Cardiovascular admissions and mortality in an inception cohort of patients with rheumatoid arthritis with onset in the 1980s and 1990s

N Goodson, J Marks, M Lunt, D Symmons

Background: There is increased cardiovascular disease mortality in rheumatoid arthritis. This may reflect an increased prevalence of cardiovascular disease or an increased case fatality in patients with rheumatoid arthritis.

Objectives: To examine whether rheumatoid patients with disease onset in the 1980s–1990s have increased mortality, and to compare cardiovascular admission rates in rheumatoid patients with those of the general population.

Methods: An inception cohort of 1010 rheumatoid patients attending Stockport rheumatology clinics between 1981 and 1996 was followed up to December 2002 through the Office for National Statistics. Standardised mortality ratios (SMR) were calculated for all-cause and cause specific mortality, using the population of Stockport as reference. Cardiovascular disease admission rates were ascertained for a subgroup of patients using national hospital episode statistics; standardised cardiovascular disease admission rates (SAR) and SMRs were calculated for this subgroup.

Results: 470 patients (48%) died during a median follow up of 11.4 years. All-cause mortality was increased in men (SMR = 1.45 (95% confidence interval, 1.22 to 1.71)) and women (SMR = 1.84 (1.64 to 2.05)), as was cardiovascular disease mortality in men (SMR = 1.36 (1.04 to 1.75) and women (SMR = 1.93 (1.65 to 2.26)). No difference in cardiovascular disease admission rates was observed in men (SAR 1.20 (0.89 to 1.58) or women (SAR = 1.10 (0.88 to 1.36)), despite excess cardiovascular disease mortality in this subgroup.

Conclusions: Patients with rheumatoid arthritis have reduced life expectancy and excess cardiovascular disease mortality. Nevertheless, standardised admission rates for cardiovascular disease were not raised. This suggests either that cardiovascular disease in rheumatoid arthritis has a higher case fatality than in the general population or that it often goes unrecognised before the fatal event.

Most mortality studies in patients with rheumatoid arthritis published since 1950 have shown an increased mortality from cardiovascular disease. There is now intense interest in verifying and quantifying this excess cardiovascular disease risk and understanding the underlying mechanisms.

Most previous mortality studies examined cohorts of patients with established rheumatoid arthritis. This may cause selection bias towards more severe progressive disease and left censorship. The study of inception cohorts of rheumatoid patients is less likely to reveal decreased survival rates because of the potential to include patients with milder disease. Several recently reported mortality studies have followed patients from early in their rheumatoid disease process. Four of these studies failed to identify any increased mortality in rheumatoid patients with disease onset during the 1980s to 1990s. One study showed that mortality from acute myocardial infarction declined in patients with onset of rheumatoid disease in more recent decades. Possible explanations for the relative improvement in survival of patients with rheumatoid arthritis include the earlier introduction of disease modifying treatments or a change in the natural history of rheumatoid arthritis, with the disease becoming less severe over recent years. It is possible that excess mortality in these rheumatoid cohorts could be identified with continued long term follow up.

However, excess mortality from cardiovascular disease was observed in a primary care based cohort of patients with early seropositive inflammatory arthritis. Data from the Mayo clinic also suggest that the excess mortality associated with rheumatoid arthritis did not fall during the 1980s to 1990s.

A study of rheumatoid patients identified from the Swedish hospital discharge register reported that, although mortality rates fell towards the end of the last century, mortality from coronary heart disease remained particularly high in rheumatoid arthritis. Thus it is difficult to know whether the survival prospects of rheumatoid patients have changed in recent years. Long term follow up of rheumatoid arthritis inception cohorts is required.

One possible explanation for the excess cardiovascular disease mortality in rheumatoid arthritis is an increased prevalence of comorbid cardiovascular disease. Studies in the USA have shown that rheumatoid patients have higher rates of cardiovascular disease events than in the general population and describe cardiovascular disease comorbidity more often in rheumatoid arthritis than in osteoarthritis. However, there have also been reports that rheumatoid patients are more likely to experience silent ischaemic heart disease and sudden cardiac death than the general population. An alternative explanation for the increased cardiovascular disease mortality in rheumatoid arthritis could be that these patients have an increased case fatality rate from cardiovascular disease events.

Our aims in this study were therefore, first, to examine whether rheumatoid patients with disease onset in the 1980s to 1990s, identified in a hospital clinic setting, have increased...
Table 1 Descriptive data and mortality data for the rheumatoid arthritis cohort

<table>
<thead>
<tr>
<th>Descriptive data</th>
<th>Women (n = 729)</th>
<th>Men (n = 281)</th>
<th>Total (n = 1010)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at presentation (years)</td>
<td>60.4 (49.8 to 69.9)</td>
<td>60.3 (51.7 to 68.3)</td>
<td>60.4 (50.4 to 69.6)</td>
</tr>
<tr>
<td>Age at diagnosis (years)</td>
<td>62.5 (49.9 to 71.9)</td>
<td>63.8 (55.1 to 72.9)</td>
<td>63.1 (51.6 to 72.5)</td>
</tr>
<tr>
<td>Age at time of diagnosis</td>
<td>59.8 (49.8 to 69.3)</td>
<td>59.6 (50.3 to 67.0)</td>
<td>59.7 (49.9 to 68.8)</td>
</tr>
<tr>
<td>RF+ at time of diagnosis</td>
<td>542 (74.3%)</td>
<td>229 (81.8%)</td>
<td>772 (76.4%)</td>
</tr>
<tr>
<td>Cause of death</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>158 (22.2%)</td>
<td>62 (23.1%)</td>
<td>220 (22.5%)</td>
</tr>
<tr>
<td>Respiratory</td>
<td>51 (7.2%)</td>
<td>26 (9.7%)</td>
<td>77 (7.9%)</td>
</tr>
<tr>
<td>Neoplastic</td>
<td>52 (7.3%)</td>
<td>36 (13.4%)</td>
<td>88 (9.0%)</td>
</tr>
<tr>
<td>Other mortality</td>
<td>67 (9.4%)</td>
<td>18 (6.7%)</td>
<td>85 (8.7%)</td>
</tr>
<tr>
<td>Total mortality</td>
<td>328 (46.1%)</td>
<td>142 (53.0%)</td>
<td>470 (48.0%)</td>
</tr>
</tbody>
</table>

Values are median (interquartile range) or n (%).
†Date of birth not known for two patients in the cohort.
‡Mortality data not available for 31 patients.
RF+/-RF+, rheumatoid factor negative/positive.

Mortality compared with the local general population; and second, to compare cardiovascular admission rates in rheumatoid patients with those of the general population.

METHODS

Setting

Stockport is a large urban area, south of Manchester, UK, with a population of nearly 285 000.

Stockport rheumatoid arthritis register

This inception cohort of 1010 patients with rheumatoid arthritis was identified from a register of all newly diagnosed cases, based on a consultant rheumatologist’s opinion, who were attending Stockport rheumatology outpatient clinics between 1981 and 1996. Patients were designated as seropositive if, at the time of diagnosis, they had either an IgM rheumatoid factor (RF) titre of 1:40 or above, or rheumatoid nodules. Many of these patients were subsequently discharged from regular rheumatology follow up. Thus, unlike many previously described hospital based cohorts,20–24 the Stockport rheumatoid arthritis cohort is composed of patients with a broad spectrum of rheumatoid disease severity and is not restricted to those under long term rheumatology follow up.

Approval for the study was given by the Stockport local research ethics committee.

Mortality study

Patients and methods

Identification data including NHS numbers, names, dates of birth, and last known address were sent to the Office for National Statistics (ONS) for flagging and notification of deaths using the NHS Central Register.25 ONS were able to match 979 of the 1010 patients (97%) and provided death drafts for all patients who died before 31 December 2002. The causes of death were coded according to World Health Organisation (WHO) rules using the International Classification of Diseases, ninth revision (ICD-9) until the 1 January 2001, when ICD-10 coding was introduced.

The Stockport district population was used as the reference group for calculating standardised mortality ratios (SMRs). ONS provided mortality rates for all causes, cardiovascular causes, respiratory causes, and neoplasia, for men and women separately in 10 year age bands, for the years 1982 to 2001. Population death rates for the year 2002 were not available at the time of analysis, and cohort mortality rates in this year were compared with those in 2001. All-cause and cause specific SMRs were calculated using indirect standardisation (STATA-7). Cox regression was used to examine whether RF status predicted mortality in the rheumatoid arthritis cohort, adjusting for age and sex. The Cox model’s proportional hazards assumptions were tested graphically using the stphplot command in STATA-7.

Cardiovascular admissions study

Patients and methods

Patients in the Stockport rheumatoid arthritis cohort who were Stockport Health Authority residents between 1994 and 2002 were identified by matching NHS numbers, dates of birth, and names. Dates of entering and leaving the health authority register were recorded. Stockport Public Health Department provided hospital episode statistics (HES)26 for
all NHS hospital admission episodes, coded with primary diagnoses of cardiovascular disease, (ICD-9 codes 390–459 and ICD-10 codes 100–199), ischaemic heart disease (ICD-9 codes 410–414 and ICD-10 codes I20–I25), and myocardial infarction (ICD-9 codes 410, 412 and ICD-10 codes I21–I22), for Stockport Health Authority residents between years 1994 and 2002. Population rates of admissions for cardiovascular disease, ischaemic heart disease, and myocardial infarction were calculated for each year using the Stockport Health Authority population estimates as the denominator (taken in October 1999 as the mid-point of the study period).

In all, 515 of the Stockport rheumatoid arthritis cohort were identified as being residents in Stockport Health Authority between 1994 and 2002. The incidence of cardiovascular disease, primary diagnosis, and admissions in the prevalent Stockport rheumatoid arthritis cohort were compared with the rate of admissions for cardiovascular disease as a primary diagnosis in the general Stockport population to calculate a standardised cardiovascular admission rate (SAR) for the rheumatoid cohort. SARs for the diagnoses of cardiovascular disease, ischaemic heart disease, and myocardial infarction for the years 1994 to 2002 were calculated using STATA-7. The start of follow up was 1 April 1994, or the date of entry to the health authority. The end of the follow up period was the date of death, the date of leaving the health authority, or the end of the study period (31 March 2002). Each patient admitted with cardiovascular disease was censored on the date of admission and a new period of follow up was started. Patients with multiple admissions contributed several periods of person years of follow up.

SMRs for this prevalent rheumatoid subgroup were calculated for all-cause, cardiovascular disease, ischaemic heart disease, and myocardial infarction mortality using the Stockport population mortality rates for these years.

RESULTS
Mortality study
Date of birth, sex, year of diagnosis, and rheumatoid factor status at diagnosis were recorded for 1008 of the 1010 patients. There were 729 women (72%) and 279 men (28%) and the median age at diagnosis was 60.4 years (interquartile range (IQR), 50.4 to 69.6) (table 1).

An attempt was made to validate the consultant diagnosis of rheumatoid arthritis. The medical records of 20 patients, selected randomly from the Stockport rheumatoid arthritis register, were reviewed, and 18 patients met American college of Rheumatology (ACR) classification criteria for rheumatoid arthritis. There was insufficient clinical information recorded to classify two patients.

The 979 patients (97%) matched by ONS were used in the initial mortality analysis. The median follow up period was 11.4 years (IQR, 7.5 to 15.4). By 31 December 2002, 470 patients (48%) had died. The median age at death was 74.9 years (68.8 to 81.0). Women died at an older age (76.0 years (68.0 to 79.9)) than men (72.2 years (66.1 to 78.9)), and patients who were RF negative died at an older age (79.6 years (71.7 to 84.8)) than RF positive patients (74.3 years (68.0 to 79.9)).

Cause of death
Cardiovascular disease was the most frequent cause of death (table 1), responsible for 48% of female and 44% of male deaths. Ischaemic heart disease was recorded as the main cause of death in 125 cases (26%) and acute myocardial infarction in 72 (15%) (table 2).

Rheumatoid arthritis was identified as the underlying cause in only 36 deaths (7.6%) and was recorded anywhere on the death certificates of 44 patients (9%).

Standardised mortality ratios
Both male and female rheumatoid patients had higher than expected mortality rates (table 3) Mortality from all cardiovascular disease causes in women were approximately twice that expected (SMR = 1.93 (95% confidence interval (CI), 1.65 to 2.26)) with a more modest increase being observed in male patients (SMR = 1.36 (1.04 to 1.75). The SMRs for ischaemic heart disease were raised in both men and women, and female rheumatoid patients had a higher mortality from myocardial infarction in female rheumatoid patients, 77 (51%) of the 149 excess deaths were caused by cardiovascular disease and 40 (27%) of the excess deaths were from ischaemic heart disease. Cardiovascular deaths were responsible for 38% of the 44 male excess deaths, and 13 of the male excess deaths (29%) were from ischaemic heart disease. Mortality from respiratory causes was increased in both male and female rheumatoid patients, and 36 respiratory deaths were coded as being caused by pneumonia, seven by interstitial lung disease, one from asthma, and the remaining respiratory deaths were from chronic obstructive airways disease. The number of deaths from neoplasia was not significantly different from that seen in the general population.

Men and women with seropositive rheumatoid arthritis had increased all-cause, cardiovascular disease, ischaemic heart disease, and myocardial infarction mortality compared with the general population (table 4). Seropositive women had twice the expected cardiovascular, ischaemic heart disease, and myocardial infarction mortality. Respiratory mortality was significantly increased in seropositive patients. Female seronegative patients had a modest increase in all-cause and cardiovascular disease mortality. Male seronegative patients had no increase in all-cause or cause specific mortality. However, the number of patients in this subgroup was small.

Predictors of mortality
Rheumatoid factor, univariately, did not predict all-cause mortality within the rheumatoid cohort. However, RF positive patients were younger at presentation and at death.
When adjusted for age at disease onset and sex, RF was a modest predictor of all-cause mortality, (hazard ratio (HR)adj = 1.55 (95% CI, 1.25 to 1.94)), cardiovascular disease mortality (HR adj = 1.37 (1.00 to 1.88)), and respiratory mortality (HR adj = 1.85 (1.04 to 3.29). The proportional hazards assumption remained true for all these models.

Cardiovascular admissions

Patient characteristics

The characteristics of the 515 rheumatoid patients who were Stockport residents between 1994 and 2002 are shown in table 5. The median disease duration was 5.8 years from the time of rheumatologist diagnosis of rheumatoid arthritis. The median age at diagnosis was 56.2 years (IQR 41.7 to 65.5).

The 515 patients accumulated 3173.3 person years of follow up between 1 April 1994 and 31 March 2002. During this time, 93 (18%) of the rheumatoid patients experienced 134 cardiovascular disease admissions and 30% of these patients had more than one admission. These patients were admitted to hospitals in Manchester, Stockport, and East Cheshire.

Standardised admission ratios

The SAR for cardiovascular disease was not raised in either male or female subjects (table 6). There were only small numbers of admissions with ischaemic heart disease and myocardial infarction. There were no significant increases in cardiovascular disease SARs for seropositive rheumatoid patients (female seropositive patients, SAR = 1.10 (95% CI, 0.54 to 2.10); male seropositive patients, SAR = 1.44 (0.95 to 3.32)).

Standardised mortality ratios in the admissions cohort

There were 182 deaths in this group of 515 prevalent rheumatoid patients, with 100 deaths from cardiovascular disease (table 7). Cardiovascular mortality was significantly increased in both male (SMR = 2.40 (95% CI, 1.64 to 3.39)) and female patients (SMR = 2.77 (95% CI, 2.15 to 3.51)). Mortality from ischaemic heart disease was also increased in both sexes, but the increase was more marked in women and seropositive patients (SMR = 2.77 (95% CI, 2.15 to 3.51)). Mortality from myocardial infarction was significantly increased in women.

Approximately 15% of all deaths in this cohort were attributed to acute myocardial infarction. There were more excess deaths from cardiovascular disease than there were excess cardiovascular disease admissions. Only 42 of the 100 patients in the rheumatoid cohort who died from cardiovascular disease had any previous cardiovascular disease admissions during the follow up period. The median time between the first cardiovascular disease event and death was 4.8 months (IQR 0.6 to 21.6). Cox regression adjusted for age and sex showed that a cardiovascular admission was a very strong predictor of subsequent all-cause mortality (HRadj = 4.2 (95% CI, 2.9 to 5.9)) and cardiovascular disease mortality (HRadj = 8.3 (5.7 to 13.6)).
Table 5  Demographics of the 515 patients with rheumatoid arthritis monitored for cardiovascular disease admissions

<table>
<thead>
<tr>
<th></th>
<th>All RA patients (n = 515)</th>
<th>Female RA patients (n = 371)</th>
<th>Male RA patients (n = 144)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>RF+ at RA diagnosis (n (%))</strong></td>
<td>388 (75.3)</td>
<td>275 (74.1)</td>
<td>113 (78.5)</td>
</tr>
<tr>
<td><strong>Age [years] start of admissions [median (IQR)]</strong></td>
<td>62.9 (53.8 to 72.3)</td>
<td>63.6 (53.5 to 72.9)</td>
<td>61.8 (54.7 to 72.9)</td>
</tr>
<tr>
<td><strong>Duration of RA [years] [median (IQR)]</strong></td>
<td>5.8 (2.8 to 9.8)</td>
<td>5.8 (2.8 to 9.8)</td>
<td>5.8 (1.8 to 8.8)</td>
</tr>
<tr>
<td><strong>SMR</strong></td>
<td>2.77</td>
<td>2.15</td>
<td>1.89</td>
</tr>
</tbody>
</table>

IGR, interquartile range; RA, rheumatoid arthritis; RF+, rheumatoid factor positive.

Table 6  Standardised admission rates (SAR) for cardiovascular disease

<table>
<thead>
<tr>
<th>Cause of admission</th>
<th>Women (n = 371): number of admissions</th>
<th>Men (n = 144): number of admissions</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Obs</td>
<td>Exp</td>
</tr>
<tr>
<td>CVD</td>
<td>83</td>
<td>75.45</td>
</tr>
<tr>
<td>IHD</td>
<td>18</td>
<td>21.66</td>
</tr>
<tr>
<td>MI</td>
<td>10</td>
<td>7.55</td>
</tr>
</tbody>
</table>

CI, confidence interval; CVD, cardiovascular disease; Exp, expected; IHD, ischaemic heart disease; MI, myocardial infarction; Obs, observed.

Table 7  Standardised mortality ratios for all cause and cardiovascular disease causes of mortality in the rheumatoid arthritis admissions group

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>RA group</th>
<th>Women (n = 371): number of deaths</th>
<th>Men (n = 144): number of deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Obs</td>
<td>Exp</td>
<td>SMR</td>
</tr>
<tr>
<td>All-cause</td>
<td>129</td>
<td>56.45</td>
<td>2.29</td>
</tr>
<tr>
<td>RF+</td>
<td>93</td>
<td>38.37</td>
<td>2.42</td>
</tr>
<tr>
<td>RF−</td>
<td>36</td>
<td>18.1</td>
<td>1.99</td>
</tr>
<tr>
<td>CVD</td>
<td>68</td>
<td>24.50</td>
<td>2.77</td>
</tr>
<tr>
<td>RF+</td>
<td>50</td>
<td>16.45</td>
<td>3.04</td>
</tr>
<tr>
<td>RF−</td>
<td>18</td>
<td>8.06</td>
<td>2.23</td>
</tr>
<tr>
<td>IHD</td>
<td>36</td>
<td>12.48</td>
<td>2.89</td>
</tr>
<tr>
<td>RF+</td>
<td>27</td>
<td>8.46</td>
<td>3.19</td>
</tr>
<tr>
<td>RF−</td>
<td>9</td>
<td>4.02</td>
<td>2.24</td>
</tr>
<tr>
<td>MI</td>
<td>19</td>
<td>7.00</td>
<td>2.71</td>
</tr>
<tr>
<td>RF+</td>
<td>15</td>
<td>4.75</td>
<td>3.15</td>
</tr>
<tr>
<td>RF−</td>
<td>4</td>
<td>2.24</td>
<td>1.78</td>
</tr>
</tbody>
</table>

CI, confidence interval; CVD, cardiovascular disease; Exp, expected; IHD, ischaemic heart disease; MI, myocardial infarction; Obs, observed; RA, rheumatoid arthritis; RF+/−, rheumatoid factor positive/negative; SMR, standardised mortality ratio.

Stockport rheumatoid arthritis cohort was managed in routine rheumatology outpatient departments and many patients were followed up in primary care.

Other studies have also observed a greater excess mortality in female than male rheumatoid patients.27 28 This sex difference may be deceptive, in that population rates of cardiovascular disease are lower in women than in men. Thus if rheumatoid arthritis had the same impact in men and women with respect to cardiovascular disease, one would expect higher SMRs for women given the smaller number of expected cases in the denominator.

In the current rheumatoid arthritis cohort, cardiovascular disease admission rates were not increased, despite the fact that cardiovascular disease mortality was twice that observed in the general population. One possible explanation could be that rheumatoid patients are less likely to experience multiple cardiovascular disease admissions than the general population. Of the rheumatoid patients admitted to hospital, 70% had only one cardiovascular disease admission during the eight years of follow up. This situation might occur if rheumatoid patients were more likely to die after or during their first cardiovascular disease admission. If rheumatoid patients are less likely to experience or interpret significant angina symptoms they would be less likely to present at secondary care for treatment. This may lead to increased rates of unrecognised myocardial infarction and sudden cardiac death.29 During the period of this study 34 (52%) of the rheumatoid patients identified as having cardiovascular disease admissions died after their first such admission. Unfortunately, the number of first cardiovascular disease admissions for the general population was not available. Thus it was not possible to compare the number of first cardiovascular disease events in the rheumatoid cohort and the general population.

The study of respiratory outcomes was not one of the main aims of this study. However, we observed increased respiratory mortality in the rheumatoid patients, and seropositive disease was a predictor of respiratory death after adjusting for age and sex. Possible explanations for the increased respiratory mortality include respiratory complications of rheumatoid arthritis, although only a few deaths were caused by interstitial lung disease, or the effects of cigarette smoking, which is identified as a risk factor for rheumatoid arthritis and seropositive disease.29 We were not able to examine associations between smoking and mortality in this study.
A strength of this study is that it was able to identify hospital admissions throughout the country for both the rheumatoid arthritis cohort and the local population controls. The study has some limitations. Inclusion on the rheumatoid arthritis register was based on consultant diagnosis rather than application of the 1987 ACR classification criteria for rheumatoid arthritis. While this method lends a degree of external validity—as a consultant rheumatologist made the clinical diagnosis of rheumatoid arthritis, which reflects common rheumatological practice—it may have introduced misclassification bias. It is likely that simply using a consultant diagnosis of rheumatoid arthritis will have included patients with inflammatory polyarthritides who did not meet the criteria of the seven ACR classification criteria. These patients might be expected to have milder disease and therefore make it more difficult to detect an association with increased cardiovascular disease mortality.

Baseline data on disease severity and disability were not collected, nor was information on smoking, social history, or comorbid conditions recorded. Opportunities to look for predictors of mortality within the cohort were therefore limited. This study relies on death certificate data for the main cause of death and hospital episode statistics for the main cause of admission. There are several limitations that can give rise to inaccuracies in mortality and admission data, although it seems that the accuracy of coding for specific admission diagnoses has improved over recent years. However, as population mortality and admission data are subject to the same inaccuracies it is unlikely that this will have introduced significant bias. Conversely if cardiovascular disease is more likely to remain clinically silent in rheumatoid arthritis, it is probable that cardiovascular disease diagnoses will be recorded less often as the primary cause of admission in this group. This may explain the similar rates of cardiovascular disease admissions observed in this study. As described by others, rheumatoid arthritis was recorded infrequently on the death certificates of these patients.

In summary, patients who have developed rheumatoid arthritis in the last two decades continue to experience excess cardiovascular mortality in the 21st century. This excess mortality is not reflected by an increase in cardiovascular disease admissions. This may reflect a high mortality during the first event or a failure to diagnose cardiovascular disease before death.

ACKNOWLEDGEMENTS
NG was supported by a clinical fellowship awarded by the Devonshire Royal Hospital Trust, Buxton. The work of the ARC Epidemiology Unit is funded by a programme grant from the Arthritis Research Campaign, UK. We are grateful for the help of the Stockport Primary Care Trust and Public Health Informatics Departments at Stepping Hill Hospital and Dr Kate Morgan, Consultant Histopathologist at Stepping Hill Hospital.

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Posture may not prevent pain with computer use

Posture may not be the key to avoiding aches and pains brought on by using computers, after all, a randomised controlled trial in the United States has suggested, so we should be cautious about encouraging any particular posture.

Incidence of musculoskeletal (MS) symptoms in hands or arms and neck or shoulders over six months was no different, nor did the time to noticing symptoms differ significantly, among three groups of newly recruited office workers in the trial. Two groups were randomly assigned to receive postural interventions, and a control group was allowed to carry on as usual. The interventions were based on protective factors derived from a previous prospective study (alternative intervention) or well known national occupational safety and health policies or private company policies (conventional intervention). The main drawback was low compliance—only 25% for the alternative and 38% for the conventional intervention—mainly because of inflexible workstations, so a potential effect cannot be ruled out.

The trial included 376 recruits in metropolitan Atlanta, Georgia, using computers for 15 hours or more a week currently and in their previous job. Demographic information, work and medical history, and computer use were collected by questionnaire at enrolment. Computer use was recorded daily by the recruits and symptoms weekly during follow up.

The use of computers has seen an escalation in work related musculoskeletal problems in the upper body. Such a potentially huge impact on public health means it is important that trials test the assumption that posture does protect.