Self-Assessment Question 1

How comfortable are you with early stroke prevention in patients with Afib?

A. Very Comfortable
B. Comfortable
C. Undecided
D. Uncomfortable
E. Very Uncomfortable

Self-Assessment Question 2

What is the first treatment priority when treating a patient with atrial fibrillation?

A. Rhythm control
B. Rate Control
C. Risk assessment for stroke/thromboembolism
Self-Assessment Question 3

What is the CHADS\textsubscript{2} score and risk of stroke for a 76 year old woman with atrial fibrillation and blood pressure of 145/100?

A. CHADS =1, over 2% per year  
B. CHADS =2, over 4% per year  
C. CHADS =3, over 6% per year  
D. CHADS =4, over 8% per year

Self-Assessment Question 4

If cardioversion was unsuccessful and a decision is made to allow atrial fibrillation to continue uncorrected, the arrhythmia is classified as:

A. Paroxysmal  
B. Persistent  
C. Permanent
Self-Assessment Question 5

If a patient who has required a stable dose of warfarin develops an INR of 3.5, what is the next step?

A. Perform a genotyping test for warfarin sensitivity
B. Monitor INR more frequently
C. Lower the dose of warfarin
D. Give the patient vitamin K

Self-Assessment Question 6

If switching a patient to a new oral direct thrombin inhibitor or Factor Xa inhibitor, when should the new agent be started after stopping warfarin?

A. 1 week after stopping warfarin
B. When INR ≤ 1.0
C. When INR ≤ 2.0
D. Same day as stopping warfarin
Self-Assessment Question 7

Is the following statement True or False: Restoring sinus rhythm enables patients to discontinue antithrombotic therapy.

A. True  
B. False

Self-Assessment Question 8

When discussing rhythm control strategies, how often should you communicate with patients the need to continue antithrombotic therapy for stroke prevention?

A. 100% of cases  
B. 75% of cases  
C. 50% of cases  
D. 25% of cases  
E. 0% of cases
Question 1

What is the first treatment priority when treating a patient with atrial fibrillation?

A. Rhythm control  
B. Rate Control  
C. Risk assessment for stroke/thromboembolism
PATHWAY TO DIAGNOSIS AND MANAGEMENT IN ATRIAL FIBRILLATION

Clinical Evaluation
- Electrocardiogram
- Assess for triggering factors
- Assess for associated medical conditions
- Triage according to clinical presentation: hemodynamic stability, patient's condition, complications
- Transthoracic echocardiogram
- Holter monitor, nocturnal test or ambulatory telemetry monitoring to assess rate control

Management
Step 1: Assess hemodynamic stability
Step 2: Control the ventricular rate
Step 3: Identify the risk of stroke/systemic embolism
Step 4: Assess risk and benefits of rhythm control

ASSESSING AND MITIGATING RISK IN STROKE PREVENTION

1. Assess for stroke risk with the CHADS2 scoring system, which captures well-validated risk factors

<table>
<thead>
<tr>
<th>Risk Criteria</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive heart failure</td>
<td>1</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1</td>
</tr>
<tr>
<td>Age (75+ years)</td>
<td>1</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1</td>
</tr>
<tr>
<td>Stroke, TIA, or systemic embolism history</td>
<td>2</td>
</tr>
</tbody>
</table>

Additional modifiers to consider in aortic valve or intermediate cases:
- Diabetes (less than 1.80 or medical therapy)
- Age (65–74 years)
- Female

2. Assess bleeding risk to make a risk-benefit decision

<table>
<thead>
<tr>
<th>FACTORS THAT INCREASE BLEEDING RISK AT ABD</th>
<th>BLOOD</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of hypertension greater than 100 systolic</td>
<td>1</td>
</tr>
<tr>
<td>Advanced age or poor functional status</td>
<td>1</td>
</tr>
<tr>
<td>Prior stroke</td>
<td>1</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>1</td>
</tr>
<tr>
<td>Age (65–74 years)</td>
<td>1</td>
</tr>
<tr>
<td>Female</td>
<td>1</td>
</tr>
<tr>
<td>Alcohol consumption (3 or more alcoholic drinks per week)</td>
<td>1</td>
</tr>
</tbody>
</table>

3. Select optimum antithrombotic therapy
- Anticoagulant therapy is preferred in high and intermediate risk patients
- Bleeding risk should be considered in choosing options antithrombotic therapy
Question 2

What is the CHADS\textsubscript{2} score and risk of stroke for a 76 year old woman with atrial fibrillation and blood pressure of 145/100?

A. CHADS =1, over 2% per year
B. CHADS =2, over 4% per year
C. CHADS =3, over 6% per year
D. CHADS =4, over 8% per year
If cardioversion was unsuccessful and a decision is made to allow atrial fibrillation to continue uncorrected, the arrhythmia is classified as:

A. Paroxysmal  
B. Persistent  
C. Permanent
CONTROL OF VENTRICULAR RATE IS THE FIRST PRIORITY IN MANAGING PATIENTS WITH AFIB, AS THIS WILL OFTEN AMELIORATE SYMPTOMS AND PREVENT HEMODYNAMIC DECOMPENSAATION.

Classifications of AFib

- Paroxysmal: Usually self-terminating within 7 days
- Persistent: Lasts longer than 7 days without documented interruption
- Permanent: AFib is accepted and allowed to continue after measures to restore sinus rhythm have either failed or are not attempted

Consider beta-blockers and non-dihydropyridine calcium channel antagonists for long-term management:

- Avoid digoxin monotherapy in patients with paroxysmal AFib
- Rather than using digoxin alone, add a beta-blocker or non-dihydropyridine calcium channel antagonist to control the rate both at rest and during exercise
- Consider rate control agents in patients with controlled heart failure
- Use amiodarone in patients where other medications are contraindicated

STROKE PREVENTION

TAKE TIME TO ASSESS AND STRATIFY RISK

- Assessing stroke risk does not eliminate need for antiplatelet therapy in patients with AFib

OPTIMIZE WARFARIN THERAPY WITH FREQUENT MONITORING

- Monitoring INR is an essential part of warfarin therapy
- When INR is in the therapeutic range (2.0–3.0), check after 1 week of stable INR
- When INR is out of therapeutic range, check every 3–5 days
- Long-term care: monitor interval 6 weeks
- Last INR before other medications should be taken 2–3 weeks

CONSIDER PATIENT RACE AND ASC WHEN DOING

- Anticoagulation
- Consideration for stroke prevention in patients with AFib
- Stroke prevention in patients with AFib

SUPRATHERAPEUTIC INR (4.0–6.0)

- Supratherapeutic INR increases the risk of hemorrhage
- Supratherapeutic INR is associated with increased risk of stroke in patients with AFib

INCREASE MONITORING FREQUENCY BEFORE RENEWAL OR 
CHANGES IN THE NOXIOUS THERAPEUTIC RANGE

- INR values in the therapeutic range require less monitoring
- INR values outside the therapeutic range require more frequent monitoring

For further information, consult a dermatologist.
Question 4

If a patient who has required a stable dose of warfarin develops an INR of 3.5, what is the next step?

A. Perform a genotyping test for warfarin sensitivity
B. Monitor INR more frequently
C. Lower the dose of warfarin
D. Give the patient vitamin K
**Question 5**

If switching a patient to a new oral direct thrombin inhibitor or Factor Xa inhibitor, when should the new agent be started after stopping warfarin?

A. 1 week after stopping warfarin  
B. When INR ≤ 1.0  
C. When INR ≤ 2.0  
D. Same day as stopping warfarin
RHYTHM CONTROL

When deciding about rhythm control, consider the pattern of AFib, symptom severity and functional status and how long-term AFib may affect the patient's overall prognosis.

Rhythm control strategy is most suited to patients who are symptomatic of the arrhythmia.

Strategies include:
- Antiarrhythmic drugs
- Cardioversion
- Catheter-based ablation

Rhythm control is not a substitute for antithrombotic therapy or a path to stopping antithrombotic therapy for patients with stroke risk factors.

Toxicity and risks must be carefully considered and discussed with the patient.

*Strategies based on evidence.
Question 6

Is the following statement True or False: Restoring sinus rhythm enables patients to discontinue antithrombotic therapy.

A. True
B. False

When deciding about rhythm control, consider the pattern of AFib, symptom severity and functional status and how long-term AFib may affect the patient's overall prognosis.

Rhythm control strategy is most suited to patients who are symptomatic of the arrhythmia.

Strategies include:
- Antiarrhythmic drugs
- Cardioversion
- Catheter-based ablation

Rhythm control is not a substitute for antithrombotic therapy or a path to stopping antithrombotic therapy for patients with stroke risk factors.

Toxicity and risks must be carefully considered and discussed with the patient.
Question 7

When discussing rhythm control strategies, how often should you communicate with patients the need to continue antithrombotic therapy for stroke prevention?

A. 100% of cases
B. 75% of cases
C. 50% of cases
D. 25% of cases
E. 0% of cases
Today’s Patient: Alex K.

Presents to ER with dizziness, right-sided headache, and loss of vision in the left eye for 1 hour

Medical History:
• 63-year-old male, executive in the banking industry
• Heart murmur since childhood
• Hypertension treated for 10 years
• 3 episodes of paroxysmal atrial fibrillation 5-7 years ago associated with palpitation, but no recurrence since he began a β-blocker
• Coronary angiography performed 4 years ago to evaluate exertional dyspnea
  ─ Nonobstructive coronary disease
  ─ Mildly elevated LVEDP (15 mmHg)
  ─ Normal LV function (ejection fraction 65%)
Today’s Patient: Alex K. (continued)

Current Medications
• Valsartan, 160 mg daily
• Hydrochlorothiazide, 25 mg daily
• Nebivolol, 10 mg daily
• Amlodipine, 5 mg daily
• Aspirin, 81 mg daily
• Vitamins

Question 1: Should this patient have been on anticoagulant therapy?

- Yes
- No
- Maybe (need more information)

Please select “Yes”, “No”, or “Maybe”
Expert Faculty Opinion

The most appropriate response is:

Yes – The patient should have been anticoagulated

Physical Examination

Appears mildly anxious but otherwise comfortable

- HR +77 bpm, irregularly irregular; BP 132/75 mmHg, afebrile
- No vascular bruits
- Chest clear
- No JVD; normal arterial pulse contour
- Precordial impulse not displaced
- 1st and 2nd heart sounds normal; soft early systolic click and grade II/VI apical mid-systolic murmur unchanged during the respiratory cycle, or with handgrip or Valsalva strain; no gallop or rub
- Pulses intact; no edema
- Neurologically intact except for equivocal Babinsky reflex (left)
Admission EKG

Lab Results

<table>
<thead>
<tr>
<th>Sodium</th>
<th>138 mEq/L</th>
<th>Cholesterol</th>
<th>138 mg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium</td>
<td>4.0 mEq/L</td>
<td>Triglycerides</td>
<td>42 mg/dL</td>
</tr>
<tr>
<td>Chloride</td>
<td>99 mEq/L</td>
<td>HDL cholesterol</td>
<td>50 mg/dL</td>
</tr>
<tr>
<td>CO₂</td>
<td>29.3 mEq/L</td>
<td>LDL cholesterol</td>
<td>80 mg/dL</td>
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<tr>
<td>Glucose</td>
<td>83 mg/dL</td>
<td>TSH</td>
<td>2.4 μIU/mL</td>
</tr>
<tr>
<td>Urea nitrogen</td>
<td>18mg/dL</td>
<td>Albumin</td>
<td>3.9 g/dL</td>
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<tr>
<td>Creatinine</td>
<td>1.1mg/dL</td>
<td>Protein total</td>
<td>6.3 g/dL</td>
</tr>
<tr>
<td>White blood cells</td>
<td>6.3x10³μL</td>
<td>Bilirubin total</td>
<td>0.2 mg/dL</td>
</tr>
<tr>
<td>Red blood cells</td>
<td>4.73x10⁶μL</td>
<td>Alk. Phosphatase</td>
<td>106 u/L</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>13.7 g/dL</td>
<td>AST (SGOT)</td>
<td>96 u/L</td>
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<tr>
<td>Hematocrit</td>
<td>42.3%</td>
<td>ALT (SGPT)</td>
<td>115 u/L</td>
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<tr>
<td>Platelets</td>
<td>221x10³μL</td>
<td>HbA1c</td>
<td>5.3%</td>
</tr>
<tr>
<td>Prothrombin time (INR)</td>
<td>11.6 sec (1.0)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
MRI

- Small acute right occipital cortical and sub-cortical infarcts
- No evidence of associated hemorrhage
- MRA: No significant arterial stenosis, occlusion or aneurysm
Echocardiogram

- Normal left ventricular systolic function; ejection fraction 60%
- Normal left ventricular size
- Normal right ventricular function
- Normal right ventricular size
- Moderate left atrial dilatation
- Mild right atrial dilatation
- Mild to moderate mitral regurgitation
- Mitral valve fibrocalcification
- Mitral annular fibrocalcification

Hospital Course

- **Stroke Service in ER decided against thrombolytic therapy since symptoms and signs had resolved**
- **Admitted to the neurological unit**
  - IV heparin initiated (aPTT 60-70 seconds) along with warfarin, 5 mg/d
  - Remained clinically stable – no further CNS symptoms
  - Afib persistent
- **Discharged on the 5th hospital day**
  - INR = 1.8
  - Warfarin, 6 mg/d
Medication Regimen at Discharge

Current Medications

• Valsartan, 160 mg daily
• Hydrochlorothiazide, 25 mg daily
• Nebivolol, 10 mg daily
• Amlodipine, 5 mg daily
• Vitamins
• Warfarin

Question 2: Was initial use of heparin reasonable?

Yes
No
Maybe (need more information)

Please select “Yes”, “No”, or “Maybe”
Expert Faculty Opinion

The most appropriate response is:

Yes – Initial use of heparin was reasonable in this case

Question 3: Was it reasonable to discharge the patient before the INR reached the therapeutic range?

Yes
No
Maybe (need more information)

Please select “Yes”, “No”, or “Maybe”
Expert Faculty Opinion

The most appropriate response is either:

N No    OR    ? Maybe (need more information)

Patient: Follow-up post discharge

• Day 2 INR = 2.3 in office
• Warfarin continued at 6 mg/d
• Week 1 INR = 2.3
Back to the Patient in Office Visit 3 Weeks Post Discharge

Alex: 3 Week Office Visit Summary

- Asymptomatic
  - no recurrence of CNS symptoms
- Pulse regular
- BP 110/75 mmHg
- Examination otherwise unchanged
- INR = 2.4
- Patient dislikes the bruising he’s noticing with warfarin
- Patient concerned about negative cognitive changes with warfarin as he’s seen in elderly family members
Alex: 3 Week Office visit

EKG

Question 4: What is the biggest concern for Alex going forward?

- Bleeding complications
- TIA/stroke recurrence
- Improving Afib control

Please select “Yes”, “No”, or “Maybe”
Expert Faculty Opinion

The most appropriate responses are:

A  Bleeding complications
B  TIA/stroke recurrence

Consider all of the following as “Maybe”:

C  Improving Afib control
Frequently Asked Questions from *Strike before Stroke: Intervening Early in Patients with Atrial Fibrillation*

1. Do we administer or not administer heparin at the time of hospitalization?

The optimal timing for initiation of anticoagulation after a stroke or TIA in a patient with AF is controversial. In short, study results are mixed, with increased bleeding risk offsetting stroke reduction when using heparin to prevent recurrent strokes within the first 14 days of symptoms. However, we need to be mindful of the substantial risk of deep vein thrombosis risk and pulmonary embolism after stroke, and use prophylaxis appropriately.

In patients with AF and minor strokes or TIAs, oral anticoagulation was initiated within 14 days of onset of symptoms, with satisfactory results (EAFT trial). With extensive hemorrhagic transformation or uncontrolled hypertension, longer delay may be appropriate. The Cerebral Embolism Study Group recommended that acute anticoagulation be withheld for at least 48 hours and for even longer if there is hemorrhagic conversion or large infarction with edema.

**Evidence Summary for Heparin during AF Hospitalization**

The low molecular weight heparin dalteparin (100 units/kg twice daily) started within 30 hours after stroke onset was associated with no reduction in the primary outcome of recurrent stroke during the first 14 days (8.5% vs 7.5%; \( P=0.73 \); odds ratio [OR] 1.13; 95% CI 0.57–2.24), relative to aspirin 160 mg. A trend emerged toward higher rates of symptomatic cerebral hemorrhage that did not reach statistical significance (2.7% vs 1.8%; \( P=0.54 \); OR 1.52; 95% CI 0.42–5.26). Advanced age, higher initial stroke severity, elevated levels of C-reactive protein (CRP), and certain hemostatic factors were associated with poorer outcomes in a post hoc subgroup analysis.

Similar findings were reported when unfractionated heparin (UFH) was used in the International Stroke Trial. The rates of recurrent stroke during the first 14 days were 2.3%, 3.4%, and 4.9% with high-dose UFH (12,500 units subcutaneously twice daily), low-dose UFH (5000 units subcutaneously twice daily), and no heparin, respectively (\( P=0.001 \)). Hemorrhagic stroke rates during this period were 2.8%, 1.3%, and 0.4% for the three treatment groups (\( P<0.0001 \)), and the combined rates of stroke or death were 18.8%, 19.4%, and 20.7% (\( P=NS \)). While heparin was effective in lowering the risk of early recurrent stroke, an increased rate of bleeding complications negated this benefit.

2. How do we decide who should receive the novel anticoagulant, and how do we initiate therapy?

The key question is whether the net clinical benefit associated with these new agents is better than warfarin for a given patient with AF. It has been suggested that patients taking warfarin with good INR control may gain little by substituting one of these new agents, while other patients with AF and risk factors for stroke could benefit. The best candidates for switching are as follows:

- Patients without access to a high-quality anticoagulation monitoring system
- Patients who are struggling and in target INR only 40% to 60% of time on warfarin owing to the inherent difficulties of this agent
To date, only one novel oral anticoagulant—dabigatran etexilate—has been approved by the U.S. Food and Drug Administration for use to prevent stroke and systemic embolism in patients with nonvalvular AF.

Some key facts about dabigatran etexilate, a direct thrombin inhibitor:
- Use a fixed dose of 150 mg twice daily in patients with adequate renal function (creatinine clearance >30 mL/min).9,10
- Dabigatran has drug interactions with P-glycoprotein inhibitors such as verapamil, amiodarone, and quinidine.
  - This interaction involves increased clearance from the gut.
- The rate of intracranial hemorrhage in the RE-LY trial was lower with dabigatran than during warfarin anticoagulation.9,10,11

Other oral anticoagulant agents currently in phase III development are selective inhibitors of factor Xa, a key protein in the coagulation cascade.12 All of the agents are administered in essentially fixed doses without monitoring of anticoagulation intensity, and some are dose-adjusted based upon renal function.

Some of these novel oral anticoagulants have clinically important antithrombotic effects and can increase the risk of hemorrhage.

Adjusted-dose warfarin is highly efficacious and relatively safe if carefully administered; hence, substantial evidence of relative safety and efficacy must be mounted to favor an alternative.

**Switching from Warfarin to Novel Anticoagulants**

- Have the patient stop their VKA antagonist and monitor INR every 1 to 2 days.
  - VKAs take several days to clear the body, but novel anticoagulants show an onset of action in a few hours, so patients have adequate coverage.
- Once INR <2.0, initiate the novel anticoagulant at the recommended dose.
  - Since dabigatran is taken twice a day, have patients start the first dose either in the morning or at night. The drug can be taken with or without food.

3. When is it appropriate to initiate genetic testing for warfarin sensitivity?

Careful patient selection and a well-organized anticoagulation management program are the most important means of achieving high-quality anticoagulation.

The future of genetic testing for warfarin management remains to be decided. Present thoughts on genetic testing are as follows:
- Use only when initiating therapy in cases where there are difficulties stabilizing the individual.
- Avoid testing in patients who are already stabilized since minimal useful information is obtained.

The cost of genetic testing and whether insurance will cover these tests can place a significant burden on patients.

The core question is whether addition of genotype data into the initial dose estimate is more or less cost-effective than more frequent INR measurements. Genetic determinants of warfarin
sensitivity are a factor that could potentially improve the quality of VKA therapy. However, it is unclear whether initial warfarin dose selection informed by pharmacogenetic data will translate into improved TTR or clinical outcomes.\textsuperscript{13,14} At least one large trial is under way to test the incremental value of testing for polymorphisms associated with warfarin sensitivity or resistance.\textsuperscript{14}

4. How do you assess a patient with atherosclerosis and atrial fibrillation for stroke risk?

Stroke rates in patients with AF who had coronary artery disease range from 2.9\% to 8.2\% per year.\textsuperscript{15,16} But, the stroke rate in those with coronary artery disease and no other recognized stroke risk factors is unknown at this time.\textsuperscript{15,17}

Limited evidence suggests that atherosclerosis in the form of complex aortic plaque in the descending aorta identified by transesophageal echocardiography independently predicts stroke risk in patients with AF.\textsuperscript{20,21} However, coronary artery disease did not independently predict stroke in three separate patient cohorts\textsuperscript{15,20,21} where CAD was defined as prior myocardial infarction or angina pectoris with or without revascularization.

The CHA\textsubscript{2}DS\textsubscript{2}VASc score builds upon the CHADS\textsubscript{2} risk stratification scheme by taking cardiovascular disease into account. In addition to the CHADS\textsubscript{2} score, the VASc criteria are as follows\textsuperscript{22}:

<table>
<thead>
<tr>
<th>Factor</th>
<th>Point Assignment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vascular disease (history of myocardial infarction, peripheral artery disease, or complex aortic plaque)</td>
<td>1 point</td>
</tr>
<tr>
<td>Age 65 to 74 years</td>
<td>1 point</td>
</tr>
<tr>
<td>Age &gt; 75 years</td>
<td>2 points</td>
</tr>
<tr>
<td>Female sex</td>
<td>1 point</td>
</tr>
</tbody>
</table>

- High stroke risk = ≥ 2 points
- Moderate stroke risk = 1 point
- Low risk = 0 points

The CHA\textsubscript{2}DS\textsubscript{2}VASc score categorizes a smaller proportion of patients into the intermediate risk group compared with the revised CHADS\textsubscript{2} approach.\textsuperscript{23,24} Ongoing independent validation studies should provide additional evidence about the CHA\textsubscript{2}DS\textsubscript{2}VASc schema regarding its performance vs existing risk schema, particularly in non-anticoagulated cohorts. The 2010 European clinical practice guidelines for management of patients with AF advocate the use of the CHA\textsubscript{2}DS\textsubscript{2}VASc score.\textsuperscript{25}

5. Which is preferable during work-up in AF: transthoracic echo or transesophageal echocardiogram?
Transesophageal echocardiography (TEE) provides high-quality images of cardiac structure and function, but is not part of the standard initial evaluation of patients with AF. However, TEE is the most sensitive and specific technique to detect sources and potential mechanisms of cardiogenic embolism. TEE offers improved visualization of the left atrial appendage, which is a common site for thrombus formation in AF.

Several TEE features have been associated with thromboembolism in patients with nonvalvular AF:
- thrombus in the left atrium (LA) or left atrial appendage (LAA)
- spontaneous echo-contrast (a marker of stasis in the LA and LAA)
- reduced LAA flow velocity
- atheromatous plaque in the thoracic segments of the aorta

Although these features are associated with cardiogenic embolism, prospective investigations are needed to compare these TEE findings with clinical and transthoracic echocardiographic (TTE) predictors of thromboembolism.

The technology is used to enhance stroke risk stratification in patients with AF and to guide cardioversion. Detection of thrombus in the LA or LAA provides convincing evidence of a cardiogenic mechanism.

- LA or LAA thrombus was detected via TEE in 5% to 15% of patients with AF and prior cardioversion.

When thrombus has been present for longer than 48 hours, a TEE-guided strategy is an alternative to the traditional approach of anticoagulation for 4 weeks before and 4 weeks after elective cardioversion; both resulted in similar rates of thromboembolism (less than 1% over 8 weeks).

Thromboembolism after conversion to sinus rhythm can occur in the absence of thrombus detected by TEE. These thromboembolic events typically occur relatively early after cardioversion in patients who were not anticoagulated, emphasizing the need for therapeutic anticoagulation in patients with AF undergoing cardioversion even when thrombus is not identified.
References


