Effect of High-Voltage Pulsed Current on Recovery After Grades I and II Lateral Ankle Sprains

Frank C. Mendel, Michael G. Dolan, Dale R. Fish, John Marzo, and Gregory E. Wilding

Context: High-voltage pulsed current (HVPC), a form of electrical stimulation, is known to curb edema formation in laboratory animals and is commonly applied for ankle sprains, but the clinical effects remain undocumented. Objective: To determine whether, as an adjunct to routine acute and subacute care, subsensory HVPC applied nearly continuously for the first 72 h after lateral ankle sprains affected time lost to injury. Design: Multicenter, randomized, double-blind, placebo-controlled trial. Setting: Data were collected at 9 colleges and universities and 1 professional training site. Participants: 50 intercollegiate and professional athletes. Interventions: Near-continuous live or placebo HVPC for 72 h postinjury in addition to routine acute and subacute care. Main Outcome Measure: Time lost to injury measured from time of injury until declared fit to play. Results: Overall, time lost to injury was not different between treated and control groups ($P = .55$). However, grade of injury was a significant factor. Time lost to injury after grade I lateral ankle sprains was greater for athletes receiving live HVPC than for those receiving placebo HVPC ($P = .049$), but no differences were found between groups for grade II sprains ($P = .079$). Conclusions: Application of subsensory HVPC had no clinically meaningful effect on return to play after lateral ankle sprain.

Keywords: soft-tissue injury, electrotherapy, return to play, time lost to injury, ligament injury

Ankle sprains are the most common injury in intercollegiate athletics and result in significant time lost from practices and games. Electrical stimulation, high-voltage pulsed current (HVPC) in particular, has long been used as a supplement to rest, ice, compression, and elevation (RICE), the near universal management strategy for ankle sprains. Our group has repeatedly demonstrated on laboratory animals that HVPC, alone or in combination with cold, effectively curbs edema formation, a cardinal sign of inflammation, if applied immediately after acute injury.
Edema formation was curbed by intermittent application of HVPC, but even more so if the treatments were continuous throughout the period when edema was actively forming. Clinicians apply RICE and HVPC to sprains, in part, to control edema in the belief that doing so eases acute symptoms, which may, in turn, affect rate of recovery of normal function. The efficacy of HVPC on ankle sprains was challenged by Michlovitz et al, who reported no effect on pain, swelling, or range of motion. Extrapolating from our animal trials, we hypothesized that HVPC would curb edema formation by reducing permeability of microvessels and thereby hasten recovery in competitive athletes after lateral ankle sprains. We designed this study to determine whether adding nearly continuous application of HVPC for the first 3 days postinjury to routine acute and subacute management practices would affect rate of recovery of competition-level function after grades I and II lateral ankle sprains in intercollegiate and professional athletes.

Methods

Study Design

Our study was a multicenter, randomized, double-blind placebo-controlled trial. The independent variable was HVPC (live or placebo) and the dependent variable was time, in days, from injury until the athlete was declared fit to play (FTP). In brief, all participating intercollegiate or professional athletes received whatever treatments their certified athletic trainers (ATs) and team physicians chose to administer for lateral ankle sprains. In addition, about half, randomly assigned, received active subsensory cathodal HVPC for 72 hours after injury, and the other half received placebo stimulation. Investigators, administering ATs, and participating athletes remained blind to which state of stimulation was provided until after the study ended. Time, in days, from injury until the athlete was declared FTP was our primary outcome. Other variables that we monitored included medications and HVPC compliance.

We recruited and trained ATs from the Buffalo Bills, a professional team in the National Football League, and from the following colleges and universities with intercollegiate teams: Alfred University, Canisius College, Central Connecticut State University, Edinboro State College, Hamilton College, Ithaca College, Marist College, Niagara University, St John Fisher College, and the University at Buffalo. Depending on the size of the institution, we trained 1 or 2 ATs to be the primary administrators and data collectors at each site. Other ATs at these sites also received training in the mechanics of the study so they could inform athletes and potentially recruit and initiate them should they sustain qualifying injuries. All ATs were compensated depending on level of involvement, directly or to their institutions. Athletes who made good-faith efforts to complete the study and who were deemed eligible by their compliance officers (COs) were also compensated via their COs. The CO is the administrative officer who ensures that the college or university is in compliance with National Collegiate Athletic Association and conference rules and regulations.

The study was conducted over 3 academic semesters beginning in the fall (August) of 2005 and concluding at the end of the fall semester (December) of 2006.
Participants

Sixty-five athletes between the ages of 17 and 33 years (mean 20.3) signed consent forms and started the protocol, and 50 of those had complete data sets. A minor and 2 athletes later found to have fractures were excluded. Eleven dropped out because they did not have the time or want to spend the time necessary to comply with the protocol. One athlete developed a mild rash, and we instructed the attending AT to terminate the protocol. Subsequently, 10 other athletes developed rashes (4 in the treated group and 6 in the control group). For these athletes, as soon as irritation was detected, electrode stockings were removed and stimulation ceased, but data collection continued. All rashes disappeared within 48 hours with no treatment.

Consent forms were approved by the institutional review board of each participating institution except the NFL team, which used the approved consent from the sponsoring institution. On injury, the AT on scene explained the study, including risks, and invited participation. If the invitation was accepted, the AT acquired and witnessed the athlete’s signature, provided a copy of the informed consent, and initiated the protocol.

To be eligible an athlete had to be a Buffalo Bill or a student-athlete at one of the participating institutions between the ages of 18 and 45 who had sustained a grade I (mild) or II (moderate) lateral ankle sprain at a scheduled practice or competition. If not in the presence of the team physician or AT at the time of injury, the athlete was required to present within 25 hours of the injury. Athletes with sprains not expected to last at least 3 days (determined on the day after injury) were excused from the study.

Procedures

As soon after injury as practically possible, all participating athletes were fitted with bipolar stocking electrodes (Silver-Thera, Prizm Medical Inc, Duluth, GA) for the next 72 hours. Nominally, this was the first 72 hours after injury, but, because not all sprains were immediately recognized, some did not begin stimulation until up to 25 hours after injury. In brief, a stocking electrode is a knee-length stocking made in part of silver-coated threads. Approximately at midlength, there is a short interval of nonconducting material, which divides the stocking into 2 large electrodes. At time of injury ATs selected electrode stockings of appropriate size (snug but not tight), immersed them in warm water, gently wrung them out, and put them on the injured limbs. All stocking electrodes were powered by miniature HVPC stimulators (Micro-Z, Prizm Medical Inc) attached via hook-and-loop fasteners at the nonconducting intervals, with cathodes (segments of stockings covering ankles and feet) as active electrodes; anodes covered most of ipsilateral legs. Next, ATs loosely applied Exoclear (Econoline Products, Inc, Charlotte, NC) as a vapor barrier over electrode stockings and stimulators. They were free to apply any form of compression over the Exoclear but were asked to leave the stimulator control surfaces exposed.

Athletes were randomly assigned to receive active or placebo stimulation as follows. Participating institutions were provided injury kits that included all instructions and gear needed to initiate athletes into the study. Included in each kit was a time-of-injury form, which provided step-by-step instructions and places for data entry, including which stimulator (numbered by institution) and channel.
were being employed. When the kits were assembled for each site, equal numbers of each channel were predesignated on the forms, and the kits were shuffled. Moreover, active and placebo channels on each stimulator were randomly set at the factory. That information was confirmed by a technician at the home institution of the investigators when the stimulators first arrived and on completion of the study. Channel designation (active or placebo) of each stimulator was not revealed to the investigators until after raw and compiled data sets were fully edited and verified. Active channels provided continuous output as selected at the controls (ie, cathodal HVPC, interpulse interval of 100 μs, at 120 pulses/s). Intensity was set by clicking on control surfaces of stimulators until stimulation could just be perceived (sensory level) and then clicking down once (~8 V) to subsensory level. Placebo channels were programmed to provide this same output, but for just 3 minutes after the final (subsensory) intensity settings were entered. Indicator lights remained illuminated regardless of actual output—units appeared to be active even if output had ramped off. To avoid accidentally changing any setting on Micro-Z stimulators, users must first depress an “on” control and, within several seconds, activate separate controls for increasing or decreasing intensity. We programmed this feature so that depressing the on control when the placebo channel was in use immediately recommenced stimulation at the intensity last set, that is, sensory-level stimulation minus 1 click. Fresh batteries were installed in stimulators each day, and electrode stockings were replaced if, after testing, intensity settings required to achieve sensory-level stimulation were more than 5 clicks more than the previous day.

To monitor compliance, we instructed athletes to record in simple diaries time (from 72-h timers started when stimulation was first initiated) and reason any change in stimulation occurred. If, for example, an athlete had to shut down stimulation to bathe or undergo treatments, he or she was instructed to first increase the intensity until sensory-level stimulation was reestablished and then record the time and number of clicks required to turn the stimulator to 0 output. Optimally, reestablishing sensory-level stimulation should have required a single click, and the number of clicks (minus 1) required to turn the stimulator to 0 output should have matched the previous number in the diary when stimulation level was last set. If, however, a lead became disconnected, or impedance at skin or electrode changed since stimulation was last started, reestablishing sensory level may have been impossible or required more than 1 click. At roughly 2-hour intervals during waking hours, athletes were instructed to check to see if their stimulators were working and if intensity levels were still appropriate. Typically, athletes had only to increase the intensity control 1 click to reestablish sensory-level stimulation and then decrease intensity 1 click to return to subsensory stimulation and then record in their diaries the time and reason the check was made. If, however, sensory-level stimulation could not be reestablished within 3 clicks or at all, athletes were instructed to note the time, shut down the system, cleanse their leg with an alcohol wipe, rewet (rinse and lightly wring out) the stocking electrode, put it back on, check all connections, reapply Exoclear, and restart stimulation, noting in their diaries the time and the intensity setting.

Athletes were instructed to remove all gear and air out the injured limb 4 or 5 times a day for 20 to 30 minutes. This was to reduce the potential of skin irritation caused by long-term contact with damp electrode stockings. Time spent bathing or receiving routine treatments and any other time the stocking electrode
was removed counted toward the goal of 4 or 5 air-out intervals per day. Air-out and rewet procedures were particularly encouraged before the athletes went to bed for the night because the system was not checked again until they awoke the next morning. When athletes went daily for evaluation and therapy, attending ATs were instructed to personally shut down stimulation by reestablishing sensory level and then counting the number of clicks necessary to turn the stimulator to 0 output, then record in the athletes’ diaries the pertinent information. This was designed as an independent check and opportunity to review with athletes the proper application of stimulation and use of diaries. ATs were also asked to daily transcribe diary entries to electronic forms to provide backups for the diaries and were encouraged to monitor compliance—determining how well athletes understood and complied with the protocol. During analysis, each diary was reviewed. Any intervals that were not properly documented were added to intervals when stimulation was not being applied (eg, when bathing, during air-outs). This total off time was divided by the total time stimulation was putatively on, that is, 72 hours, to quantify compliance (Table 1). The outcome variable for this study was total injury time measured in hours and minutes from time of injury until the athlete was declared FTP. ATs determined by their own methods, not by studywide criteria, when athletes were FTP. Dates and times these decisions were made were recorded.

We collected data on gender, age, weight, height, ankle injured, sport activity at time of injury, and grade of sprain (I or II). Near the end of the 72 hours of electrical stimulation, athletes were asked whether they thought they had received active or placebo stimulation. However, we report here (Table 1) summary statistics to demonstrate that our treated and control groups were drawn from the same population and received similar treatment other than the primary intervention, that is, live versus sham HVPC. Use of nonsteroidal anti-inflammatory drugs (NSAIDs) was calculated by dividing the number of days an athlete ingested recognized NSAID doses (eg, equivalent of 800 mg ibuprofen per day) by the total number of days injured (time of injury to time declared FTP, in days). Days on which less than anti-inflammatory doses were taken were counted as no NSAIDs.

**Statistical Methods**

Baseline demographics were tabulated by treatment group and summarized by either the mean ± SD or proportions.

All analyses were done on an intent-to-treat basis; that is, subjects were included in statistical analyses according to their randomized treatment assignment irrespective of the amount of treatment actually received. Statistical analysis of the primary study outcome—time of injury to time declared FTP—was carried out using inference procedures based on a linear model fit to the data via restricted maximum-likelihood methods. Specifically, the outcome was modeled as a function of treatment, grade of injury, and the interaction between these factors to allow for grade-specific effects of treatment. In addition, variances within the 2 grade levels were allowed to differ. Linear contrasts of the fitted model parameters were constructed to examine and test for overall and within-grade treatment differences. With the achieved sample size, calculations reveal approximately 80% power to detect treatment-group difference of 1.3 SD. To adjust for possible baseline differences, a series of secondary analyses were undertaken using the described
Table 1  Comparison of Groups Receiving Live and Sham Stimulation

<table>
<thead>
<tr>
<th>Rx</th>
<th>Age, y</th>
<th>Gender</th>
<th>Body-mass index</th>
<th>NSAIDs use (% of injury time when they were taken)</th>
<th>Noncompliance (% of time when HVPC was not applied or off)</th>
<th>Days till stimulation begins</th>
<th>Subjects who believed they received active treatment</th>
<th>Sports (# of different sports participating in when injured)</th>
<th>ATs (# who treated subjects)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Live</td>
<td>20.3 ± 2.1</td>
<td>6 F, 16 M</td>
<td>26.4 ± 5.3</td>
<td>23 ± 33</td>
<td>22 ± 18</td>
<td>0.28 ± 0.34</td>
<td>86%</td>
<td>8</td>
<td>15</td>
</tr>
<tr>
<td>Sham</td>
<td>19.8 ± 1.1</td>
<td>12 F; 16 M</td>
<td>23.8 ± 2.5</td>
<td>15 ± 29</td>
<td>20 ± 12</td>
<td>0.35 ± 0.36</td>
<td>76%</td>
<td>10</td>
<td>19</td>
</tr>
</tbody>
</table>

NSAID, nonsteroidal anti-inflammatory drug; HVPC, high-voltage pulsed current; AT, certified athletic trainer.
modeling approach. Additional predictors (variables) included gender, NSAID use, compliance (measured as a percentage of total treatment time), and time until start of treatment. Because of limited sample sizes, these additional predictors were considered 1 variable at a time. These models also included all possible interactions. To meet statistical assumptions associated with inference procedures, the log transform was applied to the outcome in all analyses, although descriptive statistics are reported in the original units. Tests were 2-sided, and a nominal significance level of .05 was used throughout. All statistical tests were performed using SAS version 9.1.3 statistical software (Cary, NC).

Results

Table 1 summarizes the characteristics of athletes randomly assigned to treatment (n = 22) and control (n = 28) groups. Also reported here is the number of sports yielding sprains by treatment group. Overall, there was no significant difference between treatment groups across grade of injury ($P = .55$; Table 2). Analyses based on inclusion of secondary predictors (see Statistical Methods), limited though they were by sample size, did not contradict the primary analysis.

Grade of injury was a consistent significant predictor ($P < .0001$) in all analyses. Table 2 summarizes the duration of injury (time of injury to time declared FTP) for treated and control groups subdivided by grade of injury. Duration of injury was not different between treated and control groups for grade II sprains ($P = .079$), but treated grade I sprains lasted longer than in the control ($P = .049$; Table 2).

Discussion

The purpose of our study was to determine whether adding nearly continuous subsensory electrical stimulation (HVPC) for the first 72 hours after lateral ankle sprain affects rate of recovery of competition-level function in intercollegiate and professional athletes. HVPC had no effect on recovery of moderate ankle sprains and prolonged recovery of mild ankle sprains by approximately 1 day.

Nearly continuous application for 3 days is considerably longer than the typical clinical protocol, which is 20 to 30 min/day. For moderate (grade II)

<table>
<thead>
<tr>
<th>Grade of injury</th>
<th>Group</th>
<th>n</th>
<th>Days until fit to play, mean ± SD</th>
<th>Grade-specific group difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>live</td>
<td>15</td>
<td>5.3 ± 1.9</td>
<td>.0498</td>
</tr>
<tr>
<td></td>
<td>sham</td>
<td>15</td>
<td>4.1 ± 1.3</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>live</td>
<td>7</td>
<td>12.6 ± 6.1</td>
<td>.07914</td>
</tr>
<tr>
<td></td>
<td>sham</td>
<td>13</td>
<td>13.9 ± 7.0</td>
<td></td>
</tr>
</tbody>
</table>

Overall group difference .55. $P$ values are based on a fitted linear model.
ankle sprains, which in this study averaged 13.4 days, 3 days of nearly continuous treatment would represent over 22% of the total injury time. Perhaps only NSAIDs and compression are typically applied over equal or greater proportions of total injury time after sprains or other soft-tissue injuries. Nonetheless, despite application over relatively large portions of total injury time resulting from moderate (grade II) lateral sprains, we saw no evidence that HVPC affected recovery time from such injuries. Mild (grade I) sprains, which in this study averaged 4.6 days in duration, would seem most likely to respond to our protocol because the putative treatment was applied for a greater portion of the total injury time. Indeed, the mean mild sprain in this study received HVPC for 65% of the total injury time. We observed a treatment effect on mild sprains, but certainly did not anticipate that recovery from mild ankle sprains treated with live stimulation would be, on average, a day longer than for those receiving sham stimulation (controls).

A possible explanation is a type I statistical error. It is possible that the 3 minutes of diminishing HVPC (ramped down from subsensory to 0 in 3 minutes) that controls received each time they adjusted or tested stimulation levels (approximately every 2 h during the day) was “more therapeutic” than the more aggressive stimulation that the treated group received. This seems to us very unlikely, but we cannot categorically reject this hypothesis. Alternatively, active stimulation may have produced harm in that time lost to injury was increased. This too seems unlikely to us because we are unaware of any evidence that low-amplitude stimulation such as applied here has ever been reported to cause damage or ill effects of any kind. Again, we have reported on the positive effects that comparable levels of HVPC have on edema formation. HVPC is thought to reduce capillary permeability, which is a plausible explanation for the positive outcomes in our series of laboratory experiments. In those studies, HVPC, cryotherapy, and ibuprofen all curbed edema formation but were applied within minutes after trauma and maintained throughout most of the inflammatory period. However, neither we nor any other group that we know of has examined effects of HVPC, or any other intervention, on time lost to injury. Michlovitz et al reported that ice and HVPC did not affect pain, swelling, or range of motion after acute lateral ankle sprains. They administered 1 treatment per day and subjects were enrolled up to 28 hours after injury, making their design of limited value for ATs in college and professional settings. Man et al also applied submotor electrical stimulation for 3 days, but only for 30 min/day, and found no difference in swelling compared with sham and motor-level stimulation.

We chose intercollegiate and professional athletes because they are prone to ankle sprains, and motivated to recovery quickly from injury. Gauging by the compliance we observed (Table 1), most athletes who consented to participate were diligent in carrying out the protocol. Intercollegiate and professional athletes are under the care and supervision of physicians and ATs who are present during most training sessions and competitions and who typically treat most soft-tissue injuries sustained by these athletes. These ATs, therefore, were positioned a priori to apprise athletes of the study; determine eligibility and initiate the protocol very soon after the qualifying injury; train athletes to self-administer the protocol; supervise and, as needed, correct athlete administration of the protocol; and collect and record required information.
Use of intercollegiate and professional athletes as the study population and dependence on ATs to administer the protocol also complicated our study. Athletes are influenced by their physicians and ATs but also make independent decisions, especially regarding use of analgesics and anti-inflammatory medications. Moreover, clinicians vary in their opinions regarding these medications: Some actively encourage their use and others actively discourage it. Therefore, we chose to allow use of prescribed and over-the-counter analgesics and NSAIDs rather than unenforceably banning them. Nineteen percent of athletes were prescribed or chose to use NSAIDs in doses known to be anti-inflammatory (equivalent to ~800 mg/d or higher of ibuprofen), and use was similar in treated and control groups (Table 1).

Maintaining electrode moisture helps overcome skin resistance to current flow. Therefore, we required athletes to periodically rewet the stocking electrodes. However, prolonged exposure to moist conditions caused 10 athletes (4 in treated group and 6 in control group) to develop itching or rashes. These dissipated quickly with no treatment but forced us to curtail further delivery of stimulation to these athletes. We asked athletes to remove the system for 20 minutes 4 or 5 times a day to reduce the incidence of rashes, but this increased the inconvenience of using the system. Although athletes who developed rashes did not receive the full 72 hours of HVPC, we left them in the data set to achieve the desired intention-to-treat analysis.

Our experiments with laboratory animals suggest strongly that electrical stimulation affects edema formation, at least when applied as the formation is occurring and only when the stimulation is “live”—there is no expectation of treatment effect after the stimulation has ceased. We attempted, therefore, to begin nearly continuous stimulation as soon after the sprain as possible and would have preferred to leave it on throughout the recovery period, or at least until edema formation ceased, but we limited application to only 3 days because of the inconvenience of the protocol. Initiating the stimulation protocol immediately after the injury turned out to be more difficult than we had anticipated. Our initial plan was to allow a delay of only 1 hour after injury before initiating stimulation, but practical constraints forced us to expand that to 25 hours because ATs simply could not quickly evaluate, recruit, and begin treatment of athletes when sprains were incurred during certain competitions, and some sprains were not recognized as such until hours after the initial injury. Some athletes began receiving HVPC within 20 minutes of their injuries and others not until 25 hours postinjury. The average delay was 7 hours and 40 minutes, well after edema formation was underway, but the active and control groups were similar in this regard (Table 1).

In laboratory animal experiments in which we showed that HVPC curbed edema formation, the amplitude of electrical stimulation was always 10% less than motor level, and that stimulation was always delivered by water-immersion technique whereby electrode adhesion, contact, and conductance were not variables. Obviously, we could not use immersion technique in this study, and to increase reliability in setting stimulation amplitudes we chose to set stimulation amplitude just below sensory threshold rather than 10% below motor level. However, we believe that we provided roughly comparable amplitudes of stimulation in this clinical trial. Our experience with the sock electrodes and stimulators used in this study suggests that sensory-level stimulation is often only 1 click (about 8 V) below motor threshold. Moreover, applying sensory-level stimulation, perceived with both live and sham stimulators, then uniformly reducing the amplitude to subsensory levels
provided a highly credible placebo effect. Indeed, the percentage of subjects who believed they were receiving active treatment was high for both groups (19/25, or 76%, for sham group and 18/21, or 86%, for live group).

Lack of an easily defined, clinically proven end point to a sprain influenced the design of our study. Lacking a verified and accepted end point, we chose the time an athlete is declared FTP. We are acutely aware that methods for determining this level of recovery are not clinically validated, enumerated, or quantifiable, but then neither is any other level of recovery that we know about. As ill defined as the process is, ATs routinely make this clinical decision. For this study we allowed participating ATs to determine fitness to play in their usual way. More than 30 (from 1 professional football team and 9 colleges and universities) participated in this study. These ATs had to decide on fitness to play in individual sports like cross-country and wrestling, as well as team sports like basketball and football, all of which have very different demands. The mix of sports and the number of ATs between treated and control groups were similar (Table 1). Our ATs and physicians used current clinical practices to determine fitness to play, and we assume those methods are representative of those used by most practicing ATs and physicians.

We are aware that the “precision” that our statistics seem to convey (ie, injury duration to the minute) is, of course, illusory. ATs typically see athletes, particularly college athletes, early and/or late in the day (before and/or after classes). Even if seen several times in 1 day, these visits are typically hours apart. Because of class or practice and game schedules, an athlete may be seen and evaluated by the AT early one day, but not again until late the following day. About as short an interval between evaluations for FTP as we could hope for would be 6 to 8 hours during the day, but more typically successive evaluations were probably closer to 24 hours apart. We originally designed our sample size to enable us to detect a 1-day difference in time to recovery between groups with a power of .9. In retrospect, this was probably unattainable regardless of our sample size, because even elite athletes are typically not evaluated more than once per day. Detecting less than a 1-day difference in time until declared FTP may be practically difficult. Clinically, however, a difference in outcome of 1 day or less could be important, especially for sports like baseball or basketball in which games are scheduled frequently and regularly.

Despite these limitations, we believe that this study enabled us to gauge whether HVPC delivered nearly continuously at just below sensory levels for 3 days beginning soon after lateral ankle sprains affected time to recovery. There is little doubt that interventions such as cold, compression, and NSAIDs can ameliorate symptoms of soft-tissue injuries, including sprains. In addition, it is not unreasonable to at least speculate that curbing symptoms might reduce recovery time. Indeed, this speculation underpins much of the rationale for applying these interventions. However, it is not at all clear that masking or reducing the severity of symptoms hastens recovery. In addition to curbing acute edema formation, low-amplitude electrical stimulation is known to promote closure of chronic ulcers and knitting of otherwise nonhealing fractures. It is possible, therefore, that electrical stimulation might somehow promote or accelerate normal healing. Nonetheless, results of this study do not support that thesis. Aside from the atypical conditions just noted, we are unaware of any intervention that accelerates normal healing. Nev-
Nevertheless, suppression of symptoms is justified to ease discomfort and as a means of initiating range-of-motion, weight-bearing, and conditioning exercises sooner than otherwise tolerated. Such exercises are known to hasten recovery of normal function compared with immobilization but are not known to hasten recovery relative to weight bearing and activity to tolerance. HVPC as applied in this study did not hasten recovery of competition-level function after grade II lateral ankle sprains and retarded recovery after grade I lateral ankle sprains in intercollegiate and professional athletes.

Conclusions

HVPC as applied in this study had no clinically meaningful effect on rate of recovery after ankle sprains in professional and intercollegiate athletes. Additional clinical trials are needed to determine the efficacy of HVPC and other acute interventions on soft-tissue injuries.

Acknowledgments

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References