Ενδιαφέροντα θέματα για ειδικούς και μη ειδικούς
Αντιπηκτική αγωγή σε κολπική ταχυκαρδία

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Θεσσαλονίκη
• **Cardioembolic stroke** accounts for approximately 15%-20% of all ischemic strokes

• patients with cardioembolic stroke exhibited the **worst prognosis**, and more than half of them died within 1.5 years

• clinically **apparent atrial fibrillation** or flutter (AF)—currently the only arrhythmias that are established stroke risk factors

• One third of ischemic strokes remain **cryptogenic**

• patients with cardioembolic-appearing cryptogenic stroke manifest nonspecific **supraventricular arrhythmias** more often than patients with other types of stroke
Atrial Tachycardias

- Adults with congenital heart disease
- Atrial flutter
- AT in general population
- Elevated supraventricular activity (ESVEA)
Atrial Tachycardia in Adults with congenital heart disease
<table>
<thead>
<tr>
<th>Complexity of CHD</th>
<th>Type of CHD</th>
<th>Prevalence (in CHD population)</th>
<th>Atrial Arrhythmia</th>
<th>Ventricular Arrhythmia</th>
<th>Other Pacing Needs</th>
</tr>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>AT</td>
<td>AF</td>
<td>Other</td>
</tr>
<tr>
<td>Simple</td>
<td>Patent ductus arteriosus</td>
<td>6-8%</td>
<td></td>
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<tr>
<td></td>
<td>Pulmonary stenosis</td>
<td>6-8%</td>
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<td></td>
<td>Ventricular septal defect</td>
<td>30-32%</td>
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<td></td>
<td>Secundum atrial septal defect</td>
<td>8-10%</td>
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<tr>
<td>Moderate</td>
<td>Aortic coarctation</td>
<td>5-7%</td>
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<tr>
<td></td>
<td>Anomalous pulmonary venous return</td>
<td>0.5-2.5%</td>
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<td></td>
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<tr>
<td></td>
<td>Atrioventricular septal defect</td>
<td>3-5%</td>
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<tr>
<td></td>
<td>Aortic stenosis</td>
<td>3-5%</td>
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<td></td>
<td>Ebstein anomaly</td>
<td>0.5-1.5%</td>
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<tr>
<td></td>
<td>Tetralogy of Fallot</td>
<td>8-10%</td>
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<tr>
<td></td>
<td>Primum atrial septal defect</td>
<td>2-5%</td>
<td></td>
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<tr>
<td>Severe</td>
<td>Truncus arteriosus</td>
<td>1.5-2%</td>
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<tr>
<td></td>
<td>Pulmonary atresia</td>
<td>2.2-5%</td>
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<tr>
<td></td>
<td>Double outlet right ventricle</td>
<td>1.5-2%</td>
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<td></td>
<td>D-transposition of the great arteries</td>
<td>6-7%</td>
<td></td>
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<tr>
<td></td>
<td>L-transposition of the great arteries</td>
<td>1.2%</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Hypoplastic left heart syndrome</td>
<td>3-4%</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Other (heterotaxy, other single ventricles)</td>
<td>7-10%</td>
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</tbody>
</table>
• **Intra-atrial reentrant tachycardia** and atrial fibrillation are the most prevalent type of arrhythmia in this patient group.

• atrial tachyarrhythmia is usually related to the **abnormal anatomy** of the underlying heart defect and often occurs as a result of surgical scar or a consequence of residual hemodynamic or electrical disturbances.
• The overall prevalence of thromboembolic complications in patients with CHD has been estimated to be 10-to100-fold higher than in age-matched controls.

• TOE prior to cardioversion of an atrial tachyarrhythmia, atrial thrombus was detected in 37%.

• The varied pathophysiology reflects diverse predisposing substrates and includes
  – dilated cardiac chambers with
  – sluggish flow,
  – intracardiac prosthetic material, pacemaker/defibrillator leads,
  – intracardiac shunts, and
  – associated hypercoagulable states
<table>
<thead>
<tr>
<th>Class</th>
<th>Recommendation</th>
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</table>
| Class I| 1. For adults with simple forms of CHD and hemodynamically stable IART or atrial fibrillation of unknown or ≥ 48-hours’ duration, therapeutic anticoagulation is recommended for at least 3 weeks prior to cardioversion, or, alternatively, a transesophageal echocardiogram may be performed to rule out intracardiac thrombus *(Level of evidence: B)*.  
2. Adults with complex CHD and sustained or recurrent IART or atrial fibrillation should receive long-term oral anticoagulation for the prevention of thromboembolic complications *(Level of evidence: B)*. |
| Class IIa| 1. For adults with moderate or complex CHD and hemodynamically stable IART or atrial fibrillation, it is reasonable to pursue therapeutic anticoagulation for at least 3 weeks prior to cardioversion or perform transesophageal echocardiography to rule out thrombus, regardless of arrhythmia duration *(Level of evidence: B)*.  
2. Long-term oral anticoagulation therapy is reasonable in adults with CHD of moderate complexity and sustained or recurrent IART or atrial fibrillation *(Level of evidence: C)*.  
3. Vitamin K antagonists can reasonably be considered the oral anticoagulant agent of choice in adults with moderate or complex CHD, pending safety and efficacy data on newer oral anticoagulants (NOACs; i.e., direct thrombin inhibitors and direct factor Xa inhibitors) *(Level of evidence: B)*. |
| Class IIb| 1. It may be reasonable for adults with IART or atrial fibrillation and simple nonvalvular forms of CHD to receive either an oral anticoagulant, aspirin, or no therapy for the prevention of thromboembolic complications on the basis of established scores for stroke risk (e.g., CHA2DS2-VASc) and bleeding risk (e.g., HAS-BLED) *(Level of evidence: B)*.  
2. In adults with simple forms of CHD and no prosthetic heart valve or hemodynamically significant valve disease, a NOAC may be a reasonable alternative to a vitamin K antagonist when anticoagulation is indicated *(Level of evidence: C)*. |
| Class III| 1. Pending future studies, there are currently insufficient pharmacokinetic/pharmacodynamic, safety, and efficacy data to endorse use of NOACs in adults with Fontan surgery *(Level of evidence: C)*.  
2. Anticoagulation is not indicated for the prevention of thromboembolic complications in adults with CHD and AV nodal reentrant tachycardia or accessory pathway-mediated tachycardia *(Level of evidence: C)*. |

**PACES/HRS Expert Consensus Statement on the Recognition and Management of Arrhythmias in Adult Congenital Heart Disease**

*Developed in partnership between the Pediatric and Congenital Electrophysiology Society (PACES) and the Heart Rhythm Society (HRS). Endorsed by the governing bodies of PACES, HRS, the American College of Cardiology (ACC), the American Heart Association (AHA), the European Heart Rhythm Association (EHRA), the Canadian Heart Rhythm Society (CHRS), and the International Society for Adult Congenital Heart Disease (ISACHD)*
Atrial Flutter
ATRIAL FLUTTER

- Echocardiographic studies reported prevalence of **thrombus** material from 0% to 38% and a prevalence of spontaneous echo contrast (SEC) from 21% to 28%

- **During cardioversion, thromboembolic event** rates varied from 0% to 6% with a follow-up from 1 week to 6 years

- One ablation study in non-anticoagulated patients reported thromboembolic events at 13.9%

- Observational studies reported an **overall elevated stroke risk** (risk ratio 1.4, 95% CI 1.35 to 1.46) and mortality risk (HR 1.9, 95% CI 1.2 to 3.1) with long time follow-up compared with a control group in both studies
  - Henrik Vadmann et al. Heart 2015
Atrial flutter: Clinical risk factors and adverse outcomes in the Framingham Heart Study

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Atrial flutter vs referents*</th>
<th>Atrial flutter vs atrial fibrillation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HR (95% CI)</td>
<td>P value</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>5.0 (3.1–8.0)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>3.1 (1.4–6.6)</td>
<td>.004</td>
</tr>
<tr>
<td>Heart failure</td>
<td>4.1 (1.9–9.0)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Stroke</td>
<td>2.2 (1.1–4.2)</td>
<td>.02</td>
</tr>
<tr>
<td>Death†</td>
<td>2.0 (1.4–2.8)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

CI = confidence interval; HR = hazard ratio.

*Referents defined as participants without atrial fibrillation or atrial flutter.
†Also adjusted for the presence or absence of prevalent cardiovascular disease.
Kaplan-Meier curves for ischemic stroke, heart failure hospitalization, and all-cause mortality in AF and AFL cohorts after 1:4 propensity score matching.
### 2016 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS

The Task Force for the management of atrial fibrillation of the European Society of Cardiology (ESC)

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Level&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Ref&lt;sup&gt;c&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>For patients with atrial flutter, antithrombotic therapy is recommended according to the same risk profile used for AF.</td>
<td>I</td>
<td>B</td>
<td>827</td>
</tr>
</tbody>
</table>
Atrial tachycardia in general population
Cumulative rates of ischemic stroke are shown according to whether or not patients had a preexisting diagnosis of paroxysmal supraventricular tachycardia (PSVT).

After excluding patients with AF, the cumulative stroke rate after a diagnosis of PSVT (0.94%; 95% CI, 0.76%–1.16%) significantly exceeded the rate among patients without PSVT (0.21%; 95% CI, 0.21%–0.22%; P<0.001, log-rank test;)

PSVT was independently associated with subsequent stroke (hazard ratio [HR], 2.10; 95% CI, 1.69–2.62).

**Supplemental Table 4.** Association Between PSVT and Ischemic Stroke Across Decades of Age.

<table>
<thead>
<tr>
<th>Age</th>
<th>Hazard Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>45-54 y</td>
<td>0.39 (0.05-2.80)</td>
</tr>
<tr>
<td>55-64 y</td>
<td>0.69 (0.26-1.85)</td>
</tr>
<tr>
<td>65-74 y</td>
<td>2.17 (1.40-3.35)</td>
</tr>
<tr>
<td>75-84 y</td>
<td>2.59 (1.81-3.71)</td>
</tr>
<tr>
<td>≥85 y</td>
<td>2.66 (1.76-4.03)</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; PSVT, paroxysmal supraventricular tachycardia.

**Univariate logistic analyses of the potential risk factors for ischemic stroke among Taiwanese adults from 1997 to 2011**

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>OR</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (per 5 years)</td>
<td>1.70</td>
<td>1.60-1.73</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Gender (male vs. female)</td>
<td>1.54</td>
<td>1.46-1.63</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Hypertension</td>
<td>21.46</td>
<td>19.90-23.22</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Diabetes</td>
<td>7.50</td>
<td>7.11-8.06</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>HBV infection</td>
<td>.72</td>
<td>0.63-0.80</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>HCV infection</td>
<td>1.77</td>
<td>1.52-2.07</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>PSVT</td>
<td>2.00</td>
<td>1.38-2.85</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Aspirin</td>
<td>.10</td>
<td>.08-.13</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

**Multiple logistic analyses of risk factors for ischemic stroke among Taiwanese adults from 1997 to 2011**

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>OR</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (per 5 years)</td>
<td>1.76</td>
<td>1.73-1.78</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Male vs. female</td>
<td>1.88</td>
<td>1.74-2.01</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>CKD</td>
<td>3.09</td>
<td>2.67-3.57</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>PSVT</td>
<td>2.05</td>
<td>1.30-3.19</td>
<td>.002</td>
</tr>
<tr>
<td>Aspirin</td>
<td>.04</td>
<td>.03-0.05</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>
Risk of Stroke in Patients With Short-Run Atrial Tachyarrhythmia

Shinya Yamada, Chin-Yu Lin, Shih-Lin Chang, Tze-Fan Chao, Yenn-Jiang Lin, Li-Wei Lo, Fa-Po Chung, Yu-Feng Hu, Ta-Chuan Tuan, Jo-Nan Liao, Abigail Louise D. Te, Yao-Ting Chang, Ting-Yung Chang, Cheng-I Wu, Satoshi Higa, Shih-Ann Chen

• retrospective, observational study

• database of Registry of 24-hour ECG monitoring at Taipei Veterans General Hospital

• short-run AT was defined as ≥3 consecutive supraventricular ectopic beats with mean P–P interval ≤600 ms and lasting <5 seconds

• 5342 patients for analysis

• There were 1595 subjects (29.8%) with short-run AT

• median FU period of the patients with and without short-run AT were 9.0 and 9.1 years
Kaplan–Meier analysis for clinical events in patients with or without short-run atrial tachyarrhythmia (AT).

Patients with short-run AT had significantly higher stroke rates compared with patients without short-run AT (11.4% versus 8.3%; P<0.001)
In patients with AF, the proposed anticoagulant treatment thresholds for balancing ischemic stroke reduction against serious bleeding were **1.7% per year** for warfarin therapy and **0.9% per year** for the use of nonvitamin K antagonist oral anticoagulants.
Kaplan–Meier analysis for new-onset stroke in short-run atrial tachyarrhythmia.
supraventricular ectopic activity (SVEA) is defined as

- SVEC per hour or
- any episode of runs of SVEC.
Excessive Atrial Ectopy and Short Atrial Runs Increase the Risk of Stroke Beyond Incident Atrial Fibrillation

The Copenhagen Holter Study
Zeynep Binici et al. Circulation. 2010

Larsen BS et al. JACC 2015
Excessive Atrial Ectopy and Short Atrial Runs Increase the Risk of Stroke Beyond Incident Atrial Fibrillation

The absolute risk of stroke in subjects with ESVEA and a CHA2DS2-VASc score ≥2 was 2.4% per year which is equivalent to patients with AF and a CHA2DS2-VASc score of 2. This is classified as a high risk, and it normally warrants anticoagulation in patients with AF.
pathophysiological mechanism by which excessive AE may contribute to increase the risk of ischemic stroke

- precedes undiagnosed incident AF
- a marker of more severe hypertension, diabetes, physical inactivity, or lipid metabolism abnormality, thus signifying an increased vascular risk profile
- interactions between coexisting risk factors such as hypertension and smoking, in addition to increased number and runs of PACs, may lead to dilation of the left atrium, stasis in the left atrial appendage, fibrosis, and endothelial dysfunction, eventually resulting in a hypercoagulable state comparable to that present in AF
Device-detected subclinical atrial tachyarrhythmias: definition, implications and management—an European Heart Rhythm Association (EHRA) consensus document, endorsed by Heart Rhythm Society (HRS), Asia Pacific Heart Rhythm Society (APHRS) and Sociedad Latinoamericana de Estimulación Cardíaca y Electrofisiología (SOLEACE)

ESVEA documented by Holter monitoring can be considered as a surrogate marker for paroxysmal AF.

General agreement and/or scientific evidence favour the usefulness/efficacy of a treatment or procedure. May be supported by randomized trials that are, however, based on small number of patients to allow a green heart recommendation.
conclusions

- AT in pts with CHD $\rightarrow$ anticoagulation (vit K antagonists)
- AFLutter $\rightarrow$ anticoagulation (as in AF)
- AT and ESVEA in general population $\rightarrow$ increased risk of thromboembolic complications (stroke), anticoagulation?
1) 3 of every 4 patients with AFL also manifest clinical AF

2) After successful ablation of the CTI to cure AFL, up to 70% of patients subsequently manifest AF, many of whom never manifested AF before the AFL ablation

3) Antiarrhythmic drugs with sodium channel-blocking properties (i.e., properties that affect conduction in the atria, such as the class IAs, class ICs, and amiodarone) are associated with a significant incidence of “converting” recurrent AF to AFL
• Accumulating data suggest that some strokes arise from forms of atrial electric dysfunction
• PSVT affects mostly older patients with a high burden of cardiovascular disease
• paroxysmal supraventricular tachycardia (PSVT) is more closely linked to cardiovascular disease than traditionally appreciated
• PSVT may also be a manifestation of acquired atrial myocardial disease in the setting of cardiovascular risk factors
• even before AF develops, atrial disease may manifest as other supraventricular arrhythmias, such as PSVT, and increase stroke risk through some combination of hypercoagulability, inflammation, endothelial injury, and structural changes
Association of Paroxysmal Supraventricular Tachycardia with Ischemic Stroke: A National Case-Control Study

Jui-Kun Chiang, MD, MSc, Hsueh-Hsin Kao, MD, Yee-Hsin Kao, MD

Journal of Stroke and Cerebrovascular Diseases
Volume 26, Issue 7, Pages 1493-1499 (July 2017)
DOI: 10.1016/j.jstrokecerebrovasdis.2017.03.005
Update: Arrhythmias (IV)

Clinical Approach to Atrial Tachycardia and Atrial Flutter From an Understanding of the Mechanisms. Electrophysiology Based on Anatomy

Francisco García-Cosío, Agustín Pastor Fuentes, and Ambrosio Núñez Angulo

Servicio de Cardiología, Hospital Universitario de Getafe, Getafe, Madrid, Spain
Given an ECG pattern of **atypical AF**, the possibilities are many, including FAT or MLAT, especially when there has been prior cardiac surgery or ablation of the atrial myocardium for the treatment of atrial fibrillation.

In patients with complex atrial lesions, not even the pattern of **typical AF** can predict the mechanism of tachycardia; FAT and MAT mechanisms may also be present.
Atrial Tachycardia Remodeling of Pulmonary Vein Cardiomyocytes
Comparison With Left Atrium and Potential Relation to Arrhythmogenesis

Tae-Joon Cha, MD; Joachim R. Ehrlich, MD; Liming Zhang, MSc; Denis Chartier, MSc; Tack Ki Leung, MD; Stanley Nattel, MD

**Background**—The pulmonary veins (PVs) are important in the pathophysiology of atrial fibrillation (AF), as is atrial tachycardia (AT) remodeling. The relative importance of AT remodeling in PVs versus other atrial sites is unknown. The present study assessed AT-induced cellular changes in PVs versus left atrium (LA) and their relationship to arrhythmogenesis.

**Methods and Results**—We studied ionic currents (single-cell patch clamp) and action potentials (APs; coronary-perfused multicellular preparations) in the PVs and LA free wall of dogs after 7-day AT pacing (400 bpm), as well as in nonpaced control dogs. In controls, rapid \( I_{Kr} \) and slow \( I_{Ks} \) delayed-rectifier currents were larger in PVs; transient-outward \( I_{in} \), inward-rectifier \( I_{K1} \), and L-type Ca\(^{2+} \) \( I_{Ca} \) currents and AP duration were smaller. AT remodeling reduced \( I_{Ca} \) and \( I_{in} \), left \( I_{Kr} \), and \( I_{Ks} \) unaltered, and increased \( I_{K1} \) in both LA and PV. AT reduced action potential duration in both LA and PV. LA–PV AP differences became smaller in AT than in control dogs. Premature extrastimuli induced atrial tachyarrhythmias at 4.5±2.8% (mean±SEM) sites in 6 control multicellular preparations compared with 64.2±7.3% sites in 9 AT-remodeled preparations (\( P<0.001 \)). Resection of all PVs failed to alter atrial tachyarrhythmia inducibility in AT-remodeled preparations (67.5±13.1%). PV resection did not significantly change tachyarrhythmia duration (mean 3.9 seconds per heart, range 0.7 to 15.7 seconds before resection; mean 7.0 seconds per heart, range 0.9 to 36.0 seconds after resection) or cycle length (120±6 ms before resection, 115±8 ms after resection).

**Conclusions**—AT produces qualitatively similar ionic remodeling in LA and PVs but reduces PV–LA AP differences. PVs are not essential for AT-induced atrial tachyarrhythmia promotion in this model, which may relate to the failure of PV isolation to prevent AF in some patient populations. *(Circulation. 2005;111:728-735.)*

**Key Words:** ion channels ■ atrium ■ fibrillation
For each increase of 10 SVEC per hour, the risk of the primary end point of death or stroke increased by 27% and the risk of atrial fibrillation by 50%.
Recent studies showed that the CHA2DS2-VASc score is useful for predicting ischemic stroke in patients with coronary artery disease without AF and cardiovascular events in patients with or without AF.5,6 Therefore, predictivity of the CHA2DS2-VASc score has been extended beyond the originally proposed AF field.
Incidence of atrial fibrillation hospitalizations per 1000 patient-years in relation to the rate of supraventricular ectopic activity and length of runs of SVEC.

For each increase of 10 SVEC per hour:

- the risk of the primary end point of death or stroke increased by 27% and
- the risk of atrial fibrillation by 50%.
Excessive Atrial Ectopy and Short Atrial Runs Increase the Risk of Stroke Beyond Incident Atrial Fibrillation

Larsen BS et al. JACC 2015

A nonlinear association between number and runs of premature atrial contractions (PACs) with stroke was observed in these patients.
ESVEA is associated with an increased risk of ischemic stroke. The study also shows a strong correlation between ESVEA and ischemic stroke beyond AF.
The Copenhagen Holter Study

- launched in 1998

- Holter recording for up to 48 hours

- ESVEA was defined as 678 healthy men and women aged between 55 and 75 years with no history of cardiovascular disease, atrial fibrillation, or stroke

- 30 supraventricular ectopic complexes (SVEC) per hour or as any episodes with runs of 20 SVEC

- Median follow-up was 6.3 years
issues still not addressed

- **What could we do when subjects with frequent PACs are identified**, even though we know they carry a higher risk of long-term unfavorable prognosis?

- Would **treatment of PACs per se**, such as radiofrequency ablation and/or anticoagulation therapy in the absence of apparent AF, add benefit for hard endpoints?

- Which **CV risk factors** have interactions with PACs; in other words, which types of patients with frequent PACs are most prone to poor outcomes?

- Prospective studies are needed to clarify the **risk factors, gene susceptibility, natural progression or disease trajectory, and clinical approach of management of frequent PACs, especially in apparently healthy subjects.**
Paroxysmal Supraventricular Tachycardia and the Risk of Ischemic Stroke

- One third of ischemic strokes remain cryptogenic because diagnostic evaluation fails to reveal an exact cause. This uncertainty prevents optimal treatment of risk factors and impedes efforts to prevent stroke. Accumulating data suggest that some strokes arise from forms of atrial electric dysfunction other than clinically apparent atrial fibrillation or flutter (AF)—currently the only arrhythmias that are established stroke risk factors.

- Before AF develops, atrial disease may manifest as other supraventricular arrhythmias, such as PSVT, and increase stroke risk through some combination of hypercoagulability, inflammation, endothelial injury, and structural changes.

- PSVT may be a heterogeneous disease with different underlying mechanisms. This assertion is supported by our finding that 91% of males, but only 50% of females, with PSVT have associated CVD.
atrial fibrillation will develop in 12% of patients with paroxysmal supraventricular tachycardia during a 1-year follow-up period.
Macro-Re-Entrant Atrial Tachycardia/Atrial Flutter
- Constant regular P wave/Flutter wave morphology
- Rate typically >250 bpm*
- Mechanism: Macro-re-entry

Focal Atrial Tachycardia
- Discrete P waves with isoelectric segment
- Rate typically 100–250 bpm*
- Mechanisms: Micro-re-entry or automaticity

Cavotricuspid Isthmus Dependent
- Right atrial re-entry dependent on conduction through the cavotricuspid isthmus
- Can be cured by ablation creating conduction block in the cavotricuspid isthmus

Typical Atrial Flutter

Not Cavotricuspid Isthmus Dependent ("Atypical Atrial Flutter")
- Re-entry that is not dependent on conduction through the cavotricuspid isthmus
- The circuit is usually defined by atrial scars from prior heart surgery, ablation, or idiopathic
- Location determines ablation approach and risks
- Multiple re-entry circuits can be present

Right Atrial
Example: Re-entry around healed surgical incision in the free wall of the right atrial after repair of congenital heart disease
- Perimtrial flutter
- Left atrial root dependent flutter
- Others

Left Atrial

ECG Atypical flutter suggested by P-wave polarity that does not fit typical atrial flutter (e.g., concurrent P-wave polarity between V1 and inferior leads)

V1 typically opposite in polarity to inferior leads

ECG flutter waves:
- Negative in II, III, aVF
- Positive in II, III, aVF
- Negative in V1
Class IIb  

1. It may be reasonable for adults with IART or atrial fibrillation and simple nonvalvular forms of CHD to receive either an oral anticoagulant, aspirin, or no therapy for the prevention of thromboembolic complications on the basis of established scores for stroke risk (e.g., CHA\textsubscript{2}DS\textsubscript{2}-VASc) and bleeding risk (e.g., HAS-BLED) (Level of evidence: B).\textsuperscript{177,197}

2. In adults with simple forms of CHD and no prosthetic heart valve or hemodynamically significant valve disease, a NOAC may be a reasonable alternative to a vitamin K antagonist when anticoagulation is indicated (Level of evidence: C).\textsuperscript{199–201,203}

Class III  

1. Pending future studies, there are currently insufficient pharmacokinetic/pharmacodynamic, safety, and efficacy data to endorse use of NOACs in adults with Fontan surgery (Level of evidence: C).

2. Anticoagulation is not indicated for the prevention of thromboembolic complications in adults with CHD and AV nodal reentrant tachycardia or accessory pathway-mediated tachycardia (Level of evidence: C).
• In moderate/complex patients with IART or AF, long-term oral anticoagulation is recommended with vitamin K antagonist (VKA) however, in simple nonvalvular CHD, the decision of anticoagulation risk can be guided by CHA2DS2-VASc score\[17,98\]. Patients with CHA2DS2-VASc score of ≥ 2 need oral anticoagulation with VKA or novel oral anticoagulants.

• Limited data is available regarding the use of NOACs in ACHD population.

• Results from further multi-centre studies are anticipated. However, it may be reasonable to consider NOAC in lieu of VKA in simple CHD and without prosthetic valves or hemodynamically significant valve disease.
Long-term oral anticoagulation is recommended in the adult with CHD of severe complexity and IART or atrial fibrillation, and appears reasonable in those with moderate forms of CHD. It is unlikely that the thromboembolic risk associated with simple non valvular forms of CHD is sufficiently high to justify long term anticoagulation as a de facto approach, such that the decision to pursue anti-platelet or anticoagulation therapy in this subgroup of patients may be guided by established risk scores for stroke (e.g., CHA2DS2-VASc) and bleeding risk (e.g., HAS-BLED).
Stroke was the second leading cause of death and seventh leading cause of disability in the year 2000 worldwide,1 and stroke prevalence increased from 16.4% in 1986 to 19.1% per 1000 persons in 2001 in Taiwan.2 Approximately 11,736 persons died of stroke (mortality rate 50.1 per 100,000 persons), making stroke the third leading cause of death in 2014 in Taiwan.3 Approximately 87% of patients experience ischemic strokes, and the most common causes are transient ischemic attack, thrombotic stroke, and embolic stroke.4 Mortality was the lowest in lacunar stroke, intermediate in atherothrombotic stroke, and highest in cardioembolic stroke.
Furthermore, patients with cardioembolic stroke exhibited the worst prognosis, and more than half of them died within 1.5 years.5 Cardioembolic stroke accounts for approximately 15%-20% of all ischemic strokes.6 In addition, the common high-risk cardioembolic conditions include atrial fibrillation (Af), recent myocardial infarction (MI), mechanical prosthetic valve, dilated myopathy, and mitral rheumatic stenosis.7 One-third of ischemic stroke events remain cryptogenic8; however, paroxysmal supraventricular tachycardia (PSVT) is a novel risk factor for cryptogenic stroke
Short episodes of atrial tachyarrhythmia (AT) are a common finding on the Holter monitoring in general population which may be associated with the development of atrial remodeling and AF inducibility. However, the relationship between short episodes of AT and the incidence of stroke is still controversial. In addition, it remains unclear whether short episodes of AT are related with the progression of AF.