Scand J Med Sci Sports 2002: 12: 136–142
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 · ISSN 0905-7188
SCANDINAVIAN JOURNAL OF
MEDICINE & SCIENCE
IN SPORTS

Iontophoresis with cortisone in the treatment of lateral epicondylalgia (tennis elbow)—a double-blind study

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Accepted for publication 29 October 2001

Lateral epicondylalgia (tennis elbow) is a common dysfunction of the arm. Because there is no agreement concerning the pathophysiology, several modes of treatments have been tried and one of the most common is local steroid injection. Iontophoresis using corticosteroids is a fairly new method recommended in the treatment of lateral epicondylalgia and has become popular owing to impression of superiority compared to local injections—noninvasive, painless and nontraumatic. The aim of this double-blind prospective, randomized study was to evaluate the short- and the long-term pain-relieving effect of corticosteroid iontophoresis in lateral epicondylalgia. Sixty-four patients suffering from lateral epicondylalgia were consecutively randomized into two groups for corticosteroid or placebo iontophoresis. The patients were treated four times during 2 weeks. Follow-ups were done the day after the final treatment and after 3 and 6 months. Twenty-three patients dropped out before the 3-month follow-up because they wanted to complement the treatment or replace it with other treatments. No significant difference between the corticosteroid group and the placebo group in relation to subjective and objective outcome could be observed after the treatment period or at the follow-ups. In fact, both groups improved throughout the study. The results of the present study do not support the use of corticosteroid iontophoresis in lateral epicondylalgia.

Lateral epicondylalgia (tennis elbow) is one of the most common dysfunctions of the arm. The first description as 'Schreibekrampfes' was attributed to Runge in 1873 (Runge, 1873), but the name is derived from Morris' description of 'lawn tennis arm' (Morris, 1882). Lateral epicondylalgia affects 1–3% of the population (Allander, 1974; Kivi, 1982; Verhaar, 1994) and occurs between 35 and 60 years with a mean age of 42–46 years. The most common cause of lateral epicondylalgia is over-use of the extensor carpi radialis brevis (ECRB) muscle (Nirschl, 1973; Priest, Braden, Gerberich-Goodwin, 1980; Nirschl, 1992) and only a small proportion (5%) refer their problems to racket sports (Coonrad & Hooper, 1973; Assendelft, Hay, Adshead, Bouter, 1996). Spontaneous recovery is known to occur within 8–12 months (Garden, 1961; Verhaar, 1994; Putnam & Cohen, 1999); however, long-lasting and severe medical consequenses and surgery are reported (Dimberg, 1987).

Even though the clinical picture is fairly uniform, there is still no agreement about the underlying pathology except for the involvement of the ECRB (Cyriax, 1936; Goldie, 1964; Coonrad & Hooper, 1973; Nirschl & Pettrone, 1979; Coonrad, 1986; Dimberg, 1987; Snijders, Volkers, Mechelse, Vleeming, 1987; Murtagh, 1988; Nirschl, 1992; Regan, Wold, Coonrad, Morrey, 1992; Thomas, Siahamis, Millicent, Boyle, 1992; Smith, Papadopolous, Mani, Cawley, 1994; Ljung, Lieber, Friden, 1999b).

Some authors suggest that the enthesis of ECRB is the key and microtears at the ECRB are the cause of the local tissue reaction (Chard & Hazleman, 1989; Nirschl, 1992; Chard, Cawston, Riley, Gresham, Hazleman, 1994). This suggestion is supported by infrared thermography which shows inflammatory hot spots over the lateral epicondyle (Binder, Parr, Thomas, Hazleman, 1983; Thomas & Savage, 1989; Thomas, 1992). The enthesis shows alterations in collagen content, reduction in cells and ground substance as well as increased lipid deposition. It is believed that such changes predispose to injury (Chard & Hazleman, 1989).

Abnormal forearm temperature gradients and significant unilateral cooling have also been reported suggesting a representation of the somatosympathetic reflex (Thomas et al., 1992).

Ljung and colloborators found that no inflammatory cell infiltrates and that few solitary mast cells were found giving further evidence to suggest that tennis elbow is not an inflammatory process in the sense of involving inflammatory cells (Ljung, Forsgren, Fridén, 1999a). Furthermore, a sympathetic dysfunction has been suggested in the pathophysiology of tennis elbow (Smith et al., 1994).

As the pathology is still obscure, there is no agreement of treatment to be used (Haker, 1993; Wright & Vicenzino, 1997; Putnam & Cohen, 1999) and no specific therapy has emerged as the 'gold standard' with superior long-term efficacy (Wright & Vicenzino, 1997).

While more than 40 different treatments—used separately or in combination—have been proposed (Labelle, Guibert, Joncas, Neuman, Fallah, Rivard, 1992), steroid injection is one of the most often reported treatments (Coonrad, 1986; Chard & Hazleman, 1989; Geoffroy, Yaffe, Rohan, 1994; Verhaar, 1994; Solveborn, Buch, Mallmin, Adalberth, 1995; Assendelft et al., 1996; Thurston, 1998; Hay, Paterson, Lewis, Hosie, Croft, 1999; Kraushaar & Nirschl, 1999) and surgical treatment is supposed to be a last resort for tennis elbow (Bosworth, 1955; Roles & Maudsley, 1972; Thurston, 1998; Putnam & Cohen, 1999).

Iontophoresis using various antiflogistic drugs is a fairly new method used in lateral epicondylalgia (Bertolucci, 1982; Demirtas & Oner, 1998). Iontophoresis, or ion transfer, uses continuous direct current of a low amperage to introduce topically applied physiologically active ions through the body surface. The principle is that an electrically charged electrode will repel a similar charged ion (Cummings, 1987). The idea of applying electrical current in order to increase the penetration of electrically charged surface tissues was first described by Veratti in 1747. According to Chien and Banga (1989), the biomedical application of electricity can be traced back to the golden age of the Greek civilization. LeDuc (1908) performed the first well-documented experiments, where he demonstrated that ions could be driven across the skin by means of an electric current, in the beginning of the 20th century. It has been shown that the corticosteroid, dexamethasone, (Decadron®) is transferred iontophoretically into all tissue layers underlying the electrode down to, and including, tendinous structures and cartilaginous tissue. This indicates that a therapeutic dose of an anti-inflammatory drug can be delivered at sufficient tissue depths (Glass, Stephen, Jacobson, 1980). Studies on humans have indicated that ions penetrate and have therapeutic effects on deeply situated structures (Kahn, 1977; Bertolucci, 1982; Harris, 1982). Iontophoresis of corticosteroids requires a local tissue concentration that is lower than those achieved with injection, but higher than those achieved with oral administration and is, therefore, considered to be both safe and effective (Glass et al., 1980). It is probable that the transportation of the drug to the intracellular compartment is increased as a result of the intense arterial vasodilation induced by galvanic current. This means that the active substance reaches a higher concentration than it would if applied orally or parenterally (Sing & Maibach, 1994). Iontophoresis gives an impression of superiority compared to local injection. Furthermore, the method permits consistent drug delivery at a low systematic dose—noninvasive, painless and nontraumatic (Bertolucci, 1982; Harris, 1982; Hasson, English, Daniels, Reich, 1988). A double-blind trial on the treatment of plantar fascitis with iontophoresis has shown good pain-relieving effects already after three treatments and with a short-term effect directly following the treatments. The long-term effect was not studied (Gudeman, Eisele, Heidt Jr, Colosimo, Stroupe, 1997). No controlled study has been found evaluating the pain-relieving effect of corticosteroid iontophoresis in lateral epicondylalgia.

The aim of this study was to evaluate the short- and the long-term pain-relieving effect of corticosteroid iontophoresis in patients suffering from lateral epicondylalgia.

Methods

Patients

Sixty-five patients suffering from lateral elbow pain were examined and evaluated by a well-trained physiotherapist between February 1998 and February 1999. All the patients were living in Boras community, Sweden, and were either self-referred or referred by their physician or physiotherapist.

The patients were informed that the pain-relieving effect of corticosteroid compared to saline using iontophoresis was to be evaluated in a double-blind study and that no fee was to be charged.

Criteria of inclusion

The diagnostic tests used were (1) palpation at the lateral epicondyle, (2) resisted wrist extension, (3) middle-finger test and (4) the vigorimeter test. Pain at the lateral epicondyle when palpating, pain produced by at least two of the tests 2, 3, or 4, and a history of pain of at least 1 month, qualified the patients for inclusion (Haker, 1993).

Criteria of exclusion

Those excluded were patients who had had any treatment during the last month for their elbow pain, patients who demonstrated dysfunction in the shoulder, neck, and/or thoracic region, local or generalized arthritis, neurological deficit, radial nerve entrapment, bilateral epicondylalgia, pregnancy and patients using a pacemaker or operated on the affected elbow.

Sixty-five patients fulfillled the criteria of inclusion and were consecutively randomized into one of two groups—the placebo group or the corticosteroid group.

Test procedure

The following tests were included in the procedure: (1) palpation at the lateral epicondyle, (2) resisted wrist extension—with the elbow extended and the forearm pronated (fig. 1)—(3) the middle-finger test (resisted extension of digitus III)—with the elbow extended and the forearm pronated (fig. 2)—and (4) the vigorimeter test measuring grip strength (Haker, 1993). In this study, the Martin vigorimeter (Gebrüder Martin, Tuttlingen, Germany) was used which is a dynamometer with a rubber balloon to be compressed in the hand. The air pressure within the balloon is registered in kilopounds per square centimeters (1 kp $cm^{-2} = 98{,}1$ kPa) on a manometer through a rubber-tube connection and a large-sized balloon was used. Patients were seated comfortably, shoulder at rest, elbow extended, forearm pronated

with 20° dorsiflexion of the wrist, holding the balloon with the connection tube protruding between thumb and index finger. They were instructed to press the balloon, and to stop pressing when any kind of pain was experienced over the lateral epicondyle (pain threshold when gripping) (fig. 3). If the mere position of the arm caused such pain, this was noted and no pressure was exerted. Otherwise, the mean value of three consecutive estimations was calculated.

The present intensity of the pain threshold when gripping was evaluated using the absolute (no scale marks on the line) Visual Analog Scale (VAS), total length 100 mm (Scott & Huskisson, 1976; Carlsson, 1983).

Treatments

In both groups, iontophoretic IOMED Trans QE medium electrodes were used. The hydrated drug electrode—negatively charged—was placed over the lateral epicondyle and the dispersive electrode—positively charged—over the extensor muscles in the forearm. The placebo group received saline and the corticosteroid group received 0.4% dexamethasone sodium phosphate. The iontophoresis was performed using the Phoresor II Iontophoresis Drug Delivery System (IOMED, Inc.). Patients were treated individually with a current of 4 mA for 10 minutes, four times (Sanderson, DeRiel, Dixon, 1989; Petelenz et al., 1992) according to a treatment schedule Monday–Wednesday–Friday–Monday or Wednesday–Friday–Monday–Wednesday.

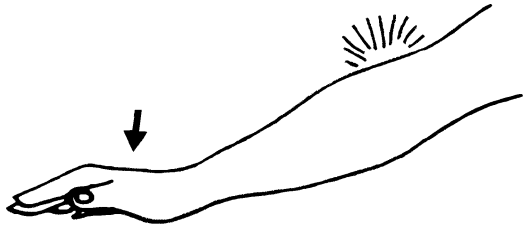

Fig. 1. Test 2. Resisted wrist extension.

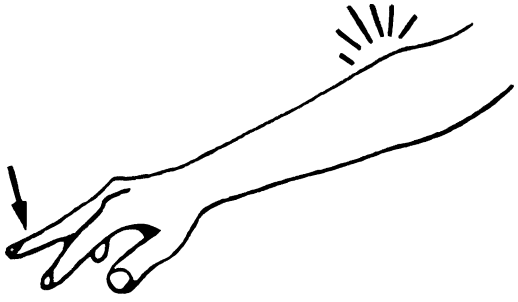

Fig. 2. Test 3. The middle finger test.

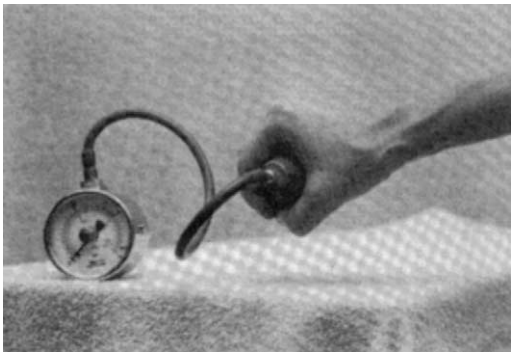

Fig. 3. Test position of the martin vigorimeter.

The first treatment was performed directly after the initial examination. A reexamination was performed the day after the final treatment and after 3 and 6 months using the same test procedure. Subjective outcome was registered by using a 7-step scale at the follow-ups in which 1 denoted completely recovered, 4 unchanged and 7 much worse.

In order to conceal study group assignments, sequentially numbered, opaque-sealed envelopes were used in the randomization of patients. The physiotherapist—the evaluator—was not aware of the treatment schedule and not involved in the treatments, examined the patients before and the day after the fourth treatment and at the follow-ups after 3 and 6 months. The assistant nurses who administered the treatments were unaware of which group the patients belonged to and so were the patients. The placebo/corticosteroid code was broken after the last 6-month follow-up.

Statistical analysis

The Mann–Whitney *U*-test of two independent samples and the Chi square test with Yates correction were used for the statistical analyses. In case of small numbers, Fisher's exact test was used. Statistical significance was assumed with $p < 0.05$.

The statistical program used was EPI-info version 6.04 B (Centre for Disease Control, Atlanta, USA).

The study was approved by the Ethics Committee, Gothenburg University, Sweden and written informed consent was obtained by the patients.

Results

Sixty-four patients completed the treatment period—31 patients in the placebo group and 33 patients in the corticosteroid group.

Twenty-eight patients in the placebo group were affected in the dominant arm compared to 19 patients in the corticosteroid group ($p = 0.007$) (Table 1). Both groups had a similar general condition regarding the cause of pain and previous treatment (Table 2). When evaluating the grip strength, the patients in the placebo group demonstrated a higher pain threshold when gripping compared to the patients in the corticosteroid group ($p = 0.04$) (Table 3).

Twenty-three patients dropped out between the trial of four treatments and the 3-month follow-up. Because of residual pain, other treatments were tried. The number of patients who dropped out—outside the control

Table 1. History of the patients who completed the treatment period

	Placebo group	Corticosteroid group
No. of patients	31	33
Male/female	20/11	21/12
Median age	45 (22–64)	50 (37–64)
Pain in dominant/nondominant arm (no.)	28/3*	19/14
Median duration of pain (months)	4 (1–36)	6 (1–48)

*$p = 0.007$.

of this study—did not differ statistically from those who completed the study except for a lower pain threshold when gripping ($p = 0.04$) (Table 4). There was a male dominance in the group that completed the study.

Forty-one patients appeared at the 3- and 6-month follow-up. No statistical difference could be observed between the outcomes of the four tests before and after the four treatments or at the follow-ups (Table 5) and no statistical differences were observed between the groups. The patients in both groups reported a subjective improvement throughout the study; however, no statistical differences could be observed between the groups (Table 6).

Table 2. Cause and previous treatment reported by the patients who completed the treatment period ($n = 64$)

	Placebo group	Corticosteroid group
Cause		
Sport	4	6
Work	15	17
Other activities	10	7
Unknown	2	3
Previous treatment	15	15
Steroids	7	7
Antiinflammatory drugs	4	2
Other treatments	4	6
Untreated	16	18

Table 3. Pretreatment values of the test procedure

	Placebo group	Corticosteroid group
No. of patients	31	33
Palpation pain lateral epicondyle (no.)	31	33
Pain-resisted wrist extension (no.)	31	32
Pain dig III test (no.)	29	30
Pain vigorimeter test (no.)	31	33
Pain threshold when gripping (kPa)	23	17*
Pain threshold when gripping (VAS)	22	24

KPa = kilopascal, median value. *$p = 0.04$. VAS = Visual Analog Scale, length 100 mm. The median value in mm.

No side effects were reported during or after the treatment period.

Discussion

In this study, no significant difference concerning the pain-relieving effect could be observed between the corticosteroid group and the placebo group. However, an identical improvement was observed in both groups throughout the study.

The great number of 'drop outs' demonstrated a significant lower pain threshold when gripping and they were also more frequently affected in the dominant arm, compared to the remaining 41 patients. The amount of dropouts may also indicate an insufficient or inappropriate pain-relieving method. As the pretreatment condition was similar in the two groups, the observed improvement in both groups probably reflects the spontaneous recovery known to occur (Garden, 1961; Verhaar, 1994; Putnam & Cohen,

Table 4. Pretreatment values of patients who dropped out after the treatment period and of patients who completed the study

	Patients who dropped out	Patients who completed the study
No. of patients	23[1]	41[2]
Male/female	11/12	30/11
Median age	45 (22–64)	50 (37–64)
Pain in dominant arm (no.)	17	30
Median duration of pain (months)	6 (range 1–36)	4 (range 1–48)
Palpation pain lateral epicondyle (no.)	23	41
Pain-resisted wrist extension (no.)	23	40
Pain dig III test (no.)	21	38
Pain vigorimeter test (no.)	23	41
Pain threshold when gripping (kPa)	16	24*
Pain threshold when gripping (VAS)	20	24

[1]Ten patients from the placebo group and 13 patients from the corticosteroid group. [2]Twenty-one patients from the placebo group and 20 patients from the coticosteroid group. kPa = kilopascal, median value. VAS = Visual Analog Scale, length 100 mm. The median value in mm. *$p = 0.04$.

Table 5. Results of the test procedure. The 41 patients who completed the study before and after the treatments, after 3 months and 6 months

	Pretreatment		Posttreatment		3 months		6 months	
	Placebo	Cortico steroid	Placebo	Cortico steroid	Placebo	Cortico steroid	Placebo	Cortico steroid
No. of patients	21	20	21	20	21	20	21	20
Palpation pain lateral epicondyle (no.)	21	20	20	18	10	9	6	7
Pain-resisted wrist extension (no.)	21	19	21	17	9	8	4	5
Pain dig III test (no.)	20	18	17	14	6	7	4	6
Pain vigorimeter test (no.)	21	20	21	17	8	10	6	5
Pain threshold when gripping (kPa)	27	20	48	40	74	70	84	79
Pain threshold when gripping (VAS)	22	29	18	9	0	5	0	0

Pretreatment = values before the first treatment. Posttreatment = the values the day after the fourth treatment. No. = number of patients. kPa = kilopascal, the values denote the median values of the two groups. VAS = Visual Analog Scale, length 100 mm. The median value in mm.

Table 6. Subjective outcome reported by the patients

	1 P/C	2 P/C	3 P/C	4 P/C	5 P/C	6 P/C	7 P/C
Posttreatment (no. of patients)	1/1	7/6	4/7	9/6	0/0	0/0	0/0
3 months (no. of patients)	7/9	10/6	2/4	2/1	0/0	0/0	0/0
6 months (no. of patients)	14/12	4/4	2/2	1/2	0/0	0/0	0/0

Post treatment = the day after the fourth treatment. P = placebo group, C = corticosteroid group. 1 = completely recovered, 2 = much better, 3 = slightly better, 4 = unchanged, 5 = slightly worse, 6 = worse, 7 = much worse.

1999) and/or the result of the direct electric current applied to both groups (Cummings, 1987) and/or the placebo effect (Wall, 1994).

The specificity of the test procedure, used in this study, corresponds very well to the results shown in a previous study (Haker, 1993) which further support the validity of the tests used.

Three to six iontophoresis treatments are recommended in the literature (Harris, 1982; Gudeman et al., 1997). In this study, four treatments were given and whether the result would have been changed by adding another two treatments is not known.

In order to minimize the specific putative effect while maintaining the psychological impact of the corticosteroid, the placebo group was administered saline, resulting in an externally identical experimental procedure (Vincent & Richardson, 1986; Richardson, 1994). Therefore, it is reasonable to conclude that the substances delivered in the two groups did not exert any pain-relieving effect or they both did.

Adding a third control group in which no treatment, but instructions were given, may have increased the quality of this study. However, it is not likely that those patients who were randomized into the third group would have accepted to wait for treatment for another 6 months. This assumption is supported by the fact that those patients who dropped out after the treatment period demonstrated a lower pain threshold when gripping.

In a well-performed study by Hay and collaborators, it was concluded that early local corticosteroid injection is effective for lateral epicondylitis. Compared to naproxen and placebo tablets, there was no difference at the 1-year follow-up (Hay et al., 1999).

The existing evidence on corticosteroid injections for the treatment of tennis elbow is not conclusive (Assendelft et al., 1996) and local steroids do have effects apart from the inflammatory reaction (Guttu, Page, Laskin, 1990). One possible effect of the steroid injection might be as a result of acute influences of the sensory nerves and the levels of substance P (SP) and calcitonin gene-related peptide (CGRP), which have been found at the origin of the ECRB muscle in patients with tennis elbow and in healthy controls (Ljung et al., 1999a).

Ljung and colloborators are also suggesting that frequent mechanical involvement may affect sensory innervation. These sensory nerve fibers not only transmit nociceptive information to the spinal cord but also have efferent effects such as involvement in vasodilation and plasma extravasion—the neurogenic inflammation (Ljung et al., 1999a). Sympathetic efferents may be activated by sensory fibers to release mediators which, in turn, overexcite the sympathetic nervous system and exacerbate pain (Levine et al., 1986). Ljung and coworkers have also recently demonstrated a morphological substrate. They found that the ECRB muscle origin is supplied with heterogeneously distributed sympathetic and sensory innervations and that there appears to be an imbalance between the vasoconstrictor and vasodilator innervations along the vascular tree in this region (Ljung et al., 1999c).

Perspectives

The results of the present study do not support the use of corticosteroid iontophoresis in the treatment of lateral epicondylalgia and should be considered as early ones. When calculating the power after finishing the study several thousands of patients would have been required to reach the 5% level of significance. The method requires more investigations to be fully evaluated.

Besides discomfort and pain, lateral epicondylalgia leads to economic consequences such as sick leave, workers' compensation claims, transfer to lower-paid jobs and even early retirement (Dimberg, 1987). For that reason, further research should be carried out to clarify the pathology of lateral epicondylalgia as well as the role of steroids and other treatments in this painful condition to promote casual and selective treatments.

Key words: corticosteroids; iontophoresis; lateral epicondylalgia; pain; tennis elbow.

Acknowledgements

Financial support was obtained from the occupational health Borashalsan and FOU-centrum in Alvsborg county. Decadron® was achieved from Merck Sharp & Dohme, Sweden. Many thanks to Dr Ronny Gunnarsson, Development Unit, Primary Health Care, Alvsborg for statistical help and to assistant nurse, Rigmor Ehnar, for skillful help in the iontophoresis treatments.

References

Allander E. Prevalence and remission rates in some common rheumatic diseases or syndromes. Scand J Rheumatol 1974; 3: 145–153.

Assendelft WJJ, Hay EM, Adshead R, Bouter L. Corticosteroid injections for lateral epicondylitis: a systematic overview. Br J General Prac 1996; 46: 209–216.

Bertolucci LE. Introduction of inflammatory drugs by iontophoresis: double-blind study. J Orthop Sports Phys Ther 1982; 2: 103–108.

Binder A, Parr G, Thomas P, Hazleman B. A clinical and thermography study of lateral epicondylitis. Br J Rheumatol 1983; 22: 77–81.

Bosworth DM. The role of the orbicular ligament in tennis elbow. J Bone Joint Surg (Am) 1955; 3: 527–533.

Carlsson AM. Assessment of chronic pain I. Aspects of the reliability and validity or the visual analogue scale. Pain 1983; 16: 87–101.

Chard MD, Cawston TE, Riley GP, Gresham GA, Hazleman BL. Rotator cuff degeneration and lateral epicondylitis: a comparative histological study. Ann Rheum Dis 1994; 53: 30–34.

Chard MD, Hazleman BL. Tennis elbow: a reappraisal. Br J Rheumatol 1989; 28: 186–190.

Chien YW, Banga AK. Iontophoretic (transdermal) delivery of drugs: overview of historical development. J Pharm Sci 1989; 78: 353–354.

Coonrad RW, Hooper WR. Tennis Elbow: its course, natural history, conservative treatment and surgical management. J Bone Joint Surg (Am) 1973; 6: 1177–1182.

Coonrad RW. Tennis elbow. J Am Acad Orthop Surg 1986; 35: 94–101.

Cummings J. In: Nelson RM, Currier DP. Clinical electrotherapy. Norvalk, CT, Appleton & Lange, 1987: 231–241.

Cyriax JH. The pathology and treatment of tennis elbow. J Bone Joint Surg (Am) 1936; 4: 921–940.

Demirtas RN, Oner C. The treatment of lateral epicondylitis by iontophoresis of sodium salicylate and sodium diclofenac. Clin Rehabil 1998; 12: 23–29.

Dimberg L. The prevalence and causation of tennis elbow (lateral humeral epicondylitis) in population of workers in an engineering industry. Ergonomics 1987; 3: 573–580.

Garden S. Tennis Elbow. Bone Joint Surg (Am) 1961; 1: 100–106.

Geoffroy P, Yaffe MJ, Rohan I. Diagnosing and treating lateral epicondylitis. Can Fam Phys 1994; 40: 73–78.

Glass J, Stephen R, Jacobson S. The quantity and distribution of radiolabeled dexamethasone delivered to tissue by iontophoresis. Int J Derm 1980; 9: 519–525.

Goldie I. Epicondylitis lateralis humeri. Act Chir Scand 1964; (Suppl) 339.

Gudeman SD, Eisele SA, Heidt Jr., Colosimo AJ, Stroupe AL. Treatment of plantar fascitis by iontophoresis of 0.4% Dexamethasone: a randomized, double-blind placebo-controlled study. Am J Sports Med 1997; 3: 312–316.

Guttu RL, Page D, Laskin DM. Delayed healing of muscle after injection bupivacaine and steroid. Ann Dent 1990; 49: 5–8.

Haker E. Lateral epicondylalgia: diagnosis, treatment and evaluation. Crit Rev Phys Rehab Med 1993; 5: 129–154.

Harris PR. Iontophoresis: clinical research in musculoskeletal inflammatory conditions. J Orthop Sports Phys Ther 1982; 2: 109–112.

Hasson SM, English SE, Daniels JC, Reich M. Effect of iontophoretically delivered dexamethasone on muscle performance in a rheumatoid arhtritic joint: a case study. Arthritis Care Res 1988; 3: 177–182.

Hay EM, Paterson SM, Lewis M, Hosie G, Croft P. Pragmatic randomised controlled trial of local corticosteroid injection and naproxen for treatment of lateral epicondylitis of elbow in primary care. BMJ 1999; 319: 964–968.

Kahn J. Acetic acid iontophoresis for calcium deposits. Phys Ther 1977; 57: 658–660.

Kivi P. The etiology and conservative treatment of humeral epicondylitis. Scand J Rehabil Med 1982; 15: 37–41.

Kraushaar BS, Nirschl RP. Tendinosis of the elbow (Tennis Elbow). J Bone Joint Surg (Am) 1999; 2: 259–278.

Labelle H, Guibert R, Joncas J, Neuman N, Fallah M, Rivard HH. Lack of scientific evidence for the treatment of lateral epicondylitis of the elbow. J Bone Joint Surg (Br) 1992; 5: 646–651.

LeDuc S. Electric Ions and Their Use in Medicine. Liverpool; Rebman Ltd 1908.

Levine JD, Dardick SJ, Roizen MF, Helms C, Basbaum AI. Contribution of sensory afferents and sympathetic efferents to joint injury in experimental arthritis. J Neurosci 1986: 6; 3423–3429.

Ljung BO, Forsgren S, Fridén J. Substance P and calcitoning gene-related peptide expression at the extensor carpi radialis muscle origin: implications for the etiology of tennis elbow. J Orthop Res 1999a; 17: 554–559.

Ljung BO, Forsgren S, Fridén J. Sympathetic and sensory innervations are heterogeneously distributed in relation to the blood vessels at the extensor carpi radialis brevis muscle origin of man. Cells Tissues Organs 1999c; 165: 45–54.

Ljung BO, Lieber RL, Fridén J. Wrist extensor muscle pathology in lateral epicondylitis. J Hand Surg 1999b; 24: 177–183.

Morris H. The Riders sprain. Lancet 1882; 29: 133–134.

Murtagh JE. Tennis elbow. Aus Fam Physician 1988; 2: 90–95.

Nirschl RP. Elbow tendinosis/Tennis elbow. Clin Sports Med 1992; 4: 851–869.

Nirschl RP, Pettrone FA. Tennis elbow. The surgical treatment of lateral epicondylitis. J Bone Joint Surg (Am) 1979; 6: 832–839.

Nirschl RP. Tennis elbow. Orthop Clin North Am 1973; 3: 787–800.

Petelenz TJ, Buttke JA, Bonds C et al. Iontophoresis of dexamethasone: laboratory studies. J Controlled Release 1992; 20: 55–66.

Priest JD, Braden V, Gerberich-Goodwin S. The elbow and tennis, Part 1: an analysis of players with and without pain. The Physician Sportsmedicine 1980; 4: 81–91.

Putnam MD, Cohen M. Painful conditions around the elbow. Orthop Clin North Am 1999; 1: 109–118.

Regan W, Wold LE, Coonrad R, Morrey BF. Microscopic histopathology of chronic refractory lateral epicondylitis. Am J Sports Med 1992; 6: 746–749.

Richardson PH. Placebo effects in pain management. Pain Rev 1994; 1: 15–32.

Roles NC, Maudsley RH. Radial tunnel syndrome. J Bone Joint Surg (Am) 1972; 3: 499–508.

Runge F. Zur Genese und Behandlung des Schreibekrampfes. Berliner Kliniche Wochenschrift 1873; 10: 245–248.

Sanderson JE, DeRiel S, Dixon R. Iontophoretic delivery of nonpeptide drugs: formulation optimization for maximum skin permeability. J Pharm Sci 1989; 78: 365–369.

Scott J, Huskisson EC. Graphic representation of pain. Pain 1976; 2: 175–184.

Singh P, Maibach HI. Iontophoresis in drug delivery: basic principles and applications. Crit Rev Ther Drug Carrier Syst 1994; 11: 161–213.

Smith RW, Papadopolous E, Mani R, Cawley MID. Abnormal microvascular response in lateral epicondylitis. Br J Rheumatol 1994; 33: 1166–1168.

Snijders CJ, Volkers ACW, Mechelse K, Vleeming A. Provacation of epicondylalgia lateralis (tennis elbow) by power grip or pinching. Med Sci Sports Exerc 1987; 5: 518–523.

Solveborn SA, Buch F, Mallmin H, Adalberth G. Cortisone injection with

anestetic additives for radial epicondylalgia (tennis elbow). Clin Orthop 1995; 316: 99–105.
Thomas D, Savage JP. Persistent tennis elbow: evaluation by infrared and nuclear medicine isotope scanning. Thermology 1989; 3: 132–136.
Thomas D, Siahamis G, Millicent M, Boyle C. Computerised infrared thermography and isotopic bone scanning in tennis elbow. Ann Rheumatol Dis 1992; 51: 103–107.
Thurston AJ. Conservative and surgical treatment of tennis elbow: a study of outcome. Aust N Z J Surg 1998; 8: 568–572.
Verhaar JAN. Tennis elbow. Anatomical, epidemiological and therapeutic aspects. Int Orthop (SICOT) 1994; 18: 263–267.
Vincent CA, Richardson PH. The evaluation of therapeutic acupuncture: concepts and methods. Pain 1986; 24: 1–13.
Wall PD. The placebo and the placebo response. In: Wall PD, Melzack R, eds, Textbook of Pain, 3rd edn. Churchill Livingstone, Edinburgh, Ch 71 1994.
Wright A, Vicenzino B. Lateral epicondylalgia II: Therapeutic Management. Phys Ther Rev; 2: 1997; 39–48.