Corticosteroid Injections: Their Use and Abuse

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Abstract

Local injections of corticosteroids are commonly used in orthopaedic practice on the assumption that they will diminish the pain of inflammation and accelerate healing. Less often considered is the possibility that their use may delay the normal repair response. Among the multitude of conditions treated with corticosteroids are acute athletic injuries, overuse syndromes, nerve compression, bone cysts, and osteoarthritis. Unfortunately, there is a paucity of well-controlled studies that provide definitive recommendations for nonrheumatologic use of corticosteroids. Also troubling are the significant potential complications that can occur with their use. The authors believe that use of corticosteroids should be limited to the few conditions that have been proved to be positively influenced by them. Their use must be accompanied by a well-orchestrated treatment plan including close follow-up, physical therapy, and limitation of activities.

J Am Acad Orthop Surg 1994;2:133-140

Since their introduction into medicine, corticosteroids have been used to treat innumerable conditions and afflictions. The injection of corticosteroids into tissues for therapeutic purposes has been popular since 1953, when the results of intra-articular injections for rheumatoid arthritis were published by Hollander.[1] Today, steroid injections are commonly used for a multitude of pathologic conditions, such as impingement syndrome, nerve compression syndromes, ligament injuries, tendinitis, bursitis, fibrositis, fasciitis, arthritis, stenosing tenosynovitis, radiculopathies, ganglions, and simple bone cysts.

Unfortunately, very little is known about the exact mechanism of action of corticosteroids for each of the various treated conditions. This is due in part to their complex interactions in the body, the multitude of target organs, and the lack of well-controlled outcome studies. There is a legitimate concern that the indiscriminate use of corticosteroids may have unwanted and unknown side effects.

To better understand the magnitude of the use of corticosteroids in orthopaedic practice, Hill et al[2] conducted a study in which 400 randomly selected orthopaedists from the American Academy of Orthopaedic Surgeons were asked to complete a questionnaire. Of the 233 (58%) who responded, 90% used steroid injections in the treatment of their patients, administering on average 150 intra-articular and 193 extra-articular injections annually. The most common extra-articular indications are shown in Table 1. The preparations most commonly used were betamethasone sodium acetate -- betamethasone phosphate (28%) and methylprednisolone acetate (22%). Eighty-nine percent of the orthopaedists had observed a complication from a steroid injection (Table 2). Despite the limitations of this type of survey data, it is clear that corticosteroid injection therapy is commonly used by practicing orthopaedists and that complications are not infrequent.

A review of the literature has led us to the conclusion that the indications, mechanism of action, recommendations for use, and methods of complication avoidance for corticosteroid therapy in orthopaedic disease have been inadequately addressed and are in need of significant clarification, supported by well-controlled studies. In this article, we will discuss what is known about the mechanism of action of corticosteroids and their effects on various types of tissue.

Mechanism of Action

Corticosteroids are naturally occurring 21-carbon steroid hormones synthesized from cholesterol and produced in the adrenal glands. They have an effect, either direct or

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Table 1
Extra-articular Conditions Treated With Corticosteroid Injection by 233 Orthopaedists

Condition	No. of Orthopaedists (%)*
Elbow epicondylitis	217 (93)
Shoulder bursitis	211 (91)
Greater trochanteric bursitis	211 (91)
de Quervain tenosynovitis	203 (87)
Shoulder bicipital tendinitis	188 (81)
Pes anserinus bursitis	181 (78)
Plantar fasciitis	170 (73)
Myofascial trigger points	163 (70)
Carpal tunnel syndrome	131 (56)
Finger tenosynovitis	120 (52)
Tarsal tunnel syndrome	86 (37)
Achilles tendinitis	78 (33)
Back pain (epidural space injection)	57 (24)

*Values are number of orthopaedists who used corticosteroid injection for a given condition, with percentage in parentheses. (Adapted with permission from Hill JJ Jr, Trapp RG, Colliver JA: Survey on the use of corticosteroid injections by orthopaedists. *Contemp Orthop* 1989;18:39-45.)

indirect, on the metabolism of most tissues in the body. As hormones, corticosteroids pass through the cell membrane and bind to cytoplasmic receptors. This steroid-receptor complex enters the cell nucleus and, via its interactions with DNA, alters RNA, which in turn alters protein synthesis, particularly enzyme synthesis.[3] Since enzymes have far-reaching effects, many functions of the body are influenced by corticosteroids (Table 3).

Table 2
Corticosteroid Complications Observed by 233 Orthopaedists

Complication	No. of Orthopaedists (%)*
Subcutaneous fat atrophy	150 (64)
Skin pigmentation changes	125 (54)
Tendon rupture	91 (39)
Cartilage damage	46 (20)
Infection	42 (18)
Foreign-body reaction	18 (8)
Sterile abscess	15 (6)
Peripheral nerve injury	14 (6)
Muscle damage	9 (4)
Anaphylaxis	5 (2)
Vascular injury	1 (0)

*Values are number of orthopaedists who observed complication, with percentage in parentheses. (Adapted with permission from Hill JJ Jr, Trapp RG, Colliver JA: Survey on the use of corticosteroid injections by orthopaedists. *Contemp Orthop* 1989;18:39-45.)

In clinical orthopaedic practice, corticosteroids are used predominantly for their potent anti-inflammatory effects. However, the potential benefit of reduced inflammation can have negative effects on healing, since inflammation is an integral part of the healing process.

The healing process is generally considered to consist of four phases. The first, or inflammation, phase is manifested clinically by the well- known signs and symptoms of heat, redness, swelling, pain, and loss of function, which is what usually brings a patient to seek medical treatment.

During the inflammatory phase, there is a complex interaction of vascular, humoral, and cellular events controlled by chemical mediators. At the cellular level, there is increased membrane permeability, which results in edema. Leukocytes are drawn to the site of injury via chemotactic mediators and are assisted by phagocytic macrophages to remove damaged cell material. Hydrolytic enzymes are released from the leukocytes, producing arachidonic acid by hydrolysis of cell membrane phospholipids. The anti-inflammatory action of corticosteroids is mediated by the inhibition of phospholipase A(2), which catalyzes the breakdown of membrane phospholipid to arachidonic acid (Fig. 1). In contrast, nonsteroidal anti-inflammatory drugs (NSAIDs) inhibit the inflammatory process at the next step, after arachidonic acid production, by blocking the enzyme cyclo- oxygenase.[4] This helps to explain the broader effects of corticosteroids, since they inhibit

Table 3
Effects of Corticosteroids

Organ System or Tissue Type	Action
Adipose tissue	Lipolytic (increase free fatty acids and glycerol)
Muscle	Catabolic (muscle wasting)
Blood	Increase neutrophils and erythrocytes; decrease lymphocytes and eosinophils
Bone	Osteoporosis
Central nervous system	Mood lability, irritability, psychoses
Lung	Produce surfactant in fetal lung; bronchodilatation
Cardiovascular system	Increase blood pressure
Liver	Anabolic (stimulate gluconeogenesis and protein production)
Immune system	Immunosuppression; stabilization of lysosomal enzymes; anti-inflammatory
Nonspecific	Analgesic

Selection

Corticosteroids are available in various preparations, each with a different plasma half-life and solubility. As a result, there is a spectrum of variations in duration of action, strength, and dose. In Table 4 we have listed the steroids most commonly used by orthopaedists and have categorized them with respect to solubility. Since plasma half-life may not be an important factor in local steroid injections, we have listed only the water solubility characteristics. As a general rule, water-soluble preparations have a shorter duration of action than water-insoluble preparations.

both the cyclo-oxygenase and lipoxygenase pathways, whereas NSAIDs inhibit only the cyclo-oxygenase pathway. Thus, steroids inhibit the synthesis of leukotrienes in addition to prostaglandins and thromboxanes, all of which further activate the inflammatory response.

In the second, or repair and regeneration, phase, cellularity, vascularity, collagen synthesis, and growth factor concentration increase. The third, or remodeling, phase is characterized by a decrease in cellularity and an increase in matrix organization. The fourth, or maturation, phase can last for an extended period of time and corresponds to attempted restoration of normal tissue function.

Throughout the healing period, critical events occur that are specific to each phase. Interference with any of the phases could theoretically have deleterious effects. Since inflammation is an important component of the normal healing process, the anti-inflammatory action of corticosteroid therapy could be detrimental.

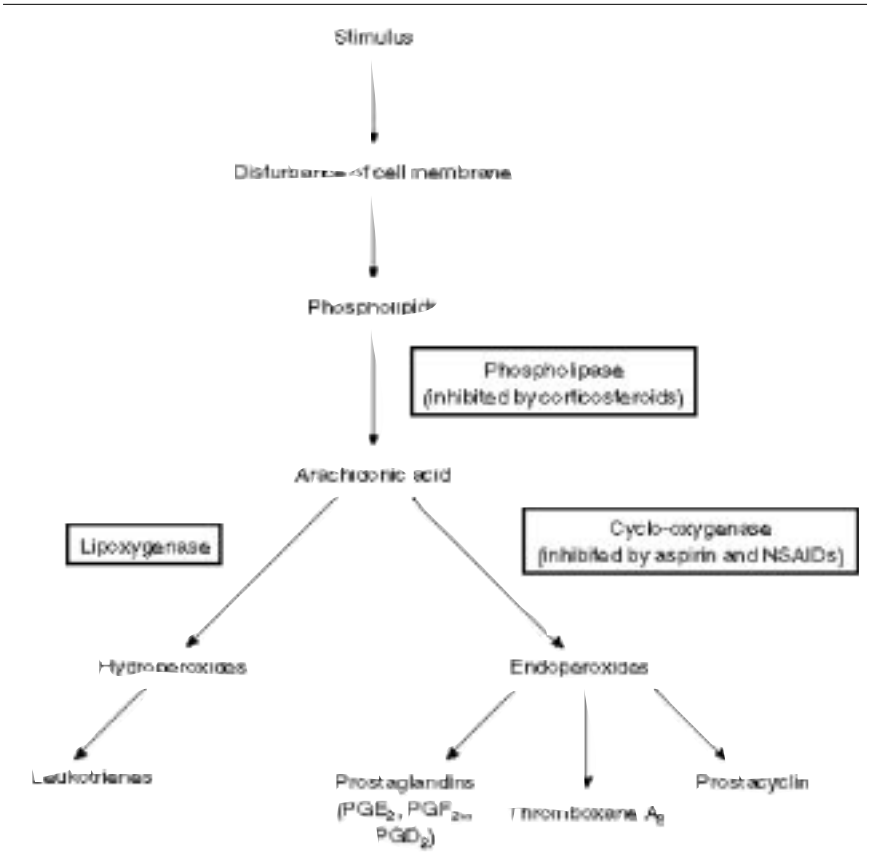

Fig. 1 Diagram of the inflammatory cascade, depicting the point of action of corticosteroids and nonsteroidal anti-inflammatory drugs (NSAIDs). (Adapted with permission from Shearn MA: Nonsteroidal anti-inflammatory agents; nonopiate analgesics; drugs used in gout, in Katzung BG [ed]: *Basic and Clinical Pharmacology*, 2nd ed. Los Altos, Calif: Lange Medical Publications, 1984, p 402.)

Table 4
Injectable Corticosteroids Commonly Used in Orthopaedic Practice

Solubility	Generic Name	Trade Name	Equivalent Dose, mg*
Most soluble	Betamethasone sodium phosphate	Celestone	0.6
Soluble	Dexamethasone sodium phosphate	Decadron	0.75
	Prednisolone sodium phosphate	Hydeltrasol	5
Slightly soluble	Prednisolone tebutate	Hydeltra-T.B.A.	5
	Triamcinolone diacetate	Aristospan Forte	4
	Methylprednisolone acetate	Depo-Medrol	4
Relatively insoluble	Dexamethasone acetate	Decadron-LA	0.75
	Hydrocortisone acetate	Hydrocortone	20
	Prednisolone acetate	Predalone	5
	Triamcinolone acetonide	Kenalog	4
	Triamcinolone hexacetonide	Aristospan	4
Combination	Betamethasone sodium phosphate–betamethasone acetate†	Celestone Soluspan	0.6

*For example, 0.6 mg of betamethasone sodium phosphate is equivalent to 0.75 mg of dexamethasone sodium phosphate, which is equivalent to 5 mg of prednisolone.
†Betamethasone acetate is slightly soluble.

There are no firm guidelines with regard to the choice of a corticosteroid; the decision is usually based on the physician's prior experience or on what is currently available in the office. Some suggested general guidelines are to select a more water-soluble preparation (e.g., betamethasone sodium phosphate) or a mixture of short- and long-acting compounds (e.g., betamethasone sodium acetate--betamethasone phosphate) for acute inflammatory conditions and to select a more water-insoluble preparation (e.g., triamcinolone hexacetonide) for suppressing chronic inflammatory conditions.

A similar lack of guidelines exists with respect to steroid dosage. Commonly accepted practice is to select the dose on the basis of the size of the area that will receive the injection, taking into account the equivalent dose of the specific steroid. For example, a large joint might receive 40 mg of Kenalog (equivalent to 7.5 mg of Decadron), while a small joint might receive 10 mg of Kenalog (equivalent to 1.88 mg of Decadron).

Effects on Various Types of Tissue

Cartilage

Intra-articular injections of corticosteroids are most commonly used for the treatment of osteoarthritis and rheumatoid arthritis. Other conditions treated by joint injection include gout, pseudogout, and adhesive capsulitis. Steroid injections are also used for analgesia.

Joint disease usually first manifests itself as synovial inflammation, which results in swelling, pain, and limitation of motion. An intra- articular injection acts on this inflammation, which subsequently decreases swelling, thereby diminishing pain and improving motion.

The results of corticosteroid therapy for osteoarthritis appear to be dependent on the joint treated. No long-term benefit has been reported for injections of the hip or the knee, possibly because the underlying disease process is biomechanical and the hip and knee are large weight-bearing joints. However, some limited short-term relief of pain and swelling does occur, which may be of benefit to the patient who is awaiting total joint arthroplasty or who wishes to postpone an operative procedure to a more convenient time. Smaller joints, such as the carpometacarpal and acromioclavicular joints, appear to have a better response to steroid injection as measured by reduction of pain and swelling. These differences are probably due to their non-weight-bearing status.

Intra-articular facet injections also are used in the treatment of chronic low back pain, on the presumption that there is an underlying degenerative condition affecting the joints. However, it has proved difficult to identify patients with facet-joint syndromes,[5] and a randomized prospective trial has failed to show any therapeutic difference between saline and methylprednisolone.[6] There is no convincing evidence that steroid facet injections have value in the treatment of chronic low back pain, with the exception of pain due to facet-joint degenerative cysts, for

which there is some evidence for their efficacy. Even more controversial and beyond the scope of this article is the hotly debated use of steroids in fibromyalgia and sacroiliac syndromes.

The results of corticosteroid therapy have been documented to be much better in rheumatoid arthritis, in which the pathophysiology of the disease centers on the synovium. Here the potent anti-inflammatory actions can suppress synovitis for a long period of time, especially with use of a relatively insoluble preparation. A review of the literature provides ample evidence to support their use in this disease. It is clear, however, that corticosteroid injection alone is not sufficient; injection must be part of a comprehensive treatment plan that includes rest, splinting, physical therapy, and use of other antirheumatic agents.

There are a number of potential complications when corticosteroids are injected into joints. A well-known but controversial complication is corticosteroid-induced arthropathy. Mankin and Conger [7] defined the biochemical lesion in a rabbit model, demonstrating a dose-dependent decrease in cartilage-matrix production following intra-articular steroid administration. Numerous other studies have been performed, with results ranging from a protective effect on the articular cartilage to the more commonly accepted deleterious effect.[8,9] There is no firm evidence in the literature to support either a benefit or a cause-and-effect association between intra-articular corticosteroid injection and arthropathy. This is predominantly because it is difficult to determine whether the arthropathy is secondary to steroid administration or is due to natural progression of the primary disease process.

In addition to possible detrimental effects on cartilage, intra-articular steroid administration may produce deleterious effects on the meniscus and intra-articular ligaments. Changes in meniscal color, fraying, and surface irregularities, in addition to histologic evidence of decreased proteoglycan content, have been demonstrated in rabbit menisci after repeated intra-articular knee injections.[10] Also, a decrease in the peak load and stiffness of the anterior cruciate bone-ligament-bone unit was observed with intra-articular steroid administration into monkey knees, with the detrimental effects occurring in a dose- and time-dependent manner;[11] in that study, no animal received more than one injection per week for 3 weeks.

Another problem with intra-articular corticosteroid injections is the potential for systemic effects due to absorption. The rate of absorption is directly proportional to the water solubility of the drug and is enhanced by multiple joint injections, due to the increased synovial surface area. The most alarming side effect is that higher doses, such as would result from simultaneous injections into two or more joints, can induce suppression of the hypopituitary-adrenal axis for 2 to 7 days. Therefore, only one large joint should be treated per visit, and injections should be spread out over as long a time period as possible.[12]

Ligament

The most common sites for steroid treatment of ligamentous injury include the medial collateral ligament of the elbow, the extra-articular knee ligaments, and the ankle ligaments. Injections are ideally not made directly into ligament substance, but rather into the periligamentous tissue. As is the case with intra-articular injections, soft-tissue injections should be performed only in conjunction with an overall treatment plan including limitation of activities, physical therapy, ice, and appropriate splinting.

The main complication associated with steroid injections is ligament rupture, even if the injection is not directly into ligament substance. This unfortunate side effect is more likely to occur in the patient who does not adhere to a program of rest. This is of particular concern because corticosteroids can mask the pain of injury, which could allow premature resumption of activity, potentially leading to further ligamentous damage. Other side effects include loss of skin pigmentation and subcutaneous atrophy, which are not always reversible.

The theory behind the use of steroids in ligamentous injury is based on their anti-inflammatory properties to limit pain, to allow earlier motion, and presumably to permit quicker healing. However, steroids are also known to inhibit collagen synthesis. Since 70% to 80% of the dry weight of ligament is collagen, it might be assumed that steroids would inhibit ligamentous healing. Nevertheless, there are clinical and laboratory studies both to support and to condemn the use of steroid injections in ligament injury.

Recent work in our laboratory has examined the effects of steroid injection on acute ligamentous injury.[13] We found that in the inflammatory and proliferative phases of healing, steroids have significant inhibitory effects on the healing process with respect to both biomechanical properties and histologic maturation. A study of the long- term effects of an injection into an acutely injured ligament demonstrated that steroid-treated ligaments possessed the same tensile strength as nontreated ligaments, but that they failed under lighter loads, possibly because of diminished cross-sectional area and histologic immaturity.[14] We have also examined the effects of a delayed steroid injection at 7 days following injury, which coincides with the end of the inflammatory phase of heal-

ing. The same deleterious effects on the healing process were found in the delayed-injection group as were seen in the ligaments that received injections immediately after injury.[15] Thus, the harmful effects of steroid injection cannot be solely attributed to interference with the inflammatory phase of healing; rather, we postulate that an additional detrimental mechanism must be present.

On the basis of extensive reviews of the literature and our own research, we believe in extremely judicious use of corticosteroids in and around an injured ligament. However, if an injection is performed, whether for an acute or a chronic injury, it should be part of a well-constructed treatment plan including rest, physical therapy, and close monitoring by the physician. Patient compliance is essential. A non-compliant patient who returns immediately to sports or other activities increases the risk of further injury.

Tendon

Tendon injuries can be classified as tendinitis or tenosynovitis. Tendinitis, the most common form, is inflammation of the tendon substance itself, usually occurring at the insertion site of tendon to bone and accompanied by a reactive inflammation in the surrounding paratenon. In contrast, tenosynovitis involves inflammation of only the paratenon. Most of the conditions commonly referred to as tendinitis are actually overuse injuries, which will be discussed later.

One problem with injection therapy for tendinitis is knowing which tissue is being treated. For example, injections performed for Achilles tendinitis are made into the peritendinous tissues, while injections for retrocalcaneal bursitis are made into the retrocalcaneal bursa, which is also a peritendinous tissue. Thus, an injection for either condition is probably affecting both structures. The same holds true for subacromial bursitis and rotator cuff tendinitis.

Some forms of tenosynovitis respond well to corticosteroid injection. Nonoperative treatment of stenosing tenosynovitis of the thumb and fingers consists of immobilization, NSAIDs, and steroid injection. Patients with single-digit involvement have had 95% satisfactory results with corticosteroid injection, and patients with symptoms of less than 4 months' duration have had 93% satisfactory results. With multiple-digit involvement or symptoms of more than 4 months' duration, the results are much less favorable.[16]

Another form of tendon entrapment, de Quervain tenosynovitis, can also be treated with corticosteroid injection. This injection is much more difficult to perform due to the variations found in the first dorsal compartment, such as multiple tendon slips of the abductor pollicis longus and a separate compartment for the extensor pollicis brevis. Satisfactory results have been reported in only 62% of patients with de Quervain tenosynovitis, regardless of symptom duration.[17]

The complications of steroid injection for tendon injury are similar to those observed with ligamentous injury, namely, tendon rupture, subcutaneous atrophy, and loss of skin pigmentation.

Bursa

A bursa is a potential space located between two structures that move against each other. Its presence serves to lower the coefficient of friction between the two gliding surfaces. With repetitive stress and, less commonly, direct trauma, the many bursae in the body can become inflamed, painful, and swollen. The production of inflammatory exudate by the synovial cells in the bursal lining is often accompanied by tendinitis.

The sites most commonly treated by steroid injection are the subacromial, greater trochanteric, olecranon, prepatellar, and retrocalcaneal bursae. The results of steroid injection are site dependent. Trochanteric and olecranon bursitis both respond well to steroid injection.[18,19] Injections into the prepatellar and retrocalcaneal bursae have had less favorable results and have been associated with tendon rupture.[20] Subacromial injections also have less favorable results with respect to pain relief. The problem with subacromial injections is determining which specific pathologic condition the injection is treating (e.g., bursitis, rotator cuff tendinitis, or impingement). In addition, when operative procedures are subsequently performed, subacromial spaces treated with steroid injection demonstrate poor quality of residual cuff tissue and inferior results compared with subacromial spaces not treated with steroid injection.[21]

Nerve

A number of peripheral nerve compression syndromes are treated by corticosteroid injection. Carpal tunnel syndrome is the most common. However, the efficacy of corticosteroids in this condition is unclear. In a prospective study in which 41 patients with carpal tunnel syndrome (50 hands) were treated with a single corticosteroid injection and 3 weeks of splinting, only 22% of hands became completely symptom free. The best results were seen in patients who had normal sensibility, normal thenar strength and mass, a 1- to 2-msec delay in distal median motor or sensory latencies, and symptoms of less than 1 year's duration.[22] Forty percent of the hands were symptom free 1 year after treatment. A more recent study examined steroid injection and splinting as the treatment for patients with mild to moderate symptoms. At 1 year, only 13% of hands were symptom free.[23]

The epidural injection of steroids for the management of sciatica due to disk herniation is commonly used in an effort to relieve the radicular pain. However, the clinical trials in support of their efficacy have produced varying results. For example, one prospective, randomized, double-blind study examined the short-term (24 hours) and long-term (20 months) results in patients with either an acute herniated lumbar disk or spinal stenosis who received injections of either 80 mg of methylprednisolone or saline. Although 39% of the steroid-treated patients with an acute herniated disk and 43.5% of those with spinal stenosis reported improvement at 24 hours, these results were not statistically different from the results in patients who received a saline injection. Long-term follow-up also failed to show any statistical improvement.[24] However, other studies have supported the efficacy of corticosteroids.

Bone

Corticosteroid injection into unicameral bone cysts has been an accepted method of treatment since Scaglietti et al[25] reported their results in 1982. Prior to that time, curettage and bone grafting was the standard of care.

Elevated prostaglandin E_2 (PGE_2) levels have been found in cyst fluid and are thought to be responsible for stimulation of the osteoclastic activating factors that perpetuate the cyst cavity. Since corticosteroids possess antiprostaglandin properties, it is postulated that corticosteroid administration into an active cyst inhibits the formation of PGE_2. This in turn inhibits osteoclastic activity, allowing resolution of the cavity.[26]

Not all cysts will respond to only a single injection; therefore, it is sometimes necessary to perform a second or third injection. Injections should be spaced approximately 6 weeks apart. If resolution of the cyst has not occurred after three injections, an alternative treatment should be used, most commonly curettage and grafting.

Overuse Syndromes

Overuse syndromes typically present as tendinitis, bursitis, and fasciitis. They are most commonly caused by athletic activities but are increasingly related to occupational activities. Less common are overuse injuries of ligaments, such as the ulnar collateral ligament of the elbow. In general, most overuse injuries are secondary to repetitive loading, which produces mechanical fatigue and consequent degeneration of the involved substance that exceeds the capability of the tissue to repair itself. However, there are no clear pathologic, biologic, and clinical markers for many of these entities.

Not surprisingly, there is considerable confusion about the success possible with the use of corticosteroid injections in this broad category of afflictions. Lateral epicondylitis (tennis elbow) is a good example of a commonly diagnosed overuse syndrome that appears to respond to corticosteroid injection. However, the term epicondylitis is actually a misnomer, since the pathophysiologic process is degenerative, not inflammatory. Histologically, acute inflammatory cells are not seen in the extensor carpi radialis brevis tendon, and chronic inflammatory cells, if present, are those of repair.[27] So why do some cases of tennis elbow improve with corticosteroid injection? At this point, we do not know, because clinical studies have included multiple forms of treatment instituted at the same time, such as avoidance of aggravating activity, bracing, physical therapy, equipment modification, nonsteroidal anti-inflammatory medication, and steroid injection. Consequently, it is difficult to determine which treatment modality is producing symptom relief. However, we are concerned about the use of steroids in these conditions because of the adverse effects on collagen synthesis.

Technique of Injection

The Occupational Safety and Health Administration regulations mandate that sterile gloves be worn when joint fluid is aspirated. Even if arthrocentesis is not performed, we use sterile gloves for all injections that involve corticosteroids. The skin is widely cleansed with an antiseptic agent and alcohol, allowing sterile palpation of landmarks. For small joints, such as those in the hand, we use a 30-gauge 0.5-inch needle. For larger or deeper joints, we use a 25-gauge 1.5-inch needle. Cutaneous anesthesia is usually obtained with xylocaine or ethyl chloride spray or a longer-acting anesthetic, such as bupivacaine. Landmarks for injections into specific sites are listed in standard texts.

Following the injection, a brief period of rest is recommended to reduce the possibility of postinjection aggravation of pain and to decrease the clearance of steroid from the area. Extremity immobilization may be considered. Once the period of rest is over, a rehabilitation program is initiated to improve flexibility, range of motion, and strength of the affected anatomic region.

Contraindications

We believe that there are several absolute contraindications to corticosteroid injection, among them infection of the proposed area of

injection, since steroids have an inhibitory effect on neutrophil function, and acute local tissue trauma, because of the catabolic effects of steroids and their interference with the normal healing response. In addition, injections should not be made directly into ligament or tendon substance. We also believe it important not to administer a corticosteroid injection to a patient who will not be compliant with other elements of treatment, particularly the associated period of rest. As a general rule, multiple injections should be avoided, but there are exceptions, such as unicameral bone cysts, rheumatoid arthritis, and selected cases of osteoarthritis.

Acknowledgment: The authors would like to thank David Efron, MD, for his critical review of the manuscript.

References

1. Hollander JL: Intra-articular hydrocortisone in arthritis and allied conditions: A summary of two years' clinical experience.*J Bone Joint Surg Am* 1953;35:983-990.
2. Hill JJ Jr, Trapp RG, Colliver JA: Survey on the use of corticosteroid injections by orthopaedists. *Contemp Orthop* 1989; 18:39-45.
3. Baxter JD, Forsham PH: Tissue effects of glucocorticoids. *Am J Med* 1972;53: 573-589.
4. Shearn MA: Nonsteroidal antiinflammatory agents; nonopiate analgesics; drugs used in gout, in Katzung BG (ed): *Basic and Clinical Pharmacology*. Los Altos, Calif: Lange Medical Publications, 1984, pp 400-417.
5. Jackson RP, Jacobs RR, Montesano PX: Facet joint injection in low- back pain: A prospective statistical study. *Spine* 1988;13:966-971.
6. Carette S, Marcoux S, Truchon R, et al: A controlled trial of corticosteroid injections into facet joints for chronic low back pain. *N Engl J Med* 1991;325:1002-1007.
7. Mankin HJ, Conger KA: The acute effects of intra-articular hydrocortisone on articular cartilage in rabbits. *J Bone Joint Surg Am* 1966;48:1383-1388.
8. Pelletier JP, Martel-Pelletier J: Protective effects of corticosteroids on cartilage lesions and osteophyte formation in the Pond-Nuki dog model of osteoarthritis. *Arthritis Rheum* 1989;32:181-193.
9. Behrens F, Shepard N, Mitchell N: Metabolic recovery of articular cartilage after intra-articular injections of glucocorticoid. *J Bone Joint Surg Am* 1976;58:1157-1160.
10. Ishikawa K: Effect of intra-articular corticosteroid on the meniscus: A histological and histochemical study in rabbit knees. *J Bone Joint Surg Am* 1981;63:120-130
11. Noyes FR, Grood ES, Nussbaum NS, et al: Effect of intra-articular corticosteroids on ligament properties: A biomechanical and histological study in rhesus knees. *Clin Orthop* 1977;123:197-209.
12. Gray RG, Gottlieb NL: Intra-articular corticosteroids: An updated assessment. *Clin Orthop* 1983;177:235-263.
13. Wiggins ME, Fadale PD, Barrach H, et al: Healing characteristics of a type I collagenous structure treated with corticosteroids. *Am J Sports Med* 1994;22: 279-288.
14. Wiggins M, Fadale P, Barrach H, et al: Long term effects of corticosteroid treatment on a healing rabbit medial collateral ligament. *Trans Orthop Res Soc* 1993;18:59.
15. Wiggins M, Fadale P, Russell M, et al: Effects of delayed steroid injection on ligament healing. *Trans Orthop Res Soc* 1994;19:633.
16. Rhoades CE, Gelberman RH, Manjarris JF: Stenosing tenosynovitis of the fingers and thumb: Results of a prospective trial of steroid injection and splinting. *Clin Orthop* 1984;190:236-238.
17. Witt J, Pess G, Gelberman RH: Treatment of de Quervain tenosynovitis: A prospective study of the results of injection of steroids and immobilization in a splint. *J Bone Joint Surg Am* 1991;73:219-222.
18. Schapira D, Nahir M, Scharf Y: Trochanteric bursitis: A common clinical problem. *Arch Phys Med Rehabil* 1986;67:815-817.
19. Smith DL, McAfee JH, Lucas LM, et al: Treatment of nonseptic olecranon bursitis: A controlled, blinded prospective trial. *Arch Intern Med* 1989;149: 2527-2530.
20. Cox JS: Current concepts in the role of steroids in the treatment of sprains and strains. *Med Sci Sports Exerc* 1984;16: 216-218.
21. Watson M: Major ruptures of the rotator cuff: The results of surgical repair in 89 patients *J Bone Joint Surg Br* 1985;67: 618-624.
22. Gelberman RH, Aronson D, Weisman MH: Carpal-tunnel syndrome: Results of a prospective trial of steroid injection and splinting. *J Bone Joint Surg Am* 1980;62:1181-1184.
23. Sachar K, Weiss APC, Gendron M: The conservative management of carpal tunnel syndrome: A re-examination of steroid and splinting. *J Hand Surg [Am]* 1994;19:410-415.
24. Cuckler JM, Bernini PA, Wiesel SW, et al: The use of epidural steroids in the treatment of lumbar radicular pain: A prospective, randomized, double-blind study. *J Bone Joint Surg Am* 1985;67: 63-66.
25. Scaglietti O, Marchetti PG, Bartolozzi P: Final results obtained in the treatment of bone cysts with methylprednisolone acetate (Depo-Medrol) and a discussion of results achieved in other bone lesions. *Clin Orthop* 1982;165:33-42.
26. Shindell R, Huurman WW, Lippiello L, et al: Prostaglandin levels in unicameral bone cysts treated by intralesional steroid injection. *J Pediatr Orthop* 1989;9:516-519.
27. Nirschl RP: Elbow tendinosis / tennis elbow. *Clin Sports Med* 1992;11:851-870.