The Effects of Spontaneous Visual and Verbal Training Versus Verbal Training Alone on the Ability to Make Positive, Lasting Changes To the Posture of People with Parkinson's Disease.

Submitted in Partial Completion of the Requirements for a Master of Arts Degree in Physical Therapy, Touro College, Barry Z. Levine, School of Health Sciences JULY 1990

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Chapter I – Introduction to the Study

Words presented throughout the text in *italics* are featured in the section titled "Definition of Related Terms" beginning on page 64

Introduction:

As Parkinson's disease progresses, the patient experiences a noticeable change in static standing posture. Clinically, there will be a tendency toward forward head and an overall kyphotic stance. The aim of this study is to compare a posture training program using conventional verbal instruction with a similar program that combines verbal instruction with simultaneous visual feedback. The comparison will focus on each method's ability to make positive changes in the patient's posture that will last over time.

Statement of the problem:

As the secondary changes of Parkinson's disease advance, the "propensity to lean forward"[1] becomes one of the most visible changes to develop, it is the major contributor to the disabilities of Parkinson's disease. Any attempt at any early intervention program involving the betterment of the classic Parkinsonian posture may serve to delay or avoid some of the most common sequelae often seen with Parkinson's disease.

Limits of the study:

Only changes in posture of the sagittal plane were explored. Specifically, this includes forward head and thoracic kyphosis.

The population was limited to Parkinson's disease patients that are independent ambulators and regularly attend the Parkinson's Therapy Center, Smithtown, N.Y.

Basic Assumptions:

It is assumed that a decrease in the linear distance the external auditory meatus and the apex of the thoracic kyphosis is directly related to an improvement of the classic "stooped-flexed" posture seen clinically in people suffering from Parkinson's disease.

The Null Hypothesis:

There are no differences between the two training methods with regard to their efficacy in improving the sagittal plane posture of Parkinson's patients.

The Need for the Study:

At the early stages of the disease, Parkinson patients tend to be "less strict than usual with preserving upright posture."[1] An early intervention program of postural awareness is essential in preventing, or postponing, the secondary changes that accompany Parkinson's disease. The clinical example of the Parkinsonian patient, breaking into a "running" or *festinating gait*, is a prime example of a secondary change related to "the propensity to bend the trunk forward."(Parkinson, 1817) The pattern of *festinating* gait also leads to an inability for the patient to stop or change direction once locomotion as been established.

An upright posture allows gravity to aid in energy conservation, but as the body bends forward, gravity plays a negative role in the conservation of energy. Thus, resulting in a drastic increase in the amount of energy required to maintain an erect stance. This especially pertains to posterior musculature, specifically of the neck, trunk and back.[2] This may explain the high clinical occurrence of low back pain in Parkinson's patients.[3]

The postural changes that occur as a result of Parkinson's disease also contribute to a decrease in lung vital capacity by restricting expansion of the thoracic cavity. This, in turn, leads to speech difficulties as well an inability to generate a productive cough. With this inability to clear secretions, there is an associated increase in the susceptibility to pneumonia;[2] this

is one of the leading causes of mortality in the Parkinsonian population.[3]

Further musculo-skeletal imbalance due to forward bending, also leads to changes in the length tension relationship of the trunk. The anterior musculature becomes shortened while the extensor musculature is lengthened. This causes the forward posture to become more fixed in nature,[2] thus having profound implications on the ability of a clinician to alter many of resulting sequelae.

Neck extension, a primary component of forward head posture, leads to an increased tension on the muscles that retract the jaw as well as a lowered resting tongue position. The increased tension consequently leads to a chronic retraction of the jaw, this along with the lowered tongue position both serve to cause difficulties with swallowing and chewing.[4]

Upper extremity range of motion is also profoundly affected by a forward flexed posture. Associated with forward head, there is an anterior rolling of the shoulders. When this occurs, it becomes increasingly difficult for the patient to achieve full range of motion, especially in motions such as shoulder flexion and abduction.[4]

Finally, the constant drive to bend forward causes the patient to continuously look down. This consequently leads to a loss of eye contact with others. The loss of this input may feed into the isolation that the Parkinsonian patient often experiences.[2]

Chapter II – Review of literature

Understanding Parkinson's Disease

Definition:

Also known as Paralysis agitans and Shaking palsy,[5] Parkinson's disease is a chronic, progressively degenerative disease of the central nervous system.[6] It is earmarked by the presence of tremor, *rigidity* and *bradykinesia*.

Anatomy and Physiology

The Basal Ganglia

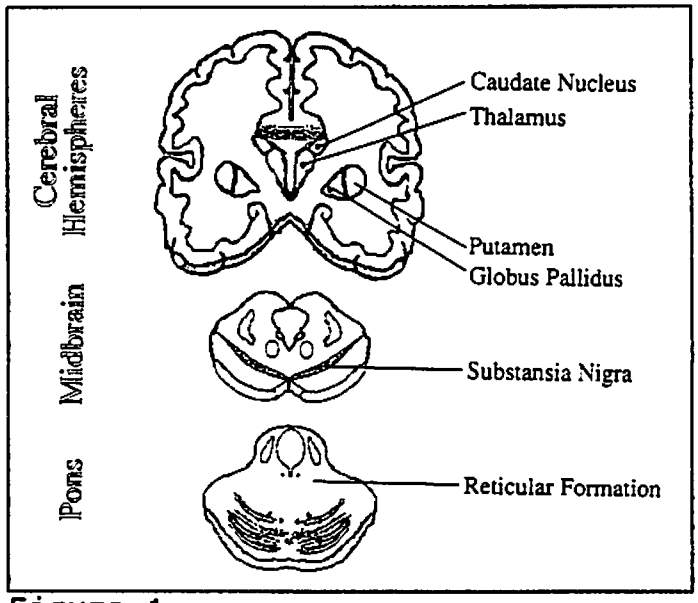

Figure 1

Currently, the term Basal Ganglia refers to three areas of gray matter constituting the base of the cerebral hemispheres. They are the Caudate nucleus, the *Putamen* and the Globus Pallidus.[7] (FIG 1) Classically, however, the term included all the gray matter areas in the *diencephalon* and *telencephalon*.[8] Other terms that may be encountered when considering the Basal Ganglia include the corpus striatum and the lentiform nucleus. Considered the primary input center of the Basal

Ganglia, corpus striatum refer to the caudate nucleus and the *putamen*. The lentiform nucleus is a term used to describe the *putamen* and Globus Pallidus collectively. The Basal Ganglia are part of the extrapyramidal system.[7]

The Substantia Nigra

The Substantia Nigra is located in the midbrain just dorsal to each *peduncle*. It extends *rostrally* from the upper border of the pons to the subthalamic region. It lies ventral to both the *red nucleus* and *medial lemniscus* and extends from the midline to the lateral boarder of the midbrain. Functionally, it is closely related to the Basal Ganglia and has been referred to as being a functional part of it.[7] The Substantia Nigra is composed of two parts, a compact zone and a reticular zone.[8] The compact zone is referred to as Substantia Nigra pars compacta (SNC).[9] It as been denoted as the "black zone" due to it's dark appearance. The dark appearance is attributed to the presence of neurons rich in a melanin-like substance; neuromelanin. Although the connection is not clear, it is postulated that this pigment is related to the production and storage of the neuro transmitter *dopamine*.[10] The reticular zone, also known as, the Substantia Nigra pars reticulata (SNpr) resides in the most ventral portion of the Substantia Nigra. It's cells contain no melanin pigment, but, are rich in iron. This gives the structure a characteristic red appearance when fresh.[8]

The Basal Ganglia's Circuit of Motor Control

The complex, finely woven fabric of connections that emanate to and from the Basal Ganglia are not fully understood. However, a brief overview of the Basal Ganglia's motor circuits would serve to aid in the understanding of the pathologies that occur there.

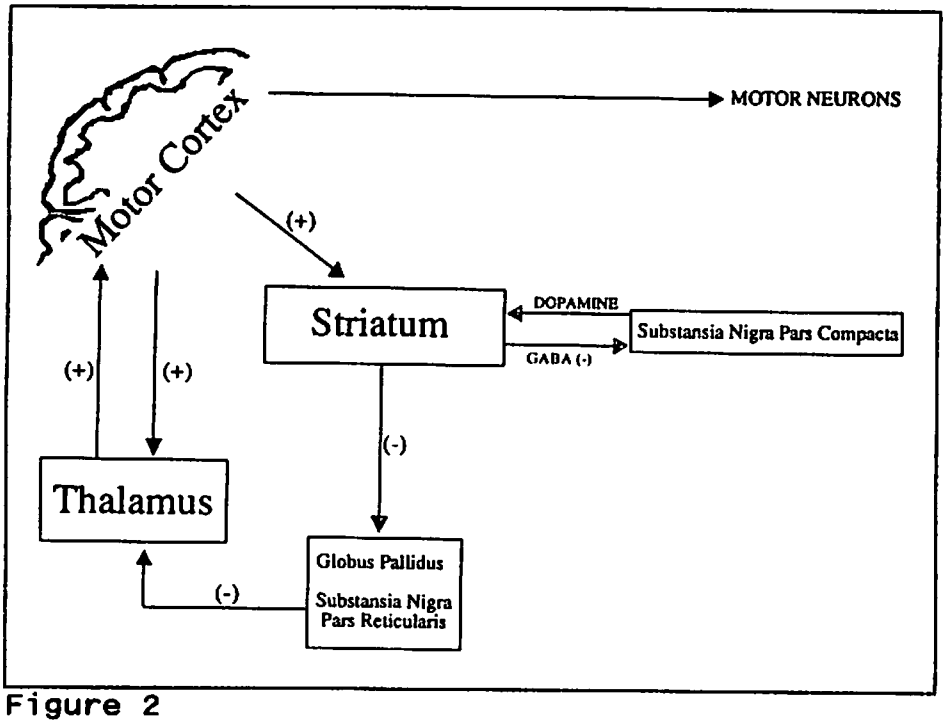

Figure 2

The "motor circuit" begins with input from the *sensorimotor cortex*, premotor cortex, and some parts of the parietal lobe, and are received by the *putamen* and caudate nucleus.[8] [11] Other input also originates from the Substantia Nigra pars compacta and the intralaminar thalamic nuclei.[7] [11] The neurons of the caudate and *putamen* project to the Globus Pallidus, which in turn projects to the ventral anterior (VA) and ventral lateral (VL) nuclei of the

thalamus. The thalamus then projects to the cerebral cortex, specifically, to the supplementary motor cortex (SMA).[8 11] (FIG.2)

An important point to recognize is the lack of any connection between the Basal Ganglia and any sensory or motor end organs. The Basal Ganglia influence movement in the absence of any direct sensory input. Furthermore, its effects on motor behavior are mediated primarily through the cerebral cortex. There are no direct connections of the Basal Ganglia with *alpha motor neurons*. The majority of it's effects are mediated through the cortico-spinal tract.[11]

The Role of Neurotransmitters in the Basal Ganglia

The striatum contains high levels of the neurotransmitters acetylcholine and *dopamine*. Acetylcholine is manufactured in the striatum and is utilized primarily by the short axons within the striatum itself.[3 7] *Dopamine*, however, is manufactured in the Substantia Nigra pars compacta and is then released to the striatum. Once available to the striatum, *dopamine* exerts an inhibition of the neurons there. This inhibition acts to decrease the inhibitive action of the striatum on the Globus Pallidus. Therefore, with a subsequent decrease in the inhibitive force on the Globus Pallidus, it is free to act as an inhibitor of the thalamus. Ultimately then, the *dopamine* of the striatum acts to inhibit the thalamus. This check on the thalamus provides the cortex with the ability to stop motor behaviors in favor of new motor behaviors. Therefore, the primary function of the

dopamergenic system is to maintain a check on the activities of the thalamus.[7]

The Relationship Between the Basal Ganglia and Parkinson's Disease.

Patients affected with Parkinson's disease have been observed to have a degeneration of the cells of the Substantia Nigra pars compacta.[3 7] This, in turn leads to a decrease in the production of *dopamine*, thus drastically compromising striatal *dopamine* levels. Logically, this reduction of *dopamine* availability leads to an imbalance toward acetylcholine activity. This imbalance results in the loss of the normal modifying influence of the Basal Ganglia on motor activities of the cortex. Therefore this breakdown of the Basal Ganglia's "mutually *antagonistic* systems"[3] leads directly to definitive Parkinsonian symptoms.

Other Factors to Consider with Regard to the Basal Ganglia

All the functions of the Basal Ganglia are not yet clear. However, current concepts suggest that the Basal Ganglia are responsible for more than simply modifying motor actions.

Marsden notes that the Basal Ganglia acts as a processing center.[11 12] It is here that complex activity can occur by the assembly of many different sub-motor programs. Simply stated, Marsden suggests that the Basal Ganglia are responsible for collecting information regarding motor patterns from other areas of the brain. It then sequences them and sends a completed motor message to the cortex.

Research also suggests that the Basal Ganglia are charged with specifying the direction and scaling the amount of volitional movement. [11] [13]

Some researchers have postulated that the Basal Ganglia also take part in the process of deciding which motor behaviors are acceptable and which should be repressed. [11] [14]

As methods of discerning neuroanatomic structures becomes more refined, more information is revealed about the complexity of the Basal Ganglia. Evarts and Wise have suggested that the Basal Ganglia links higher *cortical* function to the motor cortex. [11] [15]

Perhaps some of the most interesting notions about the function of the Basal Ganglia are those that do not deal with motor activity per se. It has been suggested that the Basal Ganglia plays a role in cognitive function as well as in fine movements of the eye. [11]

The many varied and yet to be understood functions of the Basal Ganglia should begin to give the reader some insight into how complex the pathology of Parkinson's disease is and how little is really known about the full extent of its mechanism. Research also suggests that Parkinson's disease is not simply a disease of *dopamine* production in the Substantia Nigra. Evidence now points to a complex interaction involving mesolimbic, thalamic, *cortical*, and *subcortical* structures and effecting many different types of neurotransmitters and receptors. [2] [9]

Pharmacological Treatment of Parkinson's

An in depth analysis of the pharmacological treatment of Parkinson's disease is well beyond the scope of this review. However, the drug L-Dopa does deserve some consideration. It's proper name is L-3,4-dihydroxyphenylalanine.[10] L-Dopa is the chemical precursor of *dopamine*. L-Dopa exhibits a property that makes it invaluable in the replenishment of *dopamine* to the Parkinsonian patient. L-Dopa is able to cross the blood/brain barrier, whereas *dopamine* cannot. The fact that *dopamine* cannot cross the blood/brain barrier makes the introduction of that chemical to the brain, via the blood stream, impossible. It is, however, possible for L-Dopa to be introduced to the Central Nervous System through the blood stream. L-Dopa then quickly is converted to *dopamine* and is used by the system. This makes L-Dopa an effective defense against the Central Nervous System effects of Parkinson's disease. L-Dopa, however is not without problems. As time goes on, it takes an increasingly large dose of L-Dopa to achieve the desired effect. Therefore, it seems that an accommodation process occurs during the use of this drug, thus making it increasingly less effective.

The Etiology and Classification of Parkinsonian Symptoms

A diagnosis of Parkinsonism is made upon the appearance of two or more of the following hallmark symptoms: a tremor of approximately eight *Hz*,[6] *rigidity, akinesia* and altered postural attitude.[6 10 16] With the establishment of the classic symptoms, the syndrome can be classified into Idiopathic Parkinsonism, Toxic Parkinsonism, Drug-induced Parkinsonism, Postencephalitic Parkinsonism, Arteriosclerotic Parkinsonism, and Symptomatic Parkinsonism.

Idiopathic Parkinsonism

Idiopathic Parkinsonism, as the name implies, is the classification given to the above symptoms when no direct cause can be ascertained as to their presence. After pathologic review, the Substantia Nigra shows a characteristic lack of pigmentation. *Lewy bodies*, rounded *eosinophilic intracytoplasmic inclusions*, are also noted in some affected neurons.[16] Idiopathic Parkinsonism is the most common form of Parkinsonism and is commonly referred to as "Parkinson's disease." The disease rarely appears before the age of forty and the average age of onset is at about the sixth decade of life.[10]

Toxic Parkinsonism

When the appearance of Parkinsonian symptoms can be attributed to exposure to chemical toxins, it is termed toxic Parkinsonism. Included in this category is exposure industrial poisons such as manganese, carbon disulfide, and carbon monoxide.[3]

Also found in this category is abuse of a synthetic form of heroin containing the chemical MPTP. [3] [17]

Drug-Induced Parkinsonism

A large part of the population of this classification is comprised of patients receiving drug treatment for mental illnesses such as *schizophrenia*. It seems that certain antipsychotic drugs block the action of *dopamine* and can lead to Parkinsonian symptoms. Antipsychotics are *dopamine antagonists*. Their primary function is to block the action of *dopamine*. The most common of these drugs is chlorpromazine (largactil)[3]. In the United States it's trade name is Thorazine.[10] One of the most "Parkinsonian" antipsychotics is haloperidol (Haldol). It can induce Parkinsonian symptoms with 10 minutes of injection. Reserpine and methyldopa drugs currently employed as an anti-hypertensives have also been found to cause Parkinsonian symptoms. However, the dosage required is large and the occurrence rare.[10]

With drug-induced Parkinsonism, the effects are directly related to the drugs and usually have no lasting effects. Once the drug is discontinued, it need only be completely metabolized by the body for all Parkinsonian symptoms to disappear [3] [6] [10] However, the chronic use of antipsychotics can lead to the irreversible Parkinson-like symptoms of a condition known as *Tardive Dyskinesia*.[6] [18]

Postencephalitic Parkinsonism

This is a post-infectious type of Parkinsonism that is postulated to be a result of viral encephalitis. The largest

occurrence of this virus was noted between 1917 to 1920. During that time there was an outbreak of encephalitis lethargica that reached epidemic proportions.[3 6 10 16 18] Patients suffering from this disease suffered from extreme drowsiness, difficulty swallowing and changes in personality and behavior. The survival rate was low (50% - 60%) and those who recovered remained ill for six months or more. Some patients were left with paralysis, tremors, and severe *rigidity*, while others seemed to recover completely. But, of those who recovered completely, many developed Parkinsonian symptoms later in life.[10] This type of Parkinsonism is now greatly reduced. [3 6 10 16 18]

Arteriosclerotic PseudoParkinsonism

Sometimes called arteriosclerotic Parkinsonism, the term refers to the presence of Parkinsonian-like symptoms in the post-cerebral infarct patient. The term arteriosclerotic pseudoParkinsonism has been adopted by some to suggest that the symptoms are simply sequelae of the cerebral vascular accident and not related to Parkinson's disease.[3 6 10 16 18]

Symptomatic Parkinsonism

Many different pathologies can involve the Substantia Nigra, damage the communication between the striatum and Substantia Nigra, or in some other way disturb the striatal neurotransmitter balance. This classification deals with those Parkinsonian symptoms that do not appear by themselves but occur as part of another disease process.

Clinical Signs of Parkinson's Disease

Tremor

One of the first signs of Parkinson's is tremor. They are involuntary, rhythmic, oscillating movements of *antagonistic* muscle groups. The most common tremor observed is termed the pill-rolling tremor. It is characteristic in its presentation of alternate flexion and extension of the fingers and thumb accompanied by slight turning of the forearm.[3 10] The oscillations usually occur at a rate of approximately four to eight per second. Sometimes termed resting tremors, they usually manifest only at rest and disappear with initiation of voluntary movement. However, as the disease progresses, the tremors are not always restricted to a resting state. Tremors seem to be reflective of the patients state of mind. [3] Events that serve to increase the overall stress levels of the patient seem to precipitate tremor. An increase in attentiveness such as concentrating on a mental task also serves to increase tremor. The current theories on the causes of Parkinsonian tremor attribute it to the increased action of *Basal Ganglia-thalamic-cortical* circuit.[3] Although tremors are most commonly apparent in the extremities, they may also be noted in the jaw and tongue.[3 10]

Rigidity

Often described by patients as a feeling of stiffness,[10] *rigidity* is an increased resistance to passive motion. It is not merely a symptom, it is a clinical sign that is classic of Parkinson's disease. It is the result over activation of *alpha motor neurons* and an increased sensitivity of static stretch reflexes in both *agonist* and *antagonist* muscle groups [3] *Rigidity* can effect any striated muscle. It differs from spasticity in that it is uniformed throughout the range. This is in contrast to the 'clasped knife' phenomenon that occurs with spasticity, where an abrupt release of muscular tension occurs at a threshold point of resistance.[3] Two varieties of *rigidity* exist, either cogwheel or leadpipe. Cogwheel *rigidity* is designated by it's jerky presentation. It yields to resistance a little bit at a time providing it with a 'rachet like' quality.[3] [10] The current postulations about this action attribute it to either tremor superimposed on *rigidity* or *autogenic inhibition* from the *golgi tendon organs.*[3] Leadpipe *rigidity, on the other hand,* provides a smooth pattern of resistance to passive stretch, much like bending the soft metal of a leadpipe.

Bradykinesia

Simply stated, *bradykinesia* is slowness of movement. Often referred to as 'poverty of movement,' patients suffering from *bradykinesia* show marked slowness and paucity of movement and an increase in the amount of time between intention and motor action

(reaction time). Movement in one plane is often noted as a result of loss of rotation. Fine motor tasks, such as writing, are also affected as is coordination. *Bradykinesia* is thought to be the result of impaired striatal integration of sensory information into a comprehensive motor plan.[3 9] In its extreme form, *bradykinesia* represents the complete cessation of movement. In this case it is termed akinesia, which is defined as the absence of movement.[3]

Clinical Manifestations of Parkinson's Disease

Impairments and Disabilities

When considering the complex presentation of a patient with Parkinson's disease, it is helpful to differentiate between impairments and disabilities. Impairments are conditions related directly to a pathology. In other words, impairments pertain specifically to anomalies of physical or psychological origin.[2] Specifically related to Parkinson's disease tremor, *rigidity* and *bradykinesia* would all be categorized as impairments of the central nervous system.[2 16] Disability, on the other hand, refers to the changes that occur that inhibit normal functioning.[2] Disabilities can arise in any sphere of a persons life. For this reason, disability has been classified in to four sub-groups: physical, mental, emotional and social.[2 19] Complications with gait, transfers, and dressing are all physical disabilities. Decreased ability to communicate, depression, and isolation are all considered social disabilities. An important distinction to make

is that disability does not always coexist with impairment. For instance, it is possible for a patient to experience the impairment tremor and not interfere with their normal functioning. Thus, while they do have an impairment, there is no associated disability.[2]

Direct, Indirect, and Composite Impairments

Direct impairments of Parkinson's disease are those that exist as a result of the Central Nervous System involvement. (eg. *rigidity*) The indirect impairments are those symptoms that occur as a result of central nervous system dysfunction but, are in other systems of the body (eg. musculoskeletal changes). Whereas, composite impairments are those that are an amalgamate of direct and indirect impairments that combine to form a unique set of conditions. For example, *rigidity* and *bradykinesia* (direct impairments) can lead to a characteristic flexed-stooped posture (indirect impairment). The two types of impairments together may serve to produce faulty balance (composite impairment).[2] Organizing the manifestations of Parkinson's disease in this manner helps to identify which impairments can be addressed by rehabilitative means and which cannot. Also, this system of classification serves as an organized method for predicting the maximum outcome of rehabilitative efforts. In other words, it may be unrealistic to expect to change the effects directly related to the central nervous system, but it may be possible to make positive changes in other systems.[2]

Direct Impairments

As noted earlier, the pathology of Parkinson's disease involves an upset in the balance of *dopamine* in the Basal Ganglia. The impairments of *rigidity*, tremor, decreases in coordination, and deficiencies in motor planning are all directly linked to specific losses in the central nervous system. Therefore, they represent some examples of direct impairments.[2] [19] [16] Many questions remain unanswered with regard to the full extent of Parkinson's disease. The appearance of dementia is an example. When dementia occurs in a patient with Parkinson's disease it is considered a direct effect of the disease.[3]

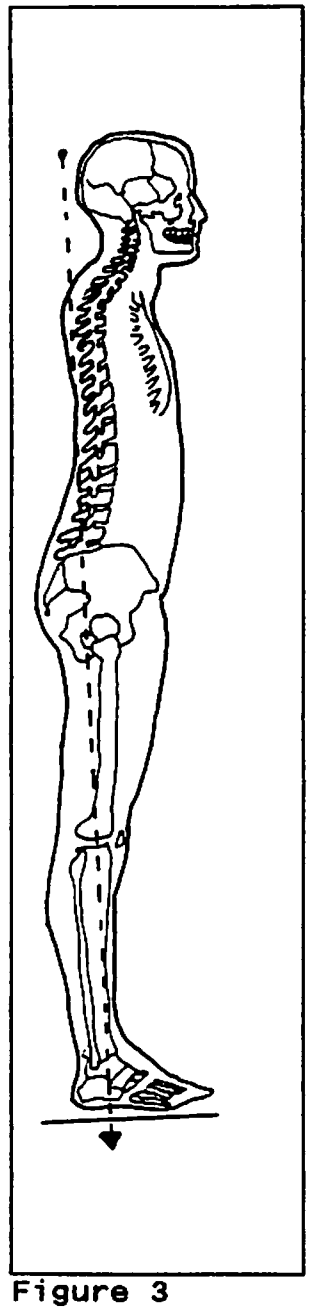

Figure 3

Indirect Impairments

Indirect impairments represent the effects of the disease on systems other than the Central nervous system. The implication here, is that a well conceived intervention plan will serve to reduce the effects of these impairments. [2]

The characteristic flexed posture,(FIG 3) so well documented in Parkinson's disease, seems to be a result of rigidity secondary to Central Nervous system dysfunction . This *rigidity*, which seems to have its greatest effect on flexor musculature, may also feed into the

tendency for the patient to remain sedentary. The latter behavior tends to lend itself to postures dominated by flexion. [2]

Although not well documented in the literature, changes in muscle length also occur in the Parkinsonian patient. Chronic postural changes then result in adaptations of the muscles that are involved. [2 20]

Musculoskeletal impairments are first seen proximally affecting contractile and noncontractile tissue of the trunk. The pelvis, and eventually the more distal structures follow.[2] Unilateral involvement is usually noted before bilateral involvement.[2 21] Specifically, these changes involve a shortening of the flexor musculature of the neck, thorax and limbs and subsequent lengthening of the extensor musculature.[2 20]

The tendency for Parkinson's patient to rely on straight-plane motions suggests an inability to call upon rotation as a component part of movement. This may be due to a generalized shortening of the flexor and extensor musculature as a result of long standing *rigidity*. This shortening leads to the clinical impairment of movement that is increasingly stiff and dominated by flexion or extension with very little lateral bending or rotation.[2 20] Without rotation the ability to roll or turn is greatly impaired. This, subtracts a major component from the overall "movement equation," and thus serves to slow the completion of movement.

The musculoskeletal impairments of Parkinson's disease can occur anywhere in the skeletal system. The pelvis, a keystone for movement, is no exception. Barriers to well coordinated movement of the pelvis lead to considerable losses in movement and decreases in weight shifting activities, in all positions.

Automatic postural adjustments could also be jeopardized by musculoskeletal dysfunction. The loss of flexibility in the lumbo-thoraco-cervical spine inhibits the proper functioning of the patient's righting reactions. Likewise, equilibrium reactions are threatened by a loss of trunk elongation and rotation.[2] These musculoskeletal factors lead to a loss of automatic postural adaptions which contribute to the high incidence of falls and subsequent fractures in people with Parkinson's disease. [2 21]

Respiratory complication is the most significant factor leading to the death of the person afflicted with Parkinson's disease.[2 21] A lack of thoracic mobility leads to a lack of thoracic expansion. This, in turn, leads to a significant decrease in vital capacity and subsequent respiratory insufficiency. For instance, a decrease in vital capacity leads to a poor inspiratory volume. This lack of sufficient lung filling, leads to ineffective coughing, the inability to clear secretions and increase the risk of contracting pneumonia and possibility death.[2]

The kyphotic posture of the Parkinsonian patient impacts greatly on the efficient functioning of the person's cardiorespiratory system. Not only is thoracic expansion reduced, but a decreased cardiac output is also clinically noted as the

disease progresses. The effects of *rigidity* and *bradykinesia* serve to compound the cardiac complications by fostering sedentary behavior which ultimately leads to generalized deconditioning. [2]

Gait deviations are synonymous with the characteristic posture of the Parkinson's patient. Impoverishment of movement predominates the locomotor patterns of the patient. Short step lengths, decreased head, neck and trunk rotation, loss of reciprocal arm swing and a lack of dorsiflexion on heel-strike are all distinctive of this "shuffling" gait pattern. [2 22] In addition, the propensity to accelerate forward is also part of the gait pattern of the Parkinson's patient. This is called *festinating* gait and makes it very difficult for the patient to stop or change directions once movement has started. A major factor contributing to this pattern, is the forward displacement of the center of gravity caused by *axial rigidity* and subsequent muscle imbalances. This displacement of the center of gravity forward may contribute to the low back pain experienced by many Parkinson's disease patients.[3] Clearly, the musculoskeletal limitations associated with Parkinson's disease contribute to the gait deviations so distinctive of the disease.

Forward protrusion of the head is a component part of the typical flexed posture seen with Parkinson's disease. In and of itself, forward head posture leads to significant impairments and can compound the total disability of the individual. Forward head posture begins as an increase in thoracic kyphosis, followed by a compensatory extension in the cervical spine. It is also

accompanied by protraction of the shoulders.[4] Early degenerative and *spondylogenic* changes are the result of the altered weight distribution of the cervical vertebra. This is especially seen in the *zygoapophyseal* joints which bear considerable weight.[4] With an increase in neck extension, the space between the cervical vertebrae is at a minimum posteriorly. This puts the greater and lesser occipital nerves at risk for impingement. Other radicular risks such as the development of traction spurs that may encroach upon nerve roots or ligamentous support structures also are associated with forward head posture. [4] [23]

In addition to specific risks within the cervical spine, the effects of forward head posture can be noted in the remainder of the axial skeleton. Changes in muscle length both in and around the cervical spine act on the axial skeleton to create an environment in which nerves are impinged and subsequent impairments occur. The development of thoracic outlet syndrome is a prime example of this. Elevation of the first and second ribs as a result of increased tension in the anterior and lateral cervical musculature serve to compress the brachial plexus as well as the subclavian artery and vein. The syndrome may manifest itself with hyperesthesias and hypoesthesias in the lateral aspect of the neck shoulder and arm.[4]

One other condition associated with forward head posture occurs from increased tension in the scalene muscles. This increased tension compromises the path of the dorsal scapular nerve. Clinically, impingement of this nerve results in weakness of the rhomboids and levator scapulae. This weakness compounds the

preexisting postural change; shoulder protraction. Shoulder girdle protraction will cause traction to be placed on the suprascapular nerve. This puts that nerve at an intense risk of compromise. Neuropathy involving this nerve can result in infraspinatus weakness as well as weakness of the glenohumeral and acromioclavicular joints.[4] [24] Prolonged shoulder protraction will tend to shorten the length of the anterior muscles of the chest. This cavitation of the chest can compress the brachial plexus, subclavian artery and vein, and thus significantly compound the effects of *Thoracic Outlet Syndrome*. A further complication of shoulder protraction is the loss of shoulder girdle range of motion that is mechanically inherent of that position. The reader can experience this by protracting their shoulders forward and then attempting to reach for an item placed far above their head.

The head, neck and shoulder posture of the Parkinson's patient is linked to changes in mandibular position and, subsequently, changes in masticatory function. With extension of the occiput, the mouth opens and the mandible retracts. An attempt at closure of the mouth in this position will result in an increased tension of the supra and infra-hyoid muscles. Owing to the intimate association of the scapula and omohyoid, scapular position will also influence the length tension relationship of the hyoid muscles.[4]

With changes in the mandibular position and the length tension relationship of the hyoids, the occlusal contact pattern, the *arthromechanics* of the temporomandibular joint, and the position of the hyoid are also altered.[4] The posterior position of the mandible in the condyle causes the superior head of the lateral pterygoid becomes stretched. This can lead to an anterior displacement of the disc. [4] Elevation of the hyoid bone may cause the tongue to assume a lower position and result in abnormal swallowing patterns.[4] [25] Abnormal swallowing patterns will then contribute to the drooling that is often associated with changes in the autonomic nervous system nuclei.[2] [16]

Impairments in the musculoskeletal and cardiopulmonary systems have been termed as indirect effects of Parkinson's disease, because their manifestations are removed from the primary Central nervous system disorder.

Composite Impairments

The composite impairments of Parkinson's disease represent a complex fabric of interrelating factors.

Composite impairments represent impairments in which the direct and indirect effects of the disease each play a role as contributing factors to the overall morbidity of the patient.

Bradykinesia is classically noted as a direct effect of Parkinson's disease.[2] It also has been proposed that *bradykinesia* is compounded by the severe musculoskeletal impairments as mentioned earlier in this paper. Clinically, it seems clear that with a loss of mobility, there would be an associated loss of movement. Therefore the impairment of *bradykinesia* may actually be part of a cycle of loss of movement - loss of mobility. This cycle then would serve to increase the severity of *bradykinesia* through increases in musculoskeletal impairments. This would then promote the notion of *bradykinesia* as a composite effect of Parkinson's disease.

As the indirect effects of Parkinson's disease are compounded by the impairments of the Central Nervous System, (direct effects) they can be defined as being composite in origin. Many of the "composite effects" of Parkinson's disease were previously mentioned as sequelae to the "indirect effects" of the disease. As mentioned earlier, drooling can be considered as a composite effect. It has been postulated that drooling results from an increase in the production of salvia caused by a disturbance in the function of the autonomic nervous system.[2] It also seems

evident, as noted earlier, that drooling is partially the result of the classic Parkinsonian forward head posture which results in poor mouth closure.

Faulty balance is also an example of a composite effect of Parkinson's disease. Faulty balance can be attributed directly to losses in the Central Nervous System, as well as losses in the musculoskeletal system.[2]

As disability begins to overcome the Parkinson's patient, it too can be a source of further impairment. For instance, the emotional disabilities such as depression and withdrawal may serve to further compound the physical impairment of *bradykinesia*.[2] As evidenced by the complex interaction of symptoms, the presentation of a patient with Parkinson's disease can vary widely among individuals at different stages of the disease.[2]

The Hoehn and Yahr Classification of Disability [3 21]

In 1967 MM Hoehn and MD Yahr devised a system of classification for the disability of Parkinson's disease. The classification is comprised of five stages with stage one as the least involved and stage five holding the greatest disability.

Stage One: A relative absence of disability, unilateral if present at all.

Stage Two: Minimal disability bilaterally or at midline. Balance is not impaired.

Stage Three: Righting reflexes impaired. Unsteadiness when turning or rising from a chair. Disability restricting some activities, but patient can maintain independent living as well as continue some forms of employment.

Stage Four: All symptoms present as severe. Patient requires some assistance with activities of daily living.

Stage Five: Bound to a bed or wheelchair unless aided.

Posture

Definition

Horak (1987) depicts posture as being a dynamic flow of movement resulting in the maintenance of an equilibrated stance against gravity. He notes a complex interaction of muscles that enable a being to continuously shift their center of gravity over their base of support.[27] While the notion of posture as being dynamic is also supported by Roaf (1977),[28] this author chose to study posture in a static state. Observation of static Posture enables an observer to obtain more objective data. Also,the notion of the neuro-developmental sequence of mobility based on stability supports a training program involving static posture.

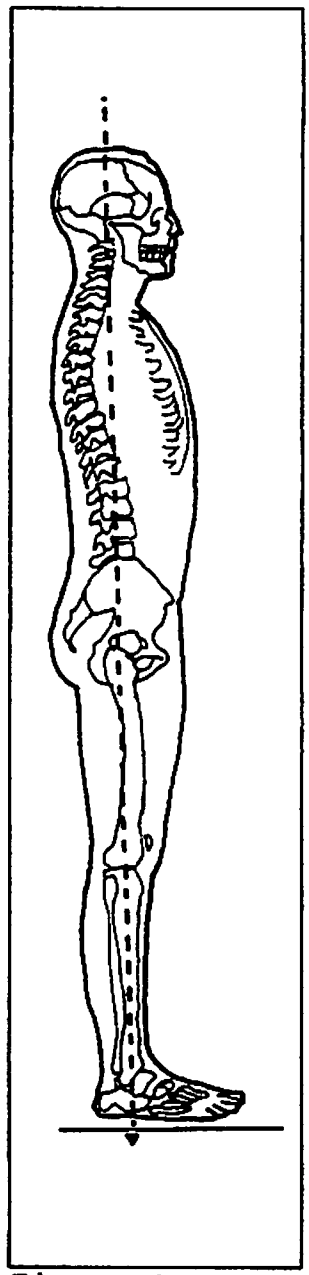

Figure 4

Anatomically, posture in the transverse plane has been described by Kendall, and McCreary. It was described in reference to a *plumb line* that bisects the external auditory meatus. It then travels to bisect the acromion and greater trochanter. It continues slightly anterior to the lateral condyle of the knee, and lateral malleolus. It ends at the foot with the bisection of the calcaneal-cuboid joint.[26] (FIG 4)

This stance requires the least amount of muscular energy to be maintained.

Study Of Posture

With regard to the evaluation of posture, Horak emphasizes the importance of quantitative measures. He advocated the use of sensitive computer equipment, as well as the use of photographs, grids for anatomical reference, video cameras, scales and rulers.[27]

Posture as a Motor Skill

Robert Roaf (1977), observed that posture is a motor skill. He postulated that, as with any skilled movement, the process of learning is one of conditioned response.[28] Basically; the close association of different inputs, followed by reinforcement, relate to make posture an integrated motor skill. These inputs that inform the body on postural status and awareness are *proprioception, exteroception,* vision and body image, to name a few.

Mental Practice

Mental practice has been described as the practice of physical activity with mental images. In a study of the effects of mental practice on balance, it was shown that improvement of balance was significant with the use of mental practice in conjunction with physical practice.[29] As noted by Garfield and Bennet (1984), "clear mental rehearsals act as neuromuscular templates or models, precisely directing your thought and feelings." Garfield and Bennet further state that, "These mental

rehearsals help you keep focused in your efforts maximizing the chances of achieving your goals in the shortest period of time with a minimal output of energy."[30] This author strongly believes that this form of training closely approximates the type of training that direct visualization is able to afford the patient. In the same manner that mental practice provides input of the proper body image through a "perfect" model, the technique of visualization used in this study makes use os an image of what actually exists and has the patient identify and correct the problem.

In further review of the literature, Warner (1988) suggests that mental practice is of the most benefit when administered over time. She goes on to state that optimal results are attainable only after a minimum of five sessions on separate days. [31]

Visual Input and Parkinson's Disease

Within the normal population, concrete visual image may play an important role in learning, but research suggests, that visual input plays an increasingly important role with a Parkinsonian population. As noted by various sources, a Parkinson's patient, normally suffering from absence of movement (akinesia), can perform certain motor activities with appropriate visual cues.[3 32] This is exemplified by clinical example of the akinetic Parkinson's patient who is only able to initiate gait by dropping a handkerchief in his path. The handkerchief provides the patient with a perceived obstacle that initiates an impulse to step over. This visual input (the handkerchief) provides information to the

brain that enables the patient to initiate movement. [3] [32] Animal studies have noted a similar reliance on visual cues in monkeys with lesions of the Globus Pallidus. The study had shown a severe impairment of motor control of the subjects upper limb when that limb had been shielded from their view.[33] In 1978 a similar study using individuals with Parkinson's disease had been reported.[34] In this study, patients were asked to perform two types of preset tracking movements with their forearm. Both sets of movements consisted of tracking between two fixed points with no mechanical stops. One being what was termed "step tracking." With step tracking, the pattern of movement was abrupt, and it changed between the points every two seconds. During the second type of movement, the patient was asked to follow tracking movements that were continuously moving between the two target points. The speeds were preset for trials at 13 degrees per second and six-point-five degrees per second. The research showed that when the study group was allowed to see their arm position, they scored comparably well as contrasted with age matched controls. A slight drift toward flexion was noted for the experimental group during step tracking. However, when sight of the moving limb was obstructed, the flexion drift was exaggerated with the experimental group in all trials. In further contrast, the control group continued to do well in all trials. Quantitatively, the overall error by the end of the trial with obstructed vision was 10 - 20 degrees, with the error occurring at a rate of approximately 0.3 - 0.5 degrees per second. The study concluded that, with visual input occluded, the

Parkinsonian patient found difficulty detecting proprioceptive displacements, and that these displacements were well in excess of normal passive malorientation.

This author, has noted the importance of what has been termed "good posture," and has identified posture as a motor skill. Furthermore, it has been demonstrated that vision and mental imagery have been shown to increase the learning potential of a motor skill, especially when the latter is linked with physical practice. Also, this author has noted evidence that supports the theory that Parkinson patients have an increased dependance on visual input to accurately execute a motor skill. It is, therefore, fond that a review of the literature suggests that visualization will aid in teaching Parkinson patients a motor skill such as posture.

Chapter III — Methods

Introduction:

The aim of this research was to determine whether direct, spontaneous visualization would be a helpful tool for a person with Parkinson's disease during the acquisition of a motor skill. The motor skill taken into consideration for this study was static, standing posture. The research was completed at the Parkinson's Therapy Center, Smithtown, N.Y.. Fifteen subjects volunteered for the project. Two people, subsequently did not finish. No incident was associated with the non completion of those subjects.

Photographic Quantification of Posture:

To record changes in the posture of the patients, a Nikon FM2 35mm camera with a 50mm lens with a "posture grid attachment" was used. The camera, lens and grid ensemble remained constant and unaltered from the start of the experiment to the finish. The posture grid attachment is a device designed by this author. When confronted with the current methods of photographic quantification of posture, they were found to be both prohibitively large and expensive. So as to decrease cost, and be able to provide a simple method of obtaining photographic quantification of posture, this author found it necessary to design an on-film measurement device.

The solution to the problem was conceived from a notion of modifying the camera in some way so that it may have some sort of built in marking system. After some careful consideration, the camera was modified by placing a "grid" at the *film plane* of the camera. In this way, the grid would be superimposed on each photograph and each box would represent a constant value; provided that the subject-to-lens and floor to lens distances remained the same and the camera was level for each exposure. To this researcher's knowledge, there was no device available that could be placed at the *film plane* for the purposes of on-film measurement. The medium best suited for the manufacture of the grid was Ektachrome 200T slide film. This is a color transparency film that is well suited to take exposures under indoor lighting conditions. Once the slides were developed, it would be possible to trim the *sprocket runners* off of the film and scotch tape the film at the *film plane*, between the film guides. (FIG. 5)

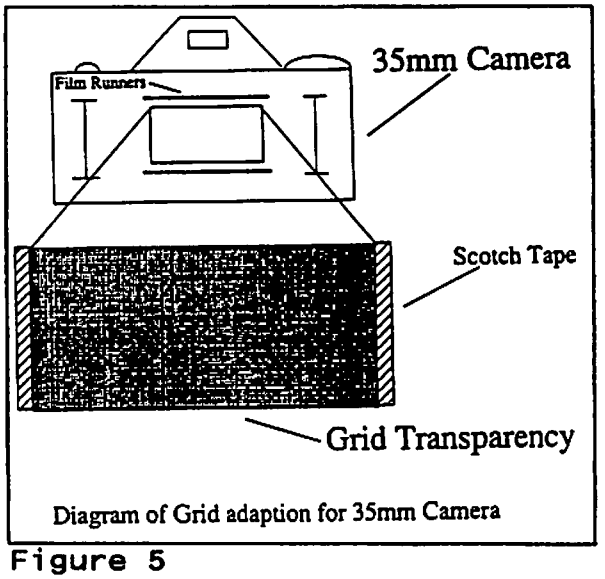

Figure 5

Exposures of ordinary graph paper yielded marginal results. The "photo blue" color used in common graph paper photographed entirely too light and was not adequate for the purpose. A graph with black lines and red cross hairs was created using an AUTO CAD (Computer Assisted Drawing) program and a pen plotter. The graph was then photographed and the resulting transparency was inserted into the camera as described above.

Test exposures were performed with Kodak VR100 print film to ascertain the optimal exposure and clearest *aperture*. The *aperture* of maximum clarity was determined to be f11. The exposure test yielded that no adjustment in exposure was needed to compensate for the grid. (See discussion.)

Calibration exposures were then taken to determine the value of each square of the grid when the distance from the lens to the subject was fixed at 400cm (4 Meters). Photographs of a ruler were taken with Ektachrome slide film at ruler-to-lens distances of 60cm, 110cm, 130cm, 140cm, and 150cm respectively. The photographs were then *digitized* into an AMIGA 2000 computer using the DIGI-VIEW software package and a Chinon color video camera. The resulting images were then digitally analyzed using the Deluxe Paint III software package. The following results were then tabulated:

Ruler-to-lens distance	Resulting Measure Per Box
60cm	5.64mm
110cm	11.28mm
120cm	11.95mm
130cm	12.70mm
140cm	13.97mm
150cm	15.24mm

Calculations were then performed to determine the size of each box at a Subject-to-lens distance of 400cm (4 Meters). The calculation used to make this determination was derived from the ratios of each Ruler-to-lens distance and result. This ratio was then compared to a ratio of the 400cm subject-to-lens measurement, and the box size at that distance (the unknown). An example of this would be the calculation of the first measurement, 60cm.

$$\frac{60cm}{5.64mm} = \frac{400cm}{X}$$

This equation then reduced to: (60cm)(X) = (400cm)(5.64mm) or
(400cm)(5.64mm)/60cm = X
X = 37.60mm

The results from subsequent calculations yielded the following results:

(400cm)(11.28mm)/110cm = 41.02mm
(400cm)(11.95mm)/120cm = 39.83mm
(400cm)(12.70mm)/130cm = 39.07mm
(400cm)(13.97mm)/140cm = 39.91mm
(400cm)(15.24mm)/150cm = 40.64mm

The Average for these calculated values was 39.68mm.

The standard deviation for these values was 1.22mm.

For the purposes of this study, however, values were rounded to the nearest millimeter. Therefore, each box of the grid measures 40mm, plus or minus 1mm at a subject-to-lens distance of 400cm(4 Meters).

Introduction of Research to Subjects:

Upon the approval of the Parkinson's Therapy Center, the topic of the study was revealed to the patients of that facility. Copies of the consent form (Appendix I) were then circulated among the group and a full explanation of the study was provided to all perspective subjects. Prior to the start of the study, fifteen subjects had confirmed their willingness to participate. A signed consent form was obtained from each participant. Each consent form was then assigned a number that corresponded to the order in which they were returned. Patients were then assigned to one of three groups by comparing the numbers on their consent form to the numbers on a random number table. After being assigned to a group, the patient received an identification code. (Appendix II) From then on, the names of the participants were not associated with the study. The first of the three groups was the Vision group. The people in this group, received instruction and exercises geared at the promotion of physiologically efficient posture. In addition, the patients would also be afforded the opportunity to view themselves, in the sagittal plane, throughout the session. This spontaneous visual feedback was provided through the use of a video camera placed four meters from the subject's right shoulder and a 64cm video monitor placed at eye level, two meters in front of the subject. The second group was the NON-Vision group. The subjects of this group were provided with the same exercise/instruction

regime, but were not afforded the spontaneous visual feedback. The final group was the Control group. This group was not given any treatment. They were evaluated at the beginning and end of the study only.

Graciously, the Parkinson's Center, was able to provide me with a room in which to conduct my study. The room had an adequate amount of space and was fully air conditioned. Before the first session, the following markings were placed on the floor with adhesive tape: (FIG. 6)

1) Two lines of tape were placed 60cm and 70cm, respectively, away from; and parallel to the west wall of the room. These taped lines represented where the patients stood during the evaluative photographs and training sessions.

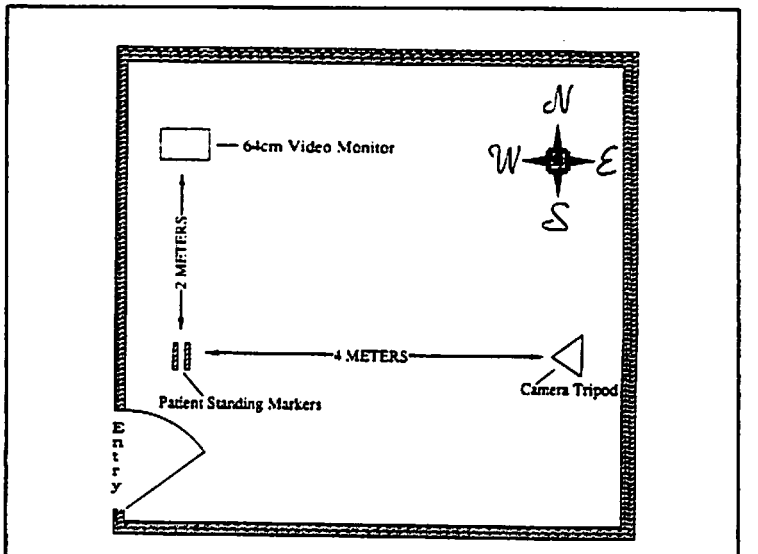

Diagram Representing the Setup of the Room in which the Study was Conducted (Dimensions of the ROOM are Approximate, Quoted Dimensions are Accurate)

Figure 6

2) Another piece of tape was placed 2 meters north of the standing markers and parallel to the north wall of the room. This register was to mark the location of the video monitor. The video screen measured 64cm diagonally and was on a stand that brought the center of the screen 110cm from the floor.

3) Another marker was placed 4 meters east of the standing markers. This point marked the location of the tripod. The tripod was modified so that a 35mm camera could be mounted vertically while at the same time, allowing the attachment of a video camera. (FIG. 7) The tripod brought the lens of the 35mm camera to 110cm above the floor. A *plumb line* and a level were used to insure the parallel relationship between the bottom of the *film plane* and the floor.

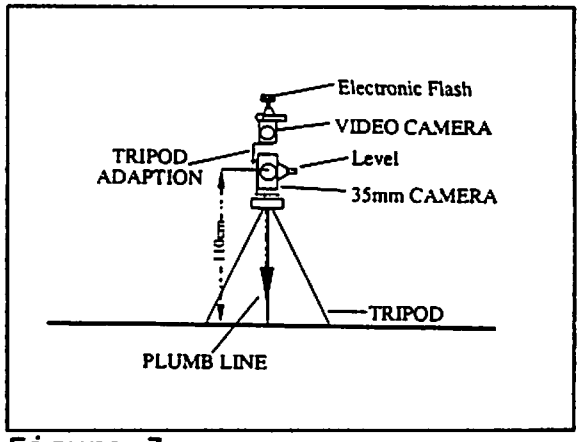

Figure 7

Description of the Subjects:

The age range of the thirteen subjects who completed the study was from 63 to 82 years old, with an average age being 71.4 years. The standard deviation was seven years. The amount of time between the onset of symptoms and the study ranged from 1 year to 8 years, with an average of 4 years and a standard deviation of 2 years. All subjects in the study were on *Sinimet*. Using the Hoehn and Yahr scale, all subjects, with the exception of two, exhibited the characteristics of either a grade two or grade three disability. While two subjects of the control group exhibited the greater disability of grade four. The male to female ratio among the group as a whole was 69% male and 31% female. Each patient was screened for past medical history that would preclude them from the study.

The age range of the Vision group alone was from 67 to 75 years with an average of 73.6 years and a standard deviation of 6.3 years. The time elapsed since onset of symptoms ranged from 3 to 5 years with an average of 4 years and a standard deviation of 1 year. The sex ratio of this group was 80% male and 20% female.

The age range of the Non-Vision group was from 63 to 82 years with an average of 72.75 years and a standard deviation of 8.4 years. The time elapsed since onset of symptoms ranged from 1 to 8 years with an average of 3 years and a standard deviation of 3.4 years. The sex ratio of this group was 75% male and 25% female.

The age range of the Control group was from 64 to 77 years with an average of 71.4 years and a standard deviation of 7 years. The time elapsed since onset of symptoms ranged from 4 to 6 years with an average of 5.25 years and a standard deviation of .96 years. The sex ratio of this group was 50% male and 50% female.

The Experiment:

To begin the first session, a pre-study photograph was taken of each of the subject individually. The subjects of the control group were released after these photographs and were not requested back until the completion of the study. The subjects of the vision and non-vision groups were asked to remain after their photographs were taken for an exercise/training session. The session lasted for five minutes and that time was standardized with the use of a kitchen timer. The only difference between the vision sessions and non-vision sessions was the use of the video camera for direct visualization by the vision group. All other aspects of the training session were held constant. After each session, a post-session photograph was taken of each person. This procedure was repeated every other day for a total of five sessions. All five sessions were attended by all subjects of the experimental groups with the exception of one person in the vision group, and one person in the non-vision group.

The exposure of the film was held constant at $1/200^{th}$ of a second and f11 with the use of an electronic flash unit.

Explanation of the Exercises Used in the Study.

Postural Awareness Exercises - Postural Awareness Exercises began with an explanation of physiologically efficient posture and it's benefits. The exercise continued by introducing the patient to the extremes of poor posture. This was accomplished by having the patient purposefully move from physiologically efficient posture

to a flexed and then extended posture. Verbal acknowledgement was given each time the proper alignment was attained. For the vision group, this was accompanied by an immediate visual image of what was considered correct and what was not.

Bilateral, Symmetrical D2 Flexion, is a movement pattern that is employed by the Proprioceptive Neuromuscular Facilitative treatment technique of Voss, Ionta, and Myers[35] - The starting position for bilateral, symmetrical D2 flexion was accomplished by having the patient hold both shoulders and elbows in extension, adduction, and internal rotation. The wrists were held in flexion. The exercise continued by having the patient slowly move in a diagonal pattern of shoulder and elbow flexion, abduction, and external rotation, with wrist extension.[35] This exercise was also accompanied by a deep inspiratory effort during the diagonal pattern. The theory behind the use of this exercise was to enhance thoracic expansion as well as to facilitate the lengthening of the anterior trunk musculature and facilitate thoracic extension. For the vision group, this also provided the further opportunity to see the extremes of posture as well as the chance to refine the mechanics of the exercise through visual reinforcement.

Shoulder Shrugs - Shoulder shrugs were executed by having the patient hold both upper extremities at their side. The shoulders were then rolled anteriorly, then superiorly, followed by posteriorly, finally inferiorly to anteriorly, to complete a

circle of glenohumeral motion in the saggital plane. Deep breathing was also used to enhance thoracic expansion during the "posterior" phase of the activity. Again, the theory behind this was to enhance thoracic expansion as well as to facilitate the lengthening of the anterior trunk musculature and encourage thoracic extension.

Bilateral Pectoral Stretch - The bilateral pectoral stretch was accomplished by having the patient touch both of their hands behind their head. The patient was then asked to bring their elbows in line with their shoulders by moving them out to the side. As they did, they were instructed to pinch their scapulae together, extend their spine and hold the position for three seconds and then relax. The theory behind this was to enhance thoracic expansion as well as to facilitate the lengthening of the anterior trunk musculature and encourage thoracic extension.

Alternate Lateral Stretching - For this activity, the patient was asked to sit on a chair and hold the seat of the chair with one hand and laterally flex the neck and the trunk to the opposite side. This activity was completed bilaterally. The theory behind this was to enhance lateral flexion of the trunk and neck.

Specifics of Each Session:

Session One:

Subjects from the control group each had one pre-study photograph taken and were released. After the pre-session photograph, the experimental groups were asked to complete the following regime:

1) Three minutes of postural awareness exercises.
2) Two minutes of Bilateral, Symmetrical D2 Flexion with Deep breathing.

Session Two:

Pre- and post-session photographs were taken of all subjects in the experimental groups. The session consisted of:

1) Two minutes of postural awareness exercises.
2) 1.5 minutes of Bilateral, Symmetrical D2 Flexion with Deep breathing.
3) 1.5 minutes of Shoulder Shrugs.

Sessions Three, Four and Five:

Pre- and post-session photographs were taken of all subjects in the experimental groups. The sessions consisted of:

1) One minute of postural awareness exercises.
2) 1.5 minutes of Bilateral, Symmetrical D2 Flexion with Deep breathing.
3) 1.5 minutes of bilateral Pectoral stretching exercises.
4) One minute of alternate lateral stretching.

Chapter IV - Presentation of Data

Introduction:

After the assignment of each subject to one of the three groups, they were given an ID code. The subjects of the Vision group were assigned a code with a "V" prefix. The ID codes for this group were: V1, V2, V3, V4, V5. The data from this group is tabulated in appendix III. The subjects of the Non-Vision group were given an ID code with an "N" prefix. The ID codes for this group were: N1, N2, N3, N4. The data from this group is tabulated in appendix IV. Members of the control group received an ID with the prefix "C". The ID codes for this group were: C1, C2, C3, C4. The data from this group is tabulated in appendix V.

Photographs of each person in the two experimental groups were taken before and after each treatment session, as well as one week after the last treatment session. Members of the control group were only photographed at the beginning and the end of the study. Each photograph was then assigned an ID code that reflected the subject in the photograph, the treatment session number, and whether it was a pre-treatment or a post-treatment exposure. (ie V1-Rx4-Pre) The abbreviation "Rx" was used as a prefix to the treatment session number. The words "Pre" and "Post" were then used to denote weather it was a pre-treatment or a post-treatment exposure. The photographs taken one week after the last session were given the word "Final" as an ID code extension. For example, if the first

person of the vision group were to have their picture taken *before* the *fourth* treatment session; it would have the code V1-Rx4-Pre. The last photograph of the study for this patient would be termed: V1-RxFinal.

Interpretation of the Data:

After all of the photographs were developed, they were *digitized* into an AMIGA 2000 computer by using the DIGI-VIEW software package. Measurements were then taken from the photographs by using the DELUXE PAINT III software package. The distance from the external auditory meatus to the next posterior grid line was taken in radians.(FIG. 8a.) A measurement of the grid box that contained the external auditory meatus was then performed in radians.(FIG. 8b.) The measurement of the meatus was then divided by the measure of the entire box in which it was contained.(a/b) This quantity was then multiplied by the real scale measure of the grid box. (4cm) The next measure was taken from the apex of the thoracic kyphosis to the next anterior line, (FIG. 8c.) and is reported in radians. A measurement of the grid box that contained the apex of the thoracic kyphosis was then performed in radians.(FIG. 8d.) The measurement of the thoracic kyphosis was then divided by the measure of the entire box in which it was contained. (c/d) This quantity was then multiplied by the real scale measure of the grid box. (4cm) The amount of boxes was then counted from the line posterior to the meatus and the line anterior to the kyphosis. (FIG. 8e.) This quantity was then multiplied by the real scale measure of the grid box. (4cm) The results of these

Figure 8

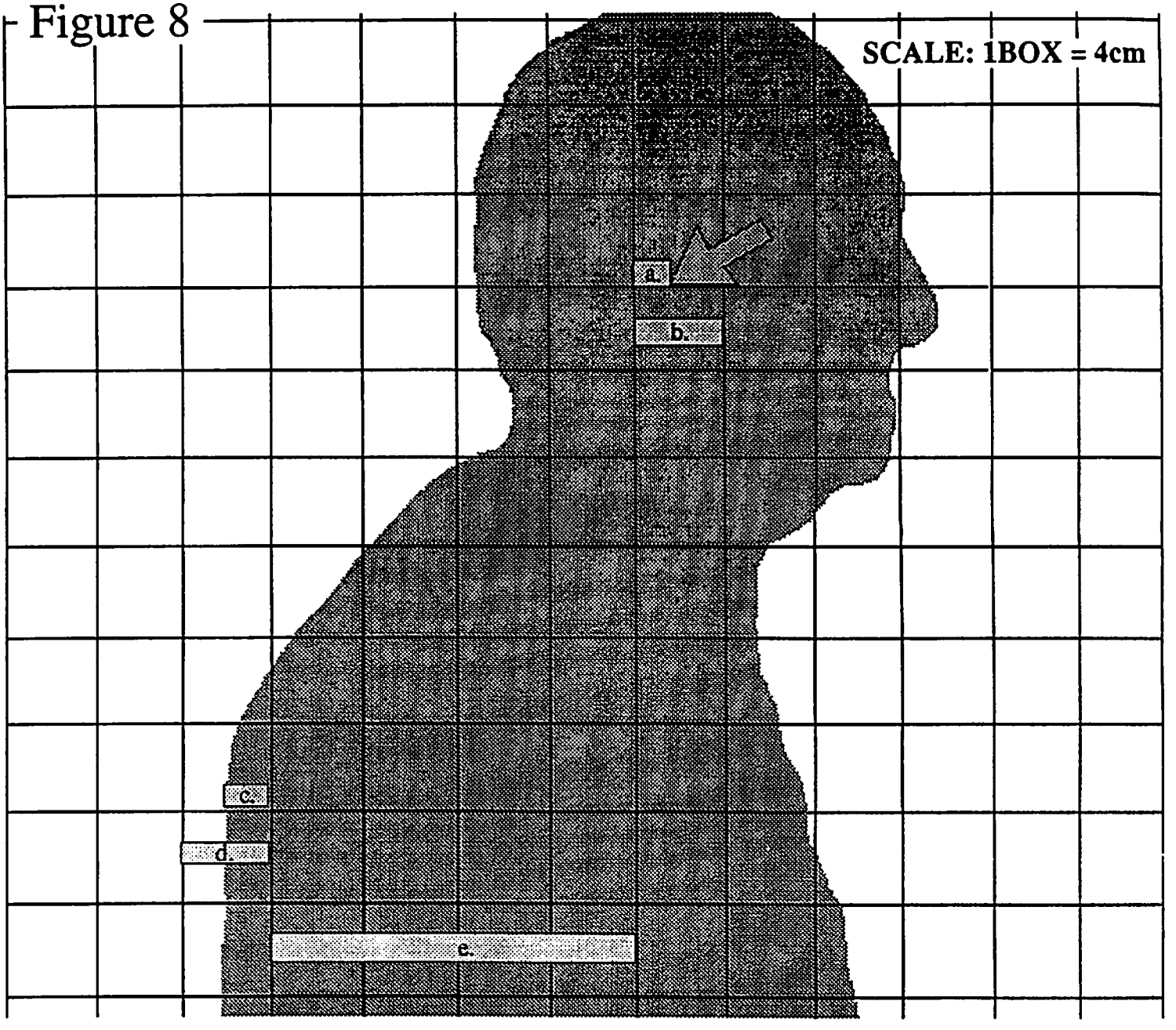

Diagram representing data acquisition photograph and measurement system.

The arrow represents the external auditory meatus.
a. represents the distance from the external auditory meatus to the next posterior grid line.
b. represents the size of the grid box that contains the external auditory meatus.
a./b. represents the decimal equivalent of a. as it relates to the size of b.
(a./b.)*4cm = the real measure equivalent of a.
c. represents the distance from the apex of the thoracic kyphosis to the next anterior grid line.
d. represents the size of the grid box that contains the apex of the thoracic kyphosis.
c./d. represents the decimal equivelant of c. as it relates to the size of d.
(c./d.)*4cm = the real measure equivelant of c.
e. represents the number of grid boxes between a. and c
(e.)*(4cm) = the real measure equivelent of e..
The equation used to derive the total forward head posture complex is:
((a./b.)*(4cm)) + ((c./d.)*(4cm) + ((e.)*(4cm)) = FHP complex (in cm)

calculations were then added together and the result represented the distance from the external auditory meatus to the apex of the thoracic kyphosis, in centimeters. This distance was then considered to represent the forward head posture measurement for that patient at that moment in time. In mathematical form, the equation would be: ((a./b.)*(4cm)) + ((c./d.)*(4cm)) + ((added boxes)*(4cm)) = FHP measurement.

Results

This section has been divided by first defining the null hypothesis that each section is addressing. The statistical trials for each section is tabulated in the appendices. To analyze null hypothesis number one (appendices VI & VII), a matched pair comparison was made to the Student's "t" distribution by computing the direct difference method to find "t." To analyze null hypotheses numbers two through six (appendices VIII & XIII), a comparison was made to the Student's "t" distribution by using the raw score formula for small or unequal samples from different populations. Each hypothesis was tested at the .100 level of significance.[36]

Null Hypothesis Number One:

"There is no change in forward head posture between the post-treatment evaluation and the pre-treatment evaluation, with regard to each subject individually." This hypothesis was tested for each subject in each group. The results revealed an acceptance of the null hypothesis in all but three cases. A significant change was noted at the .100 level of significance in two of the subjects of

the VISION group, and one subject of the NON-VISION group. The statistical analyses are tabulated in appendix VI and VII, respectively.

Null Hypothesis Number Two:

"There is no difference between the VISION and the CONTROL groups, with regard to changes in forward head posture from the post-treatment evaluation to the pre-treatment evaluation." After this hypothesis was tested statistically at the .100 level of significance (Appendix VIII), it was concluded that the null hypothesis is correct. There is no evidence to suggest that a program of verbal instruction with spontaneous visual feedback is more or less effective at the immediate improvement of the FHP of Parkinson's patients than no treatment at all.

Null Hypothesis Number Three:

"There is no difference between the VISION and the NON-VISION groups, with regard to changes in forward head posture from the post-treatment evaluation to the pre-treatment evaluation." After this hypothesis was tested statistically at the .100 level of significance (Appendix IX), it was concluded that the null hypothesis is correct. There is no evidence to suggest that a program of verbal instruction with spontaneous visual feedback is more or less effective at the immediate improvement of the FHP of Parkinson's patients than a program of verbal instruction alone.

Null Hypothesis Number Four:

"There is no difference between the NON-VISION and the CONTROL groups, with regard to changes in forward head posture from the post-treatment evaluation to the pre-treatment evaluation." After

this hypothesis was tested statistically at the .100 level of significance (Appendix X), it was concluded that the null hypothesis is correct. There is no evidence to suggest that a program of verbal instruction is more or less effective at the immediate improvement of the FHP of Parkinson's patients than no treatment at all.

Null Hypothesis Number Five:

"There is no difference between the VISION and the CONTROL groups, with regard to changes in forward head posture from the initial (first pre-treatment) evaluation to the final evaluation." After this hypothesis was tested statistically at the .100 level of significance (Appendix XI), it was concluded that the null hypothesis is correct. There is no evidence to suggest that a program of verbal instruction with spontaneous visual feedback is more or less effective at the long term improvement of the FHP of Parkinson's patients than no treatment at all.No significance was shown at the .100 confidence interval.

Null Hypothesis Number Six:

"There is no difference between the VISION and the NON-VISION groups, with regard to changes in forward head posture from the initial (first pre-treatment) evaluation to the final evaluation." After this hypothesis was tested statistically (Appendix XII), it was concluded that the null hypothesis is correct. There is no evidence to suggest that a program of verbal instruction with spontaneous visual feedback is more or less effective at the long term improvement of the FHP of Parkinson's patients than a program of verbal instruction alone.

Null Hypothesis Number Seven:

"There is no difference between the NON-VISION and the CONTROL groups, with regard to changes in forward head posture from the initial (first pre-treatment) evaluation to the final evaluation." After this hypothesis was tested statistically at the .100 level of significance,(Appendix XIII), it was concluded that the null hypothesis is correct. There is no evidence to suggest that a program of verbal instruction is more or less effective at the long term improvement of the FHP of Parkinson's patients than no treatment at all.

Conclusion:

Immediate improvement of forward head posture was determined by a decrease in the FHP measurement between an evaluative photograph taken just after each treatment to an evaluative photograph taken just before each treatment session. When considering immediate improvement within each group, significance was demonstrated by two of the five subjects of the VISION group, and one of the four subjects of the NON-VISION group. However, when each group as a whole was compared to the other no significance of this parameter was demonstrated.

Long term improvement of forward head posture was determined by a decrease in the FHP measurement between an evaluative photograph taken one week after the conclusion of the study to an evaluative photograph taken just before the first treatment session. No significant improvement of was noted after comparing the changes of this measure between all the groups of the study.

Chapter V – Discussion

Calibration of the Posture Grid:

One of the first problems of the study was encountered when assessing the test exposures of the posture grid. The photographs were taken on Kodak VR 100 Print film. When the photographs were analyzed it was determined that no exposure correction was needed to account for the density of the grid at the *film plane.* This however, was not the case. Since the test exposures were taken on Print film, exposure correction was made during the processing of the film. This resulted in a misleading representation of the correct exposure. When the experiment was done on slide film, no exposure compensation was made during processing. Subsequently, each exposure was under exposed by one f stop due to the density of the grid material at the film plane. Owing to the fact that all of the film used during the experiment was developed at once, the exposure error was carried through out the entire experiment. This error, however presented as little more than an annoyance during the analysis of the data and had little effect on the accuracy of the measurements.

Discussion of Significance of the Study

Within Groups
Immediate Change (Appendix VI & VII)

Of the five subjects in the VISION group, two showed significant (.10 CI) average improvement in FHP from the pre-evaluation to the post-evaluation photograph over the five treatment sessions. There seemed to be no correlation between this improvement and the amount of years since diagnosis or the stage of the disease. One of the subjects had been diagnosed three years prior to the study and was at a stage two disability while the other subject had been diagnosed 4 years prior to the study and was at a stage three disability. Both subjects attended all five treatment sessions.

Of the four subjects in the NON-VISION group, one showed significant (.10 CI) average improvement in FHP from the pre-evaluation to the post-evaluation photograph over the five treatment sessions. This person had been diagnosed two years prior to the study and was noted to be at a stage three disability. This subject also attended all five treatment sessions.

Between Groups
Immediate Change (Appendix VIII, IX & X)
Long Term Change (Appendix XI, XII & XIII)

No significance was shown for the immediate change in FHP when the CONTROL group was compared to the VISION group. Likewise, significance failed to be exhibited when the CONTROL group was

compared to the NON-VISION group. Nor was any significance shown when the VISION group was compared to the NON-VISION group. An identical lack of significance was shown by statistical tests performed between groups for long term changes in FHP.

Postulations and Recommendations:

The significant improvement of three subjects in the study, two from the VISION group and one from the NON-VISION group, did not seem to correlate with the experimental group to which they were assigned, nor did it correlate with the stage of the disease, and amount of years since diagnosis. This significance is most likely due to factors other than the variable of visualization vs. non-visualization.

Overall, the study yielded no significance. Limitations of the study may be attributed to either population errors or experimental errors. Population errors are problems that were unique to the experimental population. This researcher had little or no control over these errors. Experimental errors, on the other hand, are errors in the design or execution of the study. Future researchers can learn from the errors made during this study and perhaps develop a more complete design that would test the hypothesis more thoroughly.

Population Errors

Parkinson's disease is a progressive disorder with a great variety of manifestations. There is an immense amount of variability of disease manifestations among patients, making it difficult to quantify all of the component parts of the pathology. Therefore, it may be possible that the experimental condition is applicable only to patients exhibiting a specific combination of disease manifestations.

By the nature of the disease and the drugs used to combat it's symptoms, variability of the disease's manifestations exist within each person on a day to day basis. Therefore, it is possible for the disease to have completely a different presentation for a given person over a short period of time. This is evidenced by a patient's reports of "good" and "bad" days. This characteristic of the disease makes Parkinson's a difficult disease to study. By the nature of this experiment, it was necessary to "freeze" a few isolated moments out of each of these patient's lives. Realistically, there was no objective way to tell whether that moment was a "good" moment or a "bad" moment. An attempt was made, however to complete each subject's treatment session at approximately the same time each day, thus serving to lessen the effect of cyclic variations in the disease and it's drug treatment.

As Parkinson's disease progresses, the classic forward head posture becomes more fixed in nature. Perhaps with this population, a program designed to effect chronic changes in muscle length would have been in order. Such a program would have included

mobility and stretching exercises for longer sessions over a longer period of time.

Experimental Errors

One of the first problems encountered in this experiment was the small sample size. It is unlikely that a group of thirteen people, divided into three groups, is representative of the entire population of Parkinson's disease patients.

A further complication with the choice of sample was the variety in the stages of the disease as dictated by Hoehn and Yahr.[3 22] The subjects of this study varied from stage two to stage four. Perhaps this treatment technique is best received by patients exhibiting a certain stage of disability. A small sample representing a scattering of patients from each stage of the disease would be incapable of detecting that information. Therefore this study might be best done on a homologous sample as it pertains to stages of the disease.

The amount of treatment sessions, as well as the duration of each session, may have been problematic as well. Perhaps more repetition or increased amount of actual exercise time would have been able to show whether there was a true difference existed between the two experimental regimes.

The most serious implication of the study lies in the fact that, not only was there no difference between the two experimental groups, but there was also no difference when those groups were compared to the control group. In other words, the improvement of each experimental group is very similar to one in

which, no treatment was being rendered over the same *period* of time. This implies that the training regime used in the two experimental conditions was ineffective at improving posture, irrespective of the variables being tested. Perhaps a set of exercises that are inherently better suited toward the improvement of forward head posture, would better test the basic hypothesis of this study.

A lack standardization to the exercise regime may have also contributed to the overall ineffectiveness of the program. Perhaps if the exercises were read from a script each time that they were given, it would have provided a reliable method of continuity to each exercise session, thus adding to the consistency of the study.

It would be presumptuous to conclude that the posture of Parkinson's patients was unalterable. On the other hand, although the exercise program used in this study may have been inadequate, and the duration of the program may have been insufficient, there may be aspects of the Parkinson's patient's posture that is fixed and unalterable.

Conclusion

Since the sequela of Parkinson's disease are so severe and relentless, ways to prevent or retard their effects should continue to be explored. Currently, the technology to arrest the disease is unavailable, therefore a strong emphasis on improving the quality of life of these patients is essential. It is the hope of this researcher that, research continue in the area of Parkinson's disease, specifically with regard to "the propensity to lean forward." This common sequela could be considered the catalyst that sets many of the profoundly disabling effects of Parkinson's disease in motion.

Definition of Related Terms

Words in featured in this section are presented in *italics* through out the text.

Agonist - The muscle directly engaged in contraction about a specific joint,as distinguished from the muscles that oppose it.[5]

Alpha motor neurons - Cells found in the ventral horn of the spinal cord. The axons of these cells innervate skeletal muscle.[5]

Antagonist - IN RELATION TO MUSCLE: The muscle directly opposing the agonist. It is the muscle that must relax in order to allow the agonist to move the joint.
IN RELATION TO A DRUG: A drug in direct opposition to some action. A drug that has the ability to block a property is said to be the antagonistic drug of that property. [5]

Aperture - An opening or hole. In relation to photography, it is the opening behind the lens that can be changed to alter the amount of light that strikes the film plane.

Arthromechanics - Pertaining to the component movements at joints.

Autogenic inhibition - Inhibition that is produced within the organism being inhibited.

Basal Ganglia-thalamic-cortical circuit - Refers to the neuronal connection of the Basal Ganglia to the thalamus and the thalamus to the cerebral cortex.

Bradykinesia - Extreme slowness of movement. [3]

Cortical - Pertaining to the cortex, as referenced to in the text, the cerebral cortex.

Corticospinal tract - Referring to the neuronal connection between the cerebral cortex and the spinal cord.

Diencephalon - The portion of brain lying between the telencephalon and mesencephalon. It includes the epithalamus, thalamus, metathalamus, and hypothalamus. [5]

Digitization - A process in which an image is broken down into minute parts then reassembled by computer.

Dopamine antagonists - Drugs that block the action of dopamine

Dopamine - An inhibitory neurotransmitter secreted by neurons that are located in the Substantia Nigra and terminate in the striate region of the Basal Ganglia. [3]

Eosinophilic - Term given to structures that are receptive to the acidic dye eosin, in microscopic preparation.[37]

Exteroception - Pertaining to the reception of input from without, by specially adapted end organs. [5]

Festination - Abnormal and involuntary increase in speed of walking in an attempt to catch up with a displaced center of gravity due to the patient's leaning forward. [5]

Film Plane - A plane that is behind and parallel to the lens of a camera. It is the plane to which light emanating from the lens is converged and a resulting image focused. It is the plane against which the film is placed in order to record an image.

Golgi tendon organ - A sensory structure located in tendon with type Ib axons that is sensitive to tension. Increases in this structures discharge rate occur from an increase in tension. [7]

Hertz (Hz) - A unit of frequency equal to one cycle per second

Inclusion - Being enclosed or included

Intracytoplasmic - Within the cytoplasm

Lemniscus - A bundle of sensory fibers in the medulla and pons. [5]

Mesolimbic - Goes from Midbrain to Limbic structures

Peduncle - 1. A stem or stalk
2. A brachium of the brain, a band connecting parts of the brain

Plumb line - A line hung vertically with a weight, free from obstruction, hanging at it's end. It is used as an indicator of a plane that is perpendicular to the ground surface.

Proprioception - The awareness of posture, movement and changes in equilibrium and the knowledge of position, weight and resistance of objects in relation to the body. [5]

Putamen - The darker outer layer of the lenticular nucleus [5]

Red nucleus - Gray matter in the dorsal portion of the cruri cerebri of the midbrain (the tegmentum). [5]

Rigidity - Muscle stiffness or Hypertonia; Sustained contraction of muscle resulting in an inability to bend or be bent. [3]

Rostral - Toward the front or cephalic end of the body.[5]

Schizophrenia - A group of mental disorders that include psychotic features [5]

Sensorimotor cortex - Part of the cerebral cortex receiving both sensory and motor input

Sinimet - A drug used in the treatment of Parkinson's disease to raise the striatal level of dopamine in the Basal Ganglia. It is a combination of levodopa and carbidopa. [3]

Spondylogenic - Originating from the vertebrae. [5]

Sprocket runners - The perforated top and bottom of a piece of 35mm film. Designed to keep film in register at the film plane.

Subcortical - Below, or inferior to, the cortex

Tardive Dyskinesia - A disease characterized by choreiform movements buccal-lingual-facial muscles, less commonly the extremities. [18]

Telencephalon - The embryonic endbrain or posterior division of the prosencephalon from which the cerebral hemispheres, corpora striata, and rhinencephalon develop. [5]

Thalamic - Pertaining to the thalamus

Thoracic Outlet Syndrome - A group of symptoms characterized by brachial neuropathy with or with out vascular disturbance to the upper extremity. [5]

Vital capacity - Volume of air that can be expelled after full inspiration. [5]

Zygoapophyseal joint - Articulation of the processes at the neural arch of the vertebrae. [5]

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APPENDICES

I.	Consent Form
II.	Subject Code Table
III.	DATA: Vision Group
IV.	DATA: Non–Vision Group
V.	DATA: Control
VI.	Matched Pair Statistics: Vision
VII.	Matched Pair Statistics: Non–Vision
VIII.	Immediate Change: Vision Vs. Control
IX.	Immediate Change: Vision Vs. Non–Vision
X.	Immediate Change: Non–Vision Vs.Control
XI.	Long Term Change: Vision Vs. Control
XII.	Long Term Change: Vision Vs. Non–Vision
XIII.	Long Term Change: Non–Vision Vs. Control

APPENDIX I
CONSENT FORM

I,_____understand that I am participating, on a volunteer basis, in research being conducted by Russell A. L'HommeDieu, a physical therapy student at Touro College. I have been notified that the research is aimed at the improvement of the posture of people with Parkinson's disease. I have be informed of the procedure and realize that my photograph will be taken several times. I understand that my ability to stand independently has been determined, and that the experiment involves little physical contact by the researcher. I have been told that I will be asked to stand erect for approximately 5 minutes, and that the major risk is loss of balance. I have also been told that if my condition were to change, or if I experience any unusual mishaps, I should inform the researcher immediately.

I understand that I am under no obligation to follow the study through to its completion, and that I may withdraw this consent at anytime. I also understand that Russ L'HommeDieu has no obligation to me and cannot be held responsible for any injury incurred during the research period.

I allow Russ L'HommeDieu to keep, interpret, and use the results of this study under the provision that my name will not be associated with it. I have been given the right to ask questions about the study, and have them answered for me.

I have read the above statement, or have had it read to me. I have received a copy and understand its content. I therefore freely put my signature here on this day the ____ of _____,19__.

_____ witness' signature

_____ patient's signature

_____ researcher's signature

APPENDIX II

The arrangement of the completed consent forms into three groups with the use of a random number table

Table showing assignment of consent form numbers to groups and their subsequent subject ID codes.

Vision	Non-Vision	Control
07-V1	04-N1	01-C1
14-V2	10-N2	08-C2
15-V3	12-N3	11-C3
05-V4	03-N4	06-C4
13-V5	09 DISCONTINUED	02 DISCONTINUED

Appendix III - DATA: VISION

	Ear(a.) (Radians)	Box size(b.) (Radians)	Thorax(c.) (Radians)	Box size(d.) (Radians)	Added Boxes(e.) (Radians)	Result (cm)
V1-RX1-PRE	6.00	20.00	13.00	20.00	3.00	15.80
V1-RX1-POST	10.00	20.00	0.00	20.00	4.00	18.00
V1-RX2-PRE	18.00	20.00	16.00	20.00	3.00	18.80
V1-RX2-POST	12.00	20.00	6.00	20.00	4.00	19.60
V1-RX3-PRE	17.00	20.00	0.00	20.00	4.00	19.40
V1-RX3-POST	0.00	20.00	0.00	20.00	5.00	20.00
V1-RX4-PRE	4.00	20.00	13.00	20.00	4.00	19.40
V1-RX4-POST	4.00	20.00	15.00	20.00	3.00	15.80
V1-RXFINAL	20.00	50.00	0.00	20.00	5.00	21.60
V2-RX1-PRE	4.00	19.00	9.00	19.00	3.00	14.74
V2-RX1-POST	7.00	20.00	6.00	20.00	2.00	10.60
V2-RX2-PRE	18.00	20.00	14.00	21.00	2.00	14.27
V2-RX2-POST	14.00	20.00	15.00	20.00	2.00	13.80
V2-RX3-PRE	6.00	19.00	8.00	20.00	3.00	14.86
V2-RX3-POST	7.00	20.00	9.00	20.00	3.00	15.20
V2-RX4-PRE	12.00	20.00	17.00	20.00	2.00	13.80
V2-RX4-POST	5.00	19.00	15.00	20.00	2.00	12.05
V2-RX5-PRE	15.00	21.00	0.00	21.00	3.00	14.86
V2-RX5-POST	7.00	20.00	17.00	20.00	3.00	16.80
V2-RXFINAL	11.00	20.00	14.00	20.00	2.00	13.00
V3-RX1-PRE	15.00	20.00	16.00	19.00	4.00	22.37
V3-RX1-POST	7.00	20.00	11.00	20.00	4.00	19.60
V3-RX2-PRE	11.00	20.00	4.00	20.00	5.00	23.00
V3-RX2-POST	10.00	20.00	14.00	19.00	4.00	20.95
V3-RX3-PRE	8.00	21.00	16.00	20.00	4.00	20.72
V3-RX3-POST	6.00	20.00	16.00	20.00	4.00	20.40
V3-RX4-PRE	6.00	19.00	0.00	19.00	5.00	21.26
V3-RX4-POST	8.00	20.00	17.00	19.00	4.00	21.18
V3-RX5-PRE	0.00	20.00	15.00	20.00	5.00	23.00
V3-RX5-POST	7.00	20.00	4.00	20.00	5.00	22.20
V3-RXFINAL	10.00	19.00	0.00	20.00	5.00	22.11
V4-RX1-PRE	21.00	40.00	33.00	40.00	3.00	17.40
V4-RX1-POST	3.00	40.00	22.00	40.00	4.00	18.50
V4-RX2-PRE	17.00	38.00	33.00	38.00	3.00	17.26
V4-RX2-POST	15.00	40.00	26.00	40.00	3.00	16.10
V4-RX3-PRE	13.00	39.00	28.00	39.00	3.00	16.21
V4-RX3-POST	5.00	39.00	24.00	40.00	3.00	14.91
V4-RX4-PRE	17.00	37.00	32.00	39.00	3.00	17.12
V4-RX4-POST	24.00	39.00	13.00	39.00	3.00	15.79
V4-RX5-PRE	33.00	39.00	6.00	38.00	3.00	16.02
V4-RX5-POST	4.00	39.00	0.00	39.00	4.00	16.41
V4-RXFINAL	29.00	40.00	14.00	40.00	3.00	16.30
V5-RX1-PRE	28.00	39.00	0.00	39.00	4.00	18.87
V5-RX1-POST	16.00	38.00	13.00	40.00	4.00	18.98
V5-RX2-PRE	11.00	39.00	0.00	39.00	5.00	21.13
V5-RX2-POST	19.00	38.00	26.00	40.00	4.00	20.60
V5-RX3-PRE	16.00	39.00	0.00	39.00	5.00	21.64
V5-RX3-POST	0.00	39.00	0.00	39.00	5.00	20.00
V5-RX4-PRE	22.00	38.00	28.00	41.00	4.00	21.05
V5-RX4-POST	20.00	38.00	12.00	39.00	4.00	19.34
V5-RX5-PRE	28.00	39.00	25.00	40.00	4.00	21.37
V5-RX5-POST	20.00	40.00	19.00	39.00	4.00	19.95
V5-RXFINAL	29.00	40.00	21.00	39.00	4.00	21.05

Appendix IV - DATA: NON-VISION

	Ear(a.) (Radians)	Box size(b.) (Radians)	Thorax(c.) (Radians)	Box size(d.) (Radians)	Added Boxes(e.) (Radians)	Result (cm)
N1-RX1-PRE	34.00	39.00	30.00	39.00	3.00	18.56
N1-RX1-POST	28.00	39.00	33.00	39.00	3.00	18.26
N1-RX2-PRE	24.00	38.00	30.00	38.00	3.00	17.68
N1-RX2-POST	29.00	38.00	33.00	39.00	3.00	18.44
N1-RX3-PRE	30.00	39.00	0.00	39.00	4.00	19.08
N1-RX3-POST	32.00	38.00	27.00	39.00	3.00	18.14
N1-RX4-PRE	5.00	39.00	18.00	39.00	4.00	18.36
N1-RX4-POST	27.00	39.00	33.00	39.00	3.00	18.15
N1-RX5-PRE	7.00	39.00	20.00	39.00	4.00	18.77
N1-RX5-POST	19.00	38.00	0.00	38.00	4.00	18.00
N1-RXFINAL	6.00	39.00	15.00	39.00	4.00	18.15
N2-RX1-PRE	22.00	38.00	0.00	38.00	5.00	22.32
N2-RX1-POST	0.00	38.00	28.00	38.00	4.00	18.95
N2-RX2-PRE	22.00	36.00	20.00	39.00	4.00	20.50
N2-RX2-POST	24.00	35.00	27.00	39.00	4.00	21.51
N2-RX3-PRE	7.00	36.00	0.00	39.00	5.00	20.78
N2-RX3-POST	22.00	37.00	19.00	36.00	5.00	24.49
N2-RX4-PRE	28.00	35.00	13.00	39.00	4.00	20.53
N2-RX4-POST	30.00	36.00	23.00	38.00	4.00	21.75
N2-RX5-PRE	17.00	37.00	12.00	36.00	5.00	23.17
N2-RX5-POST	10.00	36.00	0.00	36.00	5.00	21.11
N2-RXFINAL	1.00	36.00	15.00	38.00	5.00	21.69
N3-RX1-PRE	28.00	37.00	9.00	37.00	3.00	16.00
N3-RX1-POST	28.00	37.00	14.00	38.00	3.00	16.50
N3-RX2-PRE	13.00	35.00	7.00	36.00	4.00	18.26
N3-RX2-POST	15.00	36.00	31.00	37.00	3.00	17.02
N3-RX3-PRE	14.00	36.00	30.00	35.00	3.00	16.98
N3-RX3-POST	28.00	37.00	17.00	35.00	3.00	16.97
N3-RX4-PRE	23.00	36.00	32.00	36.00	3.00	18.11
N3-RX4-POST	22.00	39.00	30.00	37.00	3.00	17.50
N3-RXFINAL	10.00	36.00	12.00	36.00	3.00	14.44
N4-RX1-PRE	23.00	35.00	30.00	36.00	3.00	17.96
N4-RX1-POST	0.00	35.00	0.00	35.00	4.00	16.00
N4-RX2-PRE	19.00	36.00	12.00	38.00	4.00	19.37
N4-RX2-POST	0.00	37.00	32.00	37.00	3.00	15.46
N4-RX3-PRE	25.00	36.00	0.00	36.00	4.00	18.78
N4-RX3-POST	26.00	36.00	0.00	36.00	3.00	14.89
N4-RX4-PRE	33.00	37.00	28.00	37.00	3.00	18.59
N4-RX4-POST	31.00	37.00	15.00	37.00	3.00	16.97
N4-RX5-PRE	0.00	37.00	19.00	37.00	4.00	18.05
N4-RX5-POST	18.00	36.00	32.00	37.00	3.00	17.46
N4-RXFINAL	18.00	36.00	29.00	37.00	4.00	21.14

Appendix V - DATA: CONTROL

	Ear(a.) (Radians)	Box size(b.) (Radians)	Thorax(c.) (Radians)	Box size(d.) (Radians)	Added Boxes(e.) (Radians)	Result (cm)
C1-PRE	17.00	52.00	0.00	52.00	5.00	21.31
C1-POST	28.00	37.00	10.00	37.00	4.00	20.11
C2-PRE	11.00	36.00	12.00	37.00	4.00	18.52
C2-POST	15.00	50.00	38.00	50.00	3.00	16.24
C3-PRE	8.00	36.00	18.00	37.00	3.00	14.83
C3-POST	15.00	37.00	9.00	35.00	3.00	14.65
C4-PRE	21.00	36.00	20.00	38.00	5.00	24.44
C4-POST	17.00	37.00	0.00	36.00	8.00	33.84

Appendix VI - VISION: MATCHED PAIR STATISTICS

Null Hypothesis: POST-evaluation = PRE-evaluation
Null Hypothesis Tested at .10 Confidence Interval

	PRE (cm)	POST (cm)	POST - PRE (cm)	:SQUARED	
V1-RX1	15.80	18.00	2.20	4.84	
V1-RX2	18.80	19.60	0.80	0.64	
V1-RX3	19.40	20.00	0.60	0.36	
V1-RX4	19.40	15.80	-3.60	12.96	
N	4.00	4.00	4.00	4.00	
SUM			0.00	18.80	
AVERAGE	18.35	18.35	0.00		
SD2	6.27				
SD(bar)	1.25				
				T-Score	0.00 Null Hypothesis
				Df	3 Not Rejected
V2-RX1	14.74	10.60	-4.14	17.14	
V2-RX2	14.27	13.80	-0.47	0.22	
V2-RX3	14.86	15.20	0.34	0.12	
V2-RX4	13.80	12.05	-1.75	3.06	
V2-RX5	14.86	16.80	1.94	3.76	
N	5.00	5.00	5.00	5.00	
SUM			-4.08	24.30	
AVERAGE	14.51	13.69	-0.82		
SD2	5.24				
SD(bar)	1.02				
				T-Score	-0.80 Null Hypothesis
				Df	4 Not Rejected
V3-RX1	22.37	19.60	-2.77	7.67	
V3-RX2	23.00	20.95	-2.05	4.20	
V3-RX3	20.72	20.40	-0.32	0.10	
V3-RX4	21.26	21.18	-0.08	0.00	
V3-RX5	23.00	22.20	-0.80	0.64	
N	5.00	5.00	5.00	5.00	
SUM			-6.02	12.62	
AVERAGE	22.07	20.87	-1.20		
SD2	1.34				
SD(bar)	0.52				
				T-Score	-2.32 Reject Null Hypothesis,
				Df	4 *Improvement Noted*
V4-RX1	17.40	18.50	1.10	1.21	
V4-RX2	17.26	16.10	-1.16	1.35	
V4-RX3	16.21	14.91	-1.30	1.69	
V4-RX4	17.12	15.79	-1.33	1.77	
V4-RX5	16.02	16.41	0.39	0.15	
N	5.00	5.00	5.00	5.00	
SUM			-2.30	6.17	
AVERAGE	16.80	16.34	-0.46		
SD2	1.28				
SD(bar)	0.51				
				T-Score	-0.91 Null Hypothesis
				Df	4 Not Rejected
V5-RX1	18.87	18.98	0.11	0.01	
V5-RX2	21.13	20.60	-0.53	0.28	
V5-RX3	21.64	20.00	-1.64	2.69	
V5-RX4	21.05	19.34	-1.71	2.92	
V5-RX5	21.37	19.95	-1.42	2.02	
N	5.00	5.00	5.00	5.00	
SUM			-5.19	7.92	
AVERAGE	20.81	19.77	-1.04		
SD2	0.63				
SD(bar)	0.36				
				T-Score	-2.92 Reject Null Hypothesis,
				Df	4 *Improvement Noted*

Appendix VII - NON-VISION: MATCHED PAIR STATISTICS

Null Hypothesis: POST-evaluation = PRE-evaluation
Null Hypothesis Tested at .10 Confidence Interval

	PRE (cm)	POST (cm)	POST - PRE (cm)	:SQUARED		
N1-RX1	18.56	18.26	-0.3	0.09		
N1-RX2	17.68	18.44	0.76	0.5776		
N1-RX3	19.08	18.14	-0.94	0.8836		
N1-RX4	18.36	18.15	-0.21	0.0441		
N1-RX5	18.77	18.00	-0.77	0.5929		
N	5.00	5.00	5.00	5.00		
SUM			-1.46	2.19		
AVERAGE	18.49	18.20	-0.29			
SD2	0.44					
SD(bar)	0.30					
				T-Score	-0.98	Null Hypothesis
				Df	4	Not Rejected
N2-RX1	22.32	18.95	-3.37	11.3569		
N2-RX2	20.50	21.51	1.01	1.0201		
N2-RX3	20.78	24.49	3.71	13.7641		
N2-RX4	20.53	21.75	1.22	1.4884		
N2-RX5	23.17	21.11	-2.06	4.2436		
N	5.00	5.00	5.00	5.00		
SUM			0.51	31.87		
AVERAGE	21.46	21.56	0.10			
SD2	7.96					
SD(bar)	1.26					
				T-Score	0.08	Null Hypothesis
				Df	4	Not Rejected
N3-RX1	16	16.5	0.5	0.25		
N3-RX2	18.26	17.02	-1.24	1.5376		
N3-RX3	16.98	16.97	-0.01	0.0001		
N3-RX4	18.11	17.50	-0.61	0.3721		
N	4.00	4.00	4.00	4.00		
SUM			-1.36	2.16		
AVERAGE	17.34	17.00	-0.34			
SD2	0.57					
SD(bar)	0.38					
				T-Score	-0.90	Null Hypothesis
				Df	3	Not Rejected
N4-RX1	17.96	16.00	-1.96	3.8416		
N4-RX2	19.37	15.46	-3.91	15.2881		
N4-RX3	18.78	14.89	-3.89	15.1321		
N4-RX4	18.59	16.97	-1.62	2.6244		
N4-RX5	18.05	17.46	-0.59	0.3481		
N	5.00	5.00	5.00	5.00		
SUM			-11.97	37.23		
AVERAGE	18.55	16.16	-2.39			
SD2	2.14					
SD(bar)	0.65					
				T-Score	-3.66	Reject Null Hypothesis,
				Df	4	*Improvement Noted*

APPENDIX VIII - VISION Vs. CONTROL: Immediate Change

Null Hypothesis: POST-evaluation - PRE-evaluation(NON-VISION) = POST-evaluation - PRE-evaluation (CONTROL)

Null Hypothesis Tested at .10 Confidence Interval

	POST - PRE (cm)		POST - PRE (cm)
V1-RX1	2.20	C1	-1.20
V1-RX2	0.80	C2	-2.28
V1-RX3	0.60	C3	-0.18
V1-RX4	-3.60	C4	9.40
V2-RX1	-4.14		
V2-RX2	-0.47		
V2-RX3	0.34		
V2-RX4	-1.75		
V2-RX5	1.94		
V3-RX1	-2.77		
V3-RX2	-2.05		
V3-RX3	-0.32		
V3-RX4	-0.08		
V3-RX5	-0.80		
V4-RX1	1.10		
V4-RX2	-1.16		
V4-RX3	-1.30		
V4-RX4	-1.33		
V4-RX5	0.39		
V5-RX1	0.11		
V5-RX2	-0.53		
V5-RX3	-1.64		
V5-RX4	-1.71		
V5-RX5	-1.42		
**********	**********		
SUM	-17.5900		5.7400
SUM SQUARED	309.4081		32.9476
SUM (X)SQUARED	69.82		95.03
AVERAGE	-0.73		1.43
ST.DEV	1.74		5.63
N	24.00		4.00
		S(pre-post	4.63
		T-SCORE	-0.47 Null Hypothesis
		Df	26.00 Not Rejected

APPENDIX IX - VISION Vs. NON-VISION: Immediate Change

Null Hypothesis: POST-evaluation - PRE-evaluation(NON-VISION) = POST-evaluation - PRE-evaluation (CONTROL)
Null Hypothesis Tested at .10 Confidence Interval

	POST - PRE (cm)		POST - PRE (cm)
V1-RX1	2.20	N1-RX1	-0.30
V1-RX2	0.80	N1-RX2	0.76
V1-RX3	0.60	N1-RX3	-0.94
V1-RX4	-3.60	N1-RX4	-0.21
V2-RX1	-4.14	N1-RX5	-0.77
V2-RX2	-0.47	N2-RX1	-3.37
V2-RX3	0.34	N2-RX2	1.01
V2-RX4	-1.75	N2-RX3	3.71
V2-RX5	1.94	N2-RX4	1.22
V3-RX1	-2.77	N2-RX5	-2.06
V3-RX2	-2.05	N3-RX1	0.50
V3-RX3	-0.32	N3-RX2	-1.24
V3-RX4	-0.08	N3-RX3	-0.01
V3-RX5	-0.80	N3-RX4	-0.61
V4-RX1	1.10	N4-RX1	-1.96
V4-RX2	-1.16	N4-RX2	-3.91
V4-RX3	-1.30	N4-RX3	-3.89
V4-RX4	-1.33	N4-RX4	-1.62
V4-RX5	0.39	N4-RX5	-0.59
V5-RX1	0.11		
V5-RX2	-0.53		
V5-RX3	-1.64		
V5-RX4	-1.71		
V5-RX5	-1.42		
SUM	-17.590		-14.280
SUM SQUARED	309.408		203.918
SUM (X)SQUARED	69.82		73.46
AVERAGE	-0.73		-0.75
ST.DEV	1.74		2.02
N	24.00		19.00
		S(pre-post)	2.60
		T-SCORE	0.00 Null Hypothesis
		Df	41.00 Not Rejected

Appendix X - NON-VISION Vs. CONTROL: Immediate Change

Null Hypothesis: POST-evaluation - PRE-evaluation(NON-VISION) = POST-evaluation - PRE-evaluation (CONTROL)

Null Hypothesis Tested at .10 Confidence Interval

	POST - PRE (cm)		POST - PRE (cm)		
N1-RX1	-0.30	C1	-1.20		
N1-RX2	0.76	C2	-2.28		
N1-RX3	-0.94	C3	-0.18		
N1-RX4	-0.21	C4	9.40		
N1-RX5	-0.77				
N2-RX1	-3.37				
N2-RX2	1.01				
N2-RX3	3.71				
N2-RX4	1.22				
N2-RX5	-2.06				
N3-RX1	0.50				
N3-RX2	-1.24				
N3-RX3	-0.01				
N3-RX4	-0.61				
N4-RX1	-1.96				
N4-RX2	-3.91				
N4-RX3	-3.89				
N4-RX4	-1.62				
N4-RX5	-0.59				
SUM	-14.2800		5.7400		
SUM SQUARED	203.9184		32.9476		
SUM (X)SQUARED	74.85		95.03		
AVERAGE	-0.75		1.43		
ST.DEV	2.04		5.63		
N	19.00		4.00		
			S(pre-post)	4.90	
			T-SCORE	-0.45	Null Hypothesis
			Df	21.00	Not Rejected

Appendix XI - VISION Vs. CONTROL: Long Term Change

THE NULL HYPOTHESIS:
There is no difference between the VISION GROUP and the CONTROL GROUP with regard to changes between the initial eval and the final eval
HO: AVERAGE INITAL - FINAL for VISION = AVERAGE INITIAL - FINAL for CONTROL
(CI .10)

DATA

	INITIAL EVAL (CM)	FINAL EVAL (CM)	TOTAL CHANGE (CM)
VISION: N=5.00			
SUBJECT 1	15.80	21.60	5.80
SUBJECT 2	14.74	13.00	-1.74
SUBJECT 3	22.37	22.11	-0.26
SUBJECT 4	17.40	16.30	-1.10
SUBJECT 5	18.87	21.05	2.18
CONTROL: N=4.00			
SUBJECT 1	21.31	20.11	-1.20
SUBJECT 2	18.52	16.24	-2.28
SUBJECT 3	14.83	14.65	-0.18
SUBJECT 4	24.44	33.84	9.40

STATISTICS

	TOTAL CHANGE: VISION (CM)	TOTAL CHANGE: CONTROL (CM)		
SUBJECT 1	5.80	-1.20		
SUBJECT 2	-1.74	-2.28		
SUBJECT 3	-0.26	-0.18		
SUBJECT 4	-1.10	9.40		
SUBJECT 5	2.18			
SUM(X)	4.88	5.74		
(SUM(X))SQUARED	23.81	32.95		
SUM((X)SQUARED	42.70	95.03		
AVERAGE	0.98	1.43		
ST.DEV	3.27	5.63		
N	5.00	4.00		
		S(pre-post)	5.03	
		T-SCORE	-0.09	Null Hypothesis
		Df	7.00	Not Rejected

Appendix XII - VISION Vs. NON-VISION: Long Term Change

THE NULL HYPOTHESIS:
There is no difference between the VISION GROUP and the NON-VISION GROUP with regard to changes between the initial eval and the final eval
NULL HYPOTHESIS: FINAL - INITIAL (VISION) = FINAL - INITIAL (NON-VISION)
Null Hypothesis Tested at .10 Confidence Interval
(CI .10)

DATA

	INITIAL EVAL (CM)	FINAL EVAL (CM)	TOTAL CHANGE (CM)
VISION: N=5.00			
SUBJECT 1	15.80	21.60	5.80
SUBJECT 2	14.74	13.00	-1.74
SUBJECT 3	22.37	22.11	-0.26
SUBJECT 4	17.40	16.30	-1.10
SUBJECT 5	18.87	21.05	2.18
NON-VISION: N=4.00			
SUBJECT 1	18.56	18.15	-0.41
SUBJECT 2	22.32	21.69	-0.63
SUBJECT 3	16.00	14.44	-1.56
SUBJECT 4	17.96	21.14	3.18
SUBJECT 5	24.44	33.84	9.40

STATISTICS

	TOTAL CHANGE: VISION (CM)	TOTAL CHANGE: NON-VISION (CM)		
SUBJECT 1	5.80	-0.41		
SUBJECT 2	-1.74	-0.63		
SUBJECT 3	-0.26	-1.56		
SUBJECT 4	-1.10	3.18		
SUBJECT 5	2.18	9.40		
**********	**********	**********		
SUM(X)	4.88	9.98		
(SUM(X))SQUARED	23.81	99.60		
SUM((X)SQUARED	42.70	101.47		
AVERAGE	0.98	2.00		
ST.DEV	3.27	5.04		
N	5.00	5.00		
		S(pre-post)	4.71	
		T-SCORE	-0.22	Null Hypothesis
		Df	8.00	Not Rejected

Appendix XIII - NON-VISION Vs. CONTROL: Long Term Change

THE NULL HYPOTHESIS:
There is no difference between the NON-VISION GROUP and the CONTROL GROUP with regard to changes between the initial eval and the final eval
NULL HYPOTHESIS: FINAL - INITIAL (VISION) = FINAL - INITIAL (NON-VISION)
Null Hypothesis Tested at .10 Confidence Interval
(CI .10)

DATA

	INITIAL EVAL (CM)	FINAL EVAL (CM)	TOTAL CHANGE (CM)
NON-VISION: N=4.00			
SUBJECT 1	18.56	18.15	-0.41
SUBJECT 2	22.32	21.69	-0.63
SUBJECT 3	16.00	14.44	-1.56
SUBJECT 4	17.96	21.14	3.18
CONTROL: N=3.00			
SUBJECT 1			
SUBJECT 2	21.31	20.11	-1.20
SUBJECT 3	18.52	16.24	-2.28
SUBJECT 4	14.83	14.65	-0.18
	24.44	33.84	9.40

STATISTICS

	TOTAL CHANGE: NON-VISION (CM)	TOTAL CHANGE: CONTROL (CM)		
SUBJECT 1	-0.41	-1.20		
SUBJECT 2	-0.63	-2.28		
SUBJECT 3	-1.56	-0.18		
SUBJECT 4	3.18	9.40		
**********	**********	**********		
SUM(X)	0.58	5.74		
(SUM(X))SQUARED	0.34	32.95		
SUM((X)SQUARED	13.11	95.03		
AVERAGE	0.15	1.43		
ST.DEV	2.09	5.63		
N	4.00	4.00		
		S(pre-post)	3.80	
		T-SCORE	-0.34	Null Hypothesis
		Df	6.00	Not Rejected