JACC: CLINICAL ELECTROPHYSIOLOGY
VOL. 2, NO. 4, 2016

ISSN 2405-500X/$36.00
PUBLISHED BY ELSEVIER
http://dx.doi.org/10.1016/j.jacep.2016.01.018

Incidence of Thromboembolic Complications Within 30 Days of Electrical Cardioversion Performed Within 48 Hours of Atrial Fibrillation Onset

Aatish Garg, MD,[a] Monica Khunger, MD,[a] Sinziana Seicean, MD, MPH, PhD,[b] Mina K. Chung, MD,[b] Patrick J. Tchou, MD[b]

JACC: CLINICAL ELECTROPHYSIOLOGY CME

This article has been selected as the month's *JACC: Clinical Electrophysiology* CME activity, available online at www.jacc-electrophysiology.org by selecting the CME on the top navigation bar.

Accreditation and Designation Statement

The American College of Cardiology Foundation (ACCF) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

The ACCF designates this Journal-based CME activity for a maximum of 1 *AMA PRA Category 1 Credit(s)*. Physicians should only claim credit commensurate with the extent of their participation in the activity.

Method of Participation and Receipt of CME Certificate

To obtain credit for *JACC: Clinical Electrophysiology* CME, you must:

1. Be an ACC member or *JACC: Clinical Electrophysiology* subscriber.
2. Carefully read the CME-designated article available online and in this issue of the journal.
3. Answer the post-test questions. At least 2 out of the 3 questions provided must be answered correctly to obtain CME credit.
4. Complete a brief evaluation.
5. Claim your CME credit and receive your certificate electronically by following the instructions given at the conclusion of the activity.

CME Objective for This Article: Upon completion of this activity, the learner should be able to: 1) list the guidelines recommendations for cardioversion of atrial fibrillation with onset <48 h; and 2) appraise the need for anticoagulation in patients undergoing cardioversion of atrial fibrillation with onset <48 h.

CME Editor Disclosure: CME Editor Smit Vasaiwala, MD, has nothing to declare.

Author Disclosures: All authors have reported that they have no relationships relevant to the contents of this paper to disclose.

Medium of Participation: Print (article only); online (article and quiz).

CME Term of Approval

Issue Date: August 2016
Expiration Date: July 31, 2017

Listen to this manuscript's audio summary by *JACC: Clinical Electrophysiology* Editor-in-Chief Dr. David J. Wilber.

From the [a]Department of Internal Medicine, Cleveland Clinic, Cleveland, Ohio; and the [b]Department of Cardiovascular Medicine, Heart & Vascular Institute, Cleveland Clinic, Cleveland, Ohio. All authors have reported that they have no relationships relevant to the contents of this paper to disclose.

Manuscript received July 20, 2015; revised manuscript received December 28, 2015, accepted January 28, 2016.

JACC: CLINICAL ELECTROPHYSIOLOGY VOL. 2, NO. 4, 2016
AUGUST 2016:487–94

Incidence of Thromboembolic Complications Within 30 Days of Electrical Cardioversion Performed Within 48 Hours of Atrial Fibrillation Onset

Aatish Garg, MD,[a] Monica Khunger, MD,[a] Sinziana Seicean, MD, MPH, PhD,[b] Mina K. Chung, MD,[b] Patrick J. Tchou, MD[b]

ABSTRACT

OBJECTIVES This study sought to compare the risk of thromboembolism after cardioversion within 48 h of atrial fibrillation (AF) onset in patients therapeutically versus not therapeutically anticoagulated.

BACKGROUND Although guidelines do not mandate anticoagulation for cardioversion within 48 h of AF onset, risk of thromboembolism in this group has been understudied.

METHODS Patients undergoing cardioversion within 48 h after AF onset were identified from a prospectively collected database and retrospectively reviewed to determine anticoagulation status and major thromboembolic events within 30 days of cardioversion.

RESULTS Among 567 cardioversions in 484 patients without therapeutic anticoagulation (mean CHA_2DS_2-VASc score, 2.3 ± 1.7), 6 had neurological events (1.06%), all in patients on aspirin alone. Among 898 cardioversions in 709 patients on therapeutic anticoagulation (mean CHA_2DS_2-VASc score, 2.6 ± 1.7; $p = 0.017$), 2 neurological events occurred (0.22%; OR: 4.8; $p = 0.03$), both off anticoagulation at the time of stroke. No thromboembolic events occurred in patients with CHA_2DS_2-VASc score <2 ($p = 0.06$) or in patients with postoperative AF.

CONCLUSIONS In patients with acute-onset AF, odds of thromboembolic complications were almost 5 times higher in patients without therapeutic anticoagulation at the time of cardioversion. However, no events occurred in post-operative patients and in those with CHA_2DS_2-VASc scores of <2, supporting the utility of accurate assessment of AF onset and risk stratification in determining the need for anticoagulation for cardioversion of AF <48 h in duration. (J Am Coll Cardiol EP 2016;2:487-94) © 2016 by the American College of Cardiology Foundation.

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia, affecting 1 in every 4 individuals during their lifetime (1). It independently raises stroke risk by 5-fold, which is a major cause of morbidity and mortality in these patients (2,3). This risk was shown to increase further following direct current cardioversion, possibly by stunning of the left atrium and subsequent return of mechanical function leading to clot dislodgement (4-9). This increased stroke risk persists for about a month after the procedure (10).

Patients undergoing cardioversion within 48 h of AF onset were traditionally considered to be at lower risk for thromboembolic complications, because it was thought that there is less time for left atrial thrombus formation (11). The current 2014 American Heart Association/American College of Cardiology/Heart Rhythm Society guidelines for these patients with AF onset of <48 h and with high risk of stroke recommend anticoagulation therapy as soon as possible before or immediately after cardioversion, followed by long-term anticoagulation therapy (12). The duration of this long-term anticoagulation therapy should be based on the thromboembolic risk profile. This is a Class Ia recommendation based on Level of Evidence: C. However, for AF of duration <48 h with low thromboembolic risk, anticoagulation or no antithrombotic therapy may be considered for cardioversion, without the need for post-cardioversion oral anticoagulation (13,14). This is a Class IIb recommendation with Level of Evidence: C (13).

We aimed to assess the risk of thromboembolism in patients undergoing cardioversion within 48 h of AF onset without prior therapeutic anticoagulation and to compare this risk with the risk of thromboembolism in patients who were therapeutically anticoagulated.

METHODS

STUDY POPULATION. The Cleveland Clinic Cardiac Electrophysiology Laboratory database recorded a total of 16,221 cardioversions performed between January 1996 and December 2012, of which 1,996 were performed within 48 h of AF onset. Of these, 28 cardioversions were done on patients receiving dabigatran. Because of their small number and difficulty in assessing compliance, these 28 patients were excluded from our report. After correction for duplicate entries, errors regarding timing of the onset of AF as determined by chart review, missing anticoagulation information, and lack of follow-up at the Cleveland Clinic for at least 1 month after the cardioversion, a total of 1,581 direct current cardioversions were included in our study.

SEE PAGE 495

DATA COLLECTION. A clinical database has been prospectively maintained for all patients who were cardioverted in the Cardiac Electrophysiology Laboratory at Cleveland Clinic since 1996. It includes demographic data; cardiovascular history; echocardiographic findings; smoking; alcohol intake; caffeine intake; vital signs before cardioversion; prothrombin time/international normalized ratio (INR); activated partial thromboplastin time; and medications, including aspirin, clopidogrel, heparin, warfarin, dabigatran, and antiarrhythmic medications. The duration of AF onset of <48-h duration was estimated based on the onset of patient's symptoms, electrocardiographic or telemetric evidence, and clinical examination findings. Cardioversion success or transient success and post-cardioversion rhythm were also recorded.

Besides the data collected from the electrophysiology database, an extensive electronic and paper chart review was performed for the patients included in the study. Additional baseline information was collected and included history of hypertension, congestive heart failure, diabetic status, history of prior stroke, or any vascular disease. Vascular disease was defined as presence of coronary or peripheral artery disease.

The study was approved by the Cleveland Clinic Institutional Review Board for retrospective medical records review and performed under institutional guidelines.

FOLLOW-UP. The primary outcome was any thromboembolic event, comprising any neurological event (ischemic stroke or transient ischemic attack) or any other systemic thromboembolism, within 30 days of the electrical cardioversion. To detect thromboembolic events within 30 days of cardioversion, paper and electronic charts within and beyond 30 days after cardioversion, including all discharge summaries, subsequent cardiology or primary care notes, any emergency department visits, and imaging tests, including computed tomography scans, angiograms, or magnetic resonance imaging reports, were reviewed. Dates of death, if available, and the last follow-up date at Cleveland Clinic were also recorded. Electrophysiology database information was validated, and missing information was collected during the chart review. CHA_2DS_2-VASc scores were then calculated (15,16).

All cardioversions were divided into 3 categories based on anticoagulation status at the time of the procedure. Group 1 comprised cardioversions performed either without anticoagulation or on warfarin with INR ≤1.5. These patients were considered to be nonanticoagulated at the time of cardioversion. Group 2 comprised cardioversions performed in patients on warfarin with an INR between 1.5 and 2. These were considered to be subtherapeutically anticoagulated. Group 3 comprised cardioversions where the patients were therapeutically anticoagulated at the time of the procedure on either warfarin or heparin. Therapeutic anticoagulation was defined as patients on warfarin with an INR ≥2 or patients on heparin with an activated partial thromboplastin time ≥50 at the time of cardioversion. A subgroup analysis was performed on patients who developed AF during the post-surgical period.

STATISTICAL ANALYSES. Statistical analyses were performed using Stata version 12.1 (StataCorp, College Station, Texas). Continuous variables are presented as mean ± SD, and categorical variables are presented as proportions. Shapiro-Wilk test was used to check for normality of characteristics. For normally distributed data, analysis of variance was used to compare means in 3 or more groups, whereas Kruskal-Wallis equality-of-populations rank test was used for data that were not normally distributed. All categorical variables were assessed using the chi-square test or Fisher exact test. All p values were 2-tailed, with statistical significance specified at $p < 0.05$ and confidence intervals reported at the 95% level.

ABBREVIATIONS AND ACRONYMS

AF = atrial fibrillation

INR = international normalized ratio

NOAC = new oral anticoagulants

TEE = transesophageal echocardiogram

RESULTS

In Group 1, a total of 567 cardioversions were performed in 484 nonanticoagulated patients, of which

TABLE 1 Baseline Characteristics of Patients Undergoing DCC Stratified by Their Anticoagulation Status

	No Anticoagulation	Subtherapeutic Anticoagulation	Therapeutic Anticoagulation	p Value
Number of DCC	567	116	898	
Number of patients	484	106	709	
Mean age, yrs	62.8 ± 14.0	62.1 ± 11.9	63.9 ± 13.4	0.182
Females, %	157 (27.7)	38 (32.8)	302 (33.6)	0.055
Hypertension, %	264 (46.6)	44 (37.9)	434 (48.3)	0.105
CHF, %	129 (22.8)	31 (26.7)	274 (30.6)	**0.005**
Diabetes, %	94 (16.6)	12 (10.3)	131 (14.6)	0.202
Prior stroke, %	38 (6.7)	8 (6.9)	106 (11.8)	**0.003**
Vascular disease, %	221 (39.0)	33 (28.4)	289 (32.2)	**0.01**
Mean CHA_2DS_2- VASc	2.34 ± 1.7	2.10 ± 1.6	2.62 ± 1.7	**<0.001**
Medications, %				
Aspirin alone	310 (54.7)	0	0	
Warfarin	67 (11.8)	116 (100.0)	567 (63.1)	
Heparin	0	0	331 (36.9)	

Values are n, mean ± SD, or n (%). **Bolded** values are those with statistical significance of <0.05.
CHF = congestive heart failure; DCC = direct current cardioversion.

190 (33.5%) were performed in patients on no antiplatelet or anticoagulant agents, 310 (54.7%) were receiving aspirin alone, and the remaining 67 (11.8%) were taking warfarin with an INR ≤1.5. Group 1 patients had a mean CHA_2DS_2-VASc score of 2.3 ± 1.7.

Group 2 included 116 cardioversions performed in patients who were subtherapeutic on warfarin with INR between 1.5 and 2. Mean CHA_2DS_2-VASc score in this group was 2.1 ± 1.6.

Group 3 included 898 cardioversions in 709 patients who were therapeutically anticoagulated, with 567 cardioversions (63.1%) performed in patients on warfarin having INR ≥2 and 331 (36.9%) on heparin with activated partial thromboplastin time ≥50. The mean CHA_2DS_2-VASc score was 2.6 ± 1.7. The means for CHA_2DS_2-VASc scores among the 3 groups were compared using Kruskal-Wallis equality-of-populations rank test (p values <0.001 for both). Baseline characteristics of the 3 groups are shown in Table 1.

INCIDENCE OF ANY NEUROLOGICAL EVENTS AFTER CARDIOVERSION. In Group 1, there were 6 patients (1.1%) who were found to have an ischemic neurological event within 30 days of their cardioversion. Of these, 4 were strokes, whereas 2 had transient ischemic attacks. In the 6 patients with neurological events, the mean CHA_2DS_2-VASc score was 3.3 ± 1.2 (range, 2 to 5). When compared with the patients without thromboembolic events, these 6 patients had a higher incidence of prior stroke (50.0% vs. 6.2%; p < 0.001) and presence of vascular disease (83.3% vs. 38.6%; p = 0.02). Hypertension, diabetes, and congestive heart failure were also more frequent in patients who had neurological events; however, these did not reach statistical significance, likely because of the small numbers. None of the patients in Group 1 had any other systemic thromboembolic complications or intracranial bleeds during this period. Transesophageal echocardiogram (TEE) was performed before 33 cardioversions in this group, with 6 of them reporting mild-moderate smoke. No thrombus or severe smoke was reported. Of note, there were no thromboembolic events following these 33 cardioversions.

Group 2 patients experienced no neurological or other systemic thromboembolic events within 30 days of cardioversion. TEE was performed before 11 cardioversions with 3 of them revealing mild to moderate smoke.

In Group 3, there were 2 neurological events within 30 days of cardioversion (0.22%). The first patient, who had a CHA_2DS_2-VASc score of 4, underwent surgical aortic valve replacement with pulmonary vein isolation 15 days after the cardioversion, after which the anticoagulation was held. Two days later he developed symptoms of an ischemic stroke. The other patient, who had a CHA_2DS_2-VASc score of 6, presented 19 days after cardioversion with a new facial droop and slurred speech. The INR on presentation was 1.14, therefore raising concerns about the patient's compliance with warfarin. These 2 patients had a significantly higher incidence of vascular disease compared with the patients in this group who did not have any thromboembolic complication (100% vs. 32.1%; p = 0.04). No other systemic thromboembolic events or intracranial hemorrhages were recorded in Group 3. A total of 140 of these cardioversions were preceded by TEE with 23 reporting mild to moderate smoke and 5 revealing severe smoke, whereas none of the patients had a left atrial thrombus (Table 2).

The odds of a neurological event between the 2 groups (Groups 1 and 3) was significantly different when compared in terms of number of cardioversions (p = 0.03) or based on number of patients (p = 0.047).

Table 3 shows the distribution of CHA_2DS_2-VASc scores in the 2 groups (Groups 1 and 3) where thromboembolic events were seen post-cardioversion. Although there were no neurological events in patients with CHA_2DS_2-VASc of 0 and 1, there was an increase in the incidence with an increase in the scores. None of the 8 patients in this report who had neurological events had a TEE before the cardioversion. Baseline characteristics for these 8 patients are described in Table 4.

TABLE 2 TEE Findings for Left Atrial Smoke Stratified by CHA_2DS_2 VASc Score

CHA_2DS_2 VASc	Total TEE	Smoke (%)			
		No	Mild	Moderate	Severe
0	15	14 (93.3)	0	0	1 (6.7)
1	28	26 (92.8)	1 (3.6)	0	1 (3.6)
2	43	37 (86)	5 (11.6)	0	1 (2.3)
3	45	27 (60)	14 (31.1)	3 (6.7)	1 (2.2)
4	23	19 (82.6)	3 (13)	0	1 (4.4)
5	15	13 (86.7)	2 (13.3)	0	0
6	11	6 (55.6)	5 (45.4)	0	0
7	3	3	0	0	0
8	1	1	0	0	0
9	1	1	0	0	0
Total	185	147	30	3	5

Values are n (%).
TEE = transesophageal echocardiogram.

TABLE 3 Thromboembolic Events Stratified by the CHA_2DS_2-VASc Scores of Patients Undergoing Cardioversion

CHA_2DS_2-VASc Score	Total Patients in All Groups	Total Events (%)	All Patients in Group 1 (%)	Events in Group 1 (%)	All Patients in Group 3 (%)	Events in Group 3 (%)
0	204	0	85 (41.7)	0	99 (48.5)	0
1	293	0	103 (35.2)	0	163 (55.6)	0
2	351	**2 (0.6)**	136 (38.8)	**2 (1.5)**	187 (53.3)	0
3	318	**1 (0.3)**	108 (34.0)	**1 (0.9)**	191 (60.1)	0
4	215	**3 (1.4)**	77 (35.8)	**2 (2.6)**	127 (59.1)	**1 (0.8)**
5	115	**1 (0.9)**	33 (28.7)	**1 (3.0)**	76 (66.1)	0
6	63	**1 (1.6)**	19 (30.2)	0	39 (61.9)	**1 (2.6)**
7	15	0	5 (33.3)	0	10 (67.7)	0
8	6	0	0	0	6 (100.0)	0
9	1	0	1 (100.0)	0	0	0

Values are n (%). Group 1: No anticoagulation (mean CHA_2DS_2-VASc, 2.30 ± 1.17). Group 3: Therapeutic anticoagulation (mean CHA_2DS_2-VASc, 2.62 ± 1.7). Group 2 is not described here because there were no reported events in this group. **Bolded** text indicates subjects with thromboembolic events.

POST-SURGICAL PATIENTS. We further performed a subgroup analysis on post-surgery patients where the symptom and/or electrocardiogram onset of AF were witnessed in the hospital. In this subset of patients there were 226 cardioversions performed in Group 1. Of these, 155 cardioversions (68.6%) were performed for symptom onset after the cardiothoracic surgery. The mean CHA_2DS_2-VASc score was 2.5 ± 1.6. There were only 7 cardioversions in Group 2 with a mean CHA_2DS_2-VASc score of 3.0 ± 1.2. In Group 3, a total of 108 cardioversions were performed post-operatively, of which 75 (69.4%) were performed in patients who developed AF after cardiothoracic surgery. Mean CHA_2DS_2-VASc score was 3.0 ± 1.9 ($p = 0.138$). Baseline characteristics in the post-surgical patients are shown in Table 5. There was no thromboembolic event noted within 30 days of the cardioversion, either with or without the use of therapeutic anticoagulation in this subset of post-surgical patients.

DISCUSSION

Our study demonstrates significantly greater risk of thromboembolic complications in patients who underwent electrical cardioversion within 48 h of symptom onset while not therapeutically anticoagulated. Traditionally it has been believed that some time is required for thrombus formation in AF with guidelines estimating relative safety for cardioversion within a 48-h window. There were initial studies performed in the emergency departments and inpatient settings validating this hypothesis (16–22). A prospective study performed by Weigner et al. (23) showed a very low incidence of thromboembolic disease in these patients, even without the use of anticoagulation, although the risk was not zero. At the same time, there were contradictory studies, challenging the 48-h hypothesis, because they revealed the presence of left atrial thrombus by TEE, performed in patients within 48 to 72 h of symptom onset (4,5). A recent study by Airaksinen et al. (24) revealed some stroke risk (0.7%) after cardioversion, especially in patients with higher CHA_2DS_2-VASc scores. As compared with this Finnish study, our study revealed incidence of neurological outcomes at 1.06%. In addition, we compared this incidence with the patients who were anticoagulated and found a significant decrease in the thromboembolic complications. A subgroup analysis was

TABLE 4 Characteristics of Patients Who Had Neurological Events Within 30 Days of Cardioversion

	Anticoagulation at Time of Cardioversion	INR at the Time of Cardioversion	$CHADS_2$-VASc Score	Timing of Stroke After Cardioversion (days)
1.	None	–	3	5
2.	None	–	2	17
3.	Aspirin	–	2	1
4.	Aspirin	–	5	3
5.	Aspirin	–	4	8
6.	Aspirin	–	4	27
7*	Warfarin	3.2	4	17
8†	Warfarin	2.2	6	19

*Patient underwent surgical aortic valve replacement with pulmonary vein isolation 15 days after the cardioversion, for which anticoagulation was held. †Patient was noncompliant with warfarin, with INR 1.14 at presentation.
INR = international normalized ratio.

TABLE 5 Baseline Characteristics of Post-Surgical Patients Undergoing Cardioversion Stratified by Their Anticoagulation Status

	No Anticoagulation	Subtherapeutic Anticoagulation	Therapeutic Anticoagulation	p Value
Number of DCC	226	7	108	
Number of patients	218	6	100	
Mean age, yrs	65.2 ± 12.4	65.3 ± 11.9	66.9 ± 11.2	0.496
Females, %	70 (31.0)	2 (28.6)	39 (36.1)	0.628
Mean CHA_2DS_2-VASc	2.5 ± 1.6	3.0 ± 1.2	3.0 ± 1.9	0.138
Medications				
Aspirin alone	95	0	0	
Warfarin	18	7	32	
Heparin	0	0	76	

Values are n, mean ± SD, or n (%).
Abbreviations as in Table 1.

further performed in our study in post-operative patients, revealing a significantly lower risk of thromboembolism in those patients. In our study population, the patients who were anticoagulated before cardioversion were shown to have a low risk of stroke.

In the therapeutically anticoagulated group, the 2 patients whose post-cardioversion courses were complicated by stroke were not in the therapeutic range when they presented with the neurological deficits. These results therefore highlight the importance of continuing the anticoagulation for at least 30 days after cardioversion without any interruptions, as recommended by the current guidelines (13). In these patients, elective surgery should be delayed beyond the 30-day period to prevent the interruption of anticoagulation. However, if a surgical procedure cannot be avoided during this period, all possible attempts should be made to bridge these patients for surgery and restart the anticoagulation as soon as possible after the surgery. This is especially important in patients with high CHA_2DS_2 VASc scores. The current guidelines for patients with acute-onset AF supports this caution, because they recommend the initiation of anticoagulation in patients with high risk of stroke undergoing cardioversion for acute onset AF (<48 h) "as soon as possible before or immediately after cardioversion," although there was recognition that this was based on weak evidence (grade 2C) (13).

In patients with lower CHA_2DS_2-VASc score of 0 and 1, we observed no neurological events irrespective of their anticoagulation status at the time of cardioversion. Most thromboembolic events occurred in patients with higher CHA_2DS_2-VASc score, as shown in Table 5. For this low-risk group, the guidelines are unclear regarding anticoagulation use at the time of cardioversion because they do not provide objective parameters for defining high and low thromboembolic risk in patients undergoing cardioversion within 48 h of symptom onset. Our study would support the use of anticoagulation at the time of cardioversion and during the post-cardioversion period for patients with CHA_2DS_2-VASc score of 2 and above.

With the increasing use of newer oral anticoagulants (NOAC), it would be worthwhile to evaluate a strategy of starting these patients on the NOAC at the onset of symptoms or findings indicating onset of AF and cardioverting them soon after. It should be noted, however, that these NOACs have not yet been validated in patients with valvular AF or in patients with high bleeding risk (25–28), and such patients were excluded in this study.

The duration of AF in nonhospitalized patients is usually judged on the basis of duration of symptom onset. This approach may not identify patients with chronic or paroxysmal AF, who asymptomatically go in and out of AF. Studies reveal the presence of undiagnosed asymptomatic AF in more than 5% of patients admitted with stroke or transient ischemic attack (29–31). Their symptoms may depend on periods of rapid ventricular rates caused by physical or mental stress, thereby misleading them regarding the onset of AF. The TRENDS study (31) also suggests that AF durations of <48 h may still expose the patient to higher thromboembolic risks, albeit in a paroxysmal AF population. Cardioverting such patients without achieving therapeutic anticoagulation may pose a higher risk of stroke.

A subgroup analysis was performed in our report on post-surgical patients who developed AF within the hospital. These patients are usually closely monitored, either on telemetry or by frequent vital signs evaluation by the nursing staff. Therefore, the time of AF onset is more objectively identified in these patients compared with nonhospitalized patients. There was no thromboembolic event after cardioversion either with or without anticoagulation in these patients, although they did have a lower baseline stroke risk indicated by their lower CHA_2DS_2-VASc score. It is possible that the absence of stroke events in this group is caused by a more accurate judgment of AF onset, or perhaps these patients have a different substrate, leading to a lower baseline risk of stroke.

In summary, our study supports the use of anticoagulation in patients undergoing cardioversion for acute onset AF with CHA_2DS_2-VASc score of 2 or more.

A similar benefit was not seen in patients with lower CHA_2DS_2-VASc score (0 or 1) and in post-surgical patients.

STUDY LIMITATIONS. Our study has limitations inherent to a retrospective study design, such as documentation errors and loss to follow-up. The risk of thromboembolism with AF is low in all the groups, therefore making it difficult for us to perform a multivariate analysis. The period in this study also had too few patients treated with NOACs, so no conclusions can be made regarding the impact of short- or long-term NOAC use for patients presenting within 48 h of AF onset for direct current cardioversion.

CONCLUSIONS

Our study supports the use of anticoagulation in patients undergoing cardioversion for acute onset AF with CHA_2DS_2-VASc score of 2 or more. A similar benefit was not seen in patients with lower CHA_2DS_2-VASc score (0 or 1) and in post-surgical patients.

REPRINT REQUESTS AND CORRESPONDENCE: Dr. Patrick J. Tchou, Department of Cardiovascular Medicine, Section of EP & Pacing, Cleveland Clinic, 9500 Euclid Avenue, Cleveland, Ohio 44195. E-mail: tchoup@ccf.org.

PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE: While current guidelines suggest that one may cardiovert nonanticoagulated patients within 48 h of atrial fibrillation onset, our data suggest caution in those patients with elevated CHA_2DS_2-VASc score of 2 or more. Although the total numbers were small, all of our nonanticoagulated patients in this study who suffered thromboembolic events post-cardioversions had higher CHA_2DS_2-VASc scores. Left atrial clots may even form soon after cardioversion. Transesophageal echocardiograph has revealed that stagnation of blood flow in the left atrial appendage may be the greatest immediately post-cardioversion. In the current era of fast-acting oral anticoagulants, it may be wise to initiate such anticoagulants at onset of atrial fibrillation or soon thereafter, before cardioversion, even if cardioversion is done within 48 h, so as to minimize the risk of thromboembolism after cardioversion. Further larger-scale evaluation of the efficacy of such a strategy would be helpful in confirming the utility of this approach.

TRANSLATIONAL OUTLOOK 1: This study paves way for risk stratification strategies for thromboembolism using $CHADS_2$-VASc score in patients with atrial fibrillation after direct current cardioversion.

TRANSLATIONAL OUTLOOK 2: With the advent of new oral anticoagulants, further studies investigating the use of these agents after cardioversion could be undertaken.

REFERENCES

1. Heeringa J, Kuip DA, Hofman A, et al. Prevalence, incidence and lifetime risk of atrial fibrillation: the Rotterdam Study. Eur Heart J 2006;27:949–53.

2. Wolf PA, Abbott RD, Kannel WB. Atrial fibrillation as an independent risk factor for stroke: the Framingham Study. Stroke 1991;22:983–8.

3. Lin HJ, Wolf PA, Kelly-Hayes M, et al. Stroke severity in atrial fibrillation: the Framingham Study. Stroke 1996;27:1760–4.

4. Stoddard MF, Dawkins PR, Prince CR, Ammash NM. Left atrial appendage thrombus is not uncommon in patients with acute atrial fibrillation and a recent embolic event: a transesophageal echocardiographic study. J Am Coll Cardiol 1995;25:452–9.

5. Manning WJ, Silverman DI, Waksmonski CA, Oettgen P, Douglas PS. Prevalence of residual left atrial thrombi among patients with acute thromboembolism and newly recognized atrial fibrillation. Arch Intern Med 1995;155:2193–8.

6. Bjerkelund O, Orning OM. The efficacy of anticoagulant therapy in preventing embolism related to DC electrical conversion of atrial fibrillation. Am J Cardiol 1969;23:208–16.

7. Lown B, Perlroth MG, Kaidbey S, Abe T, Harken DE. "Cardioversion" of atrial fibrillation: a report on the treatment of 65 episodes in 50 patients. N Engl J Med 1963;269:325–31.

8. Weinberg DM, Mancini J. Anticoagulation for cardioversion of atrial fibrillation. Am J Cardiol 1989;53:745–6.

9. Hansen ML, Jepsen RM, Olesen JB, et al. Thromboembolic risk in 16 274 atrial fibrillation patients undergoing direct current cardioversion with and without oral anticoagulant therapy. Europace 2015;17:18–23.

10. Berger M, Schweitzer P. Timing of thromboembolic events after electrical cardioversion of atrial fibrillation or flutter: a retrospective analysis. Am J Cardiol 1998;82:1545–7, A8.

11. Klein AL, Grimm RA, Murray RD, et al., for the Assessment of Cardioversion Using Transesophageal Echocardiography Investigators. Use of transesophageal echocardiography to guide cardioversion in patients with atrial fibrillation. N Engl J Med 2001;344:1411–20.

12. Camm AJ, Kirchhof P, Lip GY, et al. ESC Committee for Practice Guidelines. Guidelines for the management of atrial fibrillation: the Task Force for the Management of Atrial Fibrillation of the European Society of Cardiology (ESC). Eur Heart J 2010;31:2369–429.

13. January CT, Wann LS, Alpert JS, et al. 2014 AHA/ACC/HRS Guideline for the Management of Patients With Atrial Fibrillation. J Am Coll Cardiol 2014;64:e1–76.

14. You JJ, Singer DE, Howard PA, et al. Antithrombotic therapy for atrial fibrillation: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest 2012;141:e531S–75S.

15. Gage BF, Waterman AD, Shannon W, et al. Validation of clinical classification schemes for predicting stroke: results from the national registry of atrial fibrillation. JAMA 2001;285:2864–70.

16. Lip GY, Nieuwlaat R, Pisters R, Lane DA, Crijns HJ. Refining clinical risk stratification for predicting stroke and thromboembolism in atrial fibrillation using a novel risk factor-based approach: the euro heart survey on atrial fibrillation. Chest 2010;137:263–72.

17. Von Besser K, Mills AM. Is discharge to home after emergency department cardioversion safe

for the treatment of recent-onset atrial fibrillation? Ann Emerg Med 2011;58:517–20.

18. Vinson DR, Hoehn T, Graber DJ, Williams TM. Managing emergency department patients with recent-onset atrial fibrillation. J Emerg Med 2012; 42:139–48.

19. Burton JH, Vinson DR, Drummond K, et al. Electrical cardioversion of emergence department patients with atrial fibrillation. Ann Emerg Med 2004;44:20–30.

20. Jacoby JL, Cesta M, Heller MB, Salen P, Reed J. Synchronized emergency department cardioversion of atrial fibrillation saves time, money and resources. J Emerg Med 2005;28: 27–30.

21. Stiell IG, Clement CM, Perry JJ, et al. Association of the Ottawa Aggressive Protocol with rapid discharge of emergency department patients with recent-onset atrial fibrillation or flutter. CJEM 2010;12:181–91.

22. Xavier Scheuermeyer F, Grafstein E, Stenstrom R, et al. Thirty-day outcomes of emergency department patients undergoing electrical cardioversion for atrial fibrillation or flutter. Acad Emerg Med 2010;17:408–15.

23. Weigner MJ, Caulfield TA, Danias PG, Silverman DI, Manning WJ. Risk for clinical thromboembolism associated with conversion to sinus rhythm in patients with atrial fibrillation lasting less than 48 hours. Ann Intern Med 1997; 126:615–20.

24. Airaksinen KE, Grönberg T, Nuotio I, et al. Thromboembolic complications after cardioversion of acute atrial fibrillation of acute atrial fibrillation: the FinCV (Finnish Cardioversion) Study. J Am Coll Cardiol 2013;62:1187–92.

25. Ahmad Y, Lip GY, Apostolakis S. New oral anticoagulants for stroke prevention in atrial fibrillation: impact of gender, heart failure, diabetes mellitus and paroxysmal atrial fibrillation. Expert Rev Cardiovasc Ther 2012;10:1471–80.

26. Connolly SJ, Ezekowitz MD, Yusuf S, et al. Dabigatran versus warfarin in patients with atrial fibrillation. N Engl J Med 2009;361:1139–51.

27. Patel MR, Mahaffey KW, Garg J, et al. Rivaroxaban versus warfarin in nonvalvular atrial fibrillation. N Engl J Med 2011;365:883–91.

28. Granger CB, Alexander JH, McMurray JJ, et al. Apixaban versus warfarin in patients with atrial fibrillation. N Engl J Med 2011;365:981–9.

29. Grond M, Jauss M, Hamann G, et al. Improved detection of silent atrial fibrillation using 72-hour Holter ECG in patients with ischemic stroke: a prospective multicenter cohort study. Stroke 2013;44:3357–64.

30. Salvatori V, Becattini C, Laureti S, et al. Holter monitoring to detect silent atrial fibrillation in high-risk subjects: the Perugia General Practitioner Study. Intern Emerg Med 2015;10: 595–601.

31. Glotzer TV, Daoud EG, Wyse G, et al. The relationship between daily atrial tachyarrhythmia burden from implantable device diagnostics and stroke risk: the TRENDS Study. Circ Arrhythm Electrophysiol 2009;2:474–80.

KEY WORDS atrial fibrillation, cardioversion, stroke

Go to http://www.acc.org/jacc-journals-cme to take the CME quiz for this article.