occurring less than 7 days duration with a success rate of 50-70% [9,10]. The success rate increases to about 83% if initiated within 48hrs of hospitalization [1]. The effectiveness of pharmacologic cardioversion decreases with increasing duration of the AF [16,27]. In this patient, the long-standing duration of the AF may be the contributing factor to the persistent rhythm of the medication, as well as the early onset of medication side effects hampering dose escalation.

The patient was subsequently prepared for elective electrical cardioversion using 100J of direct current under 1mg/kg propofol sedation with the paddles applied anterolaterally. The patient was given a single shock and his rhythm was successfully converted to a sinus rhythm which was confirmed by a 12-lead ECG. Electrical cardioversion terminates AF in over 90% of cases [9]. It is effective both in hemodynamically stable patients and hemodynamically compromised patients. In chronic AF electrical cardioversion successfully converts the rhythm to sinus in 75% of cases [12]. Pre-treatment of AF patients with anti-arrhythmic agents such as flecainide, amiodarone, or propafenone is known to increase the success rate of electrical cardioversion [1]. Biphasic defibrillators are standard because of their superior efficacy compared with monophasic defibrillators [28].

Conclusion

Atrial fibrillation is a common cardiac arrhythmia and may be detected without any identifiable risk factor. Initiation of anticoagulation before cardioversion in a chronic or persistent AF is important to prevent thromboembolism. Electrical cardioversion is more effective in converting atrial fibrillation to sinus rhythm.

References

1. Hindricks G, Potpara T, Dagres N, et al. 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). European Heart Journal. 2021; 42: 373-4982.
2. Benjamin EJ, Muntner P, Alonso A, et al. Heart Disease and Stroke Statistics-2019 Update: A Report From the American Heart Association. Circulation. 2019; 139: e56-e528.
3. Lloyd-Jones DM, Wang TJ, Leip EP, et al. Lifetime risk for development of atrial fibrillation: The Framingham heart study. Circulation. 2004; 110: 1042-1046.
4. Mou L, Norby FL, Chen LY, et al. Lifetime Risk of Atrial Fibrillation by Race and Socioeconomic Status: ARIC Study (Atherosclerosis Risk in Communities). Circ Arrhythmia and Electrophysiology. 2018; 11: e006350.
5. Schoonderwoerd BA, Smit MD, Pen L, et al. New risk factors for atrial fibrillation: Causes of not-so-lone atrial fibrillation. Europace. 2008; 10: 668-673.
6. Brandes A, Smit MD, Nguyen BO, et al. Risk factor management in atrial fibrillation. Arrhythmia and Electrophysiology Review. 2018; 7: 118-127.
7. Menezes AR, Lavie CJ, DiNicolantonio JJ, et al. Atrial fibrillation in the 21st century: A current understanding of risk factors and primary prevention strategies. Mayo Clinic Proceedings. 2013; 88: 394-409.
8. Gutierrez C, Blanchard DG. Diagnosis and Treatment of Atrial Fibrillation. Am Fam Physician. 2016; 94: 442-452.
9. Brandes A, Crijns HJGM, Rienstra M, et al. Cardioversion of atrial fibrillation and atrial flutter revisited: Current evidence and practical guidance for a common procedure. Europace. 2020; 22: 1149-1161.

10. Savelieva I, Graydon R, Camm AJ. Pharmacological cardioversion of atrial fibrillation with vernakalant: Evidence in support of the ESC Guidelines. Europace. 2014; 16: 162-173.
11. Sears SF, Shea JB, Conti JB. Cardiology patient page How to respond to an implantable cardioverter-defibrillator shock. Circulation. 2005; 111: 380-382.
12. Aniteye E, Kotei D, Tettey M, et al. Synchronised cardioversion for chronic atrial fibrillation. Ghana medical journal. 2008; 42: 29-32.
13. Camm AJ, Kirchhof P, Lip GYH, et al. Guidelines for the management of atrial fibrillation. Europace. 2010; 12: 1360-1420.
14. Frost L. Lone atrial fibrillation: Good, bad, or ugly. Circulation. 2007; 115: 3040-3041.
15. Wyse DG, Van Gelder IC, Ellinor PT, et al. Lone atrial fibrillation: Does it exist. Journal of the American College of Cardiology. 2014; 63: 1715-1723.
16. Kirchhof P, Benussi S, Kotecha D, et al. 2016 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. Europace. 2016; 18: 1609-1678.
17. Palatini P, Fox CS, Vasan RS, et al. Parental atrial fibrillation as a risk factor for atrial fibrillation in offspring. Journal of the American Medical Association. 2004; 292: 1174-1175.
18. Ellinor PT, Shin JT, Moore RK, et al. The locus for atrial fibrillation maps to chromosome 6q14-16. Circulation. 2003; 107: 2880-2883.
19. Chen Y Han, Wang X Liang, Wang Y, et al. Mutation in Familial Atrial. Science. 2003; 299: 251-254.
20. Brugada R, Tapscott T, Czernuszewicz GZ, et al. Identification of a Genetic Locus for Familial Atrial Fibrillation. New England Journal of Medicine. 1997; 336: 905-911.
21. Lubitz SA, Yin X, Fontes JD, et al. Association between familial atrial fibrillation and risk of new-onset atrial fibrillation. Journal of the American Medical Association. 2010; 304: 2263-2269.
22. Choudhury A, Lip GYH. Atrial fibrillation and the hypercoagulable state: From basic science to clinical practice. Pathophysiology of Haemostasis and Thrombosis. 2003; 33: 282-289.
23. Lip GYH. Does atrial fibrillation confer a hypercoagulable state. The Lancet. 1995; 346: 1313-1314.
24. Atrial fibrillation: management. 2020; 1-38.
25. January CT, Wann LS, Calkins H, et al. 2019 AHA/ACC/HRS Focused Update of the 2014 AHA/ACC/HRS Guideline for the Management of Patients With Atrial Fibrillation: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. Journal of the American College of Cardiology. 2019; 74: 104-132.
26. Khatami M, Pope MK, Le Page S, et al. Cardioversion Safety - Are We Doing Enough. Cardiology. 2020; 145: 740-745.
27. Hernández-Madrid A, Svendsen JH, Lip GYH, et al. Cardioversion for atrial fibrillation in current European practice: Results of the European Heart Rhythm Association survey. Europace. 2013; 15: 915-918.
28. Mittal S, Ayati S, Stein KM, et al. Transthoracic Cardioversion of Atrial Fibrillation. Circulation. 2000; 101: 1282-1287.