greatest role will be in combination with platelet transfusions preoperatively and in the management of acute bleeding.

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Transcutaneous electrical nerve stimulation in ankylosing spondylitis: a double-blind study

To the Editor:

Inflammatory low back pain and stiffness is a characteristic feature of ankylosing spondylitis (AS). Since no drug has been shown to modify the inflammatory process per se, the aim of treatment is to ensure analgesia and normal spine mobility. Transcutaneous electrical nerve stimulation (TENS) can reduce pain in many musculoskeletal disorders, and in an open trial (1), it was reportedly effective in the treatment of unresponsive pain from AS.

We evaluated the efficacy of TENS in the management of AS lumbar pain in a controlled double-blind study of 20 patients with definite AS (New York criteria [2]), who had lumbar pain and stiffness for at least 1 month, for which treatment with nonsteroidal antiinflammatory drugs (NSAIDs) was not sufficient. NSAID therapy was discontinued 1 week before the beginning of the trial; only paracetamol was allowed for severe pain.

TENS was delivered by an Agopik A86 stimulator (Sanitec, Alessandria, Italy), through 4 electrodes placed on the skin of the lower back. Standard acupuncture points (3) were chosen as the sites of stimulation, and a low-frequency (5-Hz) current was applied to group A patients. The same stimulator with the current switched off delivered sham TENS. In order to support the placebo effect, the lights and the acoustic signal of the apparatus were demonstrated to the group B patients. Each patient received 10 treatments of 20 minutes each, over a 3-week period. Patients had been randomly assigned to 1 of the 2 treatment groups at study entry.

Assessments were performed before and after treatment by 1 of us (IO), who had no knowledge of the study group to which the patient was assigned. Pain and stiffness (0–100-mm visual analog scale), Shober's test, finger-to-floor distance in anterior and lateral flexion, and pill counts of the analgesic were performed. At the end of treatment, all patients and the blinded evaluator were asked independently to record, on a 7-point scale, their scoring of the treatment results. Statistical analysis of the data was performed using the chi-square test, Student's *t*-test, Wilcoxon's test, and the Mann-Whitney U test, as appropriate.

Table 1. Changes in pain and stiffness in 20 patients with ankylosing spondylitis after treatment with transcutaneous electrical nerve stimulation (TENS) or with sham TENS*

	Group A (active TENS)	Group B (sham TENS)
Pain (100-mm VAS)		
Before study	57.1 ± 13.7	47.6 ± 17.8
After study	35.6 ± 20.5†	41.9 ± 16.3
Difference	21.5 ± 16.3‡	5.7 ± 15.4
Stiffness (100-mm VAS)		
Before study	56.1 ± 14.4	49.8 ± 14.6
After study	31.0 ± 19.3†	43.0 ± 14.5
Difference	24.3 ± 24.4§	6.2 ± 16.1

* Pain and stiffness were scored on a visual analog scale (VAS). Values are the mean ± SD.
† $P < 0.025$ versus before study value.
‡ $P < 0.025$ versus group B.
§ $P < 0.05$ versus group B.

Significant differences ($P < 0.025$) in pain and stiffness measurements over the treatment period were found only in group A patients. There were also statistically significant differences in pain ($P < 0.025$) and, to a lesser extent, stiffness ($P < 0.05$) between the 2 treatment groups when the mean differences before and after treatment were compared (Table 1). There was no statistically significant improvement in lumbar mobility in either treatment group.

One group A patient and 5 group B patients took paracetamol; the difference was not statistically significant. The blinded evaluator scored the treatment results higher in group A patients than in group B ($P < 0.025$); the group A patients scored the results higher than did the group B patients, but the difference did not reach statistical significance. Despite the lack of significant differences between the 2 groups, individual responses to both active and placebo treatments were worthy of note. Nine patients in the active TENS group and 6 in the sham TENS group stated that their low back pain had improved mildly or greatly at the end of treatment.

Our data are consistent with the results of Nienhuis and Hoekstra's open study, and show that TENS is significantly more effective than placebo in the treatment of lumbar pain and stiffness caused by AS. Although the difference is not statistically significant, the analgesic effect of TENS may also be suggested by the lower number of analgesic pills taken by group A compared with group B during treatment. Nevertheless, the positive trend of the scores of both groups of patients reveals that the placebo effect plays a prominent role in the efficacy of TENS, as confirmed by reports in the literature (4).

These results suggest that TENS may provide a

further method of relieving pain and stiffness in patients with AS. Because AS takes decades to develop fully, a multidisciplinary effort to control the natural evolution of AS, with an intensive remedial exercise program and a suitable anti-inflammatory agent, should include TENS as a therapeutic adjunct. It is hoped that others will perform similar studies of a larger number of patients in order to obtain confirmation of the present data.

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Sustained remission of rheumatoid arthritis following hypersensitivity reaction

To the Editor:

Rheumatoid arthritis (RA) is a chronic inflammatory disease of unknown etiology. Immunologic abnormalities, including type IV hypersensitivity response abnormalities, have been described in RA (1). It is widely believed that the tissue damage is caused by the type III immune response (2,3).

The possible association of atopy with RA has been investigated in several studies, but it has been concluded that the prevalence of atopy is not increased among patients with RA (2,4). The type I hypersensitivity response in RA patients has not, however, been closely studied. Some investigators have found abnormalities in serum IgE (low or high levels) in patients with RA (4,5). It has been reported that patients with dermatitis from gold treatment experience remission or near remission of their RA (6); this is believed to be possibly related to an immunologic response to the gold. In addition, we have observed incomplete remission in 2 patients with RA and 1 with Behçet's disease, after anaphylactic reaction. In 1 of these patients, the reaction was manifested by severe urticaria.

In spite of these reports, however, a clear relationship between RA or other rheumatic conditions and type I hypersensitivity reaction has not been demonstrated. Because generalized anaphylaxis occurs only rarely in patients with RA, little is known about the effects of anaphylaxis on the RA disease course. We describe herein a patient with RA who had sustained remission after generalized anaphylaxis and anaphylactic shock.

The patient, a 45-year-old man, had erosive RA since 1969. He had been treated with steroids, various nonsteroidal antiinflammatory drugs, and D-penicillamine; all provided only temporary or limited benefit. Clinical examination in 1969 revealed swelling, stiffness, and pain bilaterally in the second metacarpophalangeal joints, the proximal interphalangeal joints, and the metatarsophalangeal joints. Synovitis of the right elbow and both knees was noted. Motion was painful and restricted in most major joints such as the shoulders, elbows, and hips. There were nodules over several joints and extensor tendons.

The results of laboratory investigations were as follows: hemoglobin 11 gm/dl, white blood cell (WBC) count 10,000/mm^3, erythrocyte sedimentation rate (ESR) 65 mm/hour, C-reactive protein 7 mg/dl, strongly positive rheumatoid factor (RF; measured qualitatively), serum IgG 1,450 mg/dl (normal 566–1,373). No tests were performed for the presence of cryoglobulins or circulating immune complexes. A node was removed from the patient's right elbow, and pathologic examination revealed features consistent with rheumatoid nodule.

In March 1979, the patient was admitted to our hospital with fever, cough, and shortness of breath of 1-week duration. On physical examination, rhonchi were heard in the left lower lung area. The WBC count on admission was 27,400/mm^3, with 98% polymorphonuclear leukocytes. Chest radiography demonstrated inflammation of the left lower lobe, with minimal effusion on the same side. There was no allergic reaction documented in his medical history. Intravenous semisynthetic penicillin was started, but, on the third day of hospitalization, anaphylactic reaction and shock occurred. He was hospitalized and resuscitated immediately, using adrenaline and methylprednisolone (total of 500 mg intravenously).

He remained in the hospital for 1 week more, without any medication except antihistamines. He was then discharged and was followed up as an outpatient by our staff. After 3 months, he was RF negative, with an ESR of 15 mm/hour. All therapy for RA was discontinued. By the second year after the anaphylactic episode, he had no nodules or clinical signs or symptoms that might be related to RA. We have followed his case for 11 years, during which time he has remained asymptomatic.

It is believed that pharmacologically active amines which are released in increased levels during the type I hypersensitivity reaction (i.e., histamine, slow-reacting substance of anaphylaxis, eosinophil chemotactic factor–anaphylaxis, serotonin, heparin, kinins, and prostaglandins) can cause some symptoms resembling RA (2–5). But the sustained remission after generalized anaphylaxis and anaphylactic shock in our patient leads us to believe that these mediators might play a preventive role in the immunologic mechanism of RA. It is doubtful that the 500-mg dose of methylprednisolone received by our patient had a role in the remission (2,7).

The hypothesis that anaphylaxis could affect the regulation of autoantibody production in RA is unproven, but this case leads us to believe that anaphylaxis might play a preventive role in some patients. We hope further studies of the mechanisms involved will be conducted, to explain the