Skin temperature recovery from cold provocation in workers exposed to vibration: a longitudinal study

M Cherniack, A Brammer, J Meyer, T Morse, D Peterson, R Fu

Background: Vibration white finger (VWF) is characterised by arterial hyperresponsiveness and vasoconstriction following cold provocation. Several years after removal from exposure, most subjects show improved finger systolic blood pressure (FSBP) under conditions of cold challenge, but continue to report cold hands and finger blanching.

Aims: To assess the underlying reasons for the persistence of cold symptoms.

Methods: A total of 204 former users of pneumatic tools with cold related hand symptoms were evaluated and re-evaluated a year later. Symptoms were evaluated using the Stockholm Workshop Scale. Finger systolic blood pressure per cent (FSBP%) was assessed by comparing digital blood pressure in a cold provoked and normalised state. Fingertip skin temperature was measured during cooling and occlusion and during rewarming and recovery.

Results: There were dramatic improvements in FSBP% (14.3 mm Hg%), modest improvement in recovered skin temperature (0.86°C), and no change in symptom stage. When the most symptomatic subjects (n = 75) were compared with the less symptomatic subjects (n = 129), there were similar inter-test improvements in FSBP%. Skin temperature recovery improved in the less symptomatic (+1.49°C), but did not change in the most symptomatic group (−0.12°C). However, the more symptomatic group had higher temperatures at the initial test, thus qualifying the result.

Conclusions: Skin temperature recovery after cold challenge in subjects with VWF remains reduced in the symptomatic subjects several years after exposure removal. This is evident even when blood pressure has increased in the setting of cold provocation. Results suggest that in VWF, the dermal circulation remains impaired, even after the restoration of arterial blood pressure in the digits.

A bnormalities in digital circulation, characterised by cold white fingers, have been a recognised hallmark of vibration induced vascular injury for more than 80 years. The clinical condition, cold induced vibration white fingers (VWF), also known as "occupational Raynaud’s phenomenon", includes components of exaggerated vasoconstriction and impaired vasodilatation. Cold provocation testing of the fingers and hands simulates the interruption of digital blood flow that is presumed to occur in cold environments. The two general approaches to cold provocation testing are blood pressure and skin temperature measurements, the latter a presumed surrogate for blood flow. For more than 20 years, the combination of digital occlusion with local cold provocation, using a water inflatable inlet cuff, and measurement of finger blood pressure or blood flow by strain gauge or sensor, has been standardised and used extensively in fieldwork. A second less standardised approach involves measurement of finger skin temperature (FST) following cold provocation. In a previous report on more than 200 vibration exposed and symptomatic subjects, who were tested and subsequently retested, we observed that: (1) finger systolic blood pressure (FSBP), assessed by cold provocation, improved following termination of vibratory exposure; (2) recovery was retarded in smokers (reversed by cessation); and (3) symptoms persisted despite improved FSBP%. Other investigators had noted the temporal discrepancy between improvement in FSBP and lack of symptom reduction following exposure reduction; one author concluding that symptomatic VWF is non-remediable.

The aim of this study was to further explore and to examine reasons for persistent cold provoked symptoms when objective test parameters improved. A different aspect of the cold response—the stability and recovery of finger skin temperature after a cold challenge—was examined. The presumption was that reduced digital skin temperatures, under ambient or cold conditions, might predict a pattern of symptomatic hyperresponsiveness to cold. The hypothesis is that the historical description of cold digits with normal arterial blood pressure may reflect measurably cold skin, possibly due to deficiencies within the superficial nutritive capillaries.

METHODS

Subjects
Two hundred and four subjects, referred to a university medical centre for VWF evaluation 1988–94, were studied. All were current, or former, skilled shipyard metalworkers from trades with documented pneumatic tool use that averaged >10 hours per week. Vascular assessment included grading of clinical symptoms using the Stockholm Workshop Scale, completion of a supplemental symptoms questionnaire, and a cold provocation test. The Stockholm Workshop Scale is a consensus rating system, developed by a hand-arm vibration expert committee for international standardisation. It classifies VWF into progressive symptom categories that range from 0 to IV. Since stage 0 indicates absence of symptoms, and stage IV represents trophic changes, which were not reported in this group, essentially all subjects fell into stages I–III. Stage III consists of frequent attacks of finger blanching.

Abbreviations: FSBP, finger systolic blood pressure; FST, finger skin temperature; FTI, finger temperature index; HAVS, hand-arm vibration syndrome; RWI, rewarming index; VWF, vibration white finger
that involve most fingers, and extends over the full finger length. Stage I consists of more occasional blanching attacks confined to the fingertips of one of more fingers. Stage II defines an intermediate blanching pattern. The differentiation between Stage I and stage II is potentially ambiguous.

Individual subject files consisted of current and past occupational history, a symptom severity questionnaire, a description of pneumatic tool use, and detailed smoking histories. Current smoking history was also obtained at the time of the test.

Finger systolic blood pressure (FSBP) experimental test

The cold provocation test has been described earlier, and follows the technique proposed by Nielsen and Lassen. Subjects were instructed to refrain from drinking caffeinated beverages and from smoking on the day of the test. Subjects were tested in a supine position following a 30 minute acclimatisation period at ambient temperature of 21°C–24°C. Hands were warmed to produce a pre-test digital skin temperature of ≥28°C. Water was circulated at 15°C, and then 10°C, through a dual inlet digit occlusion cuff. In baseline and rewarming sequences, water was circulated at 30°C. FSBP was simultaneously measured in a test and non-cooled reference finger (the thumb unless contraindicated by disease). Finger cooling was augmented by the use of a truncal cooling blanket. Results are presented as a proportion (test finger/reference finger)—then normalised to the ambient temperature, 30°C as finger systolic blood pressure per cent (FSBP%). All FSBP repeated measurements employed the same method and apparatus as in the original test (Digimatic 2000, Medimatic, Copenhagen).

For each subject, there were five separate test runs: 30°C digital cooling (test 1), 30°C with body cooling (test 2), 15°C with body cooling (test 3), 10°C with body cooling (test 4), and rewarming at 30°C (test 5). All FSBP% measurements presented in the tables compare test 4 to test 1.

FSBP% results are not normally distributed. One reason involves a possible artefact of measurement; the other is based in the probable pathophysiology of VWF. The measurement artefact reflects uncertainties in delineating FSBP measurements <32 mm Hg, because venous pressures can mask low pressure arterial flow. In addition, most investigators have observed two peaks in the distribution of measured FSBP%. Test results are either clustered at FSBP = 0, or are otherwise normally distributed. For these reasons, FSBP measurement results in this study are presented both as continuous and as grouped data. A leading contributor has argued that there are only two true categories of response: normal and FSBP = 0. The FSBP% cut-off of 30%

Skin temperature indices

The temperature indices (FTI, RWI 1, RWI 2) stand for finger temperature index and rewarming index 1 and 2, respectively. They were intended to relativise all values to a baseline. Table 1 lists the calculations for the skin temperature indices and the finger systolic blood pressure per cent. The FTI is a measure of the baseline to recovery residual, adjusted for arm temperature, and the rewarming indices describe a relation between maximum cooling and recovery fingertip temperatures. The FTI was constructed on the assumption that a comparison of the cold challenged digital temperature to an uncooled baseline—corrected for a “systemic change” in the arm temperature during testing—might be more meaningful than a simple skin temperature comparison. Similarly, the rewarming indices are the residuals and ratios of digit temperature, when the most cooled and recovery states are compared.

All 204 subjects had follow up studies performed one year after their initial study. Retesting was not mandatory; hence this was a highly selected retest cohort. The criteria for retesting were prior test abnormality on cold challenge plethysmography (FSBP% <70), or significant unabated symptom (Stockholm Scale stage III) with normal plethysmography. No subjects were included who had continuous exposure to vibratory tools after the date of their initial test. For some subjects, skin temperature monitoring data were incomplete in 1988, having been conducted prior to finalisation of the temperature monitoring protocol or because of equipment limitations. Because of the centrality of skin

Table 1

<table>
<thead>
<tr>
<th>Variable description</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Finger systolic blood pressure % (FSBP%)</td>
<td>( % \text{pressure} = 100 \left[ \frac{(P_{c30} - P_{t})(P_{ct} - P_{t})}{(P_{c30} - P_{t})^2} \right] ) where ( P_{c30} ) = FSBP of reference finger at 30°C, ( P_{ct} ) = systolic pressure of the reference finger at cooling temperature (t); ( P_{c30} ) and ( P_{ct} ) = respective values of the cooled finger</td>
</tr>
<tr>
<td>Rewarming index 1 (RWI 1)</td>
<td>( \text{FTI} = \text{FTI} - (T_{1AT1}/T_{5FT5} - T_{1AT1}/T_{5FT5}) )</td>
</tr>
<tr>
<td>Rewarming index 2 (RWI 2)</td>
<td>( \text{FTI} = \text{FTI} - (T_{1AT1}/T_{5FT5} - T_{1AT1}/T_{5FT5}) )</td>
</tr>
<tr>
<td>FTI, baseline temp (°C)</td>
<td>10 mm Hg, 10 min cooling posttest.</td>
</tr>
<tr>
<td>T1AT1, baseline arm temp (°C)</td>
<td></td>
</tr>
</tbody>
</table>
temperature to this analysis, these subjects were dropped entirely, except where there were at least two subsequent tests with complete skin temperature data. In those cases the second test became the baseline test and the subsequent test became the “follow up” test. Accordingly, the nomenclature of “initial” and “follow up test” does not always designate a minimum of two tests. In subjects where there were more than two completed tests, the first and last test was used. For all retested subjects, FSBP% was measured in the finger that was tested in the previous examination.

### Analysis

Data were analysed using the SAS statistical software package. Continuous data were summarised as means and standard deviations. Differences between group means were tested using one or two way analysis of variance and multiple comparison tests. Inter-year comparisons were made using McNemar’s test for grouped data and matched \( t \) tests for continuous data. Non-parametric tests were used for small data sets (n < 15). The relation between variables expressed as means was evaluated by linear regression. The difference between categorical data tabulated in 2 \( \times \) \( k \) contingency tables was tested by the \( \chi^2 \) test. A \( p \) value of 0.05 was chosen as the limit of statistical significance.

### RESULTS

Table 2 compares key results for all 204 subjects for the initial and follow up tests. The average age of the cohort at follow up was 43.9 years (range 25–70). There was a dramatic inter-year comparison of changes in FSBP% and finger temperature at maximum cooling, the results were significant, but they offered no advantage over the simple recovery temperature measured 10 minutes after rewarming; consequently they are dropped from later analyses. The FTI appeared to show relatively large proportional differences between tests, although the adjusted group deviation from baseline was <1°C. The variances were too large to afford meaningful comparison.

Table 3 presents comparative changes in Stockholm Workshop symptom stage between the initial and the follow up tests. There was only modest variation in symptom stage between tests, with 163 of 204 subjects (79.9%) remaining at their original stage when retested. Among the subjects who reported a stage change, 25 (12.3%) had deteriorated to a more symptomatic stage and 16 (7.8%) had improved to a less symptomatic stage. At the time of the initial test, there were 63 subjects with Stockholm Workshop Scale stage III symptoms. The number of subjects at stage III increased to 75 at the time of the follow up test. There were 129 subjects rated at Stockholm Workshop Scale stage I or II (herein, stage I–II) at follow up.

Table 4 presents 15 subjects with symptomatic improvement. Multiple regressions of changes in FSBP% and finger temperature were run on the full cohort, adjusted for initial levels and changes in clinical status. Changes in clinical status were not significant. Improvement in mean FSBP% from 47.0% to 67.8% in the improved subjects was principally due to a decrease in the proportion of subjects with FSBP% <30 from 40% to 20%, figures almost identical to the larger

### Table 2 Characteristics of all subjects comparing initial and follow up tests

<table>
<thead>
<tr>
<th>Variable name</th>
<th>Sample size</th>
<th>Initial test (95% CI)</th>
<th>Follow up test (95% CI)</th>
<th>Test difference (95% CI)</th>
<th>( p ) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>% smokers</td>
<td>204</td>
<td>61.76</td>
<td>63.80</td>
<td>-2.04</td>
<td>0.03</td>
</tr>
<tr>
<td>% subjects with FSBP% = 0</td>
<td>204</td>
<td>35.78</td>
<td>32.10</td>
<td>0.68</td>
<td>0.42</td>
</tr>
<tr>
<td>FSBP%</td>
<td>204</td>
<td>49.99 ± 5.39</td>
<td>63.40 ± 4.82</td>
<td>13.42 ± 4.96</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>FSBP% &gt;30%</td>
<td>204</td>
<td>77.38 ± 3.27</td>
<td>82.20 ± 2.41</td>
<td>-4.82 ± 3.63</td>
<td>0.01</td>
</tr>
<tr>
<td>Baseline arm temp (°C)</td>
<td>202</td>
<td>32.20 ± 0.20</td>
<td>32.10 ± 0.20</td>
<td>-0.10 ± 0.25</td>
<td>0.42</td>
</tr>
<tr>
<td>Finger temp (°C) at 10°C Cooling</td>
<td>202</td>
<td>24.65 ± 0.45</td>
<td>25.57 ± 0.49</td>
<td>0.92 ± 0.55</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Finger temp (°C) at 30°C rewarmed</td>
<td>201</td>
<td>24.64 ± 0.47</td>
<td>25.23 ± 0.51</td>
<td>0.58 ± 0.59</td>
<td>0.05</td>
</tr>
<tr>
<td>Finger temp (°C) at 10 min recovery</td>
<td>177</td>
<td>27.12 ± 0.59</td>
<td>27.99 ± 0.61</td>
<td>0.86 ± 0.65</td>
<td>0.01</td>
</tr>
<tr>
<td>Finger temperature index (FTI)</td>
<td>177</td>
<td>0.92 ± 0.47</td>
<td>0.44 ± 0.45</td>
<td>-0.47 ± 0.57</td>
<td>0.10</td>
</tr>
<tr>
<td>Rewarming index 1</td>
<td>201</td>
<td>-0.01 ± 0.20</td>
<td>0.33 ± 0.18</td>
<td>0.64 ± 0.25</td>
<td>0.01</td>
</tr>
<tr>
<td>Rewarming index 2</td>
<td>201</td>
<td>1.00 ± 0.01</td>
<td>0.99 ± 0.01</td>
<td>-0.01 ± 0.01</td>
<td>0.01</td>
</tr>
</tbody>
</table>

*The mean and standard error are calculated only for subjects with FSBP% >30.

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There was no statistically significant movement towards warmer skin temperatures in the symptomatically improved group after follow up cold challenge. The test finger temperature at 10°C cooling was on average 0.18°C warmer, improved group after follow up cold challenge. The test finger towards warmer skin temperatures in the symptomatically cohort. There was no statistically significant movement with an FSBP% 

Table 4 Characteristics of 15 subjects reporting clinical improvement, comparing initial and follow up tests

<table>
<thead>
<tr>
<th>Variable description</th>
<th>Sample size</th>
<th>Initial test ± SE</th>
<th>Follow up test ± SE</th>
<th>Test difference ± SE</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>% smokers</td>
<td>15</td>
<td>26.67</td>
<td>46.67</td>
<td>20%</td>
<td>0.38</td>
</tr>
<tr>
<td>% subjects with FSBP% = 0</td>
<td>15</td>
<td>6 (40%)</td>
<td>22 (90%)</td>
<td>–20%</td>
<td>0.38</td>
</tr>
<tr>
<td>FSBP% ≧ 30%</td>
<td>8</td>
<td>83.10 ± 5.73</td>
<td>87.28 ± 7.51</td>
<td>4.18 ± 8.63</td>
<td>0.73</td>
</tr>
<tr>
<td>Baseline arm temp (˚C)</td>
<td>15</td>
<td>32.09 ± 0.32</td>
<td>31.75 ± 0.34</td>
<td>–0.34 ± 0.43</td>
<td>1.00</td>
</tr>
<tr>
<td>Baseline finger temp (˚C)</td>
<td>15</td>
<td>29.00 ± 0.95</td>
<td>29.60 ± 0.86</td>
<td>0.60 ± 1.22</td>
<td>1.00</td>
</tr>
<tr>
<td>Finger temp (˚C) at 10°C cooling</td>
<td>15</td>
<td>25.33 ± 1.15</td>
<td>25.51 ± 0.93</td>
<td>0.18 ± 1.02</td>
<td>1.00</td>
</tr>
<tr>
<td>Finger temp (˚C) at 30°C rewarmed</td>
<td>15</td>
<td>25.95 ± 1.12</td>
<td>25.33 ± 1.90</td>
<td>–0.62 ± 1.11</td>
<td>0.58</td>
</tr>
<tr>
<td>Finger temperature index (FTI)</td>
<td>11</td>
<td>27.42 ± 1.57</td>
<td>27.13 ± 1.28</td>
<td>–0.29 ± 1.13</td>
<td>0.55</td>
</tr>
<tr>
<td>Rewarming index 1</td>
<td>15</td>
<td>–0.23 ± 0.76</td>
<td>0.21 ± 0.83</td>
<td>0.44 ± 0.68</td>
<td>1.00</td>
</tr>
<tr>
<td>Rewarming index 2</td>
<td>15</td>
<td>1.03 ± 0.018</td>
<td>1.00 ± 0.02</td>
<td>–0.03 ± 0.02</td>
<td>0.12</td>
</tr>
</tbody>
</table>

The inter-test patterns of skin temperature recovery are somewhat different. For both symptom stage groups, there were dramatic overall improvements in the FSBP%. Stage I-II subjects were actually lower for the stage III subjects compared with stage I-II subjects (59.2% versus 65.9%), the interval improvement between the tests was actually somewhat greater (15.3 versus 13.8%) for stage III subjects. None of these differences were statistically significant.

Table 5 compares subjects with stage III symptoms at follow up testing with stage I-II subjects. The purpose of this comparison was to assess any relation between the most severe symptoms and skin temperature change. In most respects, the groups were similar. Both age and years of exposure to vibratory tools did not differ between the two groups. The interval between last exposure and the follow up test was actually longer for the stage III subjects. The stage III subjects were actually somewhat lower for the stage III subjects (26.51 months), although the interval between the initial and follow up tests was virtually identical (12 versus 11 months). Current (but not former) smoking had been a significant predictor of delayed recovery of FSBP% in our earlier study, but smoking rates were virtually identical in the two groups. There were more stage III subjects at follow up with an FSBP% < 30 (25.3% versus 17.8% for stages I-II), but the proportional grouped differences between stage III and stage I-II subjects were not significant. The mean FSBP% differences between the two groups and at initial and follow up tests are interesting. Despite the modest changes in symptom stage between tests, there were dramatic overall improvements in the FSBP%.

The variations in finger skin temperature present a somewhat different picture. For both symptom stage groups there is an overall similarity in skin temperature measurements under conditions of cold challenge, and there is a mild relative improvement during the follow up test compared with the initial measurement. The two groups diverge, when the inter-test patterns of skin temperature recovery are compared. Stage I-II subjects improved on average by 1.49°C, whereas stage III subjects remained static, actually falling by 0.12°C when compared with the initial test. These group differences were not reflected in either baseline arm temperatures or test finger temperatures. This was the only inter-test parameter involving either FSBP% or skin temperature that significantly differed between the two groups. It is also notable that the stage III subjects were actually warmer at baseline initially and had a relatively larger inter-test increase in baseline digital skin temperature –0.90°C compared with 0.56°C in the comparison group. The relative inter-test improvement in all of the cold provocation related

Table 5 Comparative characteristics of symptom stage III subjects with all other subjects at the second test

<table>
<thead>
<tr>
<th>Variable description</th>
<th>Stage III (n = 75)</th>
<th>Other stages (n = 129)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at follow up test</td>
<td>44.74 ± 2.65</td>
<td>43.49 ± 2.04</td>
<td>0.47</td>
</tr>
<tr>
<td>Interval (mth) between last exposure and follow up test</td>
<td>35.68 ± 8.53</td>
<td>26.41 ± 4.86</td>
<td>0.04</td>
</tr>
<tr>
<td>Interval (mth) between last exposure and initial test</td>
<td>23.20 ± 6.39</td>
<td>15.52 ± 1.78</td>
<td>0.04</td>
</tr>
<tr>
<td>Years exposed on job</td>
<td>16.43 ± 1.96</td>
<td>15.50 ± 1.76</td>
<td>0.51</td>
</tr>
<tr>
<td>% smokers at follow up test</td>
<td>26 (34.67%)</td>
<td>45 (34.88%)</td>
<td></td>
</tr>
<tr>
<td>FSBP% ≧ 30% at follow up test</td>
<td>19 (25.3%)</td>
<td>23 (17.8%)</td>
<td></td>
</tr>
<tr>
<td>FSBP% at follow up test</td>
<td>59.16 ± 8.53</td>
<td>65.87 ± 5.76</td>
<td>0.18</td>
</tr>
<tr>
<td>FSBP% at initial test</td>
<td>43.87 ± 8.82</td>
<td>25.12 ± 6.80</td>
<td>0.16</td>
</tr>
<tr>
<td>FSBP% Δ between tests</td>
<td>15.28 ± 8.11</td>
<td>13.75 ± 6.27</td>
<td>0.82</td>
</tr>
<tr>
<td>Baseline arm temp (˚C) at follow up test</td>
<td>32.10 ± 0.33</td>
<td>32.11 ± 0.24</td>
<td>0.96</td>
</tr>
<tr>
<td>Baseline finger temp (˚C) at follow up test</td>
<td>30.48 ± 0.59</td>
<td>29.92 ± 0.55</td>
<td>0.19</td>
</tr>
<tr>
<td>Baseline finger temp (˚C) Δ between tests</td>
<td>0.90 ± 0.69</td>
<td>0.56 ± 0.67</td>
<td>0.88</td>
</tr>
<tr>
<td>Finger temp (˚C) at follow up test @ 10°C cooling</td>
<td>25.57 ± 0.82</td>
<td>25.58 ± 0.61</td>
<td>0.93</td>
</tr>
<tr>
<td>Finger temp (˚C) @ 10°C cooling Δ between tests</td>
<td>0.72 ± 1.12</td>
<td>1.16 ± 0.67</td>
<td>0.14</td>
</tr>
<tr>
<td>Finger temp (˚C) at follow up test @ 30°C rewarm</td>
<td>25.53 ± 0.88</td>
<td>25.09 ± 0.61</td>
<td>0.45</td>
</tr>
<tr>
<td>Finger temp (˚C) @ 30°C rewarm Δ between tests</td>
<td>0.46 ± 1.02</td>
<td>0.79 ± 0.33</td>
<td>0.52</td>
</tr>
<tr>
<td>Finger temp (˚C) at follow up test @ 10 min recovery</td>
<td>27.84 ± 0.92</td>
<td>28.16 ± 0.71</td>
<td>0.36</td>
</tr>
<tr>
<td>Finger temp (˚C) @ 10 min recovery Δ between tests</td>
<td>–0.12 ± 1.06</td>
<td>1.49 ± 0.73</td>
<td>0.03</td>
</tr>
</tbody>
</table>
measures—cooling, rewarming, post-rewarming—tended to be lower in stage III compared with stage I–II subjects. The overall pattern is not straightforward.

Figure 1 presents a fuller representation of the pattern of test finger skin temperature variation during the course of testing. All temperature measurements were taken after release of the occlusion cuff for the five trials described above (plus an uncuffed baseline), then at 5 minutes and 10 minutes after rewarming. Finger skin temperatures are essentially identical for the stage III and stage I–II subjects through the cooling sequences at follow up testing. In fact, only skin temperatures for stage I–II subjects at the initial test vary from the overall pattern, being lower. Stage III subjects at the initial and follow up tests had a lower FSBP% than stage I–II subjects. However, skin temperatures were actually lower for the latter group of subjects at the initial test and were identical to stage III subjects at the follow up test. In short, cooled and subsequent recovery temperatures were essentially similar for stage III subjects at the initial and follow up tests, and the large inter-test improvement for less symptomatic stage I–II subjects reflects lower skin temperatures at the initial test.

In a fuller regression model (table 6), rewarmed final temperature at the second test was regressed on age, latency from last exposure, years worked, smoking status, initial and follow up baseline digit temperatures and FSBP% measurements, and symptom status. Initial and follow up test finger baseline temperatures and initial test rewarmed final temperatures had significant associations. FSBP% was more loosely associated with rewarmed finger skin temperature; the symptom stage was not. For whatever value FST maintains as a predictor of long term symptom persistence, it is a poor cross sectional indicator of symptom stage in these workers.

**DISCUSSION**

The relation between FSBP%, symptom stage, and baseline skin temperature, and temperature recovery is complex. The juxtaposition of an improvement in digital arterial blood pressure against relative absence of skin temperature improvement is consistent with the observation that finger systolic blood pressure tests may record interruption of digital blood flow in a context of vasoconstriction, whereas skin temperature recovery tests are measures of vasodilation. By implication, while digital perfusion from arterial blood pressure and maintenance of skin temperature are associated, vibration may affect each system independently. The inability to improve skin rewarming among individuals with the most symptomatic cold hands seems to be characteristic of this population.

Several reservations specific to these particular tests require consideration. This was a hybrid test that combined elements of blood flow interruption with a cold stress. There may be advantages in the simultaneous administration of cold and mechanical stimuli. Since both FSBP and FST are outcomes of interest, cuff inflation and deflation, cooling and warming, and the induction of ischaemic intervals may create a test environment that is more representative of cold provocation under working conditions. However, skin temperature assessment is potentially contaminated and mechanical stresses cannot be isolated from temperature stresses. A second reservation applies to overly drawn inferences from temperature data featuring multiple outcome variables and small differences. The initial test results for stage III subjects (lower FSBP%, greater symptoms, and higher recovery temperature compared to stage I–II) presents an ambiguous picture. If skin temperature recovery following cold challenge was a univariate predictor of symptomatically cold hands, the most symptomatic subjects at the initial test should have had the slowest degree of skin temperature recovery after rewarming. They did not. The differential relative improvement in stage I–II participants on follow up might suggest that more moderate symptoms were related to better skin perfusion. However, this only applies in relative terms. In absolute terms, stage I–II subjects were non-differentiable from stage III subjects at follow up. Follow up was brief, however, and one year in the disease course of VWF, and improvements in FSBP might be mirrored later by skin temperature increase.

There was no unexposed control population, but published reference values also indicate a delay in recovery temperature in VWF patients. Lindsell and Griffin predicted a median digital recovery time of <240 seconds in healthy males, when 6°C was set as the recovery threshold. In our population

| Variable description | Coefficient (95% CI) | Pr>|t| |
|----------------------|----------------------|----------------|
| Test finger temp (˚C) after rewarming and an additional 10 min recovery period at initial test | 0.237 ± 0.143 | 0.0014 |
| Age at second test | 0.00205 ± 0.058 | 0.4902 |
| Interval (min) between last exposure and second test | 0.00110 ± 0.0144 | 0.8811 |
| Years exposed on job | 0.0468 ± 0.0697 | 0.1901 |
| Smoking status at second test | 0.399 ± 0.0932 | 0.4019 |
| Baseline finger temp (˚C) at initial test | -0.0927 ± 0.190 | 0.3410 |
| Baseline finger temp (˚C) at follow up test | 0.8305 ± 0.162 | <0.0001 |
| FSBP% at initial test | 0.00220 ± 0.0133 | 0.7458 |
| FSBP% at follow up test | 0.0264 ± 0.0152 | 0.0016 |
| Stockholm Workshop Scale | -0.390 ± 0.511 | 0.1359 |

Estimates of regression coefficients (standard errors) are shown.
median recovery was less than half as great at 600 seconds. However, their technique differed, involving gloving and circumferential sensor placement. An even more accelerated rewarming rate (~5.0°C/min) was described for control subjects cooled with a dual inlet cuff, similar to that used in these trials.11

Aneklo-Noblin and colleagues12 made similar cross-sectional observations that delayed skin temperature recovery was far more common than abnormal finger systolic blood pressure in symptomatic subjects. They posited that for many VWF cases, injuries were restricted to skin and its blood vessels, rather than to digital arteries. A defect in autonomic modulation of the feed capillary bed in VWF has been suggested. The measurement of vascular flux by laser Doppler, optically set at different skin depths, suggests significant differences in finger blood flow between deep and superficial vessels, with FST more closely related to the latter.12 There is also evidence that finger blood flow measured by laser Doppler diminishes progressively with decreasing external temperature.13 Moreover, in a study that has several parallels to this work, Allen and colleagues17 showed that 10.1% of a population of vibration exposed riveters were able to maintain arterial perfusion during cold challenge, measured by laser Doppler, but had prolonged reductions in capillary flux after rewarming. Symptoms were not, however, prominent, even when FSBP was abnormal.

The lack of concordance between skin temperature and symptoms has been described. The monitoring of digital skin temperature in VWF patients, after cold challenge, has been extensively reviewed.14–16 Because there is such variety in temperature recovery measurement techniques, comparisons are difficult; of more significance, there has been little obvious relation between test results and reported symptoms. A recent cross sectional evaluation of more than 20,000 subjects in the UK employed cold provocation testing using skin temperature as the principal test outcome.19 There was no correspondence with symptoms of vibration related vasospastic disease assessed by the Stockholm Workshop Scale.9 The availability of longitudinal data in the current study offers a perspective on FST maintenance and recovery that has not been available in other comparable studies.

In general, a more extreme vasoconstrictive response implies a slow pattern of skin temperature recovery. Exaggerated vasoconstriction can inhibit the normal periodic increase and decrease in skin temperature during cold exposure.20 While pathological vasoconstrictor mechanisms may account for some of the delay in FST recovery, impairments in vasodilation may also be important. There is substantial evidence that VWF is a consequence of impaired vasoregulatory function in the hand.21 The circulation in the fingers involves cholinergic, adrenergic, and serotonergic receptors with interplay between vasoregulatory systems.22 Pharmacologically demonstrable defects occur at the alpha 1-alpha 2 axis23 and effect EDRF (endothelium derived relaxation factor) controlled vascular tone.24 There are anatomic and vasoregulatory correlates. Capillary regulation occurs at the epidermal ridge, 2 mm below the skin surface, while arterial structures are deeper (the digit has very limited collateralisation). Capillary flow is primarily mediated by neurotransmitter vasoregulation, in contrast to larger arteries that are under myogenic (arterial smooth muscle) regulation. Aneklo-Noblin and colleagues12 observed that smokers with symptomatic vibration induced Raynaud’s and comparably exposed and symptomatic non-smokers both experienced a significant drop in FSBP% on cold challenge. The mechanism differed: smokers had both an arterial vasoconstrictor and peripheral cutaneous response; non-smokers only had reduction in skin circulation. Cherniack and colleagues13 made a similar observation, and hypothesised that the prolonged relative morbidity of smokers with VWF after exposure was related to arterial vasoconstriction. Based on these observations and results from the current study, it seems possible that impaired cutaneous circulation provides a general explanation for unimproved FST in the setting of improvement in FSBP. The mechanisms are not understood, but may reflect receptor effects at the level of arteriovenous anastomoses.12 Pacinian corpuscles, which are extremely responsive to vibration, are commonly located at these sites. A combination of impaired endothelium mediated vasodilation and impaired local vasoconstriction accords with results presented in this study.

Vasoconstrictor effects on delayed skin temperature recovery are, over time, potentially less important as cold hands become more directly related to impairments in vasodilatation. Allen and colleagues17 found anamnestic information to be rather unreliable. As acute dramatic vasoconstrictive episodes lessen, individuals may become more attuned to cold hands and chronic symptoms. This does not satisfactorily explain why recovery temperature was cooler in the initial test for stage I–II subjects or why symptom associations are poor. It does suggest that symptoms may coincide with competing physical events.

The results illustrate the problems posed by assuming potential associations between finger skin temperature, cold provocation, and symptoms in VWF. Under some test conditions and for selected test protocols, the recovery pattern of skin temperatures may be a tool for disease differentiation.22 However, prolonged symptom severity in the setting of improving vasospasm and diminished temperature recovery, as reported here, while potentially specific to the population and experimental technique, makes broad application conditional.

CONCLUSION

This study suggests that a sizeable fraction of subjects with exposure to vibratory tools, who report prolonged cold related vascular symptoms, have delayed recovery in finger skin temperature following cold challenge. This pattern is evident in subjects who remain symptomatic, several years after removal from exposure, despite significant improvement in cold challenged FSBP. Unlike finger systolic blood pressure, neither symptom stage nor skin temperature substantially change on follow up, although the association between symptom stage and skin temperature is weak. Stockholm Workshop vascular symptom stage does not seem to predict skin temperature or longitudinal improvement in FSBP%.

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REFERENCES


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Secondhand smoke exposure remains a risk in Massachusetts restaurants despite widespread adoption of smoking regulations

A survey of restaurant smoking regulations in the 351 towns and cities in Massachusetts found that 82% of adults and 82% of youths are not guaranteed protection from secondhand smoke in restaurants in their town of residence. The same proportion of restaurant workers and 87% of bar workers are similarly not protected.

The survey looked at the smoking regulations from each town relating to customers and employees and classified them into eight categories according to whether there were no restrictions in place, limited restrictions (such as smoking only in separate ventilated areas or bar areas) or a complete ban on smoking. The proportion of the population in each town covered by the regulations was estimated from census data. The number of bar and restaurant staff was estimated to be proportional to the town population.

Although 225 towns had adopted some type of smoking regulation only 60 (covering 17.7% of the population) completely banned smoking in restaurants. The remainder restricted smoking in some way—174 of these to bar areas or separately ventilated areas, although 35 of these still allowed for variation to the regulations.

This study shows that classifying some restaurants and bars as “smoke free” may be misleading, and argues that there may be customers, and especially restaurant and bar staff, who are still exposed to cigarette smoke. It also calls for public health workers to tighten up their implementation of smoking regulations.

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