Myofascial Pain Syndromes–Trigger Points

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During the past few months, several new studies, reviews and case studies on myofascial pain syndrome [MPS] and myofascial trigger points [TrPs] have been published. Myofascial pain syndrome and TrPs are increasingly being recognized not only in the United States, but also in Europe. Testa and colleagues [Milan, Italy] published an excellent review article detailing the clinical signs that lead to the diagnosis of MPS. Chaitow and Delany published a review article in an orthopedic journal, which may indicate an increasing acceptance of the concepts of MPS and TrPs by a medical discipline that previously has expressed little or no interest in the subject matter. Three other articles emphasized that MPS needs to be included in the differential diagnostic process of radiculopathy, dental pain, and pelvic pain, respectively.

New referred pain patterns were described from TrPs in the iliopsoas muscle causing knee pain and in the splenius capitis muscle causing tinnitus. Several articles on the use of botulinum toxin and MPS appeared in the literature. Following the integrated TrP hypothesis that suggests that excessive release of acetylcholine at the motor endplate leads to the formation of TrPs, the use of botulinum toxin is likely to increase for persistent MPS that has not responded well to other TrP therapy (1).

After 10 years of preparing this review of the pertinent literature on MPS and TrPs, David Simons welcomes the assistance of physical therapist Jan Dommerholt in preparing this review column. Each article review indicates whether it is prepared by Simons [DGS] or Dommerholt [JD].

TREATMENT


Summary

Forty subjects with a myofascial trigger point [TrP] in the trapezius muscle were randomly assigned to one of three groups. Myofascial trigger points were identified using the criteria established by Simons, Travell, and Simons (1). The demographic table indicates that the patients had pain for about three months. Patients were excluded from the study if they were treated with either TrP injections or physical therapy modalities within one year preceding the study.

Subjects in the first group received treatment with 20 minutes of transcutaneous elec-
TENS with symmetric, biphasic rectangular pulses with a 100 µsec duration, a current frequency of 60 Hz, and an intensity up to the patient’s perception of paresthesia, combined with trapezius stretches. Subjects in the second group were treated with 20 minutes of electrical muscle stimulation [EMS] with symmetric, biphasic rectangular pulses with a current frequency of 25 Hz, a pulse width of 250 µsec, a hold time of 3 sec, rest time of 6 sec and up/down ramping of 0.6 sec, also combined with trapezius stretches. For both groups, the negative electrode was placed over the TrP and the positive electrode over the acromial trapezius tendon insertion. Both groups received one session per day for two weeks. Subjects in the third group, the control group, were instructed in a home program of trapezius stretches only. The stretching program was the same for all groups and consisted of 10 stretches performed three times daily. Patients were evaluated prior to the study, immediately after the two-week treatment period, and three months after the treatment. The evaluation included a subjective pain assessment using a visual analog scale [VAS], a manual assessment of the pain threshold using a four-point scale [based on pain and local twitch responses], and goniometric range of motion [ROM] measurements of cervical lateral bending.

Prior to the study, there were no significant statistical differences between the three groups for VAS, pressure threshold, and ROM. After the two-week treatment period, subjects in group three [stretches only] did not present with any significant changes. For reported ethical reasons, the subjects were excluded from the treatment protocol and offered other treatment modalities. Group one [TENS] and group two [EMS] improved significantly for all three parameters, with the TENS group having slightly better outcomes. Three months after the treatment protocol, there were no significant differences between the two groups. The authors concluded that electrotherapy is a useful treatment modality for myofascial pain syndrome.

Comments

The authors are commended for undertaking this study. There is a paucity of studies considering the long-term effectiveness of electrotherapy for TrPs. Previous studies have only considered the immediate effect of various forms of electrotherapy (2,3). The outcome measures of VAS, ROM, and pressure threshold are appropriate. However, assessing pressure thresholds manually is highly unreliable even when using a four-point scale. The use of algometry is the current gold standard (4,5). Unfortunately, the results of this study are somewhat difficult to interpret due to several inaccuracies. For example, at one point, the authors state that “VAS [P < 0.01], ROM, and pressure threshold [P < 0.05] significantly improved in group two,” while a few sentences later, they state “only VAS was significantly improved [P < 0.05] in the group two patients.” In the table “Results of parameters at three time points” the results for TENS and EMS are grouped, making comparison between the two groups impossible. Although the authors concluded that TENS seemed to be more effective immediately after treatment, there was no statistical evidence of such. Both TENS and EMS reduced pain and increased ROM and pressure threshold and may be considered in the clinical management of persons with MPS. [JD]
distraction or mobilization, and 4. combination of number 2 and number 3 treatment programs. Blinded independent examinations before, three months after, and six months after treatment included a visual analog pain rating by the patient, the Roland-Morris Disability Questionnaire for LBP, the Short-Form Health Survey, the 71-item Minnesota Multiphasic Personality Inventory, the Confidence Score, and palpation for myofascial trigger points [TrPs] defined as a tender point with characteristic referred pain in the quadratus lumborum, gluteus maximus, gluteus medius, gluteus minimus, and piriformis muscles. Tenderness over the spinous processes and facets of the lumbar spine and of sacroiliac regions medial to the posterosuperior iliac spines identified articular dysfunction. All data were analyzed regardless of patient compliance.

All four groups showed significant reduction in pain and activity scores at three weeks with no further change at six months. Back school and combined therapy were consistently the best and the mean visual analog scores where significantly better for combined therapy than for only myofascial therapy. Otherwise there were no significant differences in outcome among therapies when compared to baseline. The marked effectiveness of back school was surprising since it was originally included as placebo treatment. When queried, participants were so satisfied because they joined the study to learn the cause of their problem, to discover how to avoid future bouts of LBP, and how to better control the pain if LBP recurred. Combined therapy was consistently but not statistically significantly better than myofascial therapy alone.

Comments

The methods section indicates that this was a study of the results of treatment of TrPs that were not clearly identified as active or latent. The muscles selected for examination and treatment may not have included ones that had TrPs contributing significantly to the patient’s pain. For pain relief, it is important to concentrate on treating the active TrPs when they are the cause of the pain. The noteworthy efficacy of back school with a targeted stretch program is consistent with the result reported by Hanten et al. (6). The design of this study does not help to identify how much more effective the treatment programs might have been if they had included the back school program also.

In addition, the results suggest that a study is needed in which the initial examinations for TrPs and articular dysfunctions are related to the relative efficacy of the chiropractic and specific TrP therapies for those patients who have either one or both of the diagnoses. The critical need is for competent scientific studies like this to address the cause of the pain, and to begin to identify which therapy or combination of therapeutic approaches is most effective for the specific causes. Specifically we need to know more about the relationship between TrPs and articular dysfunctions as causes of LBP and how they interact. [DGS]


Summary

The author summarizes his clinical experience as a massage therapist in treating patients with the symptoms of lateral epicondylitis or tennis elbow and gives detailed descriptions of how to manually inactivate myofascial trigger points in nine muscles around the elbow that refer pain and tenderness to the lateral epicondyle and may need treatment for relief. The muscles included the extensor carpi radialis longus and brevis, extensor carpi ulnaris, brachioradialis, supinator, anconeus, and the long, medial and lateral heads of the triceps brachii. The author emphasizes the importance of using a ball to apply the pressure rather than the fingers to avoid muscle overload and the importance of teaching the patient effective self treatment techniques, but makes no reference to stretch as part of the treatment.

Comments

These are valuable descriptions of how to treat these TrPs simply and effectively and for the clinician, safely. The neglect of stretching
as part of the treatment, and the statement that the extensor carpi radialis longus, brachioradialis, and supinator can be treated as one muscle reflects a disregard of basic anatomy, kinesiology, and an understanding of the etiology of TrPs. This approach may be appropriate if the only treatment being considered is application of pressure to the area with a ball. [DGS]


Summary

Twenty-six subjects were selected from a group of 84 new patients presenting to a clinic for temporomandibular disorders using the “Research diagnostic criteria for temporomandibular disorders” by Dworkin and LeResche (7). The inclusion criteria were a dull regional pain in the face persisting for more than one month and muscles tender to palpation with recognizable pain that increases by palpation. The subjects were randomly assigned to one of two groups. Both groups received structured “counseling” or education addressing normal jaw function, parafunctions, and instructions for jaw muscle relaxation. Group two started a six-week physical therapy program at the beginning of the study. Group one started the same physical therapy program two weeks later for a total of four weeks. Physical therapy consisted of five minutes of continuous ultrasound therapy, followed by a 10-minute massage of the masseter and temporalis muscles, stretching of the masseter muscle with instructions for a home stretching program, another five minutes of massage, instructions for self massage twice daily combined with an application of a warm pad and awareness of parafunctions. Prior to the study, and after two, four, and six weeks, a blinded investigator determined the pressure pain threshold over the most bulky part of the masseter and temporalis muscles, a rating on a visual analog scale, and jaw function assessed by the Mandibular Function Impairment Questionnaire. At the end of the study, subjects in both groups showed “marked improvement” for all outcome measures; however, there were no differences in outcomes between the two groups. The authors commented that there were no differences in pain pressure thresholds between the symptomatic and nonsymptomatic sides.

Comments

Dworkin and LeResche’s research diagnostic criteria attempted to classify and quantify both the physical and psychosocial components of temporomandibular dysfunction (7). Although the criteria are widely used in dental research, they are remarkable simplistic where it involves muscle dysfunction. The section Axis I, Group I, Muscle Disorders, includes only tender muscles with or without limited mouth opening as the only criteria. De Laat and colleagues expanded the Dworkin and LeResche criteria by requiring the subject’s recognition of pain with palpation. Recognition of pain is an essential component of Simons, Travell and Simons’ criteria for myofascial pain syndrome; however, the authors did not appear to consider these more specific criteria in this study (1). It should then come as no surprise that the pressure thresholds were not different between sides. No effort was made to identify the most painful region in the muscles, which would likely have identified myofascial trigger points [TrPs]. The physical therapy intervention did not address specific TrPs either. Future studies would be more interesting and clinically relevant, if one group would receive specific TrP therapy, while the other group would receive a more generic physical therapy program as described in this study. [JD]


Summary

Seventy-seven patients with refractory myofascial pain syndrome were included in this study of the effect of botulinum toxin injections. Inclusion criteria were 1. presence of
muscle spasm in the form of a taut band [a contracted group of muscle fibers] with a zone of tenderness within it; 2. pain of maneuvering or stretching; 3. existence of trigger points [TrPs] with associated referred pain [using the criteria established by Simons, Travell, and Simons (1)]; 4. failure of conservative and physical therapy to relieve myofascial pain. The physical examination included palpation of a taut band and myofascial trigger point [TrP], eliciting referred pain with palpation, and immediate pain relief following a local anesthetic injection. Treatment involved the injection of 1 ml [10 units] botulinum toxin type A in various muscles. The total dosage of the botulinum toxin per subject varied between 50 and 100 units. Needle placement was guided by multiplanar continuous fluoroscopic imaging for deep muscles, including the iliopsoas, quadratus lumborum, and piriformis, with a standardized injection site for each muscle. In superficial muscles, including the anterior and medial scalenes, trapezius, supraspinatus and brachioradialis, needle placement was electromyographically [EMG] guided by locating the motor endplate. Outcome measures included a visual analog scale, the Lattinen test for pain intensity and its impact on daily living, the Oswestry Questionnaire, and the Spanish version of the Hospital Anxiety and Depression Scale. Physical therapy treatment and pharmacological management were maintained throughout the study period. Outcome measures were assessed at 15, 30, and 90 days after the study. Improvements were noted in scores on the visual analog scale and the Oswestry, but not for the Lattinen and the Spanish version of the Hospital Anxiety and Depression Scale. Few patients reported side effects from the intervention.

Comments

The authors readily acknowledge some of the limitations of this study, especially the lack of a control group. However, several other questions arise. For example, it would be valuable to know what kind of conservative therapy and physical therapy the patients had received prior to the study. Also, the authors appear to have performed a diagnostic TrP injection with a local anesthetic prior to the botulinum toxin injection as part of the physical examination. Did the diagnostic injection elicit a local twitch response? How did the authors determine that the results of the study were in fact due to the botulinum toxin injection and not due to the diagnostic TrP injection, which could well have inactivated the TrPs? While the authors seem reasonably familiar with the current thinking about myofascial pain syndrome, it is not clear why they used a fixed injection site and videofluoroscopy for needle placement in deep muscles, and EMG only for superficial muscles. Endplate noise is thought to be a characteristic of TrPs and it would seem more logical to determine the location of the motor endplate for both superficial and deep muscles to increase specificity of the intervention (8). The authors do not explain why they decided to use a standardized dose of 10 units of botulinum toxin per injection for all muscles. Ten units may be sufficient for the anterior and medial scalenes, but are insignificant for the much larger iliopsoas and piriformis muscles. In clinical practice, botulinum toxin injections of TrPs with 25, 50, or even 100 units are common.

Regrettably, the authors use the terms “muscle spasm” and “taut band” interchangeable. As Simons and Mense have summarized, a muscle spasm is conceptually a very different phenomenon than a taut band (9). By definition, a muscle spasm is always associated with involuntary EMG activity, while a taut band is a group of muscle fibers in contracture without EMG activity. Using the terms interchangeably leads to confusion. [JD]

ETIOLOGICAL MECHANISMS


Summary

This controlled study compares the pain sensitivity in the trapezius muscle and the anterior tibialis of sixteen healthy subjects. The authors measured muscular electrical and mechanical pain thresholds, cutaneous electrical pain thresholds, and cutaneous and muscular
electrical sensory thresholds. Temporal summation was assessed by repetitive electrical stimulation. The main finding of the study was a higher pain sensitivity in the trapezius compared to the anterior tibialis muscle. In the discussion section, the authors underscored the significance of their findings for chronic myofascial pain disorders, including tension-type headaches. In previous studies from the same research group, patients with tension-type headaches commonly featured increased myofascial tenderness and hardness in the pericranial muscles (10-12). In the current study, the authors speculated about possible underlying mechanisms and suggested that referred pain patterns from the pericranial muscles may be perceived as headache. Also, the authors mentioned that ongoing nociceptive input from the pericranial myofascial tissues may result in central sensitization, myofascial pain, and chronic pain conditions.

Comment

It is interesting to see the many similarities between the discussion section of this excellent research study and the literature on myofascial trigger points [TrPs]. The findings of increased myofascial tenderness, hardness, referred pain causing headaches, and central sensitization as a result of persistent nociceptive input from muscle could easily have been copied from the Trigger Point Manual or the more recent Muscle Pain book by Mense, Simons, and Russell (1,13). Yet, this Danish research team, responsible for many other outstanding studies of migraine and tension-type headaches, has not incorporated recent TrP research in their thought process. Myofascial trigger points can certainly account for at least one mechanism for the frequent occurrence of muscle pain in the shoulder and neck regions (14,15). It is conceivable that in this study the location of stimulation of the trapezius muscle was in or near a TrP or at least a taut band in the middle of the descending trapezius muscle midway between the spinous process of C7 and the acromion. We hope that perhaps in future studies the researchers will differentiate between TrPs, taut bands and adjacent muscle tissues. [JD]

CASE REPORT


Summary

This case report describes a 33-year old female patient with an eight-year history of deep left knee pain. The pain started after a period of prolonged standing. Initial knee examinations did not reveal any abnormalities. Further studies revealed a dysplastic left hip with osteoarthritis. The patient’s symptoms were managed conservatively for several years, after which she underwent several surgical procedures, including a periacetabular osteotomy, a left femoral osteotomy, and a lateral shaft grafting procedure with bone harvested from the left anterior iliac crest. Eventually, the patient underwent a left total hip replacement that temporarily relieved her knee pain. Three months later, the patient experienced a relapse of knee pain, which she managed pharmacologically. A few months later, she experienced another exacerbation at which point she presented to an acupuncture clinic. Again, no abnormalities were observed in the left knee. However, the patient’s knee pain was provoked by left hip flexion combined with adduction or internal rotation. Pressure over a myofascial trigger point [TrP] in the left iliopsoas muscle also provoked the patient’s pain complaint. The patient was treated with deep dry needling of the TrP. The needling did not provoke the patient’s usual left knee pain and a decision was made to treat the TrP subsequently with percutaneous electrical stimulation/electroacupuncture with a stimulation frequency of 2 Hz for 15 minutes. After only two treatments, the patient had no further complaints of knee pain.

Comment

Myofascial trigger points are commonly involved in orthopedic injuries and joint pains, yet frequently overlooked. In 2001, Bajaj and colleagues already described TrPs associated with lower extremity osteoarthritis (16). This case report is significant for several reasons. First, the author used Simons, Travell,
Simons criteria for a TrP, which makes comparison to other reports and studies possible (1). Second, the author realized that the patient’s recognition of the pain is an important parameter in determining whether a TrP is clinically relevant (1,17). Third, referred pain from the iliopsoas muscle to the knee has not been described previously. Fourth, even though the patient had experienced recurrent knee pain for eight years, after proper identification and treatment of the responsible TrP, the pain complaint disappeared after only two treatments. Unfortunately, the case report did not include any longer term outcome measures. [JD]

**REVIEWS**


The awareness of myofascial pain syndrome and its hallmark feature, the myofascial trigger point [TrP] is growing not only across different disciplines, but also across different continents. From Italy comes this comprehensive meta-analysis of the literature on the clinical aspects, diagnostic tools and pathophysiology of TrP. The authors independently analyzed controlled trials and review articles included in Medline [from 1966 to December 2002] and Embase [from 1988 to December 2002]. The clinical signs of a TrP, including the taut band, jump sign, reproduction of the patient’s pain, local twitch response, referred pain, restricted range of motion, muscle weakness, and associated phenomena, are well described with multiple up-to-date references. The authors include an overview of the various interrater reliability studies of the diagnostic process and conclude that a reliable clinical assessment of TrPs can be achieved with adequate training. The minimal criteria to identify a TrP are the presence of a taut band in the muscle, a very tender point in the taut band, and the patient’s recognition of the pain. The authors state correctly that referred pain and local twitch responses are confirmatory findings.

It cannot be emphasized enough that the identification of TrPs requires training. The interrater reliability study by Gerwin and colleagues demonstrated that satisfactory reproducibility can be achieved after training (17). Other medical procedures, for example, auscultation or review of cranial nerves, require many hours of practice and are included in the medical training of all doctors. Few clinicians receive training in the accurate identification of TrPs. [JD]


The discipline of orthopedic surgery has not been overwhelmingly interested in myofascial pain syndrome, which makes this review a particular welcome step in the right direction as it was published in a respectable orthopedic journal. Chaitow and DeLany place myofascial pain syndrome in a broad context of neuromuscular therapy, that considers not only biomechanical factors, but also biochemical and psychosocial aspects of patient care. Neuromuscular therapy has its roots both in Europe and in the United States. Chaitow and Delany have combined various approaches into a comprehensive framework, which has become known as Neuromuscular Therapy, American Version. The goals of Neuromuscular Therapy, American Version include trigger point release, normalization of muscle ischemia and excessive tension in connective tissues, relief of neural entrapment or compression, correction of postural alignment, nutritional awareness, and emotional well-being. Where appropriate, they have adopted concepts from various fields not only of medicine, but also of architecture and engineering [i.e., tensegrity], biomechanics, and psychology. The authors emphasize the importance of a comprehensive history and examination and a thorough review of perpetuating factors. The authors suggest using a ten-point scale to quantify the level of applied pressure when addressing myofascial trigger points and caution against using pressure beyond mild or moderate discomfort with a score between five and seven. Five brief appendices summarizing pertinent foundations of neuromuscular therapy complete this interesting review article. [JD]

Earlier this year, the Texas Heart Institute Journal featured two articles honoring Dr. Janet G. Travell. Dr. David Simons recounts Travell’s many contributions to cardiology, while Travell’s daughter, Virginia Wilson, provides a delightful perspective and a glimpse into the life and work of her mother (18,19). Short of Travell’s autobiography “Office Hours: Day and Night,” few publications summarize Travell’s achievements as succinct, yet with a personal touch (20).

Simons starts the article with describing his first meeting with Travell in 1963, at a time when she was the White House physician to President Kennedy. He provides a fascinating historical overview of Travell’s medical career. Initially, Travell trained as a cardiologist and pharmacologist. During the 1930-ies, she was appointed as instructor in pharmacology at Cornell University Medical School and contributed multiple research papers on the use of digitalis in clinical practice. After she started developing the myofascial pain concepts, Travell continued to publish several articles applying spray and stretch techniques to painful cardiac conditions, including acute myocardial infarction, as well as myofascial trigger points. Following the 1963 meeting, Travell and Simons started a fruitful collaboration culminating in the publication of many articles and the two-volume Trigger Point Manual (1,21). Throughout the article, Simons captures the inquisitive mind of Dr. Travell, the results of which continue to benefit patients around the globe. [JD]

**BRIEF REVIEWS AND ABSTRACTS**


Awareness of myofascial trigger points [TrPs] is increasing in different medical disciplines. This abstract was published in the otorlaryngology literature. Thirty-four consecutive patients with tinnitus were evaluated for TrPs in the infraspinatus, levator scapulae, trapezius, splenius capitis, medial scalene, sternocleidomastoid, digastric, deep masseter, and anterior temporalis using the Travell and Simons criteria. The authors concluded that TrPs are very common in patients with tinnitus. About 70 percent of the patients had TrPs in at least one muscle. Tinnitus could be modulated with stimulation of TrPs in the splenius capitis, deep masseter, and sternocleidomastoid muscles. While Simons, Travell, and Simons have described tinnitus as part of the referred pain patterns of the deep masseter and sternocleidomastoid muscles, this is the first time that TrPs in the splenius capitis muscle were described in relation to tinnitus (1). This scientific poster presentation deserves consideration for publication in a peer-reviewed journal. [JD]


The authors present a brief review of the contributions of myofascial trigger points [TrPs] to various pelvis pain problems through five short case descriptions and a concise summary of the nature, diagnosis, and treatment of TrPs. One case report illustrates how visceral disease can present as a TrP in the abdominal wall, while others describe referred pain patterns from TrPs in the abdominal wall to the vagina, the impact of stressful conditions on TrPs, and the need to examine patients for the presence of TrPs in the intra-pelvic muscles, including the levator ani and obturator internus. This article may assist gynecologists and other clinicians in recognizing the implications of TrPs in the pelvic region. [JD]


This review summarizes the current understanding of the mechanisms of botulinum toxin type A in a variety of clinical pain syndromes, including cervical dystonia, spasticity, myofascial pain, and others. Dr. Lang provides a brief overview of the antinociceptive mechanisms of action, including chemode-
nervation at the nerve terminal and motor end-plate, and possible anti-inflammatory mechanisms, which to date are still poorly understood. The article emphasizes that botulinum toxin should only be used as part of a multifaceted treatment plan that includes physical therapy, reconditioning, strengthening and flexibility programs, and manual treatments. [JD]


Thant and Tan have prepared a similar review article on the use of botulinum toxin. The list of therapeutic applications is growing and includes cosmetic use for facial wrinkles; various pain syndromes, including myofascial pain syndrome; conditions associated with hypersecretion of glands, such as hyperhidrosis; and conditions associated with excessive or dyssynergic muscle contractions, such as esophageal spasm. Some of the conditions mentioned in the dyssynergic muscle contraction category may also be due to myofascial trigger points, such as bruxism and vaginismus. The authors summarize that the research of the clinical use of botulinum toxin is still limited. Studies often suffer from small sample size, methodological errors, and descriptive inaccuracies. Prospective long-term outcome studies are lacking. [JD]


A great number of musculoskeletal disorders, including myofascial pain syndrome, can generate referred pain and weakness mimicking cervical or lumbosacral radiculopathy. The author emphasizes that these disorders can be present with or without radiculopathy and need to be considered in the differential diagnosis. In addition to myofascial pain syndrome, some of the other disorders include neurologic disorders, such as herpes zoster, peripheral mononeuropathy and multiple sclerosis, but also orthopedic abnormalities, such as rotator cuff tears, epicondylitis, trochanteric bursitis, and others. The most common referred pain patterns from myofascial trigger points are illustrated throughout the article. [JD]


In the introduction to this article, the authors depict the dilemma a dentist is faced with when a patient presents with dental pain without objective clinical or radiographic findings. Rather than diagnosing this patient with a psychological diagnosis or with “atypical face pain,” the dentist should consider other non-dental causes of pain and dysfunction. After a brief discussion of odontogenic pain, the authors introduce a comprehensive list of eleven other toothache diagnoses, including cardiogenic, sinus, sialolith induced, acute otitis media induced, neoplastic, neurovascular, myofascial, neuropathic, cervical, herpetic, and psychogenic toothaches. The entire article is summarized in a “diagnostic algorithm for toothache of undetermined origin.” It is refreshing that the authors consider myofascial trigger points, rather than the more generic descriptive criteria by Dworkin and LeResche so commonly used in dentistry (7). [JD]

REFERENCES


