Neurovascular Entrapment in the Regions of the Shoulder and Posterior Triangle of the Neck

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The purpose of this article is to provide information that facilitates the critical evaluation of the thoracic outlet syndrome and other nerve entrapment syndromes in the regions of the posterior cervical triangle and the shoulder. General comments regarding entrapment are followed by discussions of specific entrapment sites. Each discussion stresses the anatomic view and includes comments on causes, symptoms, diagnostic tests, and management.

Key Words: Brachial plexus, Nerve compression syndromes, Physical therapy.

In theory, the location of the entrapment site that produces a thoracic outlet syndrome (TOS) and the resultant symptoms can be quite definitive. Most authors, however, agree with Kline et al that even more controversial is the diagnosis [for] and treatment of those patients presumed to have thoracic outlet syndrome. Both an exact cause and a specific diagnostic syndrome are usually lacking.

The understanding and treatment of TOS are complicated further by the myriad of syndromes that are considered to be either synonymous with or a part of TOS.2 The literature is confusing and in some areas conflicting. Of particular concern are specific physical therapy management strategies and their efficacy.

The purpose of this article is to discuss the potential entrapment sites of TOS from an anatomic point of view and to compare them with other nerve entrapments in the posterior cervical triangle and the region of the shoulder. Signs and symptoms and diagnostic tests are discussed, and general and specific guidelines for treatment are presented. The effects of entrapment on a nerve also are discussed.

GENERAL REVIEW OF NERVE STRUCTURE

There are three levels of organization of a nerve or nerve trunk, each unit of which is enclosed or surrounded by a connective tissue covering. The basic unit and smallest level of organization is the nerve fiber, which is the conducting component of the nerve trunk and a process of a neuron or nerve cell. Each fiber is surrounded by a delicate, but definitive, connective tissue encasement called the endoneurium. Various numbers of nerve fibers are grouped into a bundle or funiculus that is delimited by the perineurium. Finally, the funiculi forming a nerve trunk are surrounded collectively by the heaviest layer of connective tissue, the epineurium. Extensions of the epineurium extend into the substance of the nerve and, thus, separate the funiculi.

The connective tissue at all levels is classified as loose and irregular and contains the basic components of all connective tissues (ie, cells, fibers, and ground substance). Mechanically, the fibers resist tensile stresses, and the ground substance resists compression. The fibers, mostly collagen and elastic, generally are oriented longitudinally and are the most numerous in the epineurium. Stretching of the nerve, however, is thought to be resisted mainly by the fibers of the perineurium.3 The ground substance is watery and virtually without structure. As a result, the fibers—particularly those in the epineurium—provide most of the protection against compression forces. The cells of the connective tissues are particularly important. A certain group of these cells—mast cells—are integral components of the immune system and have a major role in local inflammatory reactions.

The blood supply to peripheral nerves is rich. Each nerve receives multiple nutrient arteries at various intervals, and each of the connective tissue coverings contains an extensive and freely anastomosing arterial network. Furthermore, the various arterial networks freely anastomose with one another. As a result, a nerve can be removed from its bed for considerable distances without compromising its blood supply. Local injury around a nerve, but not including the nerve, is not apt to affect the blood supply.

EFFECTS OF ENTRAPMENT

Fisher and Gorelick have crystallized the concept of entrapment rather well:

Entrapment neuropathies are lesions of individual peripheral nerves resulting from injury at vulnerable anatomic sites. Nerve compression typically occurs where a nerve passes through an opening in fibrous tissue or an osseofibrous canal. The mechanism of injury may be direct compression, angulation, stretch, or vascular compromise.4

An extension of this statement is the idea that tissues within the body are dynamic: As movement occurs, structures move relative to one another, and friction results.

I believe there are two factors to consider when evaluating the signs and symptoms that result from an entrapment neuropathy. One is local, or at the site of entrapment; the other is more distal and involves the areas and structures innervated by the affected nerves.

At the point of entrapment, either compression or friction can cause a reaction in a nerve, involving the connective tissue, that makes the nerve more susceptible to further irritation.3 The degree of such a reaction is proportional to the intensity and duration of the entrapment pressure. An initial trauma of sufficient magnitude may precipitate a
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dagnosis and treatment.

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ries according to the degree and the con­
sequences of the injury.  This type of lesion fre­
quently "precedes a destructive lesion above the first rib. An obvious potential prob­
lem is the presence of a cervical rib or a subclavian-axillary vessels pass inferior to the coracoid process and posteriorly to the insertion of the pectoralis minor muscle.

Superior Thoracic Outlet

This area is bounded by the first rib laterally, the manubrium of the sternum anteriorly, and the first one or two tho­
racic vertebrae posteriorly.  It is not an enclosure in the strict sense but rather a bony boundary against which the subclavian artery and vein and the inferior trunk of the brachial plexus must pass. Space-occupying lesions (ie, from the thyroid gland, pleura, or lung) could compress any of these structures against the first rib. If no such lesion exists, the major hazard is the first rib. The vascular structures pass medial to and then superior to the first rib; the inferior trunk may ascend somewhat as it passes above the first rib. Because the anterior portion of the first rib is considerably inferior to its posterior portion, the neurovascular structures pass above its most inferior part.

A potential problem, thus, might arise either if the first rib is elevated or if traction is applied to the neurovascular structures so that they are pulled against the first rib. An obvious potential prob­
lem is the presence of a cervical rib or a post-fixed brachial plexus (a brachial plexus that arises from spinal cord seg­
ments C6-T2). Hollinshead reported an approximate 1% occurrence of cervical rib. In either situation, the neurovas­
cular structures must ascend higher to exit from the thorax. A patient with emphysema will be predisposed to this type of compression because the ante­
rior aspect of the first rib is elevated chronically. Posture or type of body build also may be involved. The natural shoulder position tends to be lower in women than in men, and it tends to change with age; it is held lower secondary to muscle atrophy and loss of strength. This position effectively places more traction on the entire brachial plexus. The inferior trunk of the brachial plexus or the subclavian vessels, thus, are vulnerable to two mechanisms of entrapment at this location. They can be compressed against the medial aspect of the first rib or stretched (by traction) as they pass medial and then superior to the first rib.

Inferior trunk involvement may produce sensory symptoms in the fourth and fifth fingers, the medial hand, and the medial forearm. Motor symptoms are limited primarily to the intrinsic muscles of the hand. Two tests exist that logically could be used to reproduce the symptoms; however, neither test is specific enough to eliminate other potential sources of involvement. Abduction of the arm to 90 degrees and maximal lateral (external) rotation (AER test) exert considerable traction on the brachial plexus and the subclavian vessels as they are pulled under the coracoid process and pectoralis minor muscle insertion. Maintenance of this position for several minutes might reproduce the symptoms. Traction on the brachial plexus also can be increased by loading the extremity with the patient in a standing position. The traction would increase considerably if the trapezius muscles were atrophied or weakened, or both.

Suggestions for treatment using exercises are nonspecific in the literature, and reports about the usefulness of exercises are conflicting. A major emphasis should be on the strengthening of the trapezius and the levator scapulae muscles with the eventual goal of elevating the static shoulder position and, thus, removing some of the traction force on the brachial plexus. This goal can be facilitated by improving the posture of the cervical spine (flattening), thereby raising the occipital attachment of the trapezius muscle. Basmajian and Deluca have shown that the scalene muscles are the primary breathing muscles; therefore, increasing their strength would elevate the first rib and aggravate the patient's condition. Hence, breathing exercises are not indicated. The presence of a cervical rib frequently is an indication for surgery, although various results have been reported.*

**Scalene Triangle**

The scalene triangle is a gap in the muscular floor of the posterior cervical triangle that is formed by the anterior and middle scalene muscles and the first rib. From similar attachments to the transverse processes of the upper and middle cervical vertebrae, the anterior and middle scalene muscles diverge as they are followed inferiorly to their attachments on the first rib. Their rib attachments typically are, at most, 2 cm apart, so the base of the triangle is small, whereas the vertical sides are considerably longer. This triangle is narrow from anterior to posterior, and the term *scalenule groove* is more descriptive than scalene triangle. The shape of the triangle is variable, and it further varies with body build. The dimensions of this triangle are not static. Movement of the cervical spine mainly affects the antero-posterior dimension. This dimension is reduced with either extension of the cervical spine or rotation to the ipsilateral side.

The proximal portions of the brachial plexus (or the vertebral rami) and the subclavian artery pass through this triangle. The brachial plexus occupies most of the vertical dimension of the triangle; the subclavian artery occupies the scalene groove of the clavicle anterior to the lowest part of the brachial plexus. The upper roots (C5-C7) of the brachial plexus typically descend as they pass to the triangle; the inferior roots (C8 and T1) of the brachial plexus may ascend as they enter the triangle; the subclavian artery clearly ascends.

Multiple developmental variations can reduce the size of this opening, particularly its lower portion. The presence of a cervical rib can reduce the vertical dimension of the triangle substantially by elevating its base. Even though the extent of a cervical rib is variable, the result is usually the same (ie, the structures passing through the triangle must ascend higher than normal to reach the opening). The existence of a cervical rib increases the potential for neurovascular compression but does not guarantee it. Variations in the anterior or middle scalene muscles are common. Extra slips of muscle may exist that substantially narrow the width of the triangle; occasionally, an extra muscle referred to as the scalenus minimus may exist. Frequently, the first rib attachments of the muscles are widened, reducing the size of the base of the triangle. Any of these additional slips of muscle or the "normal" scalene muscles can have associated fibrous bands. The presence of sharp fibrous bands obviously increases the potential of nerve trauma. Again, the presence of a post-fixed brachial plexus may be a factor.

Scalene muscle hypertrophy has been linked to the physical demands of certain occupations. This finding is difficult to rationalize because the scalene muscles are not considered to be major motors of the shoulder. The posture of the cervical spine (ie, neck-flexion contracture), perhaps, is a more likely causative factor. Abduction and lateral rotation of the arm place a traction force on the brachial plexus. Sitting at a table or desk with the elbows on the table and the head slumped forward may, to some degree at least, produce the same traction force.

Whatever the specific cause, entrapment in the scalene groove typically affects the inferior trunk of the brachial plexus and causes symptoms consistent with C8 and T1 involvement. Symptomatic but, in reality, it was not. Symptomatic arterial occlusion (ie, coldness, pallor, and generalized pain and fatigue) seldom are present. Symptoms of venous occlusion (ie, cyanosis, venous distension, and edema) should not occur because the subclavian vein passes anterior to the anterior scalene muscle.

Tests for entrapment at this point generally are not definitive. For many years, Adson's maneuver was believed to be an important diagnostic test, and it does make sense anatomically. The radial pulse is palpated, and downward traction is applied to the upper extremity. The patient is asked to turn his head to the involved side, extend the cervical spine, and take a deep breath. These movements effectively reduce the size of the scalene groove. The loss or weakening of the pulse was believed to be diagnostic, but, in reality, it was not. This maneuver yields "positive" results in a large percentage of asymptomatic people. The AER test will reproduce the symptoms of TOS, as well loading the muscles of the upper extremity (ie, increasing the downward traction). Differentiating the diagnoses of entrapment in the superior thoracic outlet and the scalene groove is difficult and perhaps unnecessary.

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*2,5,7,12,14,17,21-25.
The presence of a cervical rib in a symptomatic patient is a strong indication for surgery. In the absence of a cervical rib, the physical therapy management should be similar to that indicated for involvement at the superior thoracic outlet. Various modalities provide temporary pain relief, but lasting results are achieved by postural changes. It makes sense anatomically that shortened scalene muscles should be stretched.

Costoclavicular Interval

The costoclavicular interval is the bony interval between the clavicle and the first rib. The superior border of the scapula is included by some authors, but its importance is questionable. Of importance is the vise-like relationship that exists between the clavicle and first rib. The sternoclavicular joint is the fulcrum around which the lateral end of the clavicle can be elevated or depressed; as the clavicle is depressed, the costoclavicular interval, which transmits the axillary vessels and the brachial plexus, is reduced in size. Although the subclavius muscle partially separates the neurovascular bundle from the clavicle, it apparently provides only minimal protection. The critical motions, in which the clavicle and first rib are approximated and the neurovascular bundle is placed in jeopardy, are depression and posterior displacement of the shoulder. These motions would occur in such activities as carrying heavy objects at the side or wearing a heavy backpack. Poor posture with drooping shoulders, which may result from muscle weakness or atrophy, also tends to reduce the size of the interval. Changes in the normal shape of either bone, such as fracture of the clavicle or an exostosis of either bone, may cause or predispose this type of entrapment.

The neurological symptoms resulting from compression at this point typically are similar to those of the more proximal entrapment sites. Definitively establishing the costoclavicular interval as the point of entrapment may be difficult but, perhaps, is not important relative to the treatment strategy. As the brachial plexus passes between the clavicle and first rib, the relative positions of the ventral rami are maintained; that is, the inferior trunk is adjacent to the first rib, and the superior trunk is just inferior to the clavicle. If the entrapment pressure is caused by approximation of the two bones, it is difficult to understand why the lower brachial plexus might be affected more than the upper brachial plexus. Perhaps a case could be made for the subclavius muscle since it separates the plexus from the clavicle but not from the first rib. In any case the reason is not clear.

Both the subclavian artery and vein are in jeopardy as they pass between the two bones. The subclavian vein especially is jeopardized because it is closer to the apex of the triangle formed between the bones. This vise-like mechanism is believed to occlude the subclavian vein in the Paget-Schroetter syndrome.

Specific physical tests designed to localize entrapment at this point probably are not available. The abduction-lateral rotation test, considered by Daskalakis to be the best test for reproducing the symptoms of TOS, appears to be of little use in substantiating costoclavicular entrapment. As the arm is abducted and rotated laterally, the lateral end of the clavicle is elevated, thus opening the interval. Heavy loading of the muscles of the axillary artery as they pass the first rib.

The symptoms resulting from entrapment at this point usually are indicative of C8 and T1 involvement. The specific test recommended for reproducing those symptoms is the abduction-lateral rotation test. I question, however, whether the coracoid-pectoralis minor loop is a true entrapment location. The physical therapy regimen should be designed to improve posture, and the patient should be advised to avoid abduction and lateral rotation of the arm.

Summary of the Thoracic Outlet Syndrome

An accurate medical history and an in-depth physical examination are of the utmost importance in determining a proper diagnosis and in prescribing a logical and effective treatment regimen. Various diagnostic maneuvers have been discussed. Various electrodiagnostic techniques (ie, electromyography, nerve-conduction studies, and somatosensory-evoked responses) are useful, but they are not foolproof. Vascular studies are used when specifically indicated.

Experience and the literature indicate that physical therapy is most effective for those patients who exhibit mild to moderate symptoms. Exercise programs to improve the posture of the vertebral column and shoulder region and to increase the strength and range of motion of the shoulder and cervical
musculature are important. Patients also should be advised about the implications of posture and about those positions that are apt to cause symptoms.

ENTRAPMENT OF BRANCHES OF THE BRACHIAL PLEXUS

 suprascapular Nerve Entrapment

After arising from the proximal part of the superior trunk of the brachial plexus, the suprascapular nerve descends across the posterior cervical triangle toward the lateral aspect of the scapula. It crosses the superior border of the scapula by passing through the scapular notch; the notch is transformed into a foramen by the taut and strong transverse scapular ligament. The nerve, thus, passes through a small fibro-osseous opening. While in the suprascapular fossa, the nerve branches to the supraspinatus muscle. The nerve continues around the neck of the spine of the scapula, providing branches to the infraspinatus muscle and to the shoulder joint.

The suprascapular nerve contains both motor and sensory fibers but innervates no cutaneous area. This nerve is thought to provide a major part of the sensory innervation of the shoulder joint, especially the posterior part of the joint capsule. Other nerves clearly innervate the joint, but the suprascapular nerve may be the major one. Some, if not most, impulses from a painful shoulder pass through the suprascapular nerve. Irritation of that nerve anywhere along its course, therefore, may produce deep pain in the region of the posterior shoulder.

The suprascapular nerve is under some tension throughout its course. As a result, movement of the scapula is accompanied by a piston-like action of the suprascapular nerve within the scapular notch. This movement is especially marked when the scapula is protracted. Even though the scapula is sliding laterally, the lateral angle is moving anteriorly and even medially.

The entrapment neuropathy affecting the suprascapular nerve appears to have various causes. The classic history is one of an insidious onset of the symptoms. Careful questioning of the patient likely will reveal a pattern of shoulder activity that includes wide and extreme excursions of scapular motion on a regular basis. During extreme shoulder ROMs, the nerve is subjected to increased motion in the foramen and to potentially increased friction against the bony and fibrous walls of the foramen. Other potential entrapment-causing events include fracture of the suprolateral aspect of the scapula in the region of the scapular notch and downward trauma that affects the nerve directly or perhaps forces the nerve against the walls of the scapular foramen. A fracture or its subsequent healing may cause a reduction in the size of the notch and roughening of its edges. The nerve, thus, may become inflamed and more susceptible to trauma as it slides in the foramen.

It is interesting to speculate on exactly how much and during which motions the nerve slides in the foramen. It would seem that a combination of glenohumeral and scapular motion is necessary to produce maximal excursion. Specifically, extreme protraction of the scapula moves the lateral angle of the scapula anteromedially and, thus, the scapular notch is closer to the origin of the suprascapular nerve. In effect, the suprascapular foramen slides proximally along the nerve. If, simultaneously, the hand is brought horizontally across the chest, a marked motion at the glenohumeral joint and resultant traction on the suprascapular nerve occur. The result is considerable movement of the nerve in the foramen; as the foramen slides proximally along the nerve, the nerve is pulled distally by the humeral motion. The patient with a true suprascapular nerve entrapment syndrome avoids most extreme motions of the shoulder, especially abduction of the arm across the chest.

Both the patient's medical history and a physical examination are important in providing a correct diagnosis. A patient's major complaints are usually a deep aching pain and weakness in the shoulder. The pain may radiate either proximally or distally, but no cutaneous involvement should occur. A muscle test might reveal weakness in abduction and lateral rotation of the arm. Atrophy of the supraspinatus and infraspinatus muscles may be detected by inspection, palpation, or both. The cross-adduction test (adducting the extended upper limb across the chest) should reproduce the shoulder pain; adduction may be limited because of either pain or tightness of the joint capsule. Of major diagnostic importance is electromyography. A delay in conduction across the entrapment site and muscle fibrillation are the most common signs. Additionally, loss of pain secondary to anesthesia of the nerve is positive diagnostically.

A connection between frozen shoulder and suprascapular nerve entrapment may exist. Some researchers believe the two syndromes are related and that one may cause the other, but their temporal relationship is unclear. Does a reduction in glenohumeral motion necessitate an increase in scapular motion (substitution) and, thus, an increase in potential nerve trauma, or does entrapment produce the shoulder pain that could lead eventually to a frozen shoulder? Perhaps suprascapular nerve entrapment is responsible for several shoulder disorders, including frozen shoulder, whose causes remain unknown.

The physical therapist has an important role in the management of patients with suprascapular nerve entrapment. Definitively diagnosed entrapment syndromes often are treated successfully with surgery. After surgery, physical therapy should be instituted to treat patients for residual pain, weakness, and loss of motion. The physical therapist frequently is involved in the treatment of many shoulder problems, such as adhesive capsulitis, that might be related to nerve entrapment. Because suprascapular nerve entrapment may be a major cause of frozen shoulder, excessive ROM may be contraindicated. If the therapist detects signs and symptoms suggesting suprascapular nerve entrapment, he should report them to the physician.

Dorsal Scapula Nerve Entrapment

The dorsal scapula nerve branches from the ventral ramus of the fifth cervical spinal nerve. It passes through the middle scalene muscle and then descends deep to the levator scapulae and rhomboid muscles and medial to the scapula. Typically, it innervates the rhomboid muscles but also may supply the levator scapulae muscles; it has no cutaneous distribution.

The entrapment occurs as the nerve passes through the middle scalene muscle. Reported symptoms include pain along the medial border of the scapula, diffuse pain in the region of the shoulder, tenderness of the middle scalene muscle, and mild winging of the scapula. Specific physical therapy management is not described in the literature.
Long Thoracic Nerve Entrapment

The long thoracic nerve is formed by branches of the ventral rami of the fifth, sixth, and seventh cervical nerves. The branches from C5 and C6 pass through the middle scalene muscle, and the nerve is formed posterior to the brachial plexus. It descends along the anterolateral aspect of the thoracic wall, passing superficial to the serratus anterior muscle, which it innervates. Movement of this nerve is restricted because it is fixed proximally to the middle scalene muscle and attached distally to each serration of the serratus anterior muscle that it passes.

The long thoracic nerve is vulnerable to injury from multiple sources. The compression of that part of the nerve that passes through the middle scalene muscle is not documented in the recent literature. The major mechanism of injury seems to be stretching of the nerve, for example, the stretching that results when the head and shoulder are separated forcibly. The symptoms include a dull aching pain in the posterior shoulder region that may extend into either the neck or arm and a variable weakness of the serratus anterior muscle. Physiological therapy options currently seem to be limited to strengthening the serratus anterior muscle.

Axillary Nerve Entrapment

Proximal to the shoulder, there are very few sites where any of the major terminal nerves of the brachial plexus are vulnerable to entrapment. With the exception of the axillary and radial nerves, it could be argued that none is formed until it reaches the level of the shoulder. Certainly, only the axillary nerve innervates any of the major muscles of the shoulder.

The axillary nerve arises from the posterior cord of the brachial plexus and passes laterally just anterior and inferior to the shoulder joint. It joins the posterior humeral circumflex vessels, and all structures pass posteriorly through the quadrangular (quadrilateral) space as they wrap around the posterior aspect of the surgical neck of the humerus. This space is formed laterally by the surgical neck of the humerus, medially by the long head of the triceps brachii muscle, superiorly by the subscapularis and the teres minor muscles, and inferiorly by the teres major muscle. Posterior to this space, the axillary nerve and vessels branch to supply the teres minor and deltoid muscles. A cutaneous branch of the axillary nerve supplies the skin superficial to the lateral aspect of the deltoid muscle.

The quadrangular space is a potential entrapment site for both the axillary nerve and the posterior humeral circumflex vessels. The quadrilateral space syndrome represents a true neurovascular entrapment because both the nerve and vessels are involved. Fibrous bands associated with the muscles forming the space are believed to cause the syndrome. Portions of the subscapularis, teres minor, and teres major muscles may be tendinous where they form the borders of this space; the long head of the triceps brachii muscle is not. Interestingly, Cahill reports that such fibrous bands have not been found in cadavers. The cases of axillary nerve entrapment that have been confirmed involved active patients in the 20- to 40-year age range. I believe that two reasonable explanations exist for the entrapment. Wide excursions of motion, presumably subjecting the nerve and vessels to considerable friction or traction, may cause the nerve to undergo the usual sequence of local irritation and inflammation. The artery, perhaps, may react either with local spasm or local inflammation, either of which may reduce the size of the vessel's lumen and, thus, blood flow to the structures supplied. Muscle hypertrophy, however, may affect nerves and vessels in the same manner. These explanations are rational but certainly conjectural.

The symptoms of this entrapment syndrome usually are vague, and their onset is gradual. Pain tends to be localized poorly around the aspects of the anterolateral shoulder, and paresthesia usually is present but follows no segmental pattern. The paresthesia may extend distally as far as the hand. Muscular involvement, if any, appears to be minimal. The vagueness of the symptoms may indicate involvement of the posterior humeral circumflex vessels and not the axillary nerve.

This syndrome may be difficult to diagnose. The pain and paresthesia essentially are nondescript. Abduction and lateral rotation of the arm frequently increase the intensity of the pain, as they do in other TOSs. According to Cahill, palpation of the quadrangular space posteriorly always produces pain in that area.

Surgery has been successful in those cases that have been definitively diagnosed. The use of anti-inflammatory and analgesic medications has produced conflicting results.

The axillary nerve is vulnerable to a stretching injury when the humeral head is dislocated anteriorly. The symptoms of this type of injury are consistent with the motor and sensory distribution of the nerve, and its onset is defined easily. Its diagnosis, therefore, should be definitive.

CONCLUSION

The anatomic basis for entrapment of the brachial plexus, as included within the diagnosis of the TOS, has been presented. The anatomic basis for entrapment of certain branches of the brachial plexus also has been discussed. The diagnosis and successful management of TOC frequently are challenging goals for the physical therapist. I hope that this article has imparted an appreciation of the various mechanisms involved in the entrapment of nerve trunks and nerves of the shoulder and posterior triangle of the neck, and that this understanding will promote continued and improved physical therapy management.

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Manuscript Reviewers

The Associate Editors and the Editor of PHYSICAL THERAPY wish to thank the following individuals for their dedicated and valuable services as manuscript reviewers during 1986.


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