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

E D Cooke MD1, M D Steinberg BEng2, R M Pearson MRCP3, C E Fleming BS, RCN1
S L Toms BS1, J A Elsade BS1, Departments of 1Medical Electronics and 2Clinical Pharmacology, St Bartholomew's Hospital, London EC1A 7BE, UK

Keywords: reflex sympathetic dystrophy; repetitive strain injury; vasomotion; thermography; blood flow

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. 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. 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±6.6 years) were included in the investigation. RSD was defined as persistent debilitating upper limb pain following injury with typical colour changes associated with algodystrophy 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. 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, microcirculatory velocity (flux) with laser Doppler flowmetry and microcirculatory volume with infrared photoplethysmography (AC-beat by beat-output). Venous occlusion strain gauge plethysmography was used to measure total blood flow in the forearm and finger. 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 laser 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.

Correspondence to: Dr E D Cooke