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CLINICAL REPORT

Monitoring Healing of Acute Charcot's Arthropathy with Infrared Dermal Thermometry

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Abstract—The purpose of this study was to describe the use of skin temperature assessment in diabetics with acute Charcot's arthropathy to monitor resolution of inflammation longitudinally throughout the course of treatment and to predict development of neuropathic ulcers.

Thirty-nine diabetic subjects presenting with acute Charcot's arthropathy received thermometric monitoring throughout their treatment course. Subjects were treated with a standard protocol involving total contact casting, removable cast walkers, and progression to therapeutic shoes.

There was a steady decrease in temperatures during the casting regimen. After temperature gradients normalized, subjects were progressed to custom therapeutic shoe gear and insoles and were followed for a mean 22.6±7.1 months. Following quiescence, 8% returned during the follow-up period with a new-onset neuropathic ulceration. Temperature gradients during taken the visit prior to ulceration were significantly higher in this group than for the rest of the population.

Elevated temperatures were strongly correlated with the location of arthropathy. Temperatures decreased in a predictable manner as acute arthropathy resolved. Additionally, increased temperature gradients may be predictive of future ulceration.

Key words: *Charcot's joint, diabetes mellitus, foot, fracture, neuropathy, skin temperature, ulcer.*

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INTRODUCTION

Neuropathic fracture of the foot and ankle is a devastating complication of diabetes that often leads to severe deformity and permanent disability. Since its first descriptions by William Musgrave nearly three centuries ago, numerous theories have been propagated regarding the pathogenesis of neuropathic osteoarthropathy (1–4). Currently, the most commonly accepted theory is an amalgam of previous neurotraumatic and neurovascular theories. It has been theorized that following the development of autonomic neuropathy (autosympathectomy), there is a generalized increase in blood flow to an affected limb, resulting in osteopenia that weakens bones of the foot and ankle (5–7). In addition to this, motor neuropathies result in muscle imbalance that places abnormal stress on the foot and ankle. Sensory neuropathy renders the person unaware of the profound bony destruction taking place during ambulation (8,9).

Signs of acute Charcot's joint generally include ill-defined unilateral pain, edema, erythema, and associated warmth to the affected area (10). As the injury moves into a postacute phase, the frank signs of inflammation resolve. However, it is often difficult to determine when complete resolution has occurred, because subjective symptoms are often absent due to sensory neuropathy. In many instances, the clinical signs of residual inflammation are subtle and difficult to objectively grade or monitor during the course of healing (11).

Several investigators have reported on the use of skin temperature monitoring as a potentially useful tool to monitor progression of Charcot's arthropathy through its acute phase (10–14). However, we are unaware of any reports in the medical literature that describe the progression of skin temperatures from initiation of treatment of acute Charcot's arthropathy to healing. The purpose of this study was to evaluate skin temperatures at the site of acute Charcot's arthropathy during and after fracture healing using the contralateral limb as a physiologic control.

MATERIALS AND METHODS

The study included 39 subjects, 20 male, 19 female, with an average age of 59.0±9.5 years, presenting for care to an outpatient, referral-based, multidisciplinary diabetic foot clinic from February 1, 1991 to July 1, 1994. All subjects were diagnosed with diabetes mellitus based on the criteria set forth by the World Health Organization (15). The mean duration of diabetes was 16.5±4.9 years. Of all diabetic subjects, 97.4 percent were type II. All subjects had palpable pedal pulses on initial evaluation and clinical loss of protective threshold using the Semmes-Weinstein monofilaments by the method and criteria described by Birke (16).

Only persons diagnosed with acute Charcot's arthropathy were selected for study. Those with concomitant osteomyelitis, chronic Charcot's arthropathy, bilateral involvement, or open reduction of the fracture were excluded from analysis. Diagnosis was based on clinical, radiographic, and dermal thermometric criteria. A preliminary (clinical) diagnosis of osteomyelitis was made, utilizing a sterile blunt surgical probe (17,18). Wounds with tracts extending to bone were given a presumptive diagnosis of osteomyelitis. This was subsequently confirmed in all cases by microbiologic and histologic analysis.

Location of Charcot's arthropathy was described using the Sanders pattern classification. Sanders describes five different patterns of Charcot's arthropathy based on anatomic location. Pattern I (2.6 percent of the study population) indicates arthropathy located in the forefoot. Pattern II (64.1 percent) refers to involvement at Lisfranc's joint. Pattern III (25.6 percent) affects the bones of the lesser tarsus. Pattern IV (7.7 percent) affects the ankle joint. Pattern V (0 percent) affects the posterior calcaneus, also known as the "posterior pillar" (19).

Subjects were treated with a standard protocol involving serial total contact casting with progression to removable cast walkers and finally to prescription therapeutic shoe gear. Total contact casts were applied using the technique described by Kominsky (20). Casts were checked at regular intervals and evaluated for proper fit. Casts of those with concomitant ulceration were changed weekly for ulcer evaluation and debridement. Cast change intervals for subjects without ulceration were dependent on cast comfort and integrity (3 weeks maximum). Casting was discontinued based on clinical signs of quiescence, radiographic signs of trabecular bridging, and dermal thermometric equilibrium with the contralateral limb. Following casting, subjects progressed to removable cast walkers and then to prescription therapeutic shoe gear (with ankle-foot orthoses, as required). Following transition into shoe gear, subjects were followed at 2-month intervals for clinical signs of recurrence.

Skin temperatures were monitored using a portable infrared thermometric probe (Exergen Model DT 1001, Exergen Products, Watertown, MA). The temperature probe displays in increments of 0.1 °F and is accurate to within ±0.2 °F (21). The probe's infrared lens measures an approximately 1.0 cm^2 area of skin. The device is held approximately 0.5 cm from the skin surface during measurement (21). Temperature measurements were made after subjects were allowed to rest for 15 min in the examination room. Ambient air temperature was thermostatically controlled between 70.0±2.0 °F during the test period. Readings were recorded from seven sites on the sole of both feet, including the hallux, first, third, and fifth metatarsal heads, first metatarsocuneiform joint, talonavicular joint, cuboid, and heel. Additionally, readings were taken at the anterior ankle. We used the contralateral limb as a physiologic control, comparing skin temperatures on the affected foot to the corresponding site, contralaterally. The resulting difference formed a skin temperature gradient, which was recorded. A positive skin temperature gradient implied that skin temperatures were greater on the foot affected with acute Charcot's arthropathy. In each case the same anatomic site on the contralateral limb was used as a control. Subjects were followed for a mean 22.6±7.1 months following return to permanent shoe gear (range 12 to 37 months).

A Mann-Whitney U test was used to compare sex and initial temperature gradient. A Student's t-test for matched samples was used to compare temperature differences from one limb to the other. A Mann-Whitney U test was used to compare temperature gradients in reulcerated subjects to the rest of the population. A Fisher's

exact test was used to compare location of maximum skin temperature elevation to radiographic and clinical location of Charcot involvement. A Pearson's correlation was performed to compare the continuous variables of age and skin temperature gradient (22). We used an alpha level of 0.05 as a cut point for significance for all tests.

RESULTS

The mean skin temperature difference for all subjects at initial presentation was 8.8±2.3 °F (range 5.1 to 14.7). The site of maximum skin temperature gradient correlated to the site of maximum Charcot arthropathy (radiographically) in 92 percent of cases. The skin temperature gradient gradually decreased during total contact cast therapy (**Figure 1**). Following transition to prescription shoe gear, the skin temperature gradient was near zero (**Figure 2**). Although the gradient was generally small during the follow-up period, the site of maximum skin temperature gradient correlated to the site of maximum Charcot osteopathy/arthropathy in 72 percent of all cases throughout the follow-up period. There was not a significant difference in temperature gradient based on sex or age either at presentation or at any period following return to shoe gear.

Seventeen subjects (44 percent of total) initially presented with a concomitant grade IA ulceration using the University of Texas diabetic wound classification: a noninfected wound through full thickness skin not involving tendon, capsule, or bone (23). Distribution of these ulcerations correlated with location of maximum deformity as described by Sanders' pattern classification in every case. There was no significant difference in skin temperature

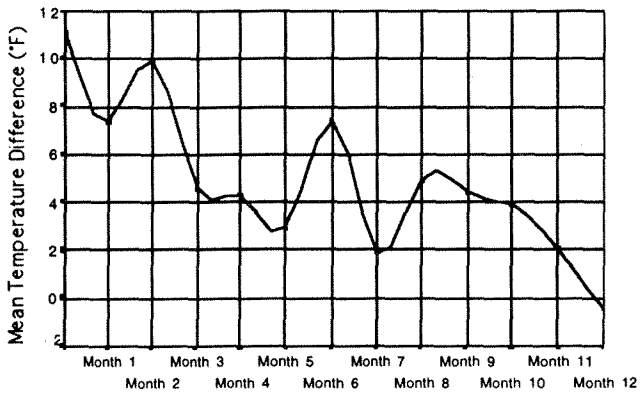

Figure 1.
Skin temperature progression during total contact casting.

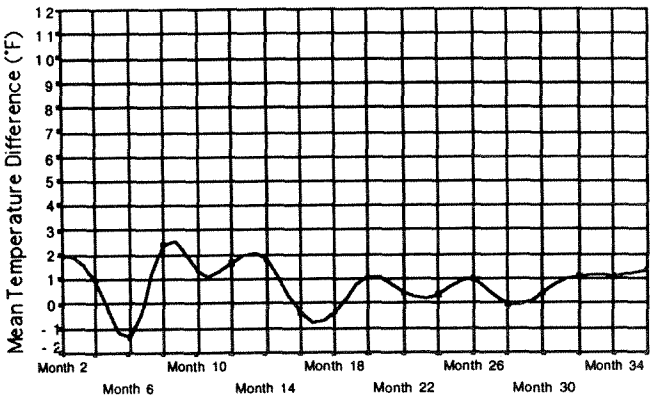

Figure 2.
Skin temperature progression following return to shoe gear: nonreulcerated.

gradients on initial presentation between ulcerated and nonulcerated subjects (1.5±1.1 vs. 2.2±1.4 °F). Additionally, there was not a significant difference in treatment duration based on whether or not the person presented with an ulcer (25.0±13.0 vs. 24.1±10.2 weeks).

Following resolution of acute Charcot's arthropathy, three subjects (7.7 percent of total population) returned during the follow-up period with a new-onset plantar neuropathic ulceration. These wounds appeared at a mean 11.0±1.7 months following return to therapeutic shoe gear. All wounds were grade IA in depth using the University of Texas wound classification (23). Interestingly, all three who ulcerated during the follow-up period had midfoot ulcerations on initial presentation with acute Charcot's arthropathy, but sites of reulceration were located in the forefoot. Skin temperature gradients (taken at the site of reulceration) for the visit prior to reulceration were significantly higher in this group than for the rest of the population at the corresponding clinic visit (4.5±0.9 vs. 0.9±0.9 °F, $P<0.001$). There was not a significant difference in skin temperature gradients at the time of initial presentation (9.7±1.6 vs. 8.7±2.3) or at the time of return to shoe gear (1.4±0.8 vs. 1.9±1.3) between reulcerated subjects and the rest of the population (**Figure 3**).

DISCUSSION

The results of this study suggest that elevated skin temperatures are directly correlated with location of acute neuropathic osteoarthropathy. These temperatures decrease in a predictable manner as acute Charcot's

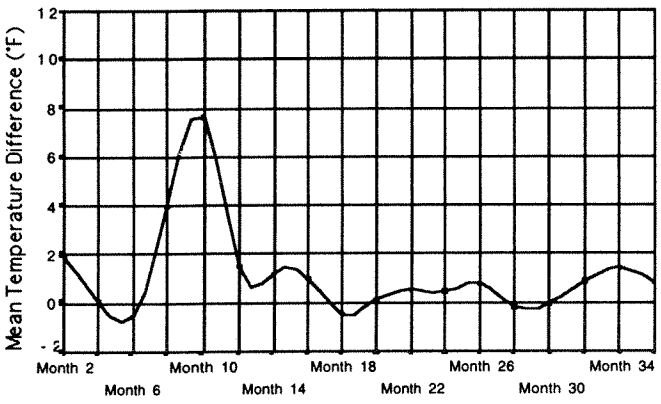

Figure 3.
Skin temperature progression following return to shoe gear: reulcerated.

arthropathy resolves into a post-acute state. Additionally, the subjects that reulcerated during the follow-up period (7.7 percent of the total population, 17.6 percent of the ulcer population) all showed significantly elevated skin temperature gradients on the visit prior to ulceration. This suggests that elevated skin temperature gradients may be predictive of future ulceration.

In the acute stage of the Charcot's arthropathy, the affected area is often grossly erythematous, edematous, and warm to the touch. In the post-acute phase, when gross signs of inflammation have resolved, premature return to activity can trigger another acute episode (24). Skin temperature measurements can be used to detect subtle temperature changes that may persist for months after a palpable difference can no longer be perceived. We use normalization of temperatures combined with clinical and radiographic signs of resolution as a benchmark to determine when guarded weightbearing should begin. Temperatures on the sole of the foot are measured and recorded at every follow-up visit and compared to the contralateral limb to monitor subtle signs of inflammation. In this study, there were no recurrences of Charcot fractures in an average follow-up period of approximately 2 years. The low rate of reinjury was probably at least in part a result of aggressive early intervention when subjects demonstrated an increase of more than 4 °F compared to the opposite foot. They were immobilized in a removable cast walker or total contact cast and instructed to sharply limit their activity. The cutoff point of 4 °F is based on our intuition at this point. Clearly, further work in this area is necessary to more firmly quantify a specific temperature range at which intervention may be warranted. Based on our observations, gradients as high as 3 or 4 °F are often difficult for a clinician or subject to perceive by palpation. Additionally, objectively quantifying temperature is problematic between visits or between clinicians. The instrument used in this study fits in the pocket of a lab coat and is simple to use, noninvasive, and relatively inexpensive. Temperatures can be measured and recorded in a matter of seconds.

We have used the opposite limb as a control because it is exposed to the same duration and control of diabetes and systemic complications as the affected limb and should represent a built-in source of comparison. Because the disease process of neuropathic fractures involves multiple factors that affect lower limb perfusion and temperature regulation, it would be difficult to identify an absolute skin temperature level that could be considered normal or one that could be used as a universal reference. In fact, the baseline temperatures for a person with Charcot's fracture may be higher than diabetics without this complication or persons without diabetes.

The protocol used in this population was very conservative, employing total contact casts and removable cast walkers for 24.5 ± 11.3 weeks on average. One of the limitations of this protocol may have been the strict policy of casting and immobilization until temperatures normalized. The true course of Charcot's arthropathy and corresponding temperature changes and reinjury could probably be appreciated better in an environment where clinical decisions were not based on subtle comparative changes of skin thermometry.

This is an area with tremendous potential, but one that will require much additional work to clarify the role of skin temperature measurements combined with clinical parameters to improve decision making. Since the prevalence of reinjury in persons with Charcot arthropathy is high (25), quantitative information to identify patients at risk could significantly improve recidivism.

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