Subclinical Atrial Fibrillation: A New Paradigm for Stroke Prevention

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Subclinical Atrial Fibrillation: A New Paradigm for Stroke Prevention

CHU-PAK LAU

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LAU: Subclinical Atrial Fibrillation: A New Paradigm for Stroke Prevention. Approximately one out of five strokes is associated with atrial fibrillation (AF). AF is often intermittent and asymptomatic. Detection of AF after cryptogenic stroke will change therapy from antiplatelet to oral anti-coagulation agents for secondary stroke prevention. A critical step is to convert "covert" AF into ECG documented AF. External rhythm recording devices have registered a high incidence of AF to occur after a cryptogenic stroke, but are limited by short duration of continuous recordings. Invasive cardiac monitoring using insertable leadless cardiac monitors (ICMs) are sensitive means to identify subclinical AF (SCAF) after cryptogenic stroke, and AF has been reported to occur in up to 30% of these patients. It will be even more attractive to identify SCAF before a stroke occurs. Recent series in pacemaker and implantable cardioverter defibrillator showed that short episodes of SCAF increased stroke risk, with odds ratio ~2.2-3.1 compared to those without SCAF recorded. However, temporal sequence of recorded SCAF and stroke occurrence was uncertain, and the overall stroke risk was lower compared with patients with clinical AF at similar risk scores. This article reviews the incidence and clinical role of using implanted devices to detect SCAF and discuss the implication of SCAF so detected in primary and secondary stroke prevention. (J HK Coll Cardiol 2015;23:10-20)

Atrial Fibrillation, Implantable Cardioverter Defibrillator, Pacemaker, Stroke

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SUBCLINICAL ATRIAL FIBRILLATION

Introduction

Epidemiological evidence suggests that atrial fibrillation (AF) increase ischaemic stroke risk by 5- to 6-fold independent of other risk factors. AF related stroke tends to be more severe, and mortality rate is higher (70-80%) compared with stroke without AF. There is also a high recurrence rate.

AF episodes are often asymptomatic, and AF may present for the first time with complications such as thromboembolism and heart failure. Indeed, paroxysmal AF has similar stroke risk compared to sustained AF. About 25% of strokes has no obvious underlying cerebrovascular disease or other stroke risk factors. AF is an important underlying mechanism for cardioembolism in these patients. Apart from overt neurological deficits, recurrent cerebral emboli can cause cognitive dysfunction and dementia.

In the absence of documented AF, secondary and primary prophylaxis of stroke relies on the use of antiplatelet agents. If AF is the cause of stroke, aspirin can reduce stroke risk by 22% compared to placebo. However, oral anticoagulation using warfarin can additional reduce this risk by 38-63% compared to aspirin. More recently, non-vitamin K oral anticoagulation agents (NOACs) have been shown to be at least non-inferior to warfarin and have a lower incidence of major haemorrhagic complications, thus improving the risk-benefits ratio of stroke prevention in AF. This background makes primary and secondary prophylaxis of AF related stroke attractive. A critical step is to document underlying AF.

AF Documentation

Due to the intermittent occurrence and often asymptomatic presentation of AF, routine ECG, 24 hour Holter and patient triggered recording devices have low detection rate of AF. Several types of external monitors with attached electrodes have allowed intermediate term continuous AF monitoring. Electrodes used include wet or dry electrodes. These provide not only patient triggered recordings, but automatic recording if AF occurs. Patient tolerability has improved with improved electrode design.

Longer term recording of cardiac rhythm is possible with implantable leadless cardiac monitors (ICMs), such as by the Medtronic Reveal XT™. As P waves are not well detected in ICMs, irregularity of RR interval is used as a surrogate for AF, the so called Lorentz plot is arithmetically used to register AF. This had been tested in the XPECT Trial. In this study, 247 patients with high AF burden received the Reveal XT™. The stored rhythm was recorded simultaneously with the ECG to an external Holter monitor for 46 h. This showed a sensitivity, specificity, positive predictive value and negative predictive value of the Reveal XT to identify AF of 96.1, 85.4, 79.3 and 97.4% respectively, with an accuracy of 98.5%. Further, false positivity is reduced by superimposing the R waves to examine for a possible P wave. The device has been further miniaturized and can be implanted with an injection mechanism (the LINQ™, Medtronic).

Obviously, cardiovascular implantable electronic devices (CIEDs) with attached atrial electrodes provide an excellent recording of AF. In the pacemaker population, Israel et al showed in CIEDs, a significantly higher sensitivity of AF detection

<table>
<thead>
<tr>
<th>Table 1. Technical aspects of AF detection using implanted atrial leads</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Closely spaced atrial bipolar lead</td>
</tr>
<tr>
<td>2. Bipolar sensing (atrial tip-atrial ring)</td>
</tr>
<tr>
<td>3. High atrial sensitivity (0.1-0.5 mV) (minimise under/intermittent sensing for AF detection)</td>
</tr>
<tr>
<td>4. Short PVARP</td>
</tr>
<tr>
<td>5. PVAB ≥25 ms to reject far field R wave</td>
</tr>
<tr>
<td>6. AEGM validation</td>
</tr>
<tr>
<td>7. Duration of AHRE to consider as AF</td>
</tr>
</tbody>
</table>

AF=Atrial fibrillation; AEGM=atrial electrogram; AHRE=Atrial high rate episode PVAB=Post ventricular atrial blanking; PVARP=Post ventricular atrial refractory period
compared to Holter recordings. Table 1 shows the technical aspect to detect AF in CIEDs. Accurate recording requires a closely spaced atrial bipole (<1 cm), an appropriate atrial sensitivity setting, post ventricular atrial refractory period (PVARP) and blanking (PVAB) adjustment.

Programming appropriate atrial rates and episode durations cut off is also critical to register AF. A low cut off rate of AF detection will increase false positivity due to inclusion of noises and far field events such as R wave, whereas a high rate cut off will miss AF episodes when atrial signals become small or fall within the PVARP and PVAB. Programming a short cut off episode duration will increase sensitivity to detect brief AF episodes, but will include false positive detection of noise, whereas a longer cut off duration will increase accuracy but miss brief AF events. A validation of the Medtronic AT500 and GEM III AT algorithms suggested sensitive AF detection if far field R waves were rejected. In 5,769 pacemaker detected "atrial high rate events" (AHREs), Kaufman et al examined the relative contribution of programmed cut off detection rate and duration on accuracy of AF detection. An increase in cut off detection rate from 190 to 250 bpm reduces false positive detection, especially if shorter AF detection rates were programmed. A cut off detection duration of >6 minutes will have a 17.3% false positive detection, compared to 3.3% if detection duration of >6 hours was programmed. It was concluded that validation by atrial electrograms (AEGMs) would be important for shorter detected AF episodes of >6 minutes, whereas this became less critical for longer episodes >6 hours. As most studies do not vigorously relate symptoms with device detected AF episodes, this review uses the term subclinical AF (SCAF) for AF detection by implanted, to distinguish them from the occurrence of clinical AF.

**Secondary Prevention in Cryptogenic Stroke**

Kishore et al summarized 32 trials which have used either external monitors or ICMs to detect AF in patients after ischaemic stroke or transient ischemic attack (TIA). There was substantial heterogeneity between studies, with an overall detection rate of any new AF occurring in 11.5%, with higher detection rate in selected (e.g. cryptogenic stroke) versus unselected patients groups (13.4 vs 6.2%). Longer duration of recording and older patient age increased the chance of AF detection. There was insufficient data on the stroke type (lacunar vs non lacunar) and the timing of starting recording after the index stroke on AF detection rate. A large prospective study recruited 572 ambulatory patients with a mean age of 55 years at a mean of 75 days after a stroke or TIA. Patients were randomized to receive either a 30-day event-triggered external recorder using dry electrodes or with another 24h Holter recording. The primary end point was detected AF >30 s, which was reached in 16.1% of patients versus only 3.2% using Holter only, and had led to an increase in oral anticoagulation use (18.6 vs 11.1%). Clinical AF was only detected in 0.5% of patients after 90 days, and AF was more often detected if the device was administered within 30 days of the index stroke. The limitations of this study are the delay in administrating AF recording, exclusion of more serious stroke and non-ambulatory patients, and lack of AF burden measurement (as only <2.5 minutes per episode can be measured). Occurrence of AF after 30 days was not determined by this study.

More prolonged monitoring using invasive recordings in a similar population was reported in the CRYSTAL AF study. In 441 patients at a mean of 38 days after a cryptogenic stroke either an external Holter or ICM (Reveal XT) was used to assess the time of first AF >30 s occurrence (Figure 1). AF was detected in 12.4% compared with 2.0% using ICM compared to Holter (p<0.001), again resulting in a higher percentage of oral anticoagulation use (14.7 vs 6.0%, p<0.007), and a trend to a lower recurrent stroke rate (7.1 vs 9.1%). At 3 years, the device projected battery life, AF was detected in 30% of patients. Most of the episodes of detected AF were asymptomatic (74% and 79% at 6 and 12 months respectively). Other prospective studies have documented a variable incidence of AF detection of
15.9%, with the results of the Stroke Prior to Diagnosis of Atrial Fibrillation Using Long Term Observation with Implantable Cardiac Monitoring Apparatus Reveal (SURPRISE) showing an incidence of new AF in 18.6% in 3 years.

Taken together, AF is a common occurrence in cryptogenic stroke and its detection will significantly affect the use of antithrombotic treatment. Since AF is detected in up to 30% of such patients in 3 years, arguably antithrombotic therapy with NOACs may be considered in such patients even without AF documentation. This is the subject of 2 prospective randomized studies in which either dabigatran or apixaban will be compared to aspirin in patients with cryptogenic stroke.

**Primary Prevention of Stroke by Early Scaf Detection Using Implanted Devices**

The strong association between clinical AF and ischaemic stroke, and the proven benefit of anticoagulation prophylaxis make early AF detection of clinical interest. Implanted CIEDs are the most reliable methods to detect AF. However, especially for shorter and asymptomatic SCAF episodes, it is uncertain if they predict clinical AF development or stroke (and other thromboembolic events) themselves. The relationship of SCAF detected by CIEDs and future clinical AF and stroke provides important background information for the clinical importance of SCAF.

**Frequency of SCAF Detected by CIED and Relation to Clinical AF**

Gillis et al reported SCAF to be detected in 68% of 231 patients with sinus node disease, and an incidence of 50.6% of SCAF was documented in 617 patients with DDD pacemakers. An incidence of 44% of AF was detected in 226 patients during a long follow up period of 7 years, with a much higher incidence of AF in

![Figure 1](image-url)
patients with a prior history of AF than those without (87 vs 22%). Independent of prior history, AF detection was associated with a 10-fold increase in incidence of persistent AF and 2.5-fold increase in major cardiovascular events. Persistent AF occurred in 22% patients. With AEGM validation, a 55% incidence of SCAF was recorded in 254 patients with 54% of them with sinus node disease.10 Several studies have also shown an incidence of SCAF in 69-79% in patients with prior history of AF,19-21 and 25-45% in patients without prior history.10,22-26

Two studies19,26 have examined the relationship of SCAF detected by device and the development of clinical AF (Table 2). In the retrospective MOST study,19 patients with an episode of SCAF (rate ≥220/min, duration > 5 minutes) increased risk of clinical AF by 5.9 times. The ASSERT study26 prospectively evaluated 2,580 patients without prior history of AF and found a 10.1% incidence of SCAF (defined as atrial rate ≥190/minute and > 6 minutes) at 3 months, after a 1 month post-implant blanking period. The presence of SCAF increases the risk of clinical AF by 5.6 times.

Taken together, SCAF is commonly recorded in patients with CIEDs, and the incidence appears to be higher in those with a prior history of AF than those without. Furthermore, in patients with a recorded SCAF episode with or without prior history of AF, future risk for clinical AF to develop would be at least 5 times higher.

### SCAF in Predicting Stroke and Other Thromboembolism

Five large studies have examined the relationship of CIED recorded SCAF and future thromboembolic risk (Table 2).19-21,25,26 With the exception of the ASSERT, all studies had included some or all patients with prior history of clinical AF. Oral anticoagulation (primarily vitamin K antagonist) was used in 18-32%, with aspirin use in over half of the remaining patients. Prior thromboembolic events occurred in 1.4-20%, in a population with an overall CHADS2 score of 1-2.2. The definition of SCAF recorded by CIEDs ranged from >5 minutes in MOST, to 24 h in Botto et al study, or SCAF burden ≥5.5 h/day in TRENDS.

The number of thromboembolic events in these studies is relatively small, with 51 events occurring in largest ASSERT trial that had recruited 2,580 patients, and only 14 events among 725 patients in Capucci’s study. The overall annual thromboembolic rate ranges from 0.89%26 to 2.5%.20 SCAF detected by the device

### Table 2. Subclinical AF recorded by implanted pacemakers and cardioverter defibrillators on the risk of development of clinical AF and stroke (and other thromboembolic events)

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Mean</th>
<th>Prior</th>
<th>Atrial</th>
<th>Duration</th>
<th>Clinical</th>
<th>Prior</th>
<th>Prior</th>
<th>CHADS2</th>
<th>No. of pts</th>
<th>TE</th>
<th>P</th>
<th>TE</th>
<th>SCAF(+)</th>
<th>SCAF(-)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>FU age (m)</td>
<td>AF activtion</td>
<td>AF</td>
<td>AF</td>
<td>AF</td>
<td>OAC</td>
<td>OAC</td>
<td>OAC</td>
<td>OAC</td>
<td>with TE</td>
<td>(RR)</td>
<td>(RR)</td>
<td>%/yr</td>
<td>TE%</td>
</tr>
<tr>
<td>MOST19</td>
<td>312</td>
<td>74</td>
<td>27</td>
<td>60%</td>
<td>AHRE &gt;5 min</td>
<td>5.9</td>
<td>0.0001</td>
<td>20%</td>
<td>24%</td>
<td>-</td>
<td>10</td>
<td>2.8</td>
<td>0</td>
<td>1.60%</td>
<td>2.20%</td>
</tr>
<tr>
<td>Capucci21</td>
<td>725</td>
<td>71</td>
<td>22</td>
<td>100%</td>
<td>AT/AF &gt;1 day</td>
<td>-</td>
<td>-</td>
<td>1.80%</td>
<td>32%</td>
<td>1.8</td>
<td>14</td>
<td>3.1</td>
<td>0.04</td>
<td>1.20%</td>
<td>-</td>
</tr>
<tr>
<td>Botto12</td>
<td>568</td>
<td>70</td>
<td>12</td>
<td>100%</td>
<td>AT/AF &gt;1 day</td>
<td>-</td>
<td>-</td>
<td>1.40%</td>
<td>25.20%</td>
<td>~1.0</td>
<td>14</td>
<td>5.3</td>
<td>-</td>
<td>2.50%</td>
<td>3.60%</td>
</tr>
<tr>
<td>TRENDS25</td>
<td>2486</td>
<td>71</td>
<td>17</td>
<td>20%</td>
<td>AT/AF ≥5.5 h/day</td>
<td>-</td>
<td>-</td>
<td>13.40%</td>
<td>20.80%</td>
<td>2.2</td>
<td>40</td>
<td>2.2</td>
<td>0.06</td>
<td>1.20%</td>
<td>2.40%</td>
</tr>
<tr>
<td>ASSERT26</td>
<td>2580</td>
<td>76</td>
<td>34</td>
<td>0%</td>
<td>AT ≥6 min</td>
<td>5.6</td>
<td>&lt;0.001</td>
<td>12.10%</td>
<td>18%</td>
<td>~1.2</td>
<td>51</td>
<td>2.5</td>
<td>0.01</td>
<td>0.89%</td>
<td>1.78%</td>
</tr>
</tbody>
</table>

**FU=follow up, OAC=oral anticoagulantion, RR=relative risk, pts=patients, SCAF=Subclinical atrial fibrillation, TE=Thromboembolism**
increased the relative risk for thromboembolism by a factor of 2.2-5.3 compared to no SCAF detected. Annual thromboembolic rates were similarly higher in those patients with detected SCAF versus those without.

The event rates for the ASSERT which included only patients without prior AF are tabulated in Table 3. SCAF detected at 3 months increased the relative risk of thromboembolic events and clinical AF to 2.81 and 5.0 respectively. Similar to other studies, no difference in total and cardiovascular mortality has been reported so far.

**SCAF Duration and CHADS2 Scores in Relation to Thromboembolism**

In a retrospective analysis, Botto el al had attempted to stratify thromboembolic risk in a cohort of 567 patients with a total number of 14 events. There was a relationship between duration of SCAF and CHADS2 score with stroke event. At a CHADS2 score of 1, only AF >24 h would increase the annual stroke rate to 4% compared to 0.6% for SCAF that lasted shorter. At a CHADS2 score of 2, any SCAF >5 minutes resulted in a 4% stroke rate. In the TRENDS study, only SCAF burden >5.5 h/day increased thromboembolic rate to 2.4%/year, in a population of CHADS2 score of 2.2. Likewise, the thromboembolic risk in ASSERT became significant only when SCAF was >17.72 h.

In a pooled analysis of over 10,000 patients, Boriani et al compared the duration of CIED recorded SCAF and stroke risk (Figure 2). The hazard ratio of SCAF >5 minutes was similar to the impact of having sustained AF, and progressively increased the SCAF duration, and the risk appeared to plateau off when SCAF reached 24 hours.

Despite these findings, the annual risk for thromboembolism in these studies are lower than expected when compared to patients with clinical AF with similar CHADS2 scores. Indeed, in the ASSERT study involving patients without prior AF, the annual rates of thromboembolic events with SCAF detected were lower than expected from published risk of CHADS2 score (Table 4). The reason for the lower stroke risk for SCAF compared to clinical AF is uncertain. Possibilities include: patients with CIEDs are different from clinical AF patients, that atrial leads might have generated a different type of AF, or SCAF may represent less severe or early AF that require time to become an establish risk. Finally, a substantial percent of patients in these studies were on anti-thrombotic therapy which would have reduced embolic risk.

**Is Subclinical AF Only a Risk Marker for Stroke?**

The traditional belief is that AF results in cardioembolic events from atrial clots due to mechanical stasis in the atrium. Indeed, in trans-esophageal

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**Table 3. Clinical outcome of patients with CHADS2 ≥2 who were recruited in the ASSERT trial**

<table>
<thead>
<tr>
<th>Event</th>
<th>Asymptomatic AF detected by device</th>
<th>SCAF present vs. absent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Present N=187</td>
<td>Absent N=1790</td>
</tr>
<tr>
<td>Ischaemic stroke or systemic embolism</td>
<td>Events %/year</td>
<td>Events %/year</td>
</tr>
<tr>
<td></td>
<td>10 2.19</td>
<td>35 0.79</td>
</tr>
<tr>
<td>Vascular death</td>
<td>16 3.51</td>
<td>137 3.10</td>
</tr>
<tr>
<td>Stroke / MI / vascular death</td>
<td>25 5.48</td>
<td>185 4.18</td>
</tr>
<tr>
<td>Clinical atrial fibrillation or flutter</td>
<td>29 6.36</td>
<td>61 1.38</td>
</tr>
</tbody>
</table>

MI = Myocardial infarction; SCAF = Subclinical AF detected (Rate >190/min and >6 mins)


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Table 4. Annual stroke (and other thromboembolic) risk (in %) at different CHADS2 scores compared to the reported risk in patients with clinical AF. While subclinical AF (SCAF) increased risk of events, the risk remained substantially lower than the occurrence of clinical AF

<table>
<thead>
<tr>
<th>CHADS2</th>
<th>&lt;2</th>
<th>2</th>
<th>&gt;2</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCAF detected</td>
<td>0.56%</td>
<td>1.29%</td>
<td>3.78%</td>
<td>P=0.35</td>
</tr>
<tr>
<td>SCAF not detected</td>
<td>0.28%</td>
<td>0.70%</td>
<td>0.97%</td>
<td>P=NS</td>
</tr>
<tr>
<td>Clinical AF in reference population</td>
<td>2.8%</td>
<td>4.0%</td>
<td>&gt;5.8%</td>
<td>–</td>
</tr>
</tbody>
</table>

echocardiographic studies, substantial risk for left atrial thrombosis occurred when AF lasted > 48 hours. This led to the guideline recommendation of prior oral anticoagulation for 4 weeks before cardioversion for AF > 48 hours. Recent observations suggest paroxysmal AF increased stroke risks as sustained AF. Indeed, even transient episodes of AF or high atrial rates induced by atrial pacing can lead to increased platelet activation and thrombin generation.

With an implanted CIED, it is possible to relate the temporal occurrence of SCAF and stroke and other thromboembolic events. Daoud et al analysed the device recordings of the 40 patients who developed an event in the TRENDS study. They documented that only half of these patients had a SCAF before the event. Of these, only about half had SCAF 30 days before the thromboembolism to suggest a causative mechanism. Overall, 29/40 (72.5%) of patients had no close temporal proximity of SCAF to stroke and may be considered to have stroke due to non-cardioembolic causes. The only factors that predicted SCAF to occur before a thromboembolic event are patients with a long duration of entry into the study (485±273 vs 251±221 days, p<0.01) and a higher mean and maximum AF burden.

In a similar analysis, the ASSERT investigators showed that only 51% (26/51) patients with stroke (or other thromboembolism) had SCAF occurring before, and only 8% of the overall cohort had SCAF within 1 month of the event (Figure 3). This suggests that in patients without prior AF, as many as 92% of the stroke may be due to non-cardiac emboli not related to SCAF.

Taken together, SCAF detected by CIEDs predicted the occurrence of clinical AF and increased the risk of stroke. However, especially in studies in which most patients did not have AF at the baseline, the annual risk for stroke (and other thromboembolism) when SCAF was detected is lower than expected from clinical AF with equal risk factors. A temporal relation...
between SCAF occurrence and stroke was plausible only in a minority of patients. These suggest non-cardioembolic causes may be more important in patients with only SCAF detected without clinical AF.

**When Should SCAF Be Anticoagulated?**

The IMPACT trial\(^34\) is a prospective randomized trial that randomized over 2000 patients with ICD or CRTD to receive vitamin K antagonists or not based on the CHADS\(_2\) score, and the presence or absence of SCAF as detected by CIED and monitored by remote monitoring. The moderate risk group (CHADS\(_2\) ≤ 4) was randomized to receive warfarin in the presence of SCAF or to terminate warfarin when SCAF became undetected. The primary end point was a composite of stroke, embolism or major bleed. Early results were presented and suggested anticoagulation guided by SCAF detected by CIED to be equal to routine clinical care. The reasons for the neutral result are not certain, and the full report is awaited.

After validation of device recorded SCAF to be accurate AF registration, it seems reasonable now to consider their thromboembolic risk in the decision for anticoagulation. In patients with prior clinical AF, they should be anticoagulated according to their CHADS\(_2\) or CHA\(_2\)DS\(_2\)-VASc scores as suggested by current guideline.\(^35\) There is no guideline for patients with only SCAF detected (Table 5). For CHADS\(_2\) = 0, SCAF requires no anticoagulant, whereas most clinicians would start oral anticoagulants for CHADS\(_2\) ≥ 3. For CHADS\(_2\) = 1, based on the ASSERT trial, the risk of stroke probably is outweighed by the risk of warfarin. At CHADS\(_2\) = 2, warfarin is likely indicated. Longer episodes of SCAF (especially close to 24 h) increase stroke risk. When NOACs are considered, it seems reasonable to initiate anticoagulation for a lower CHADS\(_2\) score or shorter AF duration. At present there is no objective cohort data or randomized data to confirm this recommendation.

**Conclusion**

In the presence of a cryptogenic stroke, SCAF of up to 30% in 3 years can be recorded by an implanted ICM. While there is no randomized data, most would consider the use of oral anticoagulation instead of aspirin therapy in secondary prevention for recurrent stroke. More controversy centered about SCAF recorded by implanted CIEDs. Recorded SCAFs predicted clinical AF. However, recorded SCAF, while increasing stroke (and other thromboembolic risk) occurred at a magnitude that is substantially less than what occurred when AF developed clinically. In addition, a temporal relation between SCAF and stroke occurred only in a minority of patients. Until more data become available, the use of oral anticoagulation in this cohort remains expert opinion, although CHADS\(_2\) score and duration of AF may help to identify a group of patients who may be such candidates.

### Table 5. Anticoagulation consideration when only subclinical AF (SCAF) is recorded by an implantable cardiac electronic device

<table>
<thead>
<tr>
<th>CHADS(_2)</th>
<th>Anticoagulation Consideration</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Warfarin not needed</td>
</tr>
<tr>
<td>1</td>
<td>Warfarin may not be needed (or start warfarin if AF ~ 24h)</td>
</tr>
<tr>
<td>2</td>
<td>Consider warfarin if AF ~ 24h (possibly shorter if NOACs are used)</td>
</tr>
<tr>
<td>≥3</td>
<td>Warfarin is indicated</td>
</tr>
<tr>
<td>Confirmed it is AF A trial electrogram validation; duration and rate programmed</td>
<td></td>
</tr>
</tbody>
</table>
References

fibrillation is associated with local cardiac platelet activation and endothelial dysfunction. J Am Coll Cardiol 2008;51:1790-3.


