Atrial Fibrillation 2017 - 2018
Quality of Life and Preventing Stroke

The 14 Clinical Challenges
<table>
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<tr>
<th>1. Presentation:</th>
<th>Simplified vs Prioritized</th>
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<tr>
<td>7. Screening</td>
<td>Screening vs No Screening</td>
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</table>
1,2). Acute & Chronic Management Of AF

1. D Kotecha et. al. . EHJ. 2016;37:2851 – **ANSD – RF - <AF Death**
2. EHJ 2016; 37:2893 - **Simple**
<table>
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<tr>
<th></th>
<th><strong>AF - CLINICAL CHALLENGES (14) – 2017 - 2018</strong></th>
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1). **Gross Mechanisms of AF**

### Focal Electrical Disease
- Risk Factor
  - Reduced LA/LA Appendage Velocities

#### Atrial Dilatation/Myopathy = Arrhythmia Burden
- Hypercoagulability
- Stasis
- Endothelial Dysfunction

#### Genetics
- Temporal Association AF & Stroke
- As needed Anticoagulation Plausible
- Focal Therapy -> Lower Risk
- Rhythm Treatments -> Lower Risk

### Systemic Disease Symptom
- **Aging**
  - Risk Marker
  - Obesity
  - Metabolic Syndrome
  - Sleep Apnea
  - Diabetes
  - Hypertension

- **Alcohol**
  - Sedentary Lifestyle

#### Arterial Stiffness
- Microvascular Dysfunction
- Diastolic Dysfunction

#### Atrial Dilatation/Fibrosis/
- Myopathy = Disease State

#### Hypercoagulable State

#### Poor Temporal Association AF & Stroke
- Systemic Therapy -> Lower Risk
- Risk Persists Despite Rhythm Treatment

---

**TJ Bunch et. al. Eur Heart J. 2016;37:2890**
**BW Calenda, V Fuster, V Reddy et. al. Nat Rev Cardiol 2016;13:549**
Obesity and AF

CJ Lavie et. al. J Am Coll Cardiol 2017;70:2022
Adipose Tissue Depots Occur Throughout The Body

Definition of Epicardial Fat
And Related Adipose Tissues

CX Wong et. al. Eur Heart J. 2017;38:1294
Bariatric Surgery and the Risk of AF

Unadjusted HR = 0.71 (95% CI, 0.60-0.83)
p < 0.001

Control Group
Surgery Group

Patients at Risk
Control 2,021 1,979 1,840 1,574 570 114
Surgery 2,000 1,955 1,853 1,615 617 134

S Jamaly et. al. J Am Coll Cardiol 2016;68:2497
2) Mechanisms of AF Initiation At The Pulmonary Veins

- Strands of fibres poorly coupled to left atrial tissue
- Abrupt changes in fibre orientation promoting conduction delays and block

PV ion currents/APs:
- Small \( I_{Na} \), reduced RMP
- Small \( I_{Ca,L} \), larger \( I_{Kf/Ks} \), reduced AP duration

PV ion currents/APs:
- Small \( I_{Kf} \), Na\(^+\) channels inactivated
- Small \( I_{Ca,L} \), larger \( I_{Kf/Ks} \), larger \( I_{Kr} \), reduced AP duration, ERP

2) Molecular Mechanisms of Focal Ectopic Firing in Paroxysmal AF

S Nattel et al. Nat Rev Cardiol. 2016; 13: 575. – Also Re-entry
Natural History of AF

AF events

AF-Maintaining Substrate

Genetic causes
Focal PV sources

AF-induced remodeling
CV disease-induced remodeling
Aging-related remodeling

CV

Age (yrs)

40
80

JB Guichard et. al. J Am Coll Cardiol 2017;70:756
LA Tissue Fibrosis on 3D LGE CMR Scans

Low Fibrosis <15%

High Fibrosis >15%

healthy tissue

fibrotic

JB King, N Marrouche et al. J Am Coll Cardiol 2017;70:1311
Fibrosis of the LA Wall, Blinded To The Treating Physicians:

Stage 1 (<10% ),
Stage 2 (≥10% -<20% ),
Stage 3 (≥20% -<30% ),
Stage 4 (≥30% ).
Risk of MACCE Utah Stage of LAG Enhancement Severity

A

Log rank p-value for trend: <0.001

Cumulative Incidence

Years

0.00 0.10 0.20 0.30 0.40 0.50 0.60

0 1 2 3 4 5

Number at Risk

Utah Stage I  424 319 237 181 136 69
Utah Stage II  509 390 296 212 128 73
Utah Stage III 235 169 141 108 74 44
Utah Stage IV  60 44 39 31 21 15

B

LA Fibrosis, %

0 5 10 15 20 25 30

Population, %

0 0.5 1.0 2.0 3.0 4.0 6.0 8.0 16.0

Hazard Ratio

JB King, N Marrouche et. al. J Am Coll Cardiol 2017;70:1311
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V Fuster, JS Chinitz, Circ. 2012; 125: 2285

Swedish AF Cohort Register (L Friberg, GYH Lip et al) Circ. 2012; 125: 2298


A/C Prevention - Emboli >>> Bleeding, Thrombosis > Bleeding
1b). To Bridge & How to Bridge for DOACs/VKAs


ORB1T=AF (BA Steinberg et al.), Circulation 2015; 131:488 – Exeptions Only
2a) Predictors of Stroke Risk Incorporated in the CHA$_2$DS$_2$-VASc Schemes - Atrial Cardiomyopathy

Clinical Predictors of Stroke Risk Associated with AF

- Heart Failure
- Hypertension
- Diabetes
- Aging
- Valve disease
- Vascular disease

Atrial cardiomyopathy

Atrial fibrillation
LAA Structure / Function – Stroke in NSR

Cardiac Imaging For Assessment

J Romero et. al. Nat Rev Cardiol. 2014;11:470
ENGAGE AF (DK Gupta et al.) EHJ 2014; 35:1457 – LA Function / NSR ?
ASSERT (M Brambatti, et al.) Circ. 2014; 129:2094- LV Function / NSR ?
IMPACT (DT Martin et al.) EHJ; 2015; 36:1660- LV Function / NSR ?
2b). **Silent Cerebral Infarcts (SCI)**

**Cardiac Disease And Procedures**

- **Cardioembolic heart disease**
  - Atrial fibrillation
  - Left ventricular thrombus
  - Cardiomyopathy
  - Patent foramen ovale

- **Cardiac procedures**
  - Left heart catheterization
  - CABG surgery
  - Transcatheter aortic valve implantation
  - Pulmonary vein isolation
  - Closure of patent foramen ovale

Silent cerebral infarct

- **Stroke**
- **Cognitive decline**
- **Dementia**
- **Depression**


F Gaita et. al. *J Am Coll Cardiol* 2013;62:1990 (Italy)

Silent Cerebral Ischemia in AF Correlation With Cognitive Function

F Gaita et. al. J Am Coll Cardiol 2013;62:1990 (Italy)
Data are drawn from the Whitehall II study, N=10,308 at study recruitment in 1985. A battery of cognitive tests was administered four times (1997-2013) to 7428 participants -414 cases of AF-, aged 45-69 years in 1997. Compared with AF-free participants, those with longer exposure to AF (5, 10, or 15 years) experienced faster cognitive decline after adjustment for sociodemographic, behavioural, and chronic diseases (\( P \) for trend=0.01). Stroke did not explain it. In adults aged 45-85 years AF is associated with accelerated cognitive decline and higher risk of dementia.
Decline In The Global Cognitive Score Function of AF

A Singh-Manoux et. al. Eur Heart J. 2017;38:2612
Mechanisms Of AF Leading To Cognitive Decline Or Dementia

HC Diener et. al. Eur Heart J. 2017;38:2619
<table>
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<th>Challenge</th>
<th>Description</th>
<th>(2)</th>
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1,2). Preventing Bleeding in Pts with AF-PCI

Group 3 - D-adjusted vitamin K antagonist plus DAPT for 1, 6, or 12 months.
Group 2 - VLD rivaroxaban (2.5 mg twice daily) plus DAPT for 1, 6, 12 Mo
Group 1 - LD rivaroxaban (15 mg once daily) plus a P2Y12 inhibitor for 12 Mo

**PIONEER AF-PCI** (CM Gibson et. al. ) **NEJM 2016;375:2423**
Group 3 - D-adjusted vitamin K antagonist plus DAPT for 1, 6, or 12 months.
Group 2 - VLD rivaroxaban (2.5 mg twice daily) plus DAPT for 1, 6, 12 Mo
Group 1 - LD rivaroxaban (15 mg once daily) plus a P2Y12 inhibitor for 12 Mo

PIONEER AF-PCI (CM Gibson et. al.) NEJM 2016;375:2423
Long-term Treatment Of Patients On NOAC Therapy After Revascularization – Elective or ACS

Elevate PCI with newer generation DES or BMS

Acute coronary syndrome

Factors to shorten combination therapy
- (Uncorrectable) high bleeding risk
- Low atherothrombotic risk (by REACH or SYNTAX score if elective?; GRACE <118 if ACS?)

Factors to lengthen combination therapy
- First-generation DES
- High atherothrombotic risk (scores as above; stenting of the left main, proximal left anterior descending, proximal bifurcation; recurrent MIs; etc.) and low bleeding risk

H Heidbuchel et al. Eur Heart J. 2017;38:2137
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    | Efficacy vs Safety (2)                 |
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    | AC vs LAA Closure (2)                  |
| 7. | Screening 
    | Screening vs No Screening (2)          |
VHD was defined as history or baseline echo evidence of at least moderate aortic/mitral regurgitation, aortic stenosis, or prior valve surgery - bioprosthesis replacement, valve repair, valvuloplasty. Patients with moderate to severe mitral stenosis or mechanical heart valves were excluded from the trial. Comparisons were made of rates of stroke/systemic embolic event (SSEE) & major bleeding. VHD increased the risk of death, major adverse CV events, and major bleeding but did not affect the relative efficacy or safety of higher-dose edoxaban versus warfarin in AF.

R De Caterina et al., J Am Coll Cardiol 2017; 69:1372
CT January et. al. J. Am. Coll. Card. 2014; 64: e1 – No Dabigatran in Mechanical HV
<table>
<thead>
<tr>
<th>Characteristics of Warfarin &amp; NOAC Agents</th>
<th>Warfarin</th>
<th>Apixaban</th>
<th>Rivaroxaban</th>
<th>Dabigatran</th>
<th>Edoxaban</th>
</tr>
</thead>
<tbody>
<tr>
<td>Renal clearance of parent drug</td>
<td>&lt;1%</td>
<td>27%</td>
<td>36%</td>
<td>80%</td>
<td>50%</td>
</tr>
<tr>
<td>Lowest CrCl drug can be prescribed per FDA label, ml/min</td>
<td>Can be used on dialysis</td>
<td>&lt;15*</td>
<td>15</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>HR (95% CI) of stroke referent to warfarin, CrCl &lt;50 ml/min</td>
<td>Reference</td>
<td>0.79 (0.55-1.14)</td>
<td>0.88 (0.65-1.19)</td>
<td>0.56 (0.37-0.85)</td>
<td>0.87 (0.65-1.18)†</td>
</tr>
<tr>
<td>HR (95% CI) of major bleeding referent to warfarin, CrCl &lt;50 ml/min</td>
<td>Reference</td>
<td>0.50 (0.38-0.66)</td>
<td>0.98 (0.84-1.14)</td>
<td>1.01 (0.79-1.30)</td>
<td>0.76 (0.58-0.98)†</td>
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## Doses Of The Different NOACs In The 4 Large Trials In AF Patients

<table>
<thead>
<tr>
<th>NOAC</th>
<th>Dose</th>
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<tbody>
<tr>
<td>Dabigatran (RELY)</td>
<td>150 mg twice daily</td>
</tr>
<tr>
<td></td>
<td>110 mg twice daily</td>
</tr>
<tr>
<td>Rivaroxaban (ROCKAT-AF)</td>
<td>20 mg once daily, protocol-mandated dose reduction to 15 mg</td>
</tr>
<tr>
<td>Apixaban (ARISTOTLE)</td>
<td>5 mg twice daily, protocol-mandated dose reduction to 2.5 mg</td>
</tr>
<tr>
<td>Edoxaban (ENGAGE-AF)</td>
<td>60 mg once daily, protocol-mandated dose reduction to 30 mg</td>
</tr>
<tr>
<td></td>
<td>30 mg once daily, protocol-mandated dose reduction to 15 mg</td>
</tr>
</tbody>
</table>

*HC Diener et. al. Eur Heart J. 2017;38:860*
Proposed Algorithm for Oral Anticoagulant Choices in Patients With Atrial Fibrillation and Chronic Kidney Disease

Patient with atrial fibrillation and chronic kidney disease

Determine stroke risk (CHA₂DS₂-VASc Score)
Consider oral anticoagulation if score is ≥ 1 in males / ≥ 2 in females

Determine bleeding risk (HAS-BLED Score)

Estimate creatinine clearance (CrCl) to determine appropriate oral anticoagulant (OAC)

<table>
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<tr>
<th>OAC options:</th>
<th>CrCl &lt; 15 ml/min or ESRD on RRT</th>
<th>CrCl 15–29 ml/min</th>
<th>CrCl 30–49 ml/min</th>
<th>CrCl ≥ 50 ml/min</th>
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<tr>
<td>Vitamin K antagonist</td>
<td>When time in therapeutic range &gt;70%</td>
<td>When time in therapeutic range &gt;70%</td>
<td>When time in therapeutic range &gt;70%</td>
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</tr>
<tr>
<td>Apixaban</td>
<td>2.5 mg, b.i.d.</td>
<td>5 mg, b.i.d.</td>
<td>5 mg, b.i.d.</td>
<td>5 mg, b.i.d.</td>
</tr>
<tr>
<td>Dabigatran</td>
<td>❌</td>
<td>75 mg, b.i.d.</td>
<td>150 or 110 mg, b.i.d.</td>
<td>150 mg, b.i.d.</td>
</tr>
<tr>
<td>Edoxaban</td>
<td>❌</td>
<td>30 mg, o.d.</td>
<td>30 mg, o.d.</td>
<td>60 mg, o.d.</td>
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<tr>
<td>Rivaroxaban</td>
<td>❌</td>
<td>15 mg, o.d.</td>
<td>15 mg, o.d.</td>
<td>20 mg, o.d.</td>
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</table>

Address bleeding risk factors, frequent follow up, and closely monitor renal function in NOAC users


HIT........(?)
2b). Initiation Or Re-initiation of Oral A/C After Intracranial Haemorrhage

ESC Working Group on Thrombosis (S Halvorsen et. al.) Eur Heart J. 2017;38:1455
H-C Diener et al. Eur Heart J 2017; 38: 860 – Bleeding GI Tract: In 1 Week NOAC or AC
Idarucizumab & Other Reversal Agents

When available, idarucizumab is likely to be the treatment of choice for patients who present with diabigatran-induced uncontrolled or life-threatening bleeding or for those who require urgent surgery or invasive procedures. Other reversal agents are in development to reverse other NOACs. These include andexanet alfa, a recombinant truncated form of enzymatically inactive factor Xa, which binds and reverses the anticoagulant action of the factor Xa inhibitors, and PER977 (ciraparantag), a synthetic small molecule that is reported to bind to all of the NOACs.

JW Eikelboom et al., Circ 2015; 132:2412 – ESC Rome, 2016 Sept
Idarucizumab for Dabigatran Reversal
Full Cohort Analysis

We performed a multicenter, prospective, open-label study to determine whether 5 g of intravenous idarucizumab would be able to reverse the anticoagulant effect of dabigatran in patients who had uncontrolled bleeding (group A) or were about to undergo an urgent procedure (group B). A total of 503 patients were enrolled: 301 in group A, and 202 in group B. The median maximum percentage reversal of dabigatran was 100%. In emergency situations, idarucizumab rapidly, durably, and safely reversed the AC effect of dabigatran.

Key Measurements Before And After The Administration of Idarucizumab

C Concentration of Unbound Dabigatran in Groups A and B

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Main Cardiac Targets of The Different Domains Of AF Management

P Kirchhof. Lancet 2017;390:1873
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<tr>
<td>Temperature-Controlled Radiofrequency Ablation for Pulmonary Vein</td>
<td>J Am Coll Cardiol 2017;69:1247</td>
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<td>Isolation in Patients With Atrial Fibrillation</td>
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<td>Left Atrial Appendage Isolation in Patients With Longstanding</td>
<td>J Am Coll Cardiol 2016;68:1929, J</td>
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<tr>
<td>Persistent AF Undergoing Catheter Ablation</td>
<td>Am Coll Cardiol 2017;69:1257</td>
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<td>BELIEF Trial</td>
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<td>Clinical Benefit of Ablating Localized Sources for Human Atrial</td>
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<td>Fibrillation</td>
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<td>The Indiana University FIRM Registry</td>
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<td>Complexity and Distribution of Drivers in Relation to Duration of</td>
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<tr>
<td>Persistent Atrial Fibrillation</td>
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1a). Cumulative Hazard Rates Of Embolic Events According To The Pattern Of AF Occurrence

T Vanassche, SJ Connolly et. al. Eur Heart J. 2015;36:281
1b). **CHA$_2$DS$_2$-VASc (Recurrent AF) in Predicting Clinical Outcomes in AF After Catheter Ablation**

T-F Chao et al., JACC 2011; 58:2380 (Japan) – 565 Pts
1c). AF Burden - After Catheter Ablation
Several Strategies (Linq Recorder etc)

El Charitos et. al. Circulation. 2012;126:806 (Luebeck, Germ.)
2a) Bleeding Risk, Ischemic Stroke Risk, Indications for Left Atrial Appendage Closure

KC Koskinas et. al. J AmColl Cardiol Intv 2016;9:1374
Primary Efficacy Outcome of Watchman LAA Closure For Embolic Protection In AF PROTECT AF Over 60 Months

- RP Whitlock et. al. Circulation. 2015;131:756
2c). Stroke Prevention in AF With LAA Closure

PREVAIL and PROTECT AF (VY Reddy et. al.) J Am Coll Cardiol 2017;70:2964
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Screening for Atrial Fibrillation

**Whom to screen**
- People aged >65
- Patients with AF who are undertreated

**Where to screen**
- Primary care or Specialist clinics (country specific)
- Opportunistic pulse then ECG
- Single time point: single-lead ECG
- Patient activated ECG (2 week) > 75 or younger if high risk

**How to screen**
- Implanted devices with enrichment
- External long term +/- enrichment

**Special cases**
- Post stroke ESUS: long term continuous

**AF-SCREEN preferred**
- Possible with further data
- Currently too expensive at scale

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