24 Stroke patients

24.1 Introduction

Recent stroke has been associated with an increased developing venous thromboembolism (VTE) 217. Reasons for this increased risk of VTE is thought to be due to the alteration in blood flow as a results of the weakness in the affected limb, possibly leading to vessel wall injury, and a resulting hypercoagulable state related to changes in the blood after stroke236. A wide range of VTE incidence has been reported for stroke patients with estimates of between 15-60%18,120. Diagnosing DVT after stroke may be difficult as symptoms may be similar to those related to the stroke such as leg swelling236. One study149 reviewed stroke patients 6 months after onset and found that patients who developed a DVT after stroke had a statistically significant poorer outcome, using a modified Rankin score, compared to those who did not develop DVT.

Stroke is divided into two main types; ischaemic stroke caused by blood clots preventing blood flow to the brain and haemorrhagic stroke caused by bleeding into/of the brain. Both types of stroke are associated with an increased risk of VTE236. NICE published guidelines in 2008 for the diagnosis and acute management of stroke and transient ischaemic attacks477.

24.2 Evidence of methods of prophylaxis

24.2.1 Summary of comparisons identified for any outcome

Seventeen (17) studies were identified that considered the interventions under consideration for stroke patients35,157,158,165,167,240,278,369,434,435,466,509,520,538,540,581,598. Of these, 2 studies were three arm trials509,520. Only studies using prophylactic-doses were included.

Only three of these studies included haemorrhagic stroke patients158,434,435. All other patients were ischaemic stroke patients.

All included RCTs were either individually critically appraised to be of a high quality (level 1+ or level 1++) or came from systematic reviews of RCTs which had been critically appraised to be of a high quality (level 1+ or level 1+++).

Although it is likely that many patients within the CLOTS study158 were treated with aspirin the authors have not provided this information within the study. For this reason the results of the study have been reported as ‘GCS vs. no prophylaxis’ rather than ‘GCS + aspirin vs. aspirin’.
### Figure 24-1: Number of studies which compared various types of prophylaxis methods.

Numbers in boxes indicate the number of RCTs for each comparison. Boxes shaded grey indicates areas where no studies were identified.

GCS – anti-embolism / graduated compression stockings; IPCD/FID – intermittent pneumatic compression devices or foot impulse devices; LMWH – low molecular weight heparin; UFH – unfractionated heparin; Asp (HD) – high dose aspirin (>300mg); Asp (LD) – low dose aspirin (<300mg); mech – mechanical prophylaxis (i.e. anti-embolism / graduated compression stockings, intermittent pneumatic compression devices or foot impulse devices); pharm – pharmacological prophylaxis

#### 24.2.2 Results from pairwise comparisons

**Table 24-1: DVT – summary of results from RCTs**

<table>
<thead>
<tr>
<th>Comparison</th>
<th>No. of studies</th>
<th>Intervention</th>
<th>Control</th>
<th>Relative risk</th>
<th>Absolute effect</th>
<th>Forest plots &amp; Evidence tables</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proph vs no proph</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GCS vs nil 158</td>
<td>1</td>
<td>205/1256</td>
<td>224/1262</td>
<td>0.92</td>
<td>(-0.01, 0.01)</td>
<td>ET: 23 FP: 1</td>
</tr>
<tr>
<td>IPCD/FID vs nil 538</td>
<td>1</td>
<td>6/13</td>
<td>6/13</td>
<td>1.00</td>
<td>(0.00, 2.00)</td>
<td>ET: 24 FP: 4</td>
</tr>
<tr>
<td>LMWH vs nil 240,540,581</td>
<td>3</td>
<td>23/89</td>
<td>36/110</td>
<td>0.74</td>
<td>(-0.08, 0.08)</td>
<td>ET: 26 FP: 13</td>
</tr>
<tr>
<td>UFH vs nil 167,434,435,520</td>
<td>4</td>
<td>41/238</td>
<td>146/243</td>
<td>0.31</td>
<td>(-0.35, -0.09)</td>
<td>ET: 27 FP: 17</td>
</tr>
<tr>
<td>Asp (high dose) vs nil 157,520</td>
<td>2</td>
<td>9/54</td>
<td>21/54</td>
<td>0.43</td>
<td>(-0.22, -0.06)</td>
<td>ET: 29 FP: 28</td>
</tr>
<tr>
<td>Pharm vs pharm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LMWH vs UFH 278</td>
<td>1</td>
<td>14/76</td>
<td>24/72</td>
<td>0.55</td>
<td>(-0.15, -0.01)</td>
<td>ET: 32 FP: 48</td>
</tr>
<tr>
<td>Comparison</td>
<td>No. of studies</td>
<td>Intervention</td>
<td>Control</td>
<td>Relative risk</td>
<td>Absolute effect</td>
<td>Forest plots &amp; Evidence tables *</td>
</tr>
<tr>
<td>------------</td>
<td>----------------</td>
<td>--------------</td>
<td>---------</td>
<td>---------------</td>
<td>-----------------</td>
<td>-------------------------------</td>
</tr>
<tr>
<td>Asp (high dose) vs UFH</td>
<td>1</td>
<td>6/35</td>
<td>7/40</td>
<td>0.98 (0.36, 2.64)</td>
<td>0.00 (-0.18, 0.17)</td>
<td>ET: 36 FP: 64</td>
</tr>
<tr>
<td>Double proph vs single</td>
<td>IPCD/FID + GCS vs GCS</td>
<td>2</td>
<td>11/181</td>
<td>17/184</td>
<td>0.65 (a) (0.15, 2.81)</td>
<td>-0.04 (-0.17, 0.09)</td>
</tr>
<tr>
<td>UFH + GCS vs GCS</td>
<td>1</td>
<td>5/120</td>
<td>6/115</td>
<td>0.80 (0.25, 2.54)</td>
<td>-0.01 (-0.06, 0.04)</td>
<td>ET: 27 FP: 141</td>
</tr>
<tr>
<td>Other strategies</td>
<td>LMWH + Asp vs UFH + Asp</td>
<td>1</td>
<td>67/666</td>
<td>118/669</td>
<td>0.57 (0.43, 0.75)</td>
<td>-0.08 (-0.11, -0.04)</td>
</tr>
<tr>
<td>IPCD/FID + GCS vs UFH + GCS</td>
<td>1</td>
<td>8/117</td>
<td>5/120</td>
<td>1.64 (0.55, 4.87)</td>
<td>0.03 (-0.03, 0.08)</td>
<td>ET: 50 FP: 199</td>
</tr>
</tbody>
</table>

* FP – forest plot number in appendix E; ET – evidence table number in appendix D

Proph - prophylaxis

(a) There is substantial statistical heterogeneity between studies for this population (I²=70.3%, χ² on 1 df = 3.37, p= 0.07)

---

**Table 24-2: Pulmonary embolism – summary of results from RCTs**

<table>
<thead>
<tr>
<th>Comparison</th>
<th>No. of studies</th>
<th>Intervention</th>
<th>Control</th>
<th>Relative risk</th>
<th>Absolute effect</th>
<th>Forest plots &amp; Evidence tables *</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proph vs no proph</td>
<td>GCS vs nil</td>
<td>1</td>
<td>13/1256</td>
<td>20/1262</td>
<td>0.65 (0.33,1.31)</td>
<td>-0.01 (-0.01,0.00)</td>
</tr>
<tr>
<td>Asp (high dose) vs nil</td>
<td>1</td>
<td>1/40</td>
<td>0/40</td>
<td>3.00 (0.13,71.51)</td>
<td>0.03 (-0.04. 0.09)</td>
<td>ET: 29 FP: 29</td>
</tr>
<tr>
<td>Pharm vs pharm</td>
<td>LMWH vs nil</td>
<td>1</td>
<td>2/106</td>
<td>4/106</td>
<td>0.50 (0.09, 2.67)</td>
<td>-0.02 (-0.06, 0.03)</td>
</tr>
<tr>
<td>LMWH vs Asp (low dose)</td>
<td>1</td>
<td>4/507</td>
<td>4/491</td>
<td>0.97 (0.24, 3.85)</td>
<td>0.00 (-0.01, 0.01)</td>
<td>ET: 52 FP: 52</td>
</tr>
<tr>
<td>Double proph vs single</td>
<td>IPCD/FID + GCS vs GCS</td>
<td>1</td>
<td>0/64</td>
<td>0/69</td>
<td>Not estimable</td>
<td>0.00 (-0.03, 0.03)</td>
</tr>
<tr>
<td>Other strategies</td>
<td>LMWH + Asp vs UFH + Asp</td>
<td>2</td>
<td>1/938</td>
<td>7/942</td>
<td>0.21 (0.04, 1.21)</td>
<td>-0.01 (-0.01, 0.00)</td>
</tr>
</tbody>
</table>

* FP – forest plot number in appendix E; ET – evidence table number in appendix D

Proph - prophylaxis

---

**Table 24-3: Major bleeding – summary of results from RCTs**

<table>
<thead>
<tr>
<th>Comparison</th>
<th>No. of studies</th>
<th>Intervention</th>
<th>Control</th>
<th>Relative risk</th>
<th>Absolute effect</th>
<th>Forest plots &amp; Evidence tables *</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proph vs no proph</td>
<td>LMWH vs nil</td>
<td>2</td>
<td>8/74</td>
<td>4/77</td>
<td>2.21(a) (0.69, 7.00)</td>
<td>0.06 (-0.08, 0.20)</td>
</tr>
<tr>
<td>UFH vs nil</td>
<td>1</td>
<td>0/35</td>
<td>0/30</td>
<td>Not estimable</td>
<td>0.00 (-0.06, 0.06)</td>
<td>ET: 27 FP: 19</td>
</tr>
</tbody>
</table>
## 24.2.3 Additional information

### 24.2.3.1 All Cause Mortality

Table 24-5: All Cause Mortality – summary of results from RCTs

<table>
<thead>
<tr>
<th>Comparison</th>
<th>No. of studies</th>
<th>Intervention</th>
<th>Control</th>
<th>Relative risk</th>
<th>Absolute effect</th>
<th>Forest plots &amp; Evidence tables</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Proph vs no proph</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GCS vs nil 158</td>
<td>1</td>
<td>122/1256</td>
<td>110/1262</td>
<td>1.11 (0.87, 1.42)</td>
<td>0.01 (-0.01, 0.03)</td>
<td>Et: 23 FP: 3</td>
</tr>
<tr>
<td>LMWH vs nil 240,540,581</td>
<td>3</td>
<td>15/102</td>
<td>5/111</td>
<td>2.75 (1.11, 6.83)</td>
<td>0.08 (0.01, 0.14)</td>
<td>Et: 26 FP: 16</td>
</tr>
<tr>
<td>UFH vs nil 167,434,435,520</td>
<td>4</td>
<td>44/235</td>
<td>64/247</td>
<td>0.83 (0.44, 1.55)</td>
<td>-0.04 (-0.14, 0.06)</td>
<td>Et: 27 FP: 20</td>
</tr>
<tr>
<td>Asp (high dose) vs nil 520</td>
<td>2</td>
<td>7/40</td>
<td>5/40</td>
<td>1.40 (0.48, 4.04)</td>
<td>0.05 (-0.11, 0.21)</td>
<td>Et: 29 FP: 31</td>
</tr>
<tr>
<td><strong>Pharm vs pharm</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LMWH vs UFH 278</td>
<td>1</td>
<td>9/106</td>
<td>8/106</td>
<td>1.13 (0.45, 2.80)</td>
<td>0.01 (-0.06, 0.08)</td>
<td>Et: 32 FP: 51</td>
</tr>
<tr>
<td>Asp (high dose) vs UFH 520</td>
<td>1</td>
<td>7/40</td>
<td>10/40</td>
<td>0.70 (0.30, 1.66)</td>
<td>-0.08 (-0.25, 0.10)</td>
<td>Et: 36 FP: 67</td>
</tr>
<tr>
<td><strong>Double proph vs single</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IPCD/FID + GCS vs GCS 369,509</td>
<td>2</td>
<td>15/191</td>
<td>24/192</td>
<td>0.65 (0.37, 1.14)</td>
<td>-0.05 (-0.41, 0.30)</td>
<td>Et: 39 FP: 118</td>
</tr>
<tr>
<td>UFH + GCS vs GCS 509</td>
<td>1</td>
<td>0/120</td>
<td>0/115</td>
<td>Not Estimable</td>
<td>0.00 (-0.02, 0.02)</td>
<td>Et: 27 FP: 144</td>
</tr>
<tr>
<td><strong>Other strategies</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LMWH + Asp vs UFH + Asp 165,598</td>
<td>2</td>
<td>69/1149</td>
<td>60/1145</td>
<td>1.15 (0.82, 1.61)</td>
<td>0.01 (-0.01, 0.03)</td>
<td>Et: 45 FP: 186</td>
</tr>
<tr>
<td>IPCD/FID + GCS vs UFH + GCS 509</td>
<td>1</td>
<td>0/114</td>
<td>0/120</td>
<td>Not Estimable</td>
<td>0.00 (-0.02, 0.02)</td>
<td>Et: 50 FP: 200</td>
</tr>
</tbody>
</table>

*FP – forest plot number in appendix E; ET – evidence table number in appendix D
Proph - prophylaxis

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There is some evidence of statistical heterogeneity (I²=36.3%, χ² on 1df = 1.57, p = 0.21)
24.2.3.2 Additional Outcomes

None of the included studies reported chronic thromboembolic pulmonary hypertension, heparin induced thrombocytopenia or post thrombotic syndrome as outcomes.

The CLOTS study\(^{158}\) reported cutaneous adverse events related to the use of GCS, ie skin breaks, ulcers, blisters or skin necrosis. The event rate in the GCS arm was 64/1256 vs 16/1262 in the control arm without GCS (RR 4.02 95% CI 2.31 to 6.91, \(p<0.001\)). Lower limb ischaemia or amputation was 7/1249 in the GCS arm and 2/1262 in the control arm (RR 3.54 95% CI 0.74 to 16.99, \(p=0.108\)).

24.3 Network meta-analysis results

No network meta-analysis was completed for this population.

24.4 Cost-effectiveness evidence

No cost effectiveness analysis was completed for this population.

24.5 Patient views

No studies investigating the experience of prophylaxis in stroke patients were found. Section 6.6 contains more information on patient views about specific prophylaxis agents.

24.6 Summary of evidence

<table>
<thead>
<tr>
<th>Intervention(s)</th>
<th>Comparison(s)</th>
<th>Intervention favoured</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>DVT</td>
</tr>
<tr>
<td>Prophylaxis vs no prophylaxis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IPCD/FID</td>
<td>No prophylaxis</td>
<td>Not sig</td>
</tr>
<tr>
<td>GCS</td>
<td>No prophylaxis</td>
<td>Not sig</td>
</tr>
<tr>
<td>Aspirin (high dose)</td>
<td>no prophylaxis</td>
<td>Asp (HD)</td>
</tr>
<tr>
<td>UFH</td>
<td>No prophylaxis</td>
<td>UFH</td>
</tr>
<tr>
<td>LMWH</td>
<td>No prophylaxis</td>
<td>Not sig</td>
</tr>
</tbody>
</table>

**Single prophylaxis vs single**

<p>| | | | | |</p>
<table>
<thead>
<tr>
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<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>LMWH</td>
<td>UFH</td>
<td>LMWH</td>
<td>Not sig</td>
<td>Not sig</td>
</tr>
<tr>
<td>Aspirin (High dose)</td>
<td>UFH</td>
<td>Not sig</td>
<td>Not sig</td>
<td>Not sig</td>
</tr>
</tbody>
</table>

**Double prophylaxis vs single**

<p>| | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>IPCD/FID + GCS</td>
<td>GCS</td>
<td>Not sig</td>
<td>No events</td>
<td>-</td>
</tr>
<tr>
<td>UFH + GCS</td>
<td>GCS</td>
<td>Not sig</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

**Other Strategies**

<p>| | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>LMWH + Asp</td>
<td>UFH + Asp</td>
<td>LMWH + Asp</td>
<td>Not sig</td>
<td>Not sig</td>
</tr>
<tr>
<td>IPCD/FID + GCS</td>
<td>UFH + GCS</td>
<td>Not sig</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

**Cost Effectiveness**

No cost effectiveness model was completed for this population.

The prophylaxis strategy which is significantly more effective in reducing DVT or PE; or resulting in significantly less major bleeding is stated in bold.
24.7 Recommendations and link to evidence

**Recommendation**

Do not offer anti-embolism stockings to stroke patients for VTE prophylaxis.

**Relative values of different outcomes**

The outcomes identified as important by the Guideline Development Group were thromboembolic events (asymptomatic and symptomatic DVT, symptomatic pulmonary embolism and fatal pulmonary embolism), bleeding events (major bleeding, fatal bleeding and stroke) and other long term events occurring as a result of VTE (chronic thromboembolic pulmonary hypertension and post thrombotic syndrome).

**Trade off between clinical benefit and harms**

Unlike pharmacological prophylaxis, mechanical methods do not increase the risk of bleeding. However, GCS have been shown to be ineffective in reducing the risk of VTE in stroke patients and were associated with an increased risk of cutaneous adverse reactions.

**Economic considerations**

No economic model was run specifically for stroke patients. GCS were found to be ineffective in reducing VTE in stroke patients and had cutaneous side effects; this is therefore not a cost-effective intervention for this population.

**Quality of Evidence**

All included RCTs were either individually critically appraised to be of a high quality (level 1+ or level 1++) or came from systematic reviews of RCTs which had been critically appraised to be of a high quality (level 1+ or level 1++).

One large multicentred RCT (CLOTS study) with more than 2500 patients compared GCS against usual care in stroke patients. This study has some minor limitations (Evidence table 23, Appendix D) but the GDG agreed that the generality of the results could be applied to the stroke population.

**Other considerations**

The CLOTS study showed that GCS are ineffective in reducing the risk of VTE in stroke patients, but are associated with an increased risk of cutaneous adverse events. This contradicts previous beliefs based on the extrapolation of efficacy observed in surgical patients that GCS may be effective at reducing VTE and challenged the notion that mechanical prophylaxis methods are harmless.

One small study (n=26) investigated the use of IPCD compared with no prophylaxis in stroke patients. There was no statistically significant difference in DVT. The GDG did not feel that the evidence was strong enough to recommend for or against the use of IPCD/FID for patients after stroke. The GDG
are aware that further research into the use of IPCD devices is currently being conducted.

In the CLOTS study, the number of patients who were using aspirin during the study was not reported but is expected to be high as currently aspirin is the standard treatment for most patients with ischaemic stroke. The GDG had considered whether this could have reduced observed efficacy of stockings, but concluded that the results of the study were still applicable as the current NICE guidelines recommend initial treatment with aspirin for ischaemic stroke\(^7\).

No patient views evidence was found specifically for this population.

<table>
<thead>
<tr>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consider offering prophylactic-dose LMWH* to patients in whom a diagnosis of haemorrhagic stroke has been excluded, the risk of bleeding (haemorrhagic transformation of stroke or bleeding into another site) is assessed to be low, and who have one or more of the following:</td>
</tr>
<tr>
<td>• major restriction of mobility</td>
</tr>
<tr>
<td>• previous history of VTE</td>
</tr>
<tr>
<td>• dehydration or comorbidities (such as malignant disease).</td>
</tr>
<tr>
<td>Continue LMWH until the acute event is over and the patient's condition is stable</td>
</tr>
</tbody>
</table>

*Some types of LMWH are not licensed for VTE prophylaxis in medical patients. Prescribers should consult the summary of product characteristics for the individual LMWH.

<table>
<thead>
<tr>
<th>Relative Values of different outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>The outcomes identified as important by the Guideline Development Group were thromboembolic events (asymptomatic and symptomatic DVT, symptomatic pulmonary embolism and fatal pulmonary embolism), bleeding events (major bleeding, fatal bleeding and stroke) and other long term events occurring as a result of VTE (chronic thromboembolic pulmonary hypertension and post thrombotic syndrome). The risk of bleeding was felt to be important in this population as ischaemic stroke patients have a risk of haemorrhagic transformation.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Trade off between clinical benefit and harms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients are likely to be relatively immobile after stroke and therefore may be predisposed to an increased risk of VTE. However, the Guideline Development Group felt that this should be balanced against the risk of bleeding, including haemorrhagic transformation which can have very serious consequences. In addition, the risk of bleeding on admission</td>
</tr>
</tbody>
</table>
may not be known and so caution should be applied before prescribing pharmacological thromboprophylaxis agents.

**Economic considerations**

No economic model was run specifically for stroke patients. The economic model for general medical patients indicated that pharmacological prophylaxis was cost effective for this broader population. Given the high risk of VTE in stroke patients, it is possible that prophylaxis is cost-effective. However, given that the consequences of bleeding are likely to be very serious for this group, drug prophylaxis is likely only to be cost-effective if the risk of intracranial bleeding is minimised. Therefore the guideline development group recommended only considering pharmacological prophylaxis to a subset of stroke patients who have been established as at increased risk for VTE and only those in whom the bleeding risks have been established as low.

**Quality of Evidence**

All included RCTs were either individually critically appraised to be of a high quality (level 1+ or level 1++) or came from systematic reviews of RCTs which had been critically appraised to be of a high quality (level 1+ or level 1++). Seven of the 17 studies (41.2%) were published prior to 2000. The treatment of stroke has changed over the last 10 years. The current NICE guidelines recommend initial treatment with aspirin for ischaemic stroke, which should not be discontinued in order to provide thromboprophylaxis. There were only two studies which compared heparin prophylaxis in addition to aspirin treatment and so the remaining studies may not be directly applicable to the current stroke population.

**Other considerations**

The current NICE guidelines recommend initial treatment with aspirin for ischaemic stroke, which should not be discontinued in order to provide thromboprophylaxis. The three conditions identified within the recommendation (major restriction of mobility, previous history of VTE, and dehydration or medical comorbidity) are taken from the stroke guideline. Adding prophylactic-dose anticoagulant agents to aspirin is likely to increase bleeding and so it is important that the bleeding risk is established as low before thromboprophylaxis is commenced.

The Department of Health has published National Stroke strategy.

### 24.7.1 Other recommendations of relevance

The specific recommendations for patients with stroke in this chapter should be read in conjunction with other relevant recommendations presented elsewhere in the guideline. These are:
• risk assessment for VTE and major bleeding (Section 5.9)
• the use of prophylaxis in general (Section 6.7)
• the provision of patient information (Section 32.5)
• patients using anticoagulants or antiplatelets for reasons other than VTE prophylaxis (Chapter 31)

24.8 Recommendations for research

The GDG recommended the following research question:

 What is the overall risk/benefit of low molecular weight heparin and/or fondaparinux sodium in respect of both stroke outcome and the development of VTE for patients with acute stroke?

Why this is important

Patients with either ischaemic or haemorrhagic stroke have a risk of both VTE and bleeding into the brain. 'Stroke: diagnosis and management of acute stroke and transient attack [TIA]' (NICE clinical guideline 68, published July 2008) recommends the use of aspirin for treatment of ischaemic stroke but does not recommend anticoagulants. There is recent evidence to suggest that prophylactic-doses of anticoagulants in addition to aspirin reduce the risk of VTE in patients with ischaemic stroke but there are no data showing an effect of these anticoagulants on the stroke itself. Do they increase the risk of haemorrhagic transformation and so increase neurological damage? This research should include patients with haemorrhagic or ischaemic strokes to identify which patients would benefit from additional pharmacological prophylaxis.

Recommended Design: RCT

Further details are provided in Appendix F

24.9 Summary of recommendations

 Do not offer anti-embolism stockings to stroke patients for VTE prophylaxis

 Consider offering prophylactic-dose LMWH* to patients in whom a diagnosis of haemorrhagic stroke has been excluded, the risk of bleeding (haemorrhagic transformation of stroke or bleeding into another site) is assessed to be low, and who have one or more of the following:

□ major restriction of mobility
□ previous history of VTE
□ dehydration or comorbidities (such as malignant disease).

Continue LMWH until the acute event is over and the patient’s condition is stable

*Some types of LMWH are not licensed for VTE prophylaxis in medical patients. Prescribers should consult the summary of product characteristics for the individual LMWH.
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