Supplementary Tables to Kirchhof et al. “Comprehensive risk reduction in patients with atrial fibrillation: Emerging diagnostic and therapeutic options. Executive summary of the report from the 3rd AFNET/EHRA consensus conference” (Thromb Haemost 2011; 106.6)

Suppl. Table 1: AFNET/EHRA classification of atrial fibrillation by etiology and suggested “type-specific” therapy.

<table>
<thead>
<tr>
<th>AF type</th>
<th>Pathophysiological Mechanism</th>
<th>Diagnostic Characteristics</th>
<th>Proposed “specific” therapy</th>
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<tbody>
<tr>
<td><strong>Inheritable</strong></td>
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<td><strong>AF A: Monogenic</strong></td>
<td>Patients with AF and inheritable cardiomyopathies (short QT, Brugada, LQTS, or hypertrophic cardiomyopathy, among others)</td>
<td>Gene-defect-related ECG-abnormalities, echo-diagnosis of inherited cardiomyopathy, family history, genetic testing</td>
<td>Therapy of underlying cardiomyopathy. Pharmacological reversal of the genetic defect (possibly, but not necessarily targeting the ion channel carrying the gene defect)</td>
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<tr>
<td><strong>Inheritable</strong></td>
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<td><strong>AF B: Polygenic</strong></td>
<td>currently under study. Manifestation as AF at young age (&lt; 65 years) with or without familial clustering</td>
<td>AF of early onset, often with some familial aggregation of AF, no evident specific underlying cardiovascular disease causing the arrhythmia</td>
<td>not yet identified.</td>
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<td><strong>Focal AF</strong></td>
<td>Localised triggers, in most cases originating from the pulmonary vein(s).</td>
<td>Pattern of frequent, but short-lasting episodes of AF with distinguishable P waves (coarse AF), atrial ectopy, and/or atrial tachycardia deteriorating in AF.</td>
<td>Isolation of the pulmonary vein(s), extended/repeated ablation procedures might be required.</td>
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<td><strong>Complex AF</strong></td>
<td>AF that is maintained by functional multiple reentrant wavelets. Complex AF is common and promoted by shortening of atrial refractoriness (e.g. tachycardia-induced atrial remodelling or enhanced parasympathetic tone) or localised conduction disturbances due to atrial fibrosis induced by structural heart disease. Complex AF is also the “final common pathway”</td>
<td>Long-lasting episodes, or persistent AF with nondistinguishable P waves (fine AF). The following therapeutic measures aim at quantification of the degree of substrate complexity: - Frequency and amplitude of P waves (primarily reflecting right atrial electrophysiological properties). - Frequency and amplitude of local wall movements recorded by tissue velocity imaging (electroechocardiography) - Incidence of complex fractionated atrial electrograms (CFAE). Noninvasive imaging: Atrial enlargement, scarring, and potentially atrial fibrosis as reflected by MRI.</td>
<td>Therapy depending on grading of the substrate complexity: Low complexity: AADs or PVI Moderate complexity: AADs and/or extended/repeated ablation procedures High complexity: Rate control (AAD and ablation ineffective). In these patients, both primary prevention of AF in patients with structural heart disease, and possibly also secondary prevention of AF by upstream therapy should be considered (unless contra-indicated).</td>
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</table>
### Postoperative AF

AF after cardiac surgery, multifactorial etiology. Acute factors: Inflammation, surgical trauma, high sympathetic tone, electrolyte changes, volume overload. Chronic predisposition: Genetic factors, atrial structural remodelling due to structural heart disease.

### Transient AF in post-operative setting

 Prevention by β-blockers, steroids, antioxidants. Treatment should consider both the transient nature of postoperative AF and the fact that postoperative AF may indicate an increased likelihood of recurrent AF in the future.

The group acknowledges that “complex AF” comprises a relatively inhomogeneous group of patients, and that further classification of this type of AF may be required to guide management better.
Suppl. Table 2: Patient groups likely to benefit (upper part) or not to benefit (lower part) from therapy with new anticoagulants, including a switch from existing therapy with OAC to one of the newer substances.

### Patients who are likely to benefit from new OACs

- Patients with poor TTR (time in therapeutic range) and INR control due to
  - innate/genetics for warfarin metabolism
  - inadequate access to monitoring, poor monitoring quality, and/or inability to self-monitor
- Patients requiring interacting concomitant medications
- Patients who have decided against anticoagulation with vitamin K antagonists despite adequate education
- Patients at low risk of gastrointestinal bleeding (dabigatran) and patients without renal dysfunction
- Patients who suffered an ischemic stroke on warfarin with adequate INR

### Patients potentially less suitable for novel OACs

- Elderly patients, especially those requiring polypharmacotherapy may be at increased risk of accumulating the newer oral anticoagulants with an associated increased risk for bleeds (see also Canadian label for dabigatran). The pharmacology suggests that patients with moderately impaired renal function (MDRD stage II-III) may be suitable for some of the Factor Xa antagonists, and MDRD II-III patients showed most benefit on therapy with dabigatran in the RELY study.
- Patients with markedly decreased renal function (MDRD IV-V)
- Patients with history of gastrointestinal bleeding
- Patients with poor TTR due to non-adherence may benefit from the regular reinforcement of therapy by monitoring needed for vitamin K antagonists therapy
- Patients at risk of progressing towards severe renal failure
- Patients with coronary artery disease with a high likelihood of requiring percutaneous revascularization until more data on combination therapy (vitamin K antagonists plus dual antiplatelet therapy) are available