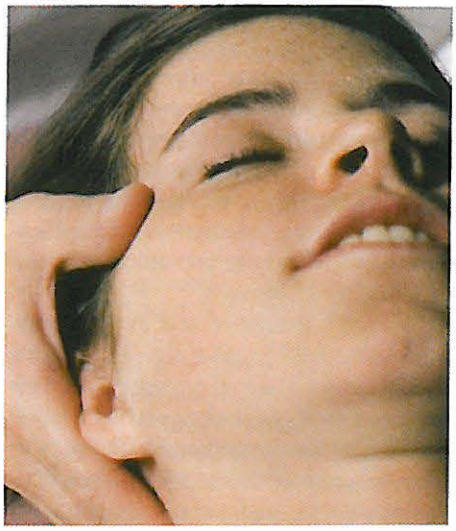
CRANIOSACRAL THERAPY & THE VISUAL SYSTEM



KENNETH I. FREY, P.T.

Abstract

Craniosacral therapy (CST) is a gentle, non-invasive, whole body evaluation and treatment approach which has been shown to be an effective method of treatment for a wide variety of problems affecting the cranial visual system. CST is based upon palpation of the craniosacral rhythm (CSR) and assessment of the craniosacral system. This paper presents the historical and conceptual background of CST and highlights the multifaceted structural relationships between the craniosacral and visual systems.

Key Words

craniosacral therapy (CST), craniosacral rhythm (CSR), craniosacral system, craniovisual system

Craniosacral therapy (CST) is a gentle, non-invasive, hands-on modality widely used by osteopathic physicians, physical and occupational therapists, chiropractors, massage therapists and other health care practitioners. The craniosacral system involves the tissues surrounding the brain, spinal cord and central nervous system. Using subtle palpation, the craniosacral therapist is able to identify restrictions anywhere in the body that potentially impact the craniovisual system.

Because of its focus on the core system in the body, clinical application has demonstrated CST effective in providing symptomatic relief to a wide variety of problems, including orthopedic and neurological disabilities.[1] CST has been instrumental in treating musculoskeletal pain syndromes, headaches, trauma,[2-6] strabismus, vertigo[2-5] developmental disorders, learning disabilities,[2,5,7] cerebral ischemia, temporo-mandibular joint dysfunction,[8] neurofascial and neurovascular impingements, scoliosis,[9] overall stress relief and general health enhancement.

Because of the anatomical, neurological and physiological interconnections and interdependency of the craniosacral system and visual systems, disturbances affecting the visual system are frequently implicated in craniosacral system dysfunctions and vice versa. Hence, craniosacral therapy can potentially support optimization of visual function and help resolve both short- and long-term visual trauma. A brief history of the evolution of this form of therapeutic intervention, and a discussion of its approach to treatment, will clarify the mutually supportive applications of craniosacral therapy and behavioral optometry.

History

CST developed from Cranial Osteopathy. William Sutherland, D.O., (1873-1954), the originator of this approach, was trained by A. T. Still (1828-1917), the father of Osteopathy. The basic principles of craniosacral therapy and of osteopathy today have increasing appeal due to the growth of alternative medicine. These principles include the fact that the body functions holistically; structure and function are interrelated; the body has a self-healing ability; and drugs may not always be the answer.

As a medical student, Sutherland, like more Western anatomists and physicians of the time, was taught that the human cranial sutures fuse by calcification at an early age, thereby prohibiting cranial vault motion. While this view of the skull, which reflects the orthodoxy of British anatomy texts, has been widely shared until fairly recently, it has by no means been universal. Students in countries following primarily Italian as opposed to British anatomy texts are taught cranial bones retain mobility. Following years of debate over this issue, Gray's Anatomy[10] acknowledges and research evidence[1-8,12-32] supports the possibility of cranial bone sutural mobility.

Craniosacral therapy is founded on the observation that cranial bone motion does occur and is inherently purposeful. As a medical student observing a disarticulated skull, Sutherland was struck by the beveled sutural surfaces of the squamous portion of the temporal bones and the greater wings of the sphenoid. He was intrigued by their structural similarity to the gills of a fish and their role providing articular mobility for respiration.[2]

Through extensive self experimentation, Sutherland became convinced of the rhythmic movement of cranial bones. This repetitive motion, initially called Cranial Rhythmic Impulse and now referred to as Craniosacral Rhythm (CSR), pulses from the cranium through the sacrum and is reflected as a subtle wavelike motion through the extremities. The rhythm of cranial bone movement is in turn compliant to the filling and emptying of Cerebral Spinal Fluid (CSF) through the cerebral ventricles.[3,11] By palpating the CSR, Sutherland was able to identify restriction impacting the craniosacral and craniovisual systems. These restrictions in turn contributed to Central Nervous System (CNS), CSF, brain and spinal cord dysfunction. Sutherland became widely known for his ability, through gentle mobilization of the craniosacral system, to treat many conditions that were unresponsive to other forms of conventional treatment.

Sutherland's ideas, based upon the principle of cranial bone motion, rapidly grew in popularity, as did opposition to these ideas. In the 1970s, the Osteopathic profession convened a commission at Michigan State University to finally prove or disprove Sutherland's theories on the movement of cranial sutural bones and the functioning of the craniosacral system. John Upledger, D.O., was a member of this investigative group. Today, Upledger is recognized internationally as the leading authority on craniosacral therapy.

Several results emerged from the work of this commission:

First, it was conclusively demonstrated that cranial sutures have the histological capacity for motion. Cranial sutures were shown to contain an abundance of collagen and elastic fibers, sharpey's fibers, vascular networks, non-myelinated nerve fiber networks and receptors.[12-16] Thus, they contained essentially all the components necessary for sutural mobility. Studying fresh sutural specimens rather than chemically preserved specimens, investigators found little evidence of the sutural ossification, which was predicted by western scientists and which would prevent movement of the cranial bones.

Second, having demonstrated that cranial bones have the potential for mobility, it was then demonstrated that cranial bones do indeed move. Radio antennae were set up on live monkeys. One antenna was embedded into each parietal bone of live anesthetized monkeys, equidistant from the sagittal suture. As a radio signal was broadcast across the two antennae, an independent motion was recorded. The craniosacral movement was distinct from either the recorded respiratory or heart rates.[16](p. 6) The CSR on three monkeys fluctuated between 8-10 cpm.[11] Since then numerous researchers have reported human craniosacral rhythmical motion of 8-12 cpm.[3,8,16-27]

Third, once evidence had been collected that demonstrated the reality of cranial bone movement and of craniosacral rhythm, the quest arose as to whether this rhythm could be objectively utilized to identify craniosacral system restrictions in the same manner by a variety of skilled practitioners. An interrater reliability and reproducibility study was performed on 25 preschool children. A 19 parameter hands-on standardized craniosacral evaluation tool was developed. Statistical analysis of the data demonstrated an overall interrater reliability of 85% agreement with a 0.5% variance allowed (71% with no variance) among four skilled craniosacral examiners on 50 examinations. The rate of CSR was compared with cardiac pulse and respiratory rates of both the subject and examiner on all examinations. The results of this study supported the existence of CSR as an independent physiological rhythm.[17]

Fourth, the question arose as to whether craniosacral rhythm imbalances might in any meaningful way be related to neurological, musculoskeletal or behavioral problems. Using the same methods derived from the interrater reproducibility study, the 19 parameter standardized CST evaluation tool was then applied by Upledger to 203 public school children.[7]

Children were categorized as normal, not normal, behavioral; motor coordination and speech problems, and learning disabilities. Following CST evaluation, an independent research technician collected historical data by personal interviews with parents. The historical categories surveyed were seizure history, head injury, obstetrical complication and ear problems. Each child's school performance and teacher assessment was evaluated by an independent statistician.

The standardized quantitative craniosacral motion examination was found to represent a practical approach to the study of relationships between craniosacral system dysfunctions and a variety of health, behavioral and performance problems. The results supported school officials' and teachers' classification of children as "normal" and "not normal." The craniosacral dysfunction scores also positively correlated with parental classifications of not normal, behavioral problems, learning disabilities, motor coordination problems and obstetrical complications. The highest scores of craniosacral restriction correlated most positively with those children suffering from multiple problems.

The Craniosacral System: How It Functions

The studies at Michigan State University resulted in a model of how the craniosacral system functions that has since become widely accepted. Called the Pressurestat Model, this approach described the craniosacral system as a semiclosed hydraulic system.[11] The rhythmical CSR fluctuations were functionally described. Retzlaff and Upledger found, that in the sagittal suture of humans there was an instrasutural nerve plexus along with a variety of neural receptors.[16] They reasoned these would function to sense both compression and stretch reception. Further, in monkeys they found a nerve tract connection between the sagittal suture and ventricular system of the brain. They theorized a signal system between the sagittal suture and ventricles relaying information on the production and reabsorption of CSF from the craniosacral system.

The Pressurestat Model describes how CSF production in the choroid plexuses fills the ventricles. The resultant increase in ventricular volume and pressure causes the parietal bones to expand in a widening motion. At the end range of motion a stretch reflex is activated in the intra

sagittal sutural nerve. The nerve sends a signal to the filled ventricles indicating that sutural expansion (stretch) is sufficient and requests cessation of CSF production. CSF ventricular production thereby ceases. Meanwhile CSF continues a constant emptying and venous reabsorption from the craniosacral system. As CSF continues to empty with no additional production, the production stoppage allows CSF volume and pressure to decrease, reversing the original sagittal sutural expansion now into a compression phase. The cycle is completed by the intrasutural compression receptors signaling the ventricular system to resume CSF production.

This rhythmic cycle of CSF filling and emptying repeats itself every six seconds. This allows three seconds for CSF filling and three seconds for emptying. The normal craniosacral rhythm is eight to 12 cycles per minute.

Dysfunctions Of The Membrane System

The most frequent and clinically significant cause of craniosacral system dysfunction is abnormal tension affecting the dural tissue membranes.[3] When dural membranes are subject to abnormal tension in a certain direction over a considerable period of time, their fibers organize themselves in the direction of the tension. This tension can be seen histologically and palpated.

Abnormal membrane tensions, along with sutural immobility, can result in distortions to the normal motion of the craniosacral system, and adversely affect free subarachnoid CSF flow, blood flow through the vascular system, neural conduction and numerous physiological functions.

The craniosacral and visual systems share an inherent anatomical relationship. Afferent visual processing from the retina to the pretectum and the lateral geniculate body to the visual cortex are enclosed in the fascial, dural and osseous housing of the craniosacral system.[33] Abnormal tissue membrane tension of the craniosacral system can negatively influence the visual system, causing structural dysfunctions influencing visual sensory and visual perceptual problems, and primary afferent visual disorders. The mechanism of these dysfunctions will now be further explored.

How Dysfunctions Are Detected

Dysfunctions within the craniosacral system can ultimately manifest themselves as subtle restrictions in the osseous and membranous portions of the craniosacral system. Moreover, since the outer dural endosteal layer is firmly attached to the inner surfaces of the cranial bones, one can use the cranial vault bones as levers to evaluate and treat intracranial structures.[3]

Craniosacral therapy's diagnostic and treatment potential, however, extends beyond the craniosacral system narrowly defined. In fact, because of the structural continuity of the entire body, CST is commonly used to identify and treat dysfunctions arising from anywhere within the body. The entire body's connective tissue is interconnected via the fascial system. Thus the craniosacral system and associated visual system can potentially become restricted from anywhere in the body. In addition, both cranial and visual problems related to adverse mechanical membrane dysfunction can be identified and may need to be alleviated from the body external to the cranium. A gentle hands-on palpation of five grams allows the craniosacral therapist to access the subtle CSR from anywhere on the body. By placing his hands on various areas of the body and palpating for symmetry, quality, range and rate of motion, the therapist can identify the source and nature of restriction involved.

It is clinically accepted that a full 80% of all intracranial restrictions, including restrictions that might affect the visual system, arise from a source external to the craniosacral system. CST can potentially serve as an adjunct to behavioral optometric treatment by acting as a diagnostic aid to identify the origins of visual problems whose source lies outside the visual system proper. Similarly, CST can help to relieve restrictions external to the craniovisual system that nonetheless impact on visual function.

The Sphenobasilar Junction

Restrictions whose origins are external to the craniosacral system frequently influence craniosacral function through their indirect impact on the sphenobasilar junction. The sphenobasilar junction is considered by many to be the keystone of the craniosacral system.[4,5] Due to its integral relationship with 17 other cranial bones and with the tentorium cerebelli, it plays a major role in visual cranial deficits.

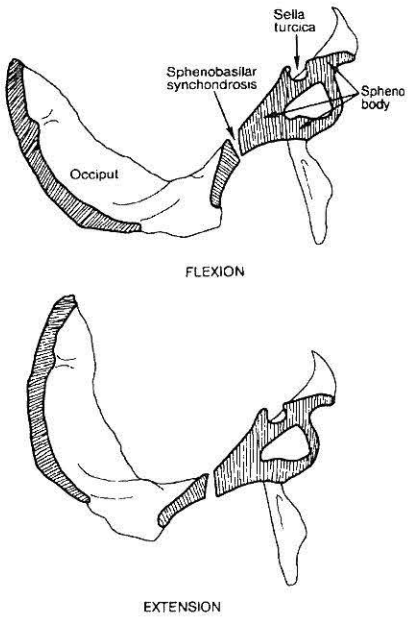


Figure 1. Flexion and Extension Phases of Craniosacral Motion at the Sphenobasilar Junction. Reprinted with permission of publisher.[3] p.104

Like other cranial bones, the sphenoid and occiput, joined together at the sphenobasilar junction, normally move in a rhythmic motion that corresponds with the flow of cerebrospinal fluid. The sphenoid and occiput move synchronously caudal direction in CSR flexion and cephalad in CSR extension (see Figure 1.).

When external restrictions impact on the sphenobasilar junction, this can result in a "lesion" or motion dysfunction which, in turn, can affect both the connected bones and the tentorium cerebelli. The tentorium cerebelli forms a horizontal membrane system which is of particular influence to the visual system function. The tentorium consists of a superior and inferior leaf. Reciprocally the superior leaf is continuous with the falx cerebri and the inferior leaf with the falx cerebelli. The tentorium bilaterally attaches to the mastoid and petrous portions of the temporal bones, at the posterior angle of the parietal bones and courses anteriorly. The superior and inferior leaves attach to the anterior and posterior clinoid processes of the sphenoid bone respectively. Therefore, for symmeterical sphenoid function these two pair of hemicranial sheaths need to be balanced.

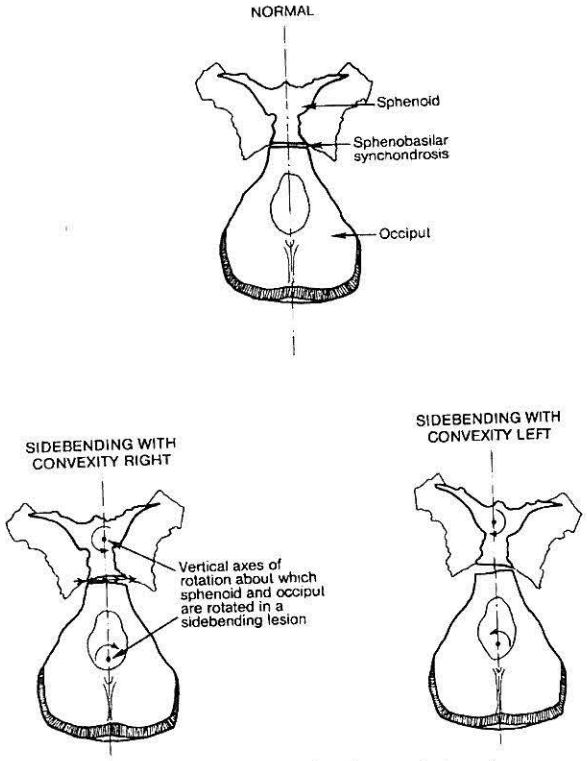


Figure 2. Sidebend Lesion. Reprinted with permission of publisher.[3] p. 106

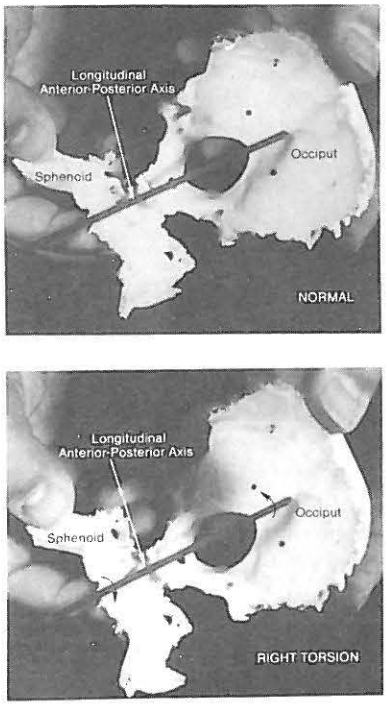


Figure 3. Torsion Lesions Reprinted with permission of publisher.[3] p.107

The oculomotor, trochlear, and abducens nerves, and the associated visual vascular support are enveloped between the two tentorial sheaths as they transverse the cranial vault.

Craniosacral lesions are named for their direction of greatest ease of motion. Thus a sphenoid which moves more easily or with greater range in an anterior/caudal or flexion motion and resists extension is called a "flexion lesion."

Visual impairments can be attributed to sidebend sphenoid lesions. This biomechanical dysfunction represents a unilateral anterior and contralateral posterior displacement of the sphenoid greater wings around a vertical axis (see Figure 2.).

Another common sphenoid dysfunction is the torsion lesion. In this case one greater wing moves cephalad, the other caudal with greater ease and range of motion (see Figure 3.). Torsion lesions are named for the greater wing which elevates more easily. In a torsion lesion the sphenoid is rotated on a longititudinal anterior to posterior axis.

Craniovisual Impairments

Sphenobasilar dysfunctions are commonly associated with both acquired and congenital strabismus as well as vergence anomalies. Abnormal extraocular muscle imbalances are frequently associated with asymmetric cranial system dural and osseous problems.

In addition to creating visual perceptual and motor disturbances, cranial base sphenobasilar distortions can create pain syndromes, headaches, endocrine disorders, visual perceptual and motor disturbances, sinusitis, nasal and upper respiratory allergies, temporomandibular joint problems, dental malocclusion and musculoskeletal imbalance problems.

Cranial base sphenoid flexion, extension, sidebend and torsion lesions are generally compensatory to some dysfunction or imbalance extrinsic to the craniosacral system. A whole body approach is therefore necessary to identify and treat the causative factors underlying these problems.

Sphenobasilar dysfunctions of intracranial origin are more serious and incapacitating. This category includes sphenoid lateral and vertical strains, and compression dysfunctions. These are usually caused by direct head traumas such as blows to the forehead and birth trauma. These dysfunctions contribute to a wide variety of severe clinical dysfunctions, including eye motor coordination problems, learning and developmental disabilities. Any biomechanical disturbance to the anterior cranial region may affect eye position and ocular alignment of visual axis.

Occipital Cranial Base Dysfunction

Occipital Cranial Base (OCB) dysfunction is another common craniosacral system structural imposition influencing vision. This occurs more frequently than realized during the birthing process, with excessive extension on the occipital atlas cervical junction, as in whiplash injuries and by postural head/neck dysfunction.[3,5]

Other cranial base visual motor disturbances can occur when the tentorium, occiput or temporal bones are shifted, causing the tentorium to change the shape of the jugular foramen, limiting the flow of venous blood drainage out of the cranium. Should the arachnoid membrane become locked down, the subarachnoid space becomes smaller and CSF flow may become impaired.[4]

Visual cortex dysfunction can occur if the angle between the falx and tentorium becomes acute and squeezes or impinges upon the visual cortex.[4] Similarly, brain stem compression can affect visual connections in the reticular formation and primary visual cortex and to the lateral geniculate process areas.

Cover photo courtesy of the Institute of Physical Therapy, New York, NY.

References

1. Fryman VM, Carney RE, Springall P. Effect of osteopathic medical management on neurologic development in children. J Am Osteopath Assoc, 1992: 6:729-44.
2. Sutherland WG. The cranial bowl. Reprinted 1994, The Osteopathic Cranial Association.
3. Upledger JE, Vredegvoogd JD. Craniosacral therapy. Seattle: Eastland Press, 1983.
4. Sutherland WG. Teachings in the science of osteopathy. Sutherland Cranial Teaching Foundation, Inc., 1990: 230.
5. Magoun HI. Osteopathy in the cranial field, Third Edition. The Cranial Academy: 1976.

6. Frymann VM. Relation of disturbances of craniosacral mechanisms to symptomatology of the newborn: study of 1,250 infants. J Am Osteopath Assoc, 1966; 65:1059-75.
7. Upledger JE. Relationship of craniosacral examination findings in grade school children with developmental problems. J Am Osteopath Assoc, 1978; 77:760-76.
8. Baker EG. Alteration in width of maxillary arch and its relation to sutural movement of cranial bones. J Am Osteopath Assoc, 1971; 70:559-64. 28.
9. Kain JB, Weiselfish S. Integrated manual therapy protocol for treatment of idiopathic scoliosis. Advance for Physical Therapists, 1992; Dec 7:8-9.
10. Goss CM. Gray's anatomy. 29th American Edition. Philadelphia: Lea & Febiger, 1973: 150.
11. Upledger JE. Craniosacral therapy I study guide. Palm Beach Gardens, FL: The Upledger Institute, 1996.
12. Retzlaff EW, et al. Nerve fiber and endings in cranial sutures. J Am Osteopath Assoc, 1978; 77:474-75.
13. Retzlaff EW, Upledger JE, Mitchell F, Jr., et al. Structure of cranial bone sutures. J Am Osteopath Assoc, 1976; 75:607-08.
14. Retzlaff EW, Roppell RM, Becker RF, et al. Craniosacral mechanism. J Am Osteopathic Assoc, 1976; 76:288-89.
15. Retzlaff EW, Upledger JE, Mitchell FL:, et al. Parieto-parietal suture in adult squirrel monkeys, saimiri sciureus. Anat Rec, 1977; 187:692.
16. Upledger JE. Research supports the existence of a craniosacral system. Palm Beach Gardens, FL: UI Enterprises, 1995.
17. Upledger JE. Reproducibility of craniosacral examination findings: a statistical analysis. J Am Osteopath Assoc, 1977; 76:890-99.
18. Upledger JE, Karni Z. Mechanoelectric patterns during craniosacral diagnosis and treatment. J Am Osteopath Assoc, 1979; 79:782-91.
19. Fryman VM. A study of rhythmic motions of the living cranium. J Am Osteopath Assoc, 1971; 70(9):928-45.
20. Temttambel M. Recording of cranial rhythmic impulse. J Am Osteopath Assoc, 1978; 78:149.
21. Herniou JC. Studies of the structures and mechanical properties of the cranium. Doctoral Thesis, Universite de Technologie de Coliegne, Paris, France.
22. Wallace, Avant, McKinney, et al. Ulstrasonic measurement of intra-cranial pulsations at nine cycles per minute. J Neurology, 1975.
23. Norton, JM. Characterization of cranial rhythmic impulse in healthy human adults. J Am Osteopath Assoc, 1992; 92:1285.
24. Poldlas H, Allen KL, Bunt EA. Computed tomography studies of human brain movements. So Africa J Surg, 1984; 22(1):57-63.
25. Roppel RM, St. Pierre N, Mitchell FL. Measurement of accuracy in bimanual perception of motion. J Am Osteopath Assoc, 1978; 77:475.
26. Adams T, Heisey RS, Smith MC, et al. Parietal bone mobility in the anesthetized cat. J Am Osteopath Assoc, 1992; 92:599-622.
27. Woods JM, Woods RM. Physical findings related to psychiatric disorder. J Am Osteopath Assoc, 1961; 60:988-93.
28. Avezaat CJ, van Eijndhoven JH. Clinic observations on the relationship between cerebrospinal fluid pulse pressure and intracranial pressure. Acta BNeurochir (Wien), 1986; 79(1):13-29.
29. Feinberg DA, Mark AS. Human brain motion and cerebrospinal fluid circulation demonstrated with MR velocity imaging. Radiology, 1987; 163:793-99.
30. Greenman PE. Roentgen findings in the craniosacral mechanism. J Am Osteopath Assoc, 1970; 1:60-71.
31. Heifetz MD, Weiss M. Detection of skull expansion with increased intracranial pressure. J Neurosurgery, 1981; 55:811-12.
32. Kostopoulos DC, Keramidas G. Changes in elongation of falx cerebri during craniosacral therapy techniques applied on the skull of an embalmed cadaver. J Cranialmandibular Practice, 1992; 10:9-12.
33. Kandel ER, Schwartz JH. Principles of neural science, Third Edition. CT: Appleton & Lange, 1991.
34. Netter FH. Atlas of human anatomy. New Jersey: CIBA-GEIGY, 1989.
35. Williams PL, et al. Gray's anatomy, 37th Edition. New York: Churchill Livingstone, 1989.

Corresponding author:
Kenneth I. Frey, P.T., Diplomate CST
Director Institute of Physical Therapy
30 West 60 Street, Suite 1C
New York, NY 10023
Date accepted for publication:
January 21, 1999