OCCUPATIONAL HEALTH: STAYING ON THE STRAIGHT AND NARROW

ALAMA AUTUMN CONFERENCE • 21-23 NOVEMBER 2018 • OULTON HALL, ROTHWELL LANE, OULTON, LEEDS LS26 8HN
Contain NUTS
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Blood thinners and working in the police force – is it safe?

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Consultant Haematologist
Bradford Royal Infirmary
Disclosures

• Bayer – sponsorship to attend conferences and to occasional talks
Summary

• What is the Police current position?

• A Case study

1. Do they need anti-coagulants?

2. Assessing the risk from injury on oral anticoagulants

3. The different anticoagulants – any difference in risk?

4. Making a decision
Police statement: Risk assessment on anticoagulants

“a police officer taking warfarin anticoagulation is not fit to perform the normal duties of a police officer due to the risk of incurring an ICH during arrest and restraint”

“should focus on the risks of head trauma, and take into consideration the wishes of the individual officer.”

• No specific research on the police
National Recruitment Standards - Medical Standards For Police Recruitment

• “unlikely to be suitable”
• “the underlying condition may also make the applicant unsuitable”

Home Office Circular 59 / 2004
Case
31 year old male police officer

- Presented to A&E with acute chest pain and SOB 2 weeks following a flight to Tenerife.
- Elevated D-dimer 1200
- CTPA – bilateral PE with right heart strain
- Commenced anticoagulation with rivaroxaban and referred to haematology
Haematology clinic questions and decisions

• Questions
  1. What is the cause of thrombosis
  2. Any persistent risk factors

• Decisions
  1. How long on anticoagulation
  2. Which anticoagulant
Case

• Questions
1. What is the cause of thrombosis – Antiphospholipid Syndrome
2. Any persistent risk factors – Antiphospholipid syndrome

• Decisions
1. How long on anticoagulation – Long term
2. Which anticoagulant - Rivaroxaban
"Hi, I am a serving PCSO in the Met police and I have been using Warfarin for 12 months now as I previously had a stroke. Although I've returned to work fit and well and my INR readings are at the required level our force medical advisor has stated I can return to duty in uniform but cannot patrol. I have 15 years police service .......I am currently in Limbo career wise... don't know what to do or where next to go?"

Due to the fact that I'm on blood thinners "Xarelta" I at this time can't be medically cleared by their medical doctor until I'm able to come off my blood thinners. Even though at the age of 43 I'm more fit than I've been in years!

"Nearing the end of my Foundation Training, mild DVT for which I was prescribed warfarin. Unfortunately, as a consequence of this I have been removed from frontline policing duties"

“I am in the process of interviewing and will eventually have to take the pre med physical. I don't want to disclose that I am taking warfarin, even though it is a small dosage. Any advice?”
Why and who are on anticoagulants?
Incidence of VTE and AF with increasing age

Overall incidence rate of first VTE: 131.5 (95% CI 130.2–132.9) per 100,000 person-years. 58.1% are unprovoked VTE.
Age of the police force

officers are getting older on average – almost a half (48.1 per cent) are now aged over 40
How many police on anticoagulants in the UK?

• 122,000 police officers in UK
• Median age around 40 years

• Would expect around 50-100 new DVT/PE per year
• Around 2500 would have AF
1. Do they need anticoagulants
AF

- **Anticoagulation NICE guidance**
  - 1.5.2 Consider anticoagulation for men with a **CHA$_2$DS$_2$-VASc** score of 1. Take the bleeding risk into account. [new 2014]
  - 1.5.3 Offer anticoagulation to people with a **CHA$_2$DS$_2$-VASc** score of 2 or above, taking bleeding risk into account. [new 2014]
  - 1.5.4 Discuss the options for anticoagulation with the person and base the choice on their clinical features and preferences. [new 2014]

<table>
<thead>
<tr>
<th>KNOW YOUR STROKE RISK</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHA2DS2-VASc Risk</td>
</tr>
<tr>
<td>---------------------</td>
</tr>
<tr>
<td>CHF or LVEF &lt;40%</td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Age &gt; 75</td>
</tr>
<tr>
<td>Diabetes</td>
</tr>
<tr>
<td>Stroke / TIA /</td>
</tr>
<tr>
<td>Thromboembolism</td>
</tr>
<tr>
<td>Vascular Disease</td>
</tr>
<tr>
<td>Age 65-74</td>
</tr>
<tr>
<td>Female</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

*CHF = congestive heart failure; TIA - transient ischemic attack; LVEF = left ventricular ejection fraction.*
VTE
Risk of VTE recurrence is highest in the first 3-4 weeks after the index DVT/PE event\textsuperscript{5}

VTE recurrence remains a persistent threat
**EINSTEIN EXT:** Primary efficacy outcome.
Continued protection against recurrent VTE
up to 360 days\(^1\)

---

**Number of subjects at risk**

<table>
<thead>
<tr>
<th></th>
<th>Rivaroxaban</th>
<th>Placebo</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>602</td>
<td>594</td>
</tr>
<tr>
<td>30</td>
<td>590</td>
<td>582</td>
</tr>
<tr>
<td>60</td>
<td>583</td>
<td>570</td>
</tr>
<tr>
<td>90</td>
<td>573</td>
<td>555</td>
</tr>
<tr>
<td>120</td>
<td>552</td>
<td>522</td>
</tr>
<tr>
<td>150</td>
<td>503</td>
<td>468</td>
</tr>
<tr>
<td>180</td>
<td>482</td>
<td>444</td>
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<tr>
<td>210</td>
<td>171</td>
<td>164</td>
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<tr>
<td>240</td>
<td>138</td>
<td>138</td>
</tr>
<tr>
<td>270</td>
<td>132</td>
<td>133</td>
</tr>
<tr>
<td>300</td>
<td>114</td>
<td>110</td>
</tr>
<tr>
<td>330</td>
<td>92</td>
<td>93</td>
</tr>
<tr>
<td>360</td>
<td>81</td>
<td>85</td>
</tr>
</tbody>
</table>

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Recurrence of VTE measured in the ITT population; all analyses were based on the first event.

VTE = Venous thromboembolism; HR = Hazard ratio; CI = Confidence interval; RRR = Relative risk; ARR = Absolute risk reduction; ITT = Intention to treat

Predicting recurrence of VTE
Provoked and unprovoked VTE

- **Transient/reversible factors**
  - e.g. surgery or hospitalisation

- **Continuing/irreversible factors**
  - e.g. cancer

- **No identifiable cause**

Proven VTE

Unprovoked (idiopathic) VTE

**ACCP** guidelines recommend at least 3 months' VKA therapy after provoked VTE, or longer after unprovoked (idiopathic) VTE

*ACCP = American College of Chest Physicians*
Virchow's triad

- Endothelial damage
  - Endothelial dysfunction
    - Smoking
    - Hypertension
  - Endothelial damage
    - Surgery
    - Catheter (PICC lines)
    - Trauma
- Hypercoagulability
  - Hereditary
    - Factor V Leiden
    - Prothrombin G20210A
    - Protein C and S deficiency
  - Acquired
    - Cancer
    - Chemotherapy
    - OCR/HRT
    - Pregnancy
    - Obesity
    - HIT
- Stasis
  - Immobility
  - Polycythemia
  - Can cause endothelial injury
VTE – prediction score

**DASH Prediction Score Derived From Cox Regression Analysis**

<table>
<thead>
<tr>
<th>DASH Predictors</th>
<th>β coefficient*</th>
<th>P-value</th>
<th>Recurrence score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. D-dimer abnormal, after stopping AC</td>
<td>0.96</td>
<td>&lt;0.0001</td>
<td>+2</td>
</tr>
<tr>
<td>2. Age &lt; 50 yr</td>
<td>0.43</td>
<td>0.002</td>
<td>+1</td>
</tr>
<tr>
<td>3. Sex - male</td>
<td>0.58</td>
<td>&lt;0.0001</td>
<td>+1</td>
</tr>
<tr>
<td>4. Hormone use at VTE onset</td>
<td>-1.05</td>
<td>0.002</td>
<td>-2</td>
</tr>
</tbody>
</table>

**DASH Prediction Rule**

- **DASH Score**
  - ≤ 1.0: 3.1%
  - 2.0: 6.4%
  - ≥ 3.0: 12.3%

*Cox regression coefficients after backward elimination and optimism correction

Extending anticoagulation in VTE
**EINSTEIN CHOICE:**

*Study design*

- Randomised, double-blind, Phase III study comparing the efficacy and safety profile of two doses of Xarelto with those of aspirin for the extended treatment of VTE for up to 1 year after the initial 6 to 12 months of therapy.

- To assess the possibility of dose reduction in treatment extension.

**Randomisation**

- Patients with symptomatic PE/DVT where there was uncertainty about the need for extension.
- Randomisation: 3,396
- Xarelto 20 mg OD (1,121)
- Xarelto 10 mg OD (1,136)
- Aspirin 100 mg OD (1,139)

Study not powered to compare Xarelto 10 mg to Xarelto 20 mg regimen.

**Previous 6–12 months anticoagulation***

- Patients with clear indication for therapeutic dose in extended treatment were excluded.

- **12 months' treatment**

  - Patients randomised after the requisite number of primary efficacy outcomes was reached were treated for ≥6 months.

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VTE = Venous thromboembolism, PE = Pulmonary embolism, DVT = Deep vein thrombosis.

EINSTEIN CHOICE:
Rationale for study arms

- **Xarelto 20 mg OD**
  - In EINSTEIN EXT, rivaroxaban 20 mg OD reduced the risk of recurrent VTE by 82% compared with placebo [5.9% ARR], with similar risk of major bleeding.

- **Xarelto 10 mg OD**
  - Rivaroxaban 10 mg OD offered effective thromboprophylaxis after elective hip or knee arthroplasty.

- **Aspirin 100 mg OD**
  - Aspirin 100 mg OD has been shown to reduce the risk of recurrent VTE by more than 30% compared with placebo, without increasing the risk of major bleeding.

VTE = Venous thromboembolism; OD = Once daily

EINSTEIN CHOICE:
Primary efficacy outcome¹

Xarelto provides superior risk reduction of recurrent VTE vs aspirin in extended treatment¹

<table>
<thead>
<tr>
<th>Xarelto 10 mg vs aspirin</th>
<th>HR = 0.26</th>
</tr>
</thead>
<tbody>
<tr>
<td>(95% CI 0.14-0.47)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Xarelto 20 mg vs aspirin</th>
<th>HR = 0.34</th>
</tr>
</thead>
<tbody>
<tr>
<td>(95% CI 0.2-0.59)</td>
<td></td>
</tr>
</tbody>
</table>

ARR = Adjusted risk reduction; RRR = Relative risk reduction; HR = Hazard ratio; VTE = Venous thromboembolism; OD = Once daily

EINSTEIN CHOICE: Primary safety outcome

Xarelto has a comparable major bleeding rate to aspirin, with a risk of <1%.1

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* EINSTEIN-CHOICE showed <1% major bleeding for extended treatment in PE/DVT patients.1

---

**Number of subjects at risk**

<table>
<thead>
<tr>
<th></th>
<th>Rivaroxaban, 20 mg</th>
<th>Rivaroxaban, 10 mg</th>
<th>Aspirin, 100 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1,107</td>
<td>1,081</td>
<td>1,053</td>
</tr>
<tr>
<td></td>
<td>1,048</td>
<td>1,036</td>
<td>1,024</td>
</tr>
<tr>
<td></td>
<td>983</td>
<td>901</td>
<td>818</td>
</tr>
<tr>
<td></td>
<td>780</td>
<td>709</td>
<td>709</td>
</tr>
<tr>
<td></td>
<td>642</td>
<td>645</td>
<td>445</td>
</tr>
</tbody>
</table>

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*In extended treatment of VTE; ‡No events after Day 360 up to Day 480.

NOAC = Novel oral anticoagulant; NE = Not significant; HR = Hazard ratio; PE = Pulmonary embolism; DVT = Deep vein thrombosis; VTE = Venous thromboembolism.

2. Assessing the risk from injury on anticoagulants
Risk from head injury in patients on warfarin

Table 3
Summary of key clinical outcomes (sTBI group)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Preinjury warfarin</th>
<th>No warfarin</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality (All patients)</td>
<td>52/700</td>
<td>13/700</td>
<td>&lt;0.010</td>
</tr>
<tr>
<td>Mortality (AIS Head ≥4)</td>
<td>34/266</td>
<td>9/169</td>
<td>&lt;0.013</td>
</tr>
<tr>
<td>HLOS [median days (Range)]</td>
<td>4 (1-49)</td>
<td>2 (0-87)</td>
<td>&lt;0.010</td>
</tr>
</tbody>
</table>

AIS Head ≥4 indicates group of patients with Abbreviated Injury Scale for Head of 4 or greater

Single centre
10 year data

65% warfarin out of range (mostly low)

sTBI warfarin group mortality 13% v 5%

Longer hospital stay

Inappropriate preinjury warfarin use in trauma patients: A call for a safety initiative

Hon HH,1,2 A Elmously,1 CD Stehly,1,2 JC Stoltzfus,3 MA Granson,4 SP Stawicki,1,2,4 and BA Hoey1,2
Mortality following traumatic ICH seems much higher in warfarin patients

- 94 patients on warfarin with head injury
- 25 has ICH
- 12/25 Died – around 50%
- Compared to 10% in age matched controls with traumatic ICH

- INR did not seem to effect outcome

- Mean INR 3.3 in alive group v 3.0 in patients that died

Complications of preinjury warfarin use in the trauma patient. Mina AA¹, Bair HA, Howells GA, Bendick PJ.
Hematoma Growth in Oral Anticoagulant Related Intracerebral Hemorrhage

Brett Cucchiara, MD; Steven Messe, MD; Lauren Sansing, MD; Scott Kasner, MD; Patrick Lyden, MD; for the CHANT Investigators

Table 2. Hemorrhage Expansion and Clinical Outcome by Group

<table>
<thead>
<tr>
<th></th>
<th>SICH (n=267)</th>
<th>OAT ICH (n=18)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICH volume change, median (IQR)</td>
<td>0.9 mL (0–5.4)</td>
<td>9.6 mL (0–19.4)</td>
<td>0.03</td>
</tr>
<tr>
<td>&gt;33% ICH expansion</td>
<td>26%</td>
<td>56%</td>
<td>0.006</td>
</tr>
<tr>
<td>Any ICH expansion</td>
<td>65%</td>
<td>78%</td>
<td>0.27</td>
</tr>
<tr>
<td>Mortality</td>
<td>17%</td>
<td>62%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>mRS 4–6</td>
<td>50%</td>
<td>90%</td>
<td>0.001</td>
</tr>
</tbody>
</table>
What about minor head injury on warfarin?

<table>
<thead>
<tr>
<th>Head injury</th>
<th>GCS</th>
<th>LOC</th>
<th>ICH rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>minimal</td>
<td>15</td>
<td>no</td>
<td>4.8%</td>
</tr>
<tr>
<td>minor</td>
<td>13-15</td>
<td>yes</td>
<td>21.9%</td>
</tr>
</tbody>
</table>

176 patients on warfarin with head injury
INR had no effect on ICH rate

Much higher rate ICH if LOC
Role of INR and risk of bleeding

<table>
<thead>
<tr>
<th>INR</th>
<th>bleeding events per patient 100 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>2-3</td>
<td>4.8</td>
</tr>
<tr>
<td>3-4.5</td>
<td>9.5</td>
</tr>
<tr>
<td>4.5 -7</td>
<td>40.5</td>
</tr>
<tr>
<td>&gt;7</td>
<td>200</td>
</tr>
</tbody>
</table>

Prospective study over 1st year of treatment in Italy – n=2745
Increased risk with age >70 years

**HAS-BLED Bleeding Risk Score**

- **H**: Hypertension ( >160 mmHg) (1 point)
- **A**: Abnormal renal / liver function (1 pt ea) (1 point)
- **S**: Stroke (usu lacunar) (1 point)
- **B**: Bleeding (hx or predisposition, anemia) (1 point)
- **L**: Labile INRs (1 point)
- **E**: Elderly (>65 yrs) (1 point)
- **D**: Drugs or Alcohol (1 pt ea) (1 point)

16% had no indication for warfarin ACCP guidelines

Inappropriate preinjury warfarin use in trauma patients: A call for a safety initiative

Hon HH,1,2  A Elmously,1  CD Stehly,1,2  JC Stoltzfus,3  MA Granson,4  SP Stawicki,1,2,4 and  BA Hoey1,2
3. Different anticoagulants- any difference?
Warfarin
What is warfarin?

- Haemorrhaging cows in 1920’s due to eating sweet clover in hay (not fresh)
- Coumarin derivative synthesised 1948
- Vitamin K antagonist – inhibits vitamin K epoxide reductase (VKOR) which is needed to activate Vit K1
- Inhibits carboxylation of FII, FVII, FIX, FX, PC, PS
- PIVKA (proteins induced in vitamin K absence) accumulate but no function (can be measured)
- Takes 3-5 days to work (while previously activated forms degrade) – FII takes longest to reduce
- PC and PS also reduced so initial “pro-thrombotic” phase covered by LMWH
How warfarin works
DOAC
## DOACs

<table>
<thead>
<tr>
<th></th>
<th>Dabigatran (IIa inhibitor)</th>
<th>Rivaroxaban (Xa inhibitor)</th>
<th>Apixaban (Xa inhibitor)</th>
<th>Edoxaban (Xa inhibitor)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Licensed for</td>
<td>SPAF Treatment and 2(^{nd}) prevention of VTE</td>
<td>SPAF Treatment and 2(^{nd}) prevention of VTE</td>
<td>SPAF Treatment and 2(^{nd}) prevention of VTE</td>
<td>SPAF Treatment and 2(^{nd}) prevention of VTE</td>
</tr>
<tr>
<td>Protein binding</td>
<td>35%</td>
<td>high</td>
<td>high</td>
<td>High</td>
</tr>
<tr>
<td>Peak effect, h</td>
<td>0.5–2</td>
<td>2–4</td>
<td>3–4</td>
<td>1–2</td>
</tr>
<tr>
<td>Excretion</td>
<td>Renal</td>
<td>Renal/hepatic</td>
<td>Renal/hepatic</td>
<td>Renal/hepatic</td>
</tr>
<tr>
<td>(T_{1/2}), h</td>
<td>11–14 (27 h CrCl &lt;30 ml/min)</td>
<td>5–13</td>
<td>~12</td>
<td>10–14</td>
</tr>
</tbody>
</table>

Non-oral anticoagulants:

Heparin
LMWH
Fondaparinux
Argatroban
DOAC v Warfarin
Reducing Ischaemic Stroke in AF

<table>
<thead>
<tr>
<th>Events per 100 person-years</th>
<th>DOAC</th>
<th>Warfarin</th>
<th>HR (95% CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apixaban</td>
<td>2.02</td>
<td>2.40</td>
<td>0.79 (0.37–1.72)</td>
<td>0.56</td>
</tr>
<tr>
<td>Dabigatran</td>
<td>1.85</td>
<td>3.10</td>
<td>0.60 (0.28–1.27)</td>
<td>0.18</td>
</tr>
<tr>
<td>Rivaroxaban</td>
<td>1.37</td>
<td>2.99</td>
<td>0.48 (0.29–0.79)</td>
<td>0.004</td>
</tr>
</tbody>
</table>
## Major bleeding rates

<table>
<thead>
<tr>
<th>Events per 100 person-years</th>
<th>DOAC</th>
<th>Warfarin</th>
<th>HR (95% CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apixaban</td>
<td>2.21</td>
<td>2.74</td>
<td>0.79 (0.38–1.64)</td>
<td>0.52</td>
</tr>
<tr>
<td>Dabigatran</td>
<td>1.69</td>
<td>2.91</td>
<td>0.58 (0.26–1.27)</td>
<td>0.17</td>
</tr>
<tr>
<td>Rivaroxaban</td>
<td>3.09</td>
<td>3.02</td>
<td>1.07 (0.71–1.61)</td>
<td>0.74</td>
</tr>
</tbody>
</table>
Intracranial Haemorrhage (ICH)
Intracranial Haemorrhage ICH

- Dabigatran 110 mg BID: $P < .001$
- Dabigatran 150 mg BID: $P < .001$
- Rivaroxaban 20 mg QD: $P = .024$
- Apixaban 5 mg BID: $P < .001$
- Edoxaban 60 mg QD: $P < 0.001$
- Edoxaban 30 mg QD: $P < 0.001$

DOAC and traumatic Head Injury

TBI in over 60 year olds admitted to ICSU
N=186
33 on DOAC
32 on warfarin mean INR 2.6

85% reversal in warfarin group v 24% in DOAC (4 PPC and 4 Idarucizumab)
<table>
<thead>
<tr>
<th>Author</th>
<th>N</th>
<th>DOAC</th>
<th>Main outcome</th>
<th>Favours</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wood et al 2017</td>
<td>81</td>
<td>Warfarin v rivaroxaban</td>
<td>Improved survival DOAC 92 v 72%</td>
<td>DOAC</td>
<td></td>
</tr>
<tr>
<td>Pozzessere et al (prior to reversal agent idarucizumab)</td>
<td>246</td>
<td>Warfarin v dabigatran</td>
<td>No difference mortality or ICH rate</td>
<td>neither</td>
<td>Confined to elderly falls and low rate of ICH</td>
</tr>
<tr>
<td>Maung et al</td>
<td>112 DOAC 373 VKA</td>
<td>Warfarin v DOAC v no ATT</td>
<td>NS 19.3%v 16.7% v 10.9%</td>
<td>Trend favouring DOAC</td>
<td></td>
</tr>
<tr>
<td>Feeney et al</td>
<td>162</td>
<td>Warfarin v DOAC</td>
<td>Reduced mortality DOAC 20.9% v 4.9%</td>
<td>DOAC</td>
<td></td>
</tr>
<tr>
<td>Kobayashi et al</td>
<td>1847 16 centres prospective</td>
<td>Warfarin v DOAC v antiplatelet</td>
<td>No difference mortality in groups 7% ICH more often in antiplatelet group 35% v 24%</td>
<td>neither</td>
<td>DOAC only 10% of cases most patients on antiplatelet treatment Possibly confounding error due to indication for treatment</td>
</tr>
</tbody>
</table>
Do antiplatelet drugs also cause increase risk with head injury?

- New York trauma centre
- 3436 cases reviewed

<table>
<thead>
<tr>
<th>Drug</th>
<th>Number</th>
<th>Risk ICH</th>
<th>Mortality with ICH</th>
</tr>
</thead>
<tbody>
<tr>
<td>aspirin</td>
<td>228</td>
<td></td>
<td>No increased risk</td>
</tr>
<tr>
<td>clopidogrel</td>
<td>43</td>
<td></td>
<td>6%</td>
</tr>
<tr>
<td>warfarin</td>
<td>91</td>
<td>30%</td>
<td>30%</td>
</tr>
<tr>
<td>Warfarin + antiplatelet</td>
<td>94</td>
<td>50%</td>
<td></td>
</tr>
</tbody>
</table>

Impact of preinjury warfarin and antiplatelet agents on outcomes of trauma patients.
Bonville DJ, Ata A, Jahraus CB, Arnold-Lloyd T, Salem L, Rosati C, Stain SC.
Reversibility of anticoagulant
4. Making a decision
Think should anticoagulation be stopped?

<table>
<thead>
<tr>
<th>CHADS2 – VASc Score</th>
<th>Stroke rate % per year</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>0</td>
</tr>
<tr>
<td>H</td>
<td>1.3</td>
</tr>
<tr>
<td>A</td>
<td>2.2</td>
</tr>
<tr>
<td>D</td>
<td>3.2</td>
</tr>
<tr>
<td>S2</td>
<td>4.0</td>
</tr>
<tr>
<td>V</td>
<td>6.7</td>
</tr>
<tr>
<td>A</td>
<td>9.8</td>
</tr>
<tr>
<td>Sc</td>
<td>9.6</td>
</tr>
<tr>
<td>0</td>
<td>6.7</td>
</tr>
<tr>
<td>1</td>
<td>15.2</td>
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</table>

<table>
<thead>
<tr>
<th>HAS-BLED Bleeding Risk Score</th>
<th>BLEEDS (per 100 pt yrs)</th>
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<tbody>
<tr>
<td>H</td>
<td>1.13</td>
</tr>
<tr>
<td>A</td>
<td>1.02</td>
</tr>
<tr>
<td>S</td>
<td>1.88</td>
</tr>
<tr>
<td>B</td>
<td>3.74</td>
</tr>
<tr>
<td>L</td>
<td>8.70</td>
</tr>
<tr>
<td>E</td>
<td>12.5</td>
</tr>
</tbody>
</table>
Reducing the risk of thrombosis

• Stop smoking
• Obesity management
• Exercise / diet advice
• Consider atrial appendage closure surgery
Reducing the risk of bleeding

• Bring back helmets?
• Avoid front line service
• Consider antiplatelet therapy instead – understanding is not as effective
• Consider DOAC usage
• Timing of DOAC medicine?
What do they want to do?

• Do they want to continue front line service?
• What do they want to do?
• What is their attitude to risk?
Think individualised patient care:

- Deep sea diver
- Caver
- Airline pilot
- International rugby player
- Fireman
- Army officer
Summary

- Individual approach to the assessment is essential

1. Can they stop anticoagulation – may need specialist Haematology or cardiology opinion
2. What is the safest anticoagulation for them
3. Can the risk of head injury be reduced
Any questions?
references

- Intracranial Bleeds after Minor and Minimal Head Injury In Patients on Warfarin, published in the journal of Emergency Medicine in 2015
- Prevalence and implications of preinjury warfarin use: an analysis of the National Trauma Databank Dossett, Lesly A; Riesel, Johanna N; Griffin, Marie R; Cotton, Bryan A; NLM.Archives of surgery (Chicago, Ill. : 1960) 146. 5: 565-70. (May 2011)
OCCUPATIONAL HEALTH: STAYING ON THE STRAIGHT AND NARROW

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