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ABNORMAL TEMPERATURE CONTROL SUGGESTING SYMPATHETIC DYSFUNCTION IN THE SHOULDER SKIN OF PATIENTS WITH FROZEN SHOULDER

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SUMMARY

In view of the possibility that sympathetically mediated pain could be responsible for frozen shoulder symptoms we compared shoulder skin vasomotor control in 11 patients with frozen shoulder and 17 similarly aged normal subjects without shoulder pain. Using computer-assisted thermography the shoulder skin temperature was assessed before and following a 'cold challenge' which consisted of a 15°C cold pack being held against the skin for 60 sec. Both prior to and immediately following the cold challenge shoulder skin temperatures tended to be lower in the patients. During a 10-min rewarming phase, however, the between-group temperature difference increased and became significant at the 0.05 level. These abnormalities of temperature control in patients clearly suggest sympathetic dysfunction in the dermatome subserving pain sensation from the affected shoulder. Whether these abnormalities are primary or secondary remains unresolved.

Key words: Frozen shoulder, Sympathetically maintained pain, Thermography.

Frozen shoulder (FS) is a common clinical condition with a characteristic triphasic natural history [1,2]. The initial phase encompasses the onset of shoulder pain. This is usually idiopathic and may be severe, causing marked sleep disturbances and impairment of 'freezing' of active shoulder joint mobility. Although many authors specifically exclude subacromial bursitis, rotator cuff calcinosis and supraspinatus and bicipital tendinitis when defining FS [2–4] others suggest that these varied conditions represent the earliest stages or FS [1,5]. During the second phase pain-induced shoulder immobility leads to fibrotic and adhesive changes within the capsule [6,7] so that both active and passive joint movements become markedly reduced or truly 'frozen'. After a variable period, which usually lasts 1–3 years, there is sometimes a gradual improvement in joint mobility forming the last, or 'thawing', phase [1].

The aetiopathology of FS is unknown but local trauma, internal derangements, immobilization, and nerve root or trunk irritation due to cervical spondylotic or supraclavicular notch entrapment syndromes have all been suggested, but not proven, as causes as reviewed by Rizk and Pinals [1] and Bland *et al.* [5]. In view of the association between FS and the clinical, radiological and thermographic features of cervical spine disease many authors suggest that autonomic dysfunction is somehow aetiologically important [1,5,8]. Such a suggestion is strengthened by the observation that FS may occur in association with a variety of disorders which could alter autonomic function [1].

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Since the cause of FS is unknown it is hardly surprising that its treatment remains empirical, including the use of local steroid injections, physiotherapy using local and mobilizing techniques, anti-inflammatory agents etc, and some authors conclude that symptomatic improvements occur irrespective of therapeutic interventions [9,10]. The condition may be protracted and very painful and disabling. In order that treatments could be made more rational, and thus hopefully more successful, further insight into the cause/s of FS is required. The present temperature study was undertaken to investigate further the role of autonomic function in patients with idiopathic or 'primary' FS.

MATERIALS AND METHODS

Experimental subjects

Eleven patients (five male) with FS were compared with 17 normal control subjects (seven male) without shoulder pain. For both groups a past history of diabetes mellitus, myocardial infarction within the preceeding 18 months, previous pulmonary tuberculosis, Raynaud's phenomenon and ingestion of drugs with a potential for altering sympathetic function all caused exclusion, although the use of simple analgesics and non-steroidal anti-inflammatory agents was permitted. For this study, which was approved by the Salford District Health Authority Ethical Committee, diagnostic criteria for FS was defined as: (a) shoulder pain of more than 6 weeks duration; (b) limitation of active and passive movements in all directions and with reduction in external rotation of at least 50%; (c) absence of other causes of shoulder arthritis on clinical, radiological and haematological examinations; (d) absence of radiological evidence for calcific periarticular tendonitis, erosive disease or other abnormalities. All patients included were new referrals to the rheumatology clinic

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