A survey of flexibility training protocols and hamstring strains in professional football clubs in England

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Objectives: To investigate the relation between current flexibility training protocols, including stretching, and hamstring strain rates (HSRs) in English professional football clubs.

Method: Questionnaire based data on flexibility training methods and HSRs were collected from 30 English professional football clubs in the four divisions during the 1998/99 season. Data were coded and analysed using cross tabulation, correlation, and multiple regression.

Results: Flexibility training protocols were characterised by wide variability, with static stretching the most popular stretching technique used. Hamstring strains represented 11% of all injuries and one third of all muscle strains. About 14% of hamstring strains were reinjuries. HSRs were highest in the Premiership (13.3 (9.4)/1000 hours) with the lowest rates in Division 2 (7.8 (2.9)/1000 hours); values are mean (SD). Most (97%) hamstring strains were grade I and II, two thirds of which occurred late during training/matches. Forwards were injured most often. Use of the standard stretching protocol (SSP) was the only factor significantly related to HSR ($r = -0.45, p = 0.031$) in the correlation analysis, suggesting that the more SSP is used, the lower the HSR. About 80% of HSR variability was accounted for by stretching holding time (SHT), SSP, and stretching technique (STE) in the multiple regression equation: $\text{HSR} = 37.79 - (0.33\text{SHT} - 10.05\text{SSP} + 2.24\text{STE}) / 2.34$. SHT (negatively correlated with HSR) was the single highest predictor, and accounted for 30% of HSR variability, and an additional 40% in combination with SSP.

Conclusions: Flexibility training protocols in the professional clubs were variable and appeared to depend on staffing expertise. Hamstring stretching was the most important training factor associated with HSR. The use of SSP, STE, and SHT are probably involved in a complex synergism which may reduce hamstring strains. Modification of current training patterns, especially stretching protocols, may reduce HSRs in professional footballers.

Muscle strains are common in sport and represent 41% of all injuries reported in the English professional football clubs. Of all the muscle strains associated with competitive sport, hamstring strains are the most common and problematic. Hamstring strain was reported to be the most common injury in Australian Rules Football, accounting for 13% of injuries, and is responsible for the most time missed after injury. The peculiar characteristics of the hamstring muscles—biarticular, made predominantly of type II fibres, and containing less titin protein—may put the muscle group at higher risk of strains. Hamstring strains are often difficult and slow to rehabilitate, and the problem is compounded by the high hamstring re-injury rate.

The causes of hamstring strain are complicated and multifactorial, involving muscle strength imbalance, inadequate warm up, lack of flexibility, muscle fatigue, and previous strain/inadequate rehabilitation. Extremes of flexibility have been associated with injury, and one specific area of attention is the impact of suboptimal flexibility on hamstring injury. The role of stretching in enhancing flexibility and reducing injury remains contentious. Methodological inconsistencies as well as varied subject populations and research designs have often limited such research. In fact, most studies that have indicated a relation between stretching and injury were retrospective, with questionable exposure and in some cases no statistical tests performed. Other studies have found no relation between flexibility training and injury.

On the other hand, inappropriate stretching interacting with other factors has been suggested to be partially responsible for injury. This is supported by the fact that studies that showed a positive relation between stretching and injury were those that used multiple interventions. As a consequence of such data, guidelines for stretching and flexibility training have been proposed, including the concept of three to five stretching sessions a week, with four to five repetitions per hamstring muscle group and a holding time of 15–30 seconds. These approaches significantly increased flexibility with only minimal additional benefits when holding times were extended for 1–2 minutes. The choice of the ideal stretching techniques to achieve such increases in flexibility and potentially reduce injury is contentious. Static stretching has been advocated in combination with proprioceptive neuromuscular facilitation (PNF), whereas ballistic stretching is considered potentially dangerous. In addition, optimal fitness and strength training including eccentric hamstring strength training have been advocated, as well as stretching with the anterior pelvic tilt position.

Given the contentious nature of research that has assessed the link between hamstring flexibility and injury and the broad range of stretching techniques and strategies advocated in the scientific and popular literature, it is somewhat surprising that there is a relative lack of literature on stretching and flexibility training that occurs in professional sport. Few studies have reported the kind of flexibility training used by athletes in general, and little or no descriptive research exists on the possible link between...
flexibility training protocols and injury rates in professional footballers. The purpose of this study therefore was to determine the current protocols of flexibility training, including stretching protocols, in the English professional football clubs and whether any relation exists with hamstring strain rates (HSRs).

METHODS
This study was conducted as a follow up to one involving 30 professional football clubs. However, only 20 of the original clubs responded, hence a further 16 clubs selected randomly from all four divisions were invited to participate. Of these, 10 clubs responded. In all, 30 out of 46 clubs took part in this study, eight from the Premiership, 10 from Division 1, and six each from Divisions 2 and 3. Team doctors, physiotherapists, managers/coaches, and fitness trainers who work with the first teams of their respective clubs provided information based on the 1998/99 soccer season. Self administered questionnaires with a mixture of open ended and closed questions were mailed to staff of clubs who agreed to participate in the study. The questionnaire was designed with the help of the Division of Public Health Medicine and Epidemiology (University of Nottingham), and was based on a review of the relevant literature in the area of study. The questionnaire contained clear definitions of words such as injury and reinjury, and classification of hamstring strains, etc to facilitate completion. It also provided guidelines for completion of specific sections by the most appropriate support staff.

Pertinent areas of interest in relation to the study included: staffing complement, demands of the football year, fitness training procedures, warm up and warm down procedures, flexibility training procedures, and injury information. In this study, injury refers to “a physical impairment received during a competitive match or training session which prevented a player from being available for selection for the next competitive game”. Hamstring reinjury in this survey refers to a second injury occurring at the same site during the 1998/99 football season. Hamstring strains were classified as grade I (minor injury with normal, but painful contraction), II (moderate, partial tear with abnormal contraction), and III (complete tear with weak to non-existent contraction) and diagnosed by the team doctors. Clubs that took part in this survey used on average two doctors working full time or part time. About 45% of these doctors have MSc/Diploma in Physiotherapy as qualification, the others being orthopaedic surgeons (14%), general practitioners (17%), and osteopaths (24%). On average, the head doctor of the clubs had experience in sports medicine ranging from 6.5 years (Division 3) to 9.8 years (Premiership). The timing of hamstring strain incidence was also noted as early, mid, and late onset during the first, middle, or last third of the season and expressed per 1000 hours. The data were screened for variance of individual data, including tests for skewness and kurtosis. Four reports on frequency of hamstring stretching from three Division 1 clubs and one Division 3 club were considered outliers and excluded from the analysis. The distribution of hamstring strains, muscle strains, and total injuries were cross tabulated, and the relation with training practices assessed using Pearson’s correlation and then stepwise multiple regression. One-way analysis of variance was used to determine the main and interaction effects of stretching factors on HSR after the tests of association. $p<0.05$ was considered significant (SPSS).

RESULTS
Thirty clubs returned the study questionnaires, which consisted of six sections; however, only 19 clubs completed all six sections. Section 6 of the questionnaire had a series of questions on injury information, the completion of which required retrieving stored information. Therefore the ability to complete this section depended on record keeping and storage of information. Thus section 6 was the least fully completed section, with 23 respondents. Although seven clubs failed to complete section 6 fully, the total number of responses about the incidence of hamstring strains for example was 27. Overall, the total number of responses declined for questions that demanded details of
distribution/timing of hamstring strains or subclassification of specific training programmes. This led to differential totals included in the respective analyses conducted: 21 responses for flexibility training compared with 27 for hamstring strains or 30 for number of physiotherapists employed. Again, although the number of responses about hamstring strains was 27, data on hamstring strain rates/1000 hours could only be calculated for 23 clubs because some clubs failed to provide information on their training duration.

**Staffing complement**

Clubs in the Premiership employed more support staff than all other divisions (table 1). Most Premiership clubs employed at least two doctors compared with one for all other divisions. Most clubs in all divisions employed at least two physiotherapists, with the widest range in the Premiership.2–7 Sports scientists and fitness trainers were only sporadically employed, with at least one sports scientist exclusively employed in the Premiership (table 1). The number of players employed in the professional football league increased from Division 2 and 3 clubs to a maximum in the Premiership, which probably accounted for the additional support staff needed.

**Training patterns**

The training modalities used by the professional clubs included endurance training, strength/resistance training, and flexibility training. The training patterns were characterised by very wide inter/intradivision variability (table 2). The pattern of training modalities could not be reported by some of the clubs (table 2), explaining the differences in response numbers. However, on average, clubs in Divisions 1 and 2 seemed to devote more time per week to endurance training, whereas the Premiership clubs devoted slightly less time, and Division 3 clubs devoted the least time.

Strength/resistance training was also characterised by wide inter/intradivision variability, although half of Division 2 and 3 clubs failed to report, and two Premiership clubs and one Division 1 club also failed to report. The Premiership spent more time on strength/resistance training, whereas only about half this time was devoted by Division 3 clubs, with clubs in Division 1 and 2 spending about two thirds of this time on strength/resistance training.

Unlike with endurance and strength/resistance training where some responses were similar, flexibility training exhibited distinct patterns for each division. The most time spent on flexibility training per week was by Division 1 clubs, followed by the Premiership clubs, then Division 2, and the least by Division 3. This pattern in flexibility training was similar to the trend in total training times for all three modalities.

The Premiership clubs devoted almost 40% of total training time to flexibility training, with about 30% on endurance and 30% on strength/resistance training. Division 3 clubs also exhibited similar training protocol distributions. In Division 1, nearly 50% of training time was devoted to flexibility training, with about 30% and 20% to endurance and strength/resistance training respectively. Division 2 clubs used the least flexibility training, representing a third of total training time a week, while almost 40% of time was used for endurance training.

**Warm up and warm down procedures**

All clubs reported using warm up protocols before training sessions and matches. Table 3 summarises the characteristics of warm up and warm down protocols. Whereas almost two thirds of all clubs used mainly active warm up, over two thirds of Division 2 clubs used both active and passive warm up protocols. In addition, five out of six Division 2 clubs and seven out of 10 Division 1 clubs reported using warm down protocols both after training and after matches. In the Premiership and Division 3 clubs, the use of warm down protocols after a match fell to half (four out of eight Premiership clubs) and a third (two out of six Division 3 clubs) respectively compared with its use after training. There were no differences in the duration of warm up use across all divisions, with warm ups lasting 22–25 minutes, although Division 2 clubs spent less time on warm down compared with the other divisions. Overall Division 3 clubs used warm down protocols the least, and Division 2 clubs the most. Stretching before warm up was not popular in all divisions except Division 2 where half (three out of six) used stretching before warming up. However, seven out of eight Premiership clubs stretched both during and after warm up, with seven out of 10 Division 1 and four out of six Division 3 clubs doing likewise.

**Hamstring stretching**

Static stretching was reported as the most popular stretching technique used among all clubs in this study. In most cases,

### Table 1  Staffing complement employed by professional football clubs in England

<table>
<thead>
<tr>
<th>Staffing Type</th>
<th>Premiership (N = 8)</th>
<th>Division 1 (N = 10)</th>
<th>Division 2 (N = 6)</th>
<th>Division 3 (N = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Doctors</td>
<td>2 (1–5)</td>
<td>1 (1–3)</td>
<td>1 (1–4)</td>
<td>1 (1–4)</td>
</tr>
<tr>
<td>Physiotherapists</td>
<td>2 (2–7)</td>
<td>2 (1–3)</td>
<td>2 (1–3)</td>
<td>2 (1–2)</td>
</tr>
<tr>
<td>Fitness trainers</td>
<td>1 (0–2)</td>
<td>0 (0–1)</td>
<td>1 (0–2)</td>
<td>0 (0–3)</td>
</tr>
<tr>
<td>Sports scientists</td>
<td>1 (1–3)</td>
<td>0 (0–1)</td>
<td>0 (0–1)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Football players</td>
<td>35 (31–37)</td>
<td>28 (22–40)</td>
<td>20 (20–35)</td>
<td>20 (20–26)</td>
</tr>
</tbody>
</table>

Values are staff numbers (range).

### Table 2  Distribution of weekly training in professional football clubs

<table>
<thead>
<tr>
<th>Training modality</th>
<th>Premiership (N = 8)</th>
<th>Division 1 (N = 10)</th>
<th>Division 2 (N = 6)</th>
<th>Division 3 (N = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endurance</td>
<td>605 (516) (n = 5)</td>
<td>678 (439) (n = 9)</td>
<td>654 (373) (n = 5)</td>
<td>352 (289) (n = 3)</td>
</tr>
<tr>
<td>Strength/resistance</td>
<td>614 (172) (n = 7)</td>
<td>403 (204) (n = 9)</td>
<td>440 (165) (n = 3)</td>
<td>317 (274) (n = 3)</td>
</tr>
<tr>
<td>Flexibility</td>
<td>735 (458) (n = 7)</td>
<td>978 (1084) (n = 8)</td>
<td>537 (234) (n = 3)</td>
<td>421 (239) (n = 3)</td>
</tr>
<tr>
<td>Total</td>
<td>1954 (814) (n = 5)</td>
<td>2058 (1134) (n = 8)</td>
<td>1631 (202) (n = 3)</td>
<td>1090 (529) (n = 3)</td>
</tr>
</tbody>
</table>

Proportion of flexibility training (%): 37.6 47.5 32.9 38.6

Values are mean [SD] (minutes/week). N represents the number of clubs in a particular division participating in the study. n represents the number of clubs that responded to a particular mode of training.
static stretching was used in combination with PNF stretching (table 4). Most clubs in all divisions used an SSP. However, Division 2 (four out of six) appear to have a lower tendency to adhere to the protocol at all/most sessions whereas all Premiership and Division 1 and Division 3 (five out of six) clubs adhered more strictly to stretching protocols. Hamstring SHTs exhibited wide variability, with the least in Division 3 clubs. The longest SHT reported was about 30 seconds by the Premiership clubs, whereas players in Division 3 clubs stretched for only about half this time, with Division 1 and 2 clubs holding stretches for about 20 seconds. Players in the Premiership and Division 2 in Division 2 on average stretched with fewer repetitions per session than those in Divisions 1 and 3. These reports on HSF exclude reports from three Division 1 clubs and one Division 3 club which reported HSFs of 10, 17, 20, and 25, which were considered unrealistic, hence outliers.

### Distribution of muscle and hamstring strains

During the 1998/99 football season, a total of 1435 injuries were recorded in all 30 clubs that took part in the study, of which 479 were muscle strains and 158 hamstring strains. Hamstring strains thus represented 11% of all injuries and muscle strains 33%. Overall the highest total number of injury and muscle strains were in Division 3 closely followed by the Premiership and Divisions 1 and 2 (table 5). However, HSRs were highest in the Premiership, followed by Divisions 3, 1, and 2 respectively.

Out of the 158 hamstring strains recorded, 156 were classified into grades; 151 were of grade I and II severity, and about 14% of these were reinjuries. The Premiership recorded the highest numbers of both grade II and III injuries (27 and 3 respectively) compared with 13 and 0 respectively for Division 2, while Divisions 1 and 3 recorded 25 and 1, and 9 and 1 respectively. In the Premiership, four out of 46 hamstring strains were injuries, as were seven out of 56 in Division 1, three out of 23 in Division 2, and seven out of 33 in Division 3. Thus hamstring reinjury rates were lowest in the Premiership (9%) and highest in Division 3 (21%). Forwards had the highest risk, sustaining 49 out of 122 hamstring strains, whereas goalkeepers were at very low risk (three out of 122). Defenders (36 out of 122) and midfielders (34 out of 122) had similar risks. Thus, compared with goalkeepers, the incidence ratio of hamstring strain in the playing positions was 16:1 for forwards, 12:1 for defenders, and 11:1 for midfielders. About two thirds of hamstring strains occurred during matches (77 of 122) compared with training (45 of 122). Similarly two thirds occurred late during activity, whether training (25 of 45) or in a match (49 of 77). The numbers of strains occurring early in and midway through training were the same (10 of 45), whereas the corresponding numbers in matches were 12 and 16 out of 77. Almost all hamstring strains were managed conservatively, with only 1.6% (three of 158) being managed surgically.

### HSRs and training factors

The use of an SSP was the only training factor that correlated significantly and negatively with HSR (r = −0.53, p = 0.01; table 6), suggesting that the use of SSPs is associated with lower HSR.

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**Table 3** Characteristics of warm up and warm down procedures used by the professional football clubs

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Premiership (N = 8)</th>
<th>Division 1 (N = 10)</th>
<th>Division 2 (N = 6)</th>
<th>Division 3 (N = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active*</td>
<td>5 (n = 6)</td>
<td>6 (n = 9)</td>
<td>2 (n = 6)</td>
<td>4 (n = 5)</td>
</tr>
<tr>
<td>Passive†</td>
<td>1 (n = 6)</td>
<td>0 (n = 9)</td>
<td>0 (n = 6)</td>
<td>0 (n = 5)</td>
</tr>
<tr>
<td>Active and passive</td>
<td>2 (n = 7)</td>
<td>4 (n = 6)</td>
<td>4 (n = 5)</td>
<td>2 (n = 5)</td>
</tr>
<tr>
<td>Stretching before warm up</td>
<td>1 (n = 6)</td>
<td>3 (n = 9)</td>
<td>3 (n = 6)</td>
<td>2 (n = 5)</td>
</tr>
<tr>
<td>Stretching during/after warm up</td>
<td>7 (n = 6)</td>
<td>7 (n = 9)</td>
<td>3 (n = 6)</td>
<td>4 (n = 5)</td>
</tr>
<tr>
<td>Duration of warm up (minutes)</td>
<td>23.1 (4.4)</td>
<td>24.9 (8.3)</td>
<td>22.5 (4.2)</td>
<td>25.8 (4.1)</td>
</tr>
<tr>
<td>Duration of warm down (minutes)</td>
<td>17.2 (11.5)</td>
<td>14.4 (12.7)</td>
<td>10.1 (3.6)</td>
<td>14.6 (3.7)</td>
</tr>
<tr>
<td>Regular use of warm down protocol (after training)</td>
<td>6 (n = 6)</td>
<td>7 (n = 9)</td>
<td>5 (n = 6)</td>
<td>3 (n = 5)</td>
</tr>
<tr>
<td>Regular use of warm down protocol (after match)</td>
<td>4 (n = 6)</td>
<td>7 (n = 9)</td>
<td>5 (n = 6)</td>
<td>2 (n = 5)</td>
</tr>
</tbody>
</table>

Where applicable, values are mean (SD).

*Active warm up involves gaining body temperature through one’s own physical activity. †Passive warm up involves gaining body temperature through the use of an external heat source (application of hot material or use of additional clothing to retain heat).

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**Table 4** Characteristics of hamstring stretching in the professional football clubs

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Premiership (N = 8)</th>
<th>Division 1 (N = 10)</th>
<th>Division 2 (N = 6)</th>
<th>Division 3 (N = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Use of SSP</td>
<td>6 (n = 6)</td>
<td>9 (n = 9)</td>
<td>5 (n = 6)</td>
<td>4 (n = 5)</td>
</tr>
<tr>
<td>Number of clubs strictly adhering to SSP at most/all sessions</td>
<td>6 (n = 6)</td>
<td>9 (n = 9)</td>
<td>5 (n = 6)</td>
<td>4 (n = 5)</td>
</tr>
<tr>
<td>SHT (s)*</td>
<td>31.3 (14.6)</td>
<td>23.0 (7.90)</td>
<td>21.7 (11.3)</td>
<td>13.3 (5.0)</td>
</tr>
<tr>
<td>HSF per session†</td>
<td>3 (2–5) (n = 7)</td>
<td>5 (2–10) (n = 6)</td>
<td>3 (2–6) (n = 5)</td>
<td>5 (3–6) (n = 4)</td>
</tr>
</tbody>
</table>

N represents the number of clubs in a particular division participating in the study.

r represents the number of clubs that responded to the questionnaire on the stated component of stretching.

*Values are mean (SD).

†Values are mean (range).

HSF, hamstring stretching frequency or repetitions per session; PNF, proprioceptive neuromuscular facilitation; SHT, static stretching holding time; SSP, standard stretching protocol; STE, stretching technique employed.
In addition, there was a significant interactive effect between SHT and STE (analysis of variance; $F = 7.1, p = 0.04$) without significant individual main effects (SHT: $F = 2.8, p = 0.71$; STE: $F = 1.7, p = 0.3$) (table 7).

**Prediction of HSR**

Stepwise multiple regression analysis using the specific training factors as independent variables indicated that stretching was the most important factor associated with HSR (table 8). SHT was the single most important predictor, accounting for nearly 30% of variability of HSR ($r^2 = 0.29$). SSP in combination with SHT adds 40% to the variability of training ($r^2 = 0.69$). Almost 80% of the variability of HSR was accounted for by three independent variables, SHT, the use of SSP, and STE (table 8). The multiple regression equation of hamstring strain is:

$$
\text{HSR} = 37.79 - (0.33 \times \text{SHT} + 10.05 \times \text{SSP} + 2.24 \times \text{STE}) + 2.34
$$

**DISCUSSION**

The flexibility training protocols of 30 English professional football clubs during the 1998/99 season were studied, with the aim of identifying any training factors that may be associated with HSR. There were no differences in training protocols within or between divisions that could be attributed to the fact that people of different backgrounds and experience are recruited to manage and supervise training. Moreover, the desired impact of sports scientists/fitness trainers is probably not being achieved because of the low numbers of responses that responded to the questionnaire on the stated component of injury. HSR, Hamstring strain rate; MSR, muscle strain rate; TIR, total injury rate.

**Table 5** Distribution of injury rates in the professional football clubs

<table>
<thead>
<tr>
<th>Source</th>
<th>Type I sum of squares</th>
<th>df</th>
<th>Mean square</th>
<th>F</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>1509.665</td>
<td>1</td>
<td>1509.665</td>
<td>136.588</td>
<td>0.000</td>
</tr>
<tr>
<td>HSF</td>
<td>8.171</td>
<td>2</td>
<td>4.085</td>
<td>0.370</td>
<td>0.712</td>
</tr>
<tr>
<td>SHT</td>
<td>92.945</td>
<td>3</td>
<td>30.982</td>
<td>2.803</td>
<td>0.072</td>
</tr>
<tr>
<td>STE</td>
<td>56.482</td>
<td>3</td>
<td>18.827</td>
<td>1.703</td>
<td>0.230</td>
</tr>
<tr>
<td>HSF+ SHT</td>
<td>0.000</td>
<td>0</td>
<td></td>
<td>0.000</td>
<td>1.000</td>
</tr>
<tr>
<td>HSF+ STE</td>
<td>0.000</td>
<td>0</td>
<td></td>
<td>0.000</td>
<td>1.000</td>
</tr>
<tr>
<td>SHT+ STE</td>
<td>235.756</td>
<td>3</td>
<td>78.585</td>
<td>7.110</td>
<td>0.044</td>
</tr>
<tr>
<td>HSF+ SHT+ STE</td>
<td>0.000</td>
<td>0</td>
<td></td>
<td>0.000</td>
<td>1.000</td>
</tr>
<tr>
<td>Error</td>
<td>44.211</td>
<td>4</td>
<td>11.053</td>
<td>1.000</td>
<td>0.474</td>
</tr>
<tr>
<td>Total</td>
<td>2692.283</td>
<td>22</td>
<td></td>
<td></td>
<td>1.000</td>
</tr>
<tr>
<td>Corrected total</td>
<td>622.954</td>
<td>21</td>
<td></td>
<td></td>
<td>0.357</td>
</tr>
</tbody>
</table>

Analysis of variance; dependent variable hamstring strain rate. $R^2 = 0.292$ (adjusted $R^2 = 0.267$).

HSR, Hamstring stretching frequency (repetitions per session); SHT, stretching holding time; SRT, strength/resistance training.

**Table 8** Model summary for multiple regression for hamstring strain rate

<table>
<thead>
<tr>
<th>Model variables included</th>
<th>$R$</th>
<th>$R^2$</th>
<th>SE Est</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.54</td>
<td>0.29</td>
<td>3.97</td>
</tr>
<tr>
<td>2</td>
<td>0.83</td>
<td>0.69</td>
<td>2.74</td>
</tr>
<tr>
<td>3</td>
<td>0.89</td>
<td>0.79</td>
<td>2.34</td>
</tr>
</tbody>
</table>

The model components include: 1. One predictor: (constant); hamstring stretching holding time (SHT) (seconds). 2. Two predictors: (constant), SHT (seconds) and use of standard stretching protocol (SSP). 3. Three predictors: (constant), SHT (seconds), SSP, and stretching technique employed (STE). (1) SHT indicates the extent to which the stretching holding time used influences the tendency for injury prevention; (2) SSP indicates the extent to which the use of standard stretching protocols used influences the tendency for injury prevention; (3) STE indicates which stretching techniques employed influence the tendency for injury prevention.
15–30 seconds is also advocated.20 21 Up to four repetitions per stretching session is thought to be adequate.22 In a more recent study,23 15 seconds holding time was found to be more effective in enhancing active flexibility but not passive flexibility compared with five seconds. These findings suggest that gains in flexibility may be linearly related to SHTs up to 30 seconds. In our study, but for the wide intra and inter division variability, SHTs would otherwise be within acceptable ranges. Consistency in the use of such SHTs in protocols using the sound techniques may hold the key for benefits of stretching in injury prevention. Arguments based on the basic scientific evidence that stretching could cause injury have been suggested,23 appear laudable, and have not been disputed. However, if investigations into stretching fail to consider consistent use of appropriate STEs and SHTs and their interaction with other training factors, the results are unlikely to reveal any relation with injury. Authors of such studies are likely to report conflicting findings, and this raises concern about experimental designs, whether randomised clinical trials or cohort, and their interpretations.23 In a previous review article,23 the only studies showing a link between HSR and stretching were those that used multiple intervention. In the present study, the simple relation between HSR and stretching could not be established by correlation analysis, but only by stepwise multiple regression analysis. Moreover SHT and STE showed no single main effects, but rather a significant interaction effect on HSR. This therefore implies that stretching is only beneficial if held for an optimal length of time—for example, 15–30 seconds—as suggested by the literature. These findings indicate that the causes of injury in general, and hamstring strains in particular, are likely to be complex, interactive, and multifactorial24 involving flexibility, strength, warm up, and fatigue. It has been suggested that stretching must therefore interact with other training factors to have an injury preventing effect.24 Our findings clearly suggest that the current stretching practices of professional footballers are not detrimental, and an improvement in the quality and consistency of use of more appropriate stretching may possibly further reduce HSR.

In this study, muscle and hamstring strains accounted for 33% and 11% of all injuries respectively. This compares with another study of four English professional football clubs, in which 41% of injuries were reported as muscle strains,1 and a study of Australian rules football, in which hamstring strains represented 13% of all injuries.4 Hamstring strains occur when strong concentric quadriceps contractions generate forces that the eccentric strength of the hamstrings cannot withstand. They are therefore prevalent in sports requiring sudden bursts of speed.45 46 In the present study, forwards, who “take off” more often with sudden bursts of speed, had the highest relative risk (16:1) with respect to goalkeepers compared with defenders (12:1) and midfielders (11:1). Even though it has been reported that midfielders do more running and are thus more prone to injury,45 46 the literature suggests that high instantaneous speed demands are associated with hamstring strains. Our study supports such a mechanism. In fact, it has been reported that hamstring strains are more common in faster athletes.45 In this survey, goalkeepers had the lowest HSRs. This can be explained by the relatively rare demand for sudden bursts of speed in this position rather than them having the greatest flexibility. Grade III hamstring strains were rare, and most of the few that occurred were in the Premiership. Tiredness has been suggested to contribute to hamstring strains.17 The fact that most hamstring strains in our study occurred late on during activity supports this. In all the 122 hamstring strains that were timed, 74 occurred late during the activity (training or matches), 26 occurred mid-activity, and the remaining 22 occurred during early activity.

In this survey, reinjuries represented 14% of hamstring strains and appeared to increase the lower the division: from 9% in the Premiership to 21% in Division 3. In Australian Rules football, hamstring reinjuries have been reported as 34%, more than double the average rate in all divisions of the football league in England. Apart from individual susceptibility, inadequate rehabilitation44 and premature return to competition have been mentioned as reasons for reinjury. This survey was a follow up to a previous study which investigated the use of physical profiling for guiding return to unrestricted training after injury, and 20 clubs out of the 30 in this survey took part in that study.39 It is therefore not surprising to note the relatively low rates of hamstring reinjury after the increased awareness in benchmarking to guide return to training after injury. Another reason could be the availability of sports scientists and physiotherapists who are responsible for rehabilitation of injured players. This survey shows that the number of physiotherapists and sports scientists decrease from the Premiership to the lower divisions, and this may explain the trend in hamstring reinjury rates.

The major limitation of this study is that not all clubs in the football league took part, which was further compounded by the failure of some clubs to complete certain sections of the questionnaire, particularly those dealing with injury types and classifications. This may simply mean that such fine details of injury information are not routinely recorded. Another limitation is that training protocols were investigated in clubs and not in individual players. A study of individual players and their training practices may produce more realistic results. Similarly, although hamstring strains and the various grades were clearly defined, diagnosis and classification could only be assumed to be correct, given the experience of doctors and physiotherapists employed by the football clubs. However, there may be slight disparities in diagnosis and classification. Despite these limitations, however, the study shows the contribution of stretching to hamstring strains in professional footballers in England, and a modification of current training protocols is suggested. Further research is needed to determine ideal stretching protocols, particularly the most appropriate techniques and holding times for the prevention of hamstring strains.

In conclusion, this study suggests a relation between flexibility training protocols in professional footballers in England and HSR, and that STE and SHT are the most important components of a standard protocol that may have potential for prevention of hamstring strains.

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REFERENCES
34. Eales M. Physical profiling in English professional football and its use to guide return to unrestricted training following injury. Unpublished MSc Sports Medicine Thesis, Centre for Sports Medicine, University of Nottingham, 1999.
Sodium ingestion and the prevention of hyponatraemia during exercise

The study of Twernbold et al. is important for a number of reasons, not all of which may have been emphasised sufficiently by the authors.

Firstly, it confirms that a rate of fluid intake of 1000 ml/h is too high for a group of female runners running at ~ 10 km/h and who would therefore complete a 42 km marathon in about 4.25 hours. As the athletes drank 4 litres and gained 2 kg during the trial, their average rate of weight loss (as opposed to sweat rate) was about 500 ml/h. As not all of the weight lost during exercise is sweat and as much as 1–3 kg of this weight loss may result from fuel and water losses that do not contribute to dehydration, the absolute maximum rate at which these athletes should have ingested fluid during exercise was probably even less than 500 ml/h. This is substantially less than the drinking guidelines of the American College of Sports Medicine and the Gatorade Sports Science Institute, which recommend that athletes should have ingested fluid during exercise at a rate of up to 1200–1800 ml/h. As there is no evidence that gaining weight during exercise improves performance, there is good evidence that athletes who lose no weight or who gain weight during exercise are increasingly likely to (a) have an impaired performance, (b) develop troubling gastrointestinal symptoms, or (c) finish the race with serum sodium concentrations below about 128 mmol/l causing hyponatraemic encephalopathy. It is not immediately clear why the authors chose such high rates of fluid intake in these athletes. Except, perhaps, if they wished to prove the value of sodium ingestion during exercise. I note, for example, that the study was funded by a commercial company that, I am informed, markets a sports drink containing sodium chloride.

For it seems highly probable that if athletes overdrink so that they retain fluid and gain weight, then the extent to which their serum sodium concentration falls will be influenced, albeit to a quite limited extent, by the sodium content of the ingested fluids. This indeed was shown by the results of this study. But whether that finding has relevance to the sodium requirements of athletes who are specifically advised not to overdrink during exercise to ensure that they do not develop hyponatraemic encephalopathy is an entirely different question, which cannot be answered with the study design chosen by these authors.

For example, the presence of a control group who drank according to the dictates of thirst (“ad libitum”) and not according to the guidelines of influential sports medical and commercial organisations, so that they may be less prone to overdrink and so to gain weight during exercise, would have established that athletes who lose more than 1–3 kg during exercise do not develop symptomatic hyponatraemic encephalopathy (even though they are both dehydrated and sodium deficient. Rather, they are more likely to finish such races with raised serum sodium concentrations.

I would rather argue that a fundamental feature of all prospective trials that aim to evaluate a novel intervention such as the role of sodium ingestion in the prevention of hyponatraemia during exercise should be to compare the new intervention with the currently established best practice.

As the currently established best practice is not to ingest fluid at such high rates that weight is gained during exercise, because this practice can produce a fatal outcome, so this study design should, in retrospect, not have been sanctioned. Rather, the control group in the study should have ingested fluid according to guidelines based on the strongest body of current information. It is, for obvious reasons, my biased opinion that the guidelines that come closest to a defendable evidence base are those that have been recently accepted by the United States Track and Field and the International Marathon Medical Interest Group.

Fortunately the data of Twernbold et al. do allow some calculations to estimate the likely value of the extra sodium that was ingested by two of their groups. Thus, the athletes in their study had a mean weight of 58 kg. According to the formula of Montain et al. their predicted extracellular fluid (ECF) volume would be about 14.5 litres (25% of body weight). As the starting serum (and ECF) sodium concentration ([Na⁺]) in the three groups of runners was ~137 mmol/l (table 3 of their article), the average total ECF Na⁺ content of the three experimental groups was 1909–1993 mmol at the start of the race. As weights increased by 1.8–2.1 kg in the three groups during exercise (table 3 of their article), the increases in ECF volume would have been 450–525 ml in the respective groups, assuming that the ECF increased in proportion to the increase in total body water (TBW). Multiplying this new ECF volume by the serum Na⁺ after the race gives the new total ECF Na⁺ content after the race. As shown in table 1, the total ECF Na⁺ content increased by 34 mmol in the group that ingested the high salt drink (H) during the race, but fell by 23 mmol in the group drinking water (W). As all groups ran for about four hours, according to these calculations fluid loss during exercise would have varied from 6 to 21 mmol/h, giving a sweat [Na⁺] of 12–42 mmol/l in the W and H groups respectively (as their total sweat losses were ~2 litres in each group). The clear paradox identified by the calculations in table 1 is that (a) the total Na⁺ loss apparently increases with increased Na⁺ intake and (b) the estimated Na⁺ loss in the group who ingested only water during the race (W) is less than one third of that in the group who ingested the most Na⁺ (H) during the race.

As these calculations are based on two real measurements (body weight changes and changes in plasma [Na⁺]), this apparently ludicrous conclusion can only be explained if (a) Na⁺ ingestion during exercise increases whole body Na⁺ losses in sweat and urine or (b) the estimated ECF volume in the W group after exercise is less than the value calculated. That is, specifically in the W group, the ECF volume contracted despite an increase in TBW of 1.9 litres. Indeed, this response is to be expected. There is consistent evidence that the response of the ECF and the intracellular fluid (ICF) volumes to fluid ingestion during prolonged exercise are influenced by the Na⁺ content of the ingested fluid, so that the ECF (and ICF) volume is likely to fall less if an isotonic Na⁺ drink is ingested, to fall less if either water or a dilute Na⁺ drink is ingested, or to expand if a concentrated (50–100 mmol/l) Na⁺ drink is ingested at the same rate that body weight is lost during exercise. In the latter case, any reduction in the TBW appears to come from a reduction in the ICF.

For example, if each group did indeed lose 84 mmol Na⁺ as did group H (table 1), a value that seems eminently reasonable as it equates to a quite reasonable sweat [Na⁺] of ~40 mmol/l, then the true ECF volume in the W group after the race would have been 14.5 litres—that is, it is unchanged from the starting value. This value (expressed in litres) is calculated as: (pre-race ECF Na⁺ content – 84) in mmol divided by post-race serum [Na⁺] in mmol/l.

Indeed, if subjects in the W group did lose 84 mmol Na⁺ during the race but also had a post-race ECF volume expanded to 14.95 litres, then their post-race [Na⁺] would have been even lower (128 mmol/l) than that actually measured after the race (132 mmol/l; table 1). It is interesting to note that although the Na⁺ content of the group who ingested Na⁺ (“H”), they would have exhibited the early symptoms of hyponatraemic encephalopathy. That they did not have such low serum [Na⁺] indicates the importance of small changes in ECF volume (in this case only 450 ml or ~3% of the total ECF volume) in determining the extent to which the serum [Na⁺] changes during prolonged exercise in which subjects both sweat and ingest fluid to excess.

Unfortunately, the vital importance of these small changes in ECF volume in determining whether hyponatraemic encephalopathy will develop in those who overdrink during exercise can only be ignored by those who argue incorrectly that it is the Na⁺ deficit that determines the extent to which the serum [Na⁺] falls in those who develop hyponatraemia during exercise. This calculation elegantly shows why small changes in ECF volume determine whether or not hyponatraemic...
encephalopathy will develop in those who overdrink, regardless of whether or not they also incur a Na⁺ deficit during exercise. A recent paper confirms these predictions by showing that mathematical modelling supports the argument that changes in TBW exert a much greater effect on serum [Na⁺] than does whole body Na⁺ content in those who overdrink and hence gain weight during exercise. Perhaps the point of these calculations is to show that it is not possible to calculate the state of Na⁺ balance in athletes during exercise and so to determine whether or not athletes have developed a Na⁺ “deficit” simply by measuring serum [Na⁺]. This is because the ECF volume will not be the same before, during, and after exercise and will change depending on the nature of the fluid ingested and the extent of any fluid deficit or excess that develops during exercise. But more importantly, these calculations clearly show why the regulation of the TBW and the ECF volume will have a much greater influence on serum [Na⁺] than will either the expected Na⁺ losses in sweat or the amount of Na⁺ ingested from sodium-containing sports drinks.

For example, a 1 litre (7%) reduction in the ECF volume would “release” 140 mmol Na⁺ into the contracted ECF volume. This means that it is possible to lose 140 mmol Na⁺ in sweat and urine without any change in serum [Na⁺] provided that the ECF volume were to contract by only 7%. If sweat [Na⁺] is about 40 mmol/l, as appears to have been the case in this study of Twerenbold et al., then this 140 mmol is the equivalent of the Na⁺ content of about 3.5 litres of sweat.

As athletes in this study sweated at a maximum rate of only 500 ml/h when running at 10 km/h, this means that simply by reducing their ECF volume by 1 litre, those athletes could have maintained their pre-race serum [Na⁺] while running for seven hours and drinking just sufficient water to allow for a 1 litre reduction in ECF volume and without requiring any Na⁺ replacement whatever. The simple calculation explains why those endurance athletes who, before about 1969, were advised either not to drink at all, or only sparingly during exercise, always finished the races with raised serum [Na⁺] despite having incurred what might have been quite sizeable Na⁺ deficits.

In contrast, athletes in this study who believed the incorrect advice that ingesting Na⁺ in high rates is essential to maintain a normal serum [Na⁺] during exercise, so they overdrank sufficiently to increase their ECF volume by 1 litre, would need to ingest and retain at least an additional 140 mmol Na⁺ in addition to the ~80 mmol lost in sweat (table 1). This is equivalent to the Na⁺ content of 1.24 litres of the low and 7.5 litres of the high sodium drinks respectively in this trial. To maintain fluid balance in this four hour trial when drinking at those high rates and sweating at about 500 ml/h, they would then need to urinate at rates of 1375–2600 ml/h. Both of these rates exceed the maximum at which human kidneys are able to produce urine at rest, yet alone during and after prolonged exercise. Drinking at such rates would therefore only lead to progressive fluid accumulation and ultimately death from hyponatraemic encephalopathy.

In summary, these calculations explain why contraction of the ECF in athletes who lose body weight during exercise will maintain the serum [Na⁺] even in the face of quite large and unreplaceable Na⁺ loss in sweat, and why the ingestion of sodium-containing sports drinks in the vain hope of matching the rates of Na⁺ loss in sweat can only lead to fluid retention and progressive hyponatraemia, as elegantly shown by this study. Indeed if this inappropriate behaviour is approached with sufficient vigour, ultimately the result will be death from hyponatraemic encephalopathy, which, as these calculations and this study again show, cannot occur without the presence of distinct fluid overload.

Finally, it is important to note that, even though Na⁺ ingestion marginally increased serum [Na⁺] in the group that ingested the most concentrated Na⁺ drink, this practice was without benefit as running performances were unaltered by Na⁺ ingestion, and the incidence of symptoms was no different between the groups as no athletes reportedly developed symptoms. However, the symptoms of mild hyponatraemic encephalopathy are mild and may not have been sought with sufficient diligence. For example, all subjects, myself included, in our study in which mild hyponatraemia was induced by fluid overload at rest, developed quite disabling symptoms at serum [Na⁺] of ~136 mmol/l or lower. Indeed it would have been most interesting to determine whether the presence of sublethal mental symptoms was different in the three groups in this study, as all had similar degrees of fluid overload despite different serum [Na⁺]. If the symptoms in this condition are due purely to fluid overload, then the incidence of symptoms should have been the same in all groups despite different serum [Na⁺]. Alternatively, if the symptoms are related to the degree of hyponatraemia, then they should have been most obvious in the W group, who finished with the lowest post-race serum [Na⁺]. My bias would be to expect that the extent of any symptoms are more likely related to the degree of fluid overload, and hence the increase in the ECF, than to the level to which the serum [Na⁺] has been reduced.

Table 1 Sodium balance calculations for three groups of runners running at ~10 km/h for four hours while ingesting solutions with different [Na⁺]

<table>
<thead>
<tr>
<th>Pre-race</th>
<th>Pre-race ECF volume (litres)</th>
<th>Pre-race ECF Na⁺ content (mmol)</th>
<th>Post-race</th>
<th>Post-race ECF volume (litres)</th>
<th>Post-race ECF Na⁺ content (mmol)</th>
<th>Post-race Na⁺ balance (litres)</th>
<th>Amount of Na⁺ ingested (mmol)</th>
<th>Apparent amount (mmol)</th>
<th>Rate of Na⁺ loss during exercise (mmol/h)</th>
</tr>
</thead>
<tbody>
<tr>
<td>H</td>
<td>137.2</td>
<td>14.50*</td>
<td>1991</td>
<td>134.8</td>
<td>15.02</td>
<td>2025</td>
<td>+34</td>
<td>1182</td>
<td>84/21</td>
</tr>
<tr>
<td>L</td>
<td>137.2</td>
<td>14.50</td>
<td>1989</td>
<td>132.8</td>
<td>14.95</td>
<td>1985</td>
<td>-4</td>
<td>71</td>
<td>76/19</td>
</tr>
<tr>
<td>W</td>
<td>137.5</td>
<td>14.50</td>
<td>1993</td>
<td>131.8</td>
<td>14.95</td>
<td>1970</td>
<td>-23</td>
<td>0</td>
<td>24/6</td>
</tr>
</tbody>
</table>

H, High sodium intake; L, low sodium intake; W, water during exercise.

*Based on 25% of mean body weight of 57.7 kg for the total group of runners. Weights for different groups were not reported.

†From table 2 of Twerenbold et al. to convert mg sodium (table 2) into mmol sodium, divide by the molecular weight of sodium (22.99).

References

BOOK REVIEWS

Tennis

It is widely recognised that each sport has its own unique demands and injuries. Therefore the IOC, IFF, ATP, WTA, and Society for Tennis Medicine and Science should be congratulated on producing, in this publication, a comprehensive overview of tennis sports medicine. Together they have assembled an impressive array of experts in this field to write succinct and relevant chapters.

Every aspect of tennis is covered to cater for a broad range of readers, including players themselves. Some areas are covered in a high level of technical detail to please the biomechanists, in particular. However, some of the sports medicine is basic in concept and lacking significant evidence based validity.

Nevertheless, I would highly recommend this book to any health professional who treats a large number of tennis players. Most chapters provide a link between common sports medicine problems and their occurrence in tennis, including conditions that are unique to this sport. At times, some authors are somewhat optimistic with their views of recovery time from surgery—for example, three weeks for arthroscopic debri-dement of the infrapatellar fat pad.

Overall it is well presented with relevant and useful photographic and diagrams to aid the reader, and each chapter gives a list of further recommended reading. Unfortunately the book does not provide an answer to where 14 million tennis balls go, imported each year into Australia, as discussed by the editor recently!

Dying to win

Dying to win gives an eye opening account of the extent to which drugs play a major role in sport. Doping is not new and has been used in sport since ancient Olympic times; it is just that drug use in modern times is at such a level of sophistication, it is now an industry in its own right. The book describes the privileged position sport holds in society, having appeal for both the participant and the spectator. This has led to the massive media interest, commercialism, professional-ism, and governmental regulation and manipulation. Economic pressure in the industrialised world and governmental propaganda in the former East Germany, and more recently China, pave the way for increasing pharmaceutical intervention in sport. With the fall of the GDR, the world saw for the first time what it had long suspected, the extent of systematic doping on a State run basis, and the most interesting fact is that the East Germans never won any records! Further, the book takes a look at the next big issue surrounding drugs in sport—genetic engineering.

Dying to win does not just describe the evolution of doping. It explains the complex relation between anti-doping policy, implementation of those policies, and the role of governments, the IOC, and international and national sporting organisations. With the ever increasing involvement of the legal profession, a vicious circle occurs: it becomes too costly for sporting organisations to fight court battles, with their reliance on Government funding depending on results and punishments set in accordance with what will stand up in courts. This all leads to the relative inertia of the governing bodies to be pro-active in the anti-drugs campaign.

The inception of the World Anti-Doping Agency (WADA) after the 1998 Winter Olympics in France drugs fiasco provided a way forward to standardise and implement anti-doping policy across the world by an independent body.

Problems and solutions to anti-doping policy are addressed. The major problem is inadequate definition of doping—to quote Arthur Gold “The definition lies not in words but in integrity of character.” It is interesting to note that those behind the athlete, namely coach, administrators, medical profession, and scientists, all seem to lose perspective along with their ethics and “integrity of character” when the race for “gold” is on. Dying to win suggests that these people should be held just as accountable as the athletes themselves. Another unfortunate aspect of anti-doping policy is the difficulty in detecting some abused drugs and the fact that these strategies often lag behind the ability of the pharmaceutical industry to develop new drugs, often for genuine medical reasons but with the unfortunate ability to enhance performance. Education is proposed as a key aspect to anti-doping policy, and parallels with its success in the use of recreational drugs are made. Governments also play a role in limiting supply, decreasing demand for drugs, and the implementation of independent bodies to carry out testing. The success of anti-doping policy is also hard to measure. Fewer positive tests may simply suggest that these people along with their ethics and “integrity of character” have been compromised, despite the efforts of the pharmacy, sport, and Government. This all leads to the problem of doping in sport and the difficulty in finding solutions to the problem. It makes interesting reading for all those involved in sport, from the athlete and coach to the sport administrator, the medical profession, and governments.
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BASEM Conference 2006
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CORRECTIONS

doi: 10.1136/bjsm.2004.03921corr1

doi: 10.1136/bjsm.2004.000044corr1
Dadebo B, White J, George K P. A survey of flexibility training protocols and hamstring strains in professional football clubs in England (Br J Sports Med 2004;38:388–94). The multiple regression equation within the Abstract section of this paper was published incorrectly. The correct equation is:

\[
\text{HSR} = 37.79 - (0.335 \times H1 + 1.08 \times SS + 2.24 \times STE) \pm 2.34
\]

We apologise for this error.

doi: 10.1136/bjsm.2004.010876corr1
Sran M M. To treat or not to treat: new evidence for the effectiveness of manual therapy (Br J Sports Med 2004;38:521–5). The volume number for reference 23 (Sran et al) was incorrectly published as 24; the correct volume number is 29.

In Table 2 the results for Giles and Muller should read: Greater short term benefit for back pain with manipulation, but not for neck pain. Acupuncture more effective for neck pain.

In the section “Definitions and search strategy” the first line of paragraph 2 should read: I searched Medline, Cinahl, and Embase databases for randomised clinical trials comparing manual therapy, including spinal joint mobilisation (with or without manipulation) or manipulation only with other conservative treatments for back or neck pain.

We apologise for these errors.