Reflex sympathetic dystrophy and repetitive strain injury: temperature and microcirculatory changes following mild cold stress

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Summary

Temperature and blood flow studies were performed in the upper limbs of six patients with reflex sympathetic dystrophy (RSD), nine patients with repetitive strain injury (RSI) and 12 control subjects using thermography, laser Doppler flowmetry, infrared photoplethysmography and venous occlusion strain gauge plethysmography. The contralateral responses of the symptomatic and asymptomatic limbs were examined after being subjected, separately, to mild cold stress (20°C for 1 min). Altered thermoregulation and haemodynamics were evident in RSD. Though the pattern of response to contralateral cold challenge is similar to normal in RSI, vasodilatation and reduced vasomotion appears to be characteristic in this condition. Such changes may assist in distinguishing between RSD and RSI from other causes of chronic upper limb pain.

Introduction

We have shown that upper limb temperature and microcirculatory dynamics are altered in reflex sympathetic dystrophy (RSD) in response to mild cold stress of the unaffected limb[1,2]. Since RSD can complicate any penetrating or non-penetrating injury to a limb, including soft tissue injuries such as repetitive strain injury (RSI), in clinical practice it is important to distinguish these conditions[3]. To determine if temperature and microcirculatory abnormalities also occur in RSI the response to cold challenge was evoked and compared in patients with RSD, RSI and control subjects.

Methods

Six patients (one man, 5 women; mean age 39.0±11.7 years) with RSD, nine patients (three men, six women; mean age 39.8±13.6 years) with RSI and 12 control subjects (5 men, seven women; mean age 30.1±8.6 years) were included in the investigation. RSD was defined as persistent debilitating upper limb pain following injury with typical colour changes associated with allodynia and hyperpathia, with or without swelling and with or without limitation of joint movement. RSI was defined as chronic upper limb pain occurring in otherwise fit patients performing repetitive tasks, for example keyboard operating or playing a musical instrument, and in whom a specific lesion of joints, tendons or muscle had been confidently excluded. Apart from their index condition all of the patients were otherwise fit. None were hypertensive or being treated with drugs with a potential action on the cardiovascular system, haemostasis or blood rheology. In particular, none were being treated with aspirin, or any drug with similar activity, for more than a week prior to testing.

On the study day patients were requested to have a light breakfast, to avoid alcoholic or caffeinated drinks and to refrain from vigorous exercise. Those who smoked were asked not to do so from the day before the study.

The method of study has been described previously[1,2,5]. In brief, the patients were seated, lightly clad, in a temperature and humidity controlled laboratory (24±1°C; RH 30-40%). The forearms were exposed and supported on a frame so that the hands were held at heart level with palms uppermost. Mean hand temperature was measured using thermography[1,4-6], microcirculatory velocity (flux) with laser Doppler flowmetry[7-9] and microcirculatory volume with infrared photoplethysmography (AC-beat by beat-output)[10,11]. Venous occlusion strain gauge plethysmography was used to measure total blood flow in the forearm and finger[12,13]. The laser Doppler probe was attached to the pulp of the middle finger of the symptomatic hand and the photoplethysmography probes to the pulps of the fourth fingers of both hands. The finger strain gauge was applied to the distal phalanx of the index finger of the symptomatic limb and the inflatable cuff wrapped around the proximal phalanx of the same finger. The arm strain gauge was applied just below the elbow on the forearm of the symptomatic limb and the corresponding cuff around the same upper arm. Measurements at baseline were recorded for a minimum of 10 min to obtain steady-state temperature and blood flow. Once equilibrium had been achieved, the asymptomatic hand was covered in a fine polythene glove, to avoid wetting the hand, and subjected to mild cold challenge by immersion in water at 20°C for 1 min. The glove was removed and the hand returned to the frame, in its original position and a further 10 min of data was recorded.

After a second period of equilibration, the same procedure was repeated, but with the instrument probes connected to the asymptomatic hand and the symptomatic hand subjected to the same cold stress. With the control subjects, since neither limb was affected the first hand stressed was randomized across the group.

All data were collected and recorded using a data acquisition program running on an IBM personal computer.

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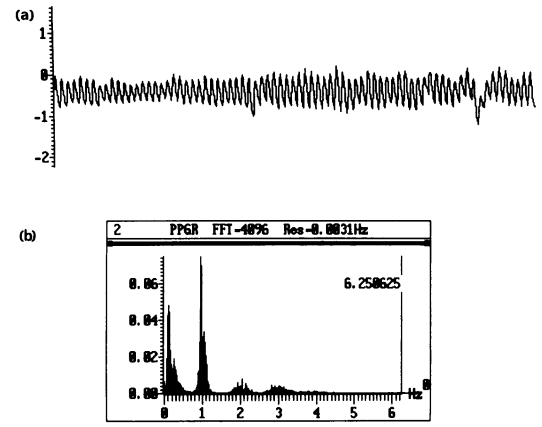

Figure 1. (a) Circulatory harmonics obtained by fast Fourier transform (FFT) of the photoplethysmography (PPGR) complex. (b) Vasomotion occurs between 0 and 0.10 Hz

Analysis

All data were analysed using 'Minitab' (version 7.1). The average value of the last 5 min of the raw data of each epoch for each parameter, before and after cold stress, was used for statistical analysis. Circulatory harmonics (Figure 1) were obtained by spectral analysis of the photoplethysmographic (PPG) complexes using a fast Fourier transform (FFT). Vasomotion, the spontaneous constriction and relaxation of arterioles[14], occurs in the frequency spectrum around six cycles per minute[15] and its power was calculated as a percentage of the total PPG signal power[16]. Baseline, post stress and percentage change from baseline for each variable were compared using the Mann-Whitney *U*-test for non-parametric data. Statistical significance was set at the 5% level.

Results

There was no significant difference in the sex distribution of the groups, or the duration of disease (RSD 26.6±17.1 months; RSI 17.1±20.5 months); the RSD group (1 man, 5 women; mean age 39.0±11.7 years) was significantly older ($P<0.04$) compared to the controls (five men, seven women; 30.1±8.6 years). One patient with RSD, four patients with RSI and two controls smoked cigarettes. Analysis showed that smoking had no significant influence on the results, which are summarized in Table 1 and Figure 2.

Mean hand temperature

Though the difference in mean hand temperature between the groups, pre- or post-cold stress was not statistically significant, the equilibrated mean temperature of the symptomatic hand in RSD was 2°C cooler than controls and 1.6°C cooler than the RSI group (Figure 2a). Also, cold stress of the asymptomatic hand was associated with an increase in mean temperature of the symptomatic hand which rose 0.7% from baseline, whilst in RSI and controls the hands cooled by 1.8% and 3%, respectively; though consistent, these differences were not statistically significant.

Microcirculatory flux

Absolute values of microcirculatory flux in the symptomatic hand in RSD were not significantly different from controls either pre- or post-cold stress. However, the percentage change following cold stress, a 32% increase in RSD compared to a 12% decrease in controls was significant ($P<0.05$). The RSI group had significantly higher basal and post stress microcirculatory flux in the symptomatic hand ($P<0.05$) but showed a non-significant increase after cold challenge (Figure 2 b).

Table 1. Hand temperature and blood flow (median±1 SD) in the upper limb of controls and patients with reflex sympathetic dystrophy (RSD) and repetitive strain injury (RSI) with the percentage change following mild cold challenge (20°C for 1 min) of the contralateral hand

	RSD	*RSI*	*Controls*
Mean hand temperature (°C)			
Baseline	29.1±1.7	30.7±2.4	31.1±2.3
Post cold challenge	29.3±1.9	30.3±2.0	30.6±2.4
Change (%)	+0.7±4.4	−1.8±4.4	−2.9±3.7
Microcirculatory flux (voltage output)			
Baseline	1.37±0.46	3.19±1.23*	2.01±0.91
Post cold challenge	1.53±0.61	3.17±1.25	1.72±0.89
Change (%)	+32.4±29.7*	+0.3±23.6	−12.2±22.4
Microcirculatory volume (arbitrary units)			
Baseline	2.02±2.33	5.86±3.04	2.75±5.66
Post cold challenge	1.84±1.52	4.63±3.30	2.10±5.07
Change (%)	−4.2±45.3	−10.0±31.8	−16.5±39.5
Total arm blood flow (ml/min/100 g)			
Baseline	2.90±0.78	2.65±1.09	3.37±3.00
Post cold challenge	3.43±1.42	2.75±2.08	2.59±2.70
Change (%)	+18.3±26.0	+17.5±33.5	−17.4±40.0
Total finger blood flow (ml/min/100 g)			
Baseline	5.10±2.54*	8.07±5.82	−12.45±7.76
Post cold challenge	4.15±5.64	11.78±4.47	10.89±8.04
Change (%)	+0.2±51.7	+5.6±53.4	−22.7±40.7
Vasomotion (%)			
Baseline	3.27±2.00	1.40±2.10*	3.64±29.94
Post cold challenge	1.44±30.07*	1.98±2.54*	5.85±33.40
Change (%)	−11.6±104.5*	+39.0±158.0	+39.1±336.2

*Significant differences (SD) from controls ($P<0.05$)

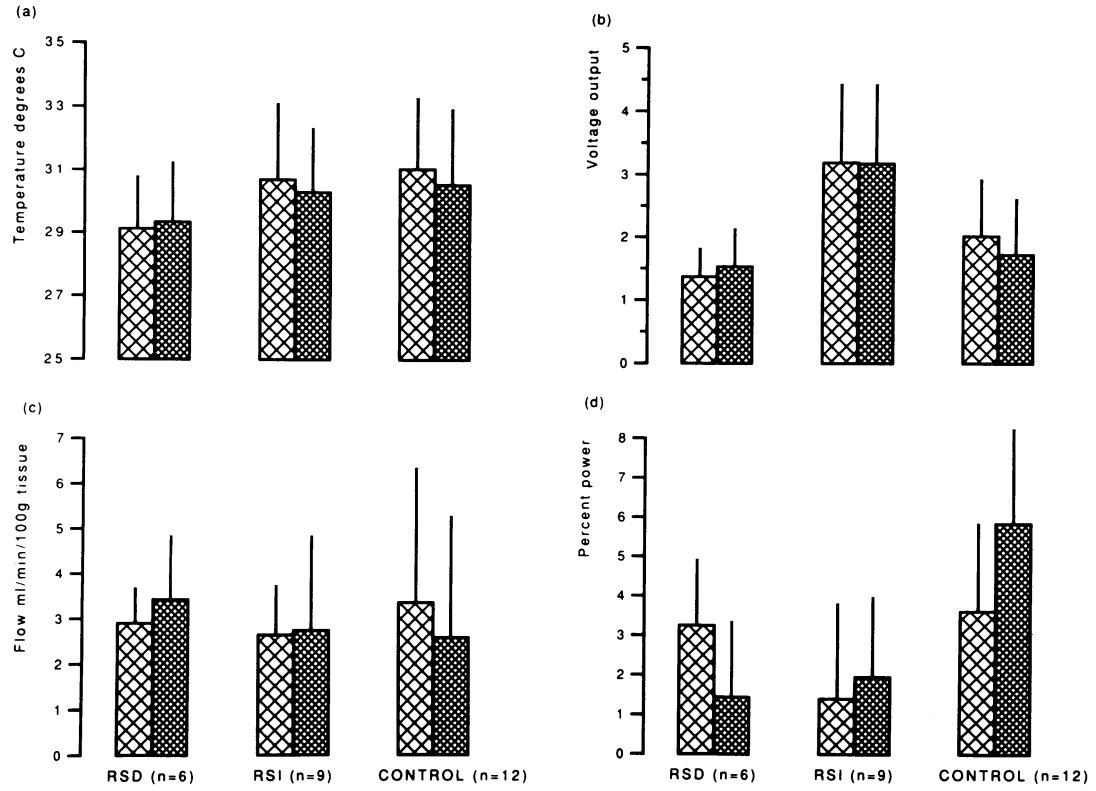

Figure 2. Showing the patterns of change. (a) In hand temperature: note the lower baseline temperature and difference in direction of the response of reflex sympathetic dystrophy (RSD) after cold challenge of the contralateral (asymptomatic) hand. (b) In microcirculatory flux: note a similar pattern of response in RSD and the high flux in repetitive strain injury (RSI) unchanged following contralateral cold challenge. (c) In total arm blood flow: note the increase in total flow in RSD after contralateral cold challenge and the fall in controls. Values are unchanged in RSI. (d) Vasomotion: note the low basal value in RSI and the increase in RSI and controls after contralateral cold challenge in contrast to the fall in RSD. The apparent discrepancies between this graphical presentation of data and the calculated percentage changes (Table 1) are due to the use of median values. The overall pattern of the response is unchanged. ☒ =Baseline; ▩ =post cold stress

Microcirculatory volume

Median microcirculatory volume in RSI at equilibration was approximately double that in RSD and controls but did not reach statistical significance.

Total arm blood flow

Total arm blood flow, increased by 18.3% in RSD and by 17.5% in RSI after contralateral cold challenge in contrast to a 17.4% decrease in the control group (Figure 2c). These differences were, however, not statistically significant.

Total finger blood flow

At equilibration, total blood flow in the index finger of the symptomatic hand was significantly lower in RSD than controls ($P<0.05$), but not in RSI. After cold challenge neither patient group differed significantly from controls.

Vasomotion

The basal content of vasomotion was significantly different from controls in RSI ($P<0.05$) but not in RSD. After contralateral cold stress, both RSD and RSI showed significantly lower vasomotor activity than controls ($P<0.05$); however, whilst RSI and controls both showed a marked increase after contralateral cold stress, RSD showed a significant decrease ($P<0.05$; Figure 2d).

Measurements of temperature and blood flow in the ipsilateral hand at both baseline or on direct cold challenge, or of the unaffected hand before and after cold challenge of the affected hand, yielded no useful information.

Discussion

The expected (normal) contralateral response to the vasoconstrictive stimulus of cold, namely a fall in hand temperature and blood flow, occurred in the control subjects. Therefore, it is reasonable to describe a reversed response, an immediate and persistent rise in hand temperature with an increased blood flow, as occurred in RSD, as paradoxical. Moreover, though only the reduction in vasomotion and increase in microcirculatory flux were statistically significant (Table 1), the pattern of change in the other parameters (Figure 2b-c), mirror these, confirming our previous observations[1,2].

RSI at baseline showed vasodilatation with microcirculatory flux significantly higher and remaining largely unchanged on contralateral cooling. Similarly microcirculatory volume at baseline was approximately double that in RSD and controls. The latter value did not reach statistical significance probably because of the variability of this parameter in controls (see SD, Table 1). Basal vasomotor activity in RSI was significantly lower than controls and on contralateral cooling increased following the normal pattern (Figure 2d). Thus the response to contralateral cold challenge in RSI either followed the normal pattern or remained unchanged, as might be expected if the persistent vasodilatation was due to an inflammatory process or to the conversion of, at least some, peripheral α-adrenoceptors to β (vasodilatory) characteristics[17,18]. This implies that there is no thermoregulatory dysfunction in RSI in contrast to the abnormal thermoregulatory behaviour in RSD where the stimulus appears to be misinterpreted.

Considering the clinical presentation of RSD, the observation of a lower temperature than normal associated with vasoconstriction, or indeed altered vasomotion, is not unexpected. More surprising was the increase in basal microcirculatory flux and reduced vasomotion in RSI which would not be suspected from the usual clinical presentation of these cases. However the preservation of the normal thermoregulatory response after cold challenge suggests that these changes are likely to be reversible.

This is, as far as we are aware, the first description of abnormal vasomotion in RSI and RSD.

We recognize that vasodilatation[19] occurs early in RSD and is followed weeks or months later by vasoconstriction, the stage which all the RSD cases in this study had reached before referral. Unfortunately, it is still rare to have patients referred soon after the onset of the disease. However, experience indicates that whether vasodilated or vasoconstricted the 'paradoxical' response to cold challenge is characteristic.

These observations indicate that RSD and RSI are distinct entities, a view strengthened by the fact that the patients were assessed at roughly the same time after the onset of their disability. Clearly, differences in the thermoregulatory response may assist in distinguishing between these conditions and other causes of chronic upper limb pain.

Present experience suggests that patients with RSI are unlikely to develop RSD but this remains unproved.

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