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HYPOTHESIS

Cranial osteopathic treatment in conjunction with exercise will produce an improvement in the balance of subjects with Parkinson's disease, as measured using the Berg Balance Scale. This difference will be greater ($p<.05$) than that of the control group using exercise only.

ABSTRACT

Parkinson's disease is the second leading neurodegenerative disease in the adult population in Canada. It is characterized by cardinal motor symptoms which include tremor, rigidity, bradykinesia and postural instability. Postural instability predisposes Parkinson's patients to an increased risk of falls. Falling in Parkinson's patients is associated with reduction in quality of life, expense to the individual and the healthcare system, and morbidity.

Postural instability in Parkinson's is now believed to be rooted within the basal ganglia in the central nervous system. The treatment of postural instability has thus far been limited to medication and physiotherapy. Cranial osteopathy, in that it focuses on the mobility of the cranial bones, may provide a unique, non-invasive, therapeutic alternative that could result in improvement in balance in Parkinson's patients.

The objective of this pilot study was to determine the effect of cranial osteopathic treatment on balance in patients with Parkinson's disease. Balance was measured using a functional performance test, known as the Berg Balance Scale (BBS).

The study design followed a single blind between-group design. A mixed gender group with ages ranging from 55- 82 with a diagnosis of idiopathic Parkinson's disease were studied. Subjects were randomly assigned to either a control or experimental group. The experimental group consisted of seven subjects and the control group consisted of four subjects with idiopathic Parkinson's disease. A power analysis determined that a sample size of n=16 was needed to have statistical significance on the Berg Balance Scale within this population.

The control group participated in a four week exercise program designed to increase stability and balance. The experimental group received four cranial osteopathic

treatments in addition to participating in the four week exercise program. A pre-test and post-test assessment using the Berg Balance Scale was administered on initiation and completion of the study at two levels of severity of Parkinson's as measured on the Hoehn and Yahr rating scale. To provide added insight, an osteopathic assessment of the severity of lesions was conducted.

The result of this preliminary study showed a greater improvement in the Berg Balance Scale score for the group receiving osteopathic treatment compared to the control group participants who received only exercise ($p=.028$). This result was not dependent on level of severity of Parkinson's as measured on the Hoehn and Yahr scale ($p=.87$).

Additionally, 17 of 38 variables measured pre-treatment and post-treatment on the lesion severity scale and 29 of 38 variables measured pre-treatment and post-treatment on the vitality scale showed improvement for the group receiving osteopathic treatment, while control group participants showed no change on either measure.

While the results are interpreted as promising for the use of osteopathic treatment in improving balance in Parkinson's patients, the research calls for caution in interpretation given the small sample size which did not meet the requirements of the power analysis. This paper concludes with a call for further research on the efficacy of cranial osteopathy in treatment of Parkinson's.

SOMMAIRE EN FRANCAIS

La maladie de Parkinson est la deuxième maladie neurodégénérative leader chez les adultes au Canada. Il se caractérise par des symptômes moteurs cardinales qui comprennent des tremblements, rigidité, bradykinésie ainsi qu'une instabilité posturale. Cette instabilité posturale prédispose les patients atteints de la maladie de Parkinson à un risque accru de chutes. Chutes chez les patients atteints de la maladie de Parkinson est associée à la morbidité, une réduction de la qualité de vie ainsi qu'un stress financière chez la personne et le système de soins de santé.

On suppose maintenant que la cause de l'instabilité posturale est enracinée dans les noyaux gris centrales du système nerveux central. Le traitement de l'instabilité posturale a jusqu'à présent été limité aux médicaments ainsi que la physiothérapie. L'ostéopathie crânienne, en ce qu'il se concentre sur la mobilité des os crâniens, peut-être fournir une alternative thérapeutique unique et non invasive, qui pourrait se traduire par une amélioration de l'équilibre chez les patients atteints de la maladie de Parkinson.

L'objectif de cette étude pilote était de déterminer l'effet du traitement d'ostéopathique crânien sur l'équilibre des patients atteints de la maladie de Parkinson. L'equilibre a été mesurée à l'aide d'un test de performance fonctionnelle : l'échelle d'équilibre de Berg.

L'etude a suivit une methodologie utilisant « single-blind » avec un groupe d'hommes et de femmes âges de 55-82 ans avec un diagnostic de la maladie de Parkinson idiopathique. Par tirage au sort, nous avons constitués un groupe contrôle et un groupe expérimental. Le groupe expérimental se composait de sept sujets et le groupe de contrôle se composait de quatre sujets atteints de la maladie de Parkinson idiopathique.

Une analyse a détermine qu'un pouvoir de n = 16 était nécessaire pour que l'étude aye une signification statistique.

Le groupe de contrôle a participé à un programme d'exercice pendant quatre semaines, qui a été conçu pour accroître la stabilité et l'équilibre. Le groupe expérimental a reçu quatre traitements d'ostéopathie crâniens en plus de participer au programme d'exercice. Une évaluation pré et post-test a été administre en utilisant l'échelle d'équilibre Berg. Ce test a été administrée a l'initiation et a l'achèvement de l'étude à deux niveaux de gravité de la maladie de Parkinson, mesurée par l'échelle de Hoehn et Yahr. Pour fournir de l'information supplémentaire, une evaluation de la gravité des lésions d'ostéopathie a été effectuée.

Le résultat de cette étude préliminaire a montré une grande amélioration dans les resultats du test de l'Echelle de Berg dans le groupe recevant les traitements ostéopathique comparé aux participants de groupe de contrôle (p =. 028) quelle que soit le niveau de gravité de la maladie de Parkinson, mesurée sur l'échelle de Hoehn et Yahr (p =. 87)

En outre, 17 de 38 variables mesurées pré et post traitement sur l'échelle de gravité de lésion et 29 de 38 variables mesurées pré et post traitement sur l'échelle de vitalité a montré une amélioration pour le groupe experimentale, tandis que il y avait aucun changement sur chaque mesure avec les sujets du groupe contrôle.

Bien que les résultats sont interprétés comme prometteur pour l'utilisation du traitement ostéopathique dans l'amélioration de l'équilibre chez les patients atteints de la maladie de Parkinson, la recherche appelle à la prudence dans l'interprétation des donnée, car la taille de l'échantillon ne respectait pas les exigences. Cet article se termine par un

appel de poursuivre les recherches sur l'efficacité de l'ostéopathie crânienne dans le traitement de la maladie de Parkinson.

TABLE OF CONTENTS

TABLE OF FIGURES

TABLE OF TABLES

1 CHAPTER ONE: INTRODUCTION

The objective of this research was to assess the effects of cranial osteopathy on balance in people with Parkinson's disease.

Parkinson's disease is the second leading neurodegenerative condition, second only to Alzheimers, in the adult population within Canada (Martin, Suchowersky, Kovacs Burns, & Jonsson, 2010). In Canada, approximately 100,000 people are living with Parkinson's disease. Although Parkinson's can affect anyone during their adult life, the incidence of Parkinson's disease increases with age. One in every 1000 people will develop Parkinson's, with that figure increasing to 1 in 100 between the ages of 60 and 80 (De Lau & Breteler, 2006).

This is of particular importance as the geriatric population in Canada continues to grow. Statistics Canada ("Population estimates and projections ", 2008) estimated that the population greater than sixty-five years of age will increase from 4.4 million today to almost 7 million by the year 2021. It has also been projected by Statistics Canada that between 1991 and 2016, there will be a 92% increase in the number of people over the age of 65 living with Parkinson's (Canada, 2011).

Parkinson's disease is clinically defined as a progressive disorder characterized by tremor, rigidity, bradykinesia and postural instability. Pathologically, there is neuronal loss within the substantia nigra of the basal ganglia, which affects dopamine production. (Martin, et al., 2010; Martinez-Martin, 1994; Nutt, Hammerstad, & Gancher, 1992). Parkinson's disease is characterized by an impairment of postural reflexes, thereby reducing stability and leading to disturbances in balance. Balance issues predispose Parkinson's patients to falling which threatens injury, immobility and loss of independence (Ashburn, Stack, Pickering, & Ward, 2001).

The risk of falling is a major concern for those living with Parkinson's disease. Ashburn et al. (2001) found incidence of falling in the subjects with Parkinson's disease was three times higher than that reported among the age matched control group. An analysis of six studies on falling in Parkinson's disease, determined that occurrence of falls in this population is between 40%-70% (Pickering et al., 2007). Subjects with Parkinson's that had fallen in the past 12 months were twice the proportion of that within the general population (Ashburn, et al., 2001).

Falls are associated with morbidity, reduced quality of life, mortality, and expense to healthcare systems and the individual. Falls in the general elderly population account for two thirds of all injury-related hospital admissions and three quarters of injury-related days of hospital care (Jaglal, Sherry, & Schatzker, 1996). The cost of caring for elderly patients due to falls is between $10 and $20 billion dollars (U.S.) per year (Tibbits, 1996).

One common result of falling is hip fractures, which leads to restrictions of mobility and activity, and eventually, to a loss of independence. In Parkinson's, hip fractures due to falling occur more frequently. One study concluded that Parkinson's patients suffered 66% more hip fractures compared to healthy controls (Johnell, Melton, Atkinson, O'Fallon, & Kurland, 1992). Taggart and Crawford (1995) found a 10% lower bone mineral density in Parkinson's subjects compared to healthy age matched controls. U.S. statistics estimated that hip fractures due to falls cost $7.3 billion U.S. in 1989 (Jaglal, et al., 1996). Canada reported an annual cost of $650 million from hip fracture implications with an estimated rise to $2.4 billion by 2041 (Wiktorowicz, Goeree, Papaioannou, Adachi, & Papadimitropoulos, 2001).

Balance control, which was once thought of as a single system of a fixed set of reflexes, is now seen as a complex motor skill derived from the interaction of many sensorimotor processes. This evolution of knowledge has changed our understanding of postural instability in Parkinson's disease. There is now a strong alternative view that postural instability in Parkinson's disease is not a result of dysfunctional peripheral reflexes, but rather is caused by neuronal loss within the basal ganglia.

Cranial osteopathy is a unique form of treatment because of its believed effects on the central nervous system. It is for this reason that this study endeavored to assess the effects of cranial osteopathy on Parkinson's disease. The following study was designed as a single blinded between-group study. The original study proposal intended to employ a fully powered study of 16 subjects. However, despite considerable efforts and time, a full sample was not possible to generate. With the permission of the thesis committee's chair, Jane Stark, DOMP, this thesis was re-titled as a pilot study.

The Berg Balance scale was employed to assess the balance of Parkinson's subjects pre-intervention and post-intervention. Both the control and experimental groups participated in an exercise program prescribed by the Parkinson's Society of Canada and administered by an independent physiotherapist. In addition, the experimental group also received four cranial osteopathic treatments. The results of this study demonstrated a positive effect of cranial osteopathy on balance in Parkinson's patients. However, one must caution that the power was small and the groups uneven. Thus these positive results would indicate further study is warranted.

2 CHAPTER TWO: LITERATURE REVIEW

Balance can be defined as the ability to maintain the center of gravity over the base of support. One of the roles of the basal ganglia is to maintain the neurons within the motor cortex in a state of readiness for action. This enables the postural muscles to engage in order to maintain the center of gravity during movement. In Parkinson's disease, the inability to maintain balance is thought to be rooted in these motor cortex neurons. The dysfunction of these neurons disrupts anticipatory postural muscles by decreasing the timing and size of muscle activation (Smithson, Morris, & Iansek, 1998)

2.1 DIAGNOSIS OF IDIOPATHIC PARKINSON'S DISEASE

There is currently no reliable test to diagnose Parkinson's disease. Instead, the diagnosis of Parkinson's disease is primarily based on clinical symptoms. The patient must present with at least two of the following symptoms: bradykinesia, resting tremor, rigidity and postural instability. The onset of symptoms appears asymmetric, presenting in one limb and spreading to the other limb unilaterally. When they are available, PET and CT scans are used to determine a pathological diagnosis which includes degeneration of the substantia nigra and the presence of Lewy bodies within the substantia nigra. However, these diagnostic tests are not always readily available and therefore it is common to prescribe levodopa if Parkinson's disease is suspected (De Lau & Breteler, 2006). A positive response to this medication is thought to be a verification of diagnosis.

The Lewy body is an eosinophilic cytoplasmic inclusion body that is found within remaining neurons of effected nuclei (Nutt, 1992). They differ in size and shape depending on their location however all contain a filamentous cytoskeleton with a dense eosinophilic core and a surrounding halo (Forno, 1996). Lewy bodies are thought to form when substantial cellular degradation is present and there is an accumulation of protein

(Jenner & Olanow, 1998). Although Lewy bodies have been studied extensively there is no consensus as to their role in Parkinson's disease. One possible theory is that they may aid in the removal of damaged protein (Martin, et al., 2010).

The presence of Lewy bodies is not limited to Parkinson's disease as they appear in other disorders involving neuronal loss. However the distribution of Lewy bodies is specific to Parkinson's disease. Lewy bodies are associated with Parkinson's disease when found within the substantia nigra, the hypothalamus, and the mesolimbic and mesocortical pathways. More specifically, Lewy bodies are present in the dorsal motor nucleus of the vagus, the hypothalamus, the nucleus basalis of Meynert (NBM), the locus ceruleus (LC), the Edinger-Westphal nucleus in the midbrain, the raphe nuclei, cerebral cortex, and autonomic ganglia (Jager, Hartog, & Bethlem, 1960).

Parkinsonism is a description of symptoms and although idiopathic Parkinson's disease is the primary cause there are other conditions that cause Parkinsonism. Other conditions that are considered when determining diagnosis include: essential tremor, multiple system atrophy, progressive supranuclear palsy, cortico-basal ganglionic degeneration, dementia with Lewy bodies, and vascular Parkinson's (Martin, et al., 2010).

A report of the Quality Standards Subcommittee of the American Academy of Neurology concluded that if any of a number of clinical features appear at early onset a diagnosis of idiopathic Parkinson's disease is unlikely. The clinical features include: falls during initial appearance of symptoms, poor response to levodopa, symmetrical clinical symptoms, rapid progression, lack of tremor, or dysautonomia (Suchowersky, Reich, et al., 2006).

2.2 ETIOLOGY OF PARKINSON'S DISEASE

Although the prevalence of Parkinson's disease is extensive, the causes are still relatively unknown. Current popular theory is that Parkinson's disease is not caused by one single factor but rather by a multitude of factors. The following is a review of some of the most prevalent theories.

2.2.1 GENETICS

A monogenetic cause has been widely refuted in recent years. Although a variety of mutated genes have been discovered, they only account for 10% of Parkinson's cases (Hardy, Cookson, & Singleton, 2003). However, a genetic predisposition is widely accepted as a high risk factor for Parkinson's disease. Various studies have been conducted on familial etiology of Parkinson's disease, and there is an accepted theory that some genes, in combination with environmental factors make individuals more susceptible to Parkinson's (De Lau & Breteler, 2006).

Thirteen genetic loci have been identified in Parkinson's disease (De Lau & Breteler, 2006). These genetic loci are considered to be Mendelian, meaning that Parkinson's disease results from changes to a single gene that is inherited from the previous generation. One example of this is the PARK1 gene that encodes a particular pre-synaptic protein that can be neurotoxic if not formed properly (Gwinn-Hardy, 2002).

In conjunction with these thirteen genetic loci, five areas have been identified to be associated with susceptibility to Parkinson's disease. These include chromosomes 5,6,8,9 and 17 (Scott et al., 2001).

In the future, genetics may play a role in early intervention, or perhaps pre-symptomatic neuroprotective treatment (Gwinn-Hardy, 2002).

2.2.2 ENVIROMENTAL/NEUROTOXINS

Support for the neurotoxic theory was promoted in 1983 when a group of heroin users injected a synthetic form of heroin that caused Parkinsonism. An extensive study on these patients concluded that the causative substance was 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) (Langston & Ballard, 1984). The pathological examination showed destruction of the dopamine neurons of the substantia nigra. All of the subjects progressed into an idiopathic Parkinson's state within 10 years of initial onset (Parkes, 1986).

MPTP is unique in the specificity of its destructive actions which differs from other toxic exposure. Other toxic substances cause degeneration throughout the brain with slightly varying symptoms. MPTP also differs in its responsiveness to treatment as seen with levodopa treatment. A lower dosage of levodopa, produces stronger and more prevalent side effects. Another, differing factor from idiopathic Parkinson's disease is the absence of Lewy bodies.

MPTP studies on primates have replicated the destruction of the nigro-striatal dopamine system. They also displayed all the cardinal signs of idiopathic Parkinson's disease, as well as effectively responding to levodopa. Administering MPTP to rodents also produced destruction of the nigro-striatal system, but without corresponding motor impairments (Javitch, D'Amato, Strittmatter, & Snyder, 1985; Parkes, 1986). These studies weren't meant to prove the hypothesis of toxicity as a causative factor in Parkinson's disease. However, this toxicity hypothesis has been refuted because it doesn't replicate the underlying progressive path of human Parkinson's disease.

2.2.3 OXIDATIVE STRESS

Oxidative stress occurs when the burden of oxyradicals is greater than the body's antioxidant ability. The mitochondria produce ATP and generate oxygen used within individual cells. The byproduct of this process is oxyradicals which are highly reactive and have the potential to destroy tissue. In normality, the mitochondria have antioxidant defenses, however in Parkinson's disease there is an abnormal increase in oxyradicals that could possibly overtax the mitochondria (Jenner, 2003). The potential side effects of this pathogenic mechanism is two-fold: firstly, an increase in oxyradicals will cause an increase in cellular death, and secondly, a potential decrease in ATP production will decrease cellular oxygen. It is important to note, however, that research has been inconclusive as to whether oxidative stress is due to mitochondrial dysfunction or vice versa (Jenner & Olanow, 1998).

2.3 PATHOGENESIS OF IDIOPATHIC PARKINSON'S DISEASE

Parkinson's disease is characterized as a movement disorder resulting from deficiency of dopamine in the motor control pathways of the central nervous system.

Dopamine is produced in the substantia nigra of the basal ganglia. Dopamine affects two motor pathways within the basal ganglia. These pathways control the activity of the globua pallidus interna, which has inhibitory connections to the thalamus. The thalamus has excitatory influence over the pathways to the motor cortex. Dopamine reduces the inhibitory activity of the globua pallidus interna, which in turn allows for the thalamus to facilitate movement (Fix, 2009; Martin, et al., 2010).

In Parkinson's disease, the neurons that produce dopamine degenerate. This loss of dopamine neurotransmitter results in an imbalance of the indirect and direct motor pathways. This imbalance causes the globua pallidus interna to be in a constant excitatory

state, which inhibits the thalamic outflow and therefore inhibit movement (Forno, 1996; Lang & Lozano, 1998).

2.4 PHARMACOLOGICAL TREATMENT OF PARKINSON'S DISEASE

All pharmaceutical options to date treat Parkinson's disease symptomatically and therefore can be quite limiting. Although, drug treatment is extremely effective at treating many of the cardinal symptoms, it has little effect on balance and postural stability (Koller, Glatt, Vetere-Overfield, & Hassanein, 1989). There have been no breakthroughs in finding a treatment to slow the degeneration of the neurons that produce dopamine (Rocchi, Chaiari, & Horak, 2002). However, advancements in pharmaceutical treatment have provided information on the pathogenesis of Parkinson's disease. This is most prevalent with the discovery of the dopamine precursor levodopa. Still the most common medication prescribed for Parkinson's disease, it substantiated the theory of the substantia nigra degeneration causing dopamine deficiency (Forno, 1996; Martin, et al., 2010).

2.4.1 DOPAMINE PRECURSOR/LEVODOPA

Parkinson's disease is a pathology characterized by the depletion of dopamine. However, administering dopamine does not effectively replenish levels because it cannot cross the blood-brain barrier. Pharmaceutical research discovered that levodopa, the metabolic precursor to dopamine, does cross the blood-brain barrier. In order to prevent systemic and peripheral conversion to dopamine, levodopa is normally administered in conjunction with a decarboxylase inhibitor such as carbidopa. Carbidopa does not cross the blood-brain barrier and therefore does not interfere with the conversion of levodopa to dopamine within the CNS. In Canada, this combination is offered in a single pill called Sinemet. Sinemet "reduces the amount of levodopa required for optimum therapeutic benefit by about 75-80%, permits an earlier response to therapy, reduces the incidence of

nausea, vomiting and cardiac arrhythmias" (*Compendium of pharmaceuticals and specialties: The Canadian drug reference for health professionals*, 2009, p. 2138).

Levodopa has been well recognized as the most effective treatment for Parkinson's disease since the late 1960's (Goetz, Poewe, Rascol, & Sampaio, 2005; Martin, et al., 2010). It significantly reduces bradykinesia, rigidity and tremor but has no effect on postural stability (Koller, et al., 1989) . In a more recent study, the effects of levodopa on postural sway were measured in Parkinson's patients during quiet stance. The subjects had all received deep brain stimulation as well as levodopa treatment. They were measured under four conditions: off levodopa and off stimulation; deep brain stimulation only; levodopa only; and, finally, both levodopa and electrode stimulation. This study concluded that postural sway abnormalities increase with levodopa as opposed to the other three conditions (Rocchi, et al., 2002).

Further substantiation that levodopa is ineffective at improving balance in Parkinson's disease can be seen in post-encephalitic Parkinson's, where the initial symptom is falling. Post-encephalitic Parkinson's is characterized by degeneration of the globus pallidus, the area of the basal ganglia responsible for the righting reflex. Patients with post-encephalitic Parkinson's are unresponsive to levodopa, suggesting that this medication is not effective in treating the postural instability component of Parkinson's (Koller, et al., 1989).

Although levodopa is currently the most effective treatment for the cardinal signs of Parkinson's disease, there are some significant side effects. The most troublesome are the motor complications such as on-off phenomenon and dyskinesia, which tend to be brought on from long term use. One study concluded that the prevalence of these side

effects is 50% of those who have taken levodopa for five years (Lang & Lozano, 1998). The on-off phenomenon is the alteration between an effective response to the medication and little or no response. As the disease progresses this phenomenon can be quite sudden, causing a postural instability or loss of balance (Nutt, et al., 1992).

Dyskinesia usually presents as a twisting or writhing motion during peak dosage when Parkinson's motor symptoms are minimal (Martin, et al., 2010). This may occur as a response to excessive stimulation of dopamine receptors. With disease progression this dyskinesia is sometimes present throughout the entire dosage. In addition to dyskinesia the non-motor side effects include nausea, sudden onset of sleep, vomiting, depression, and psychotic episodes (*Compendium of pharmaceuticals and specialties: The Canadian drug reference for health professionals*, 2009).

2.4.2 DOPAMINE AGONISTS

Dopamine agonists work to stimulate the dopamine receptors. Initially dopamine agonists were prescribed as an adjunct to levodopa, primarily for those who experience motor complications from the levodopa. Dopamine agonists are now also prescribed as a monotherapy but a review by the American Academy of Neurology noted that levodopa was more effective in treating the motor symptoms of Parkinson's disease (Suchowersky, Gronseth, et al., 2006). Dopamine agonists are more effective in the treatment of rigidity and bradykinesia, but similar to levodopa it has also shown little effect on postural instability (Suchowersky, Gronseth, et al., 2006).

The original dopamine agonists, bromocriptine and peroglide, were ergot derivatives and had severe side effects such as retroperitoneal or pleural fibrosis and cardiac valvulopathy. Peroglide has since been taken off the market in North America (Martin, et al., 2010). Two new forms of agonist, ropinirole and pramipexole, are now

available in Canada with fewer severe side effects than the originals. However, these recent additions are associated with a greater frequency of side effects then levodopa. The most common side effects include: hallucinations, somnolence, leg edema and dizziness (Suchowersky, Gronseth, et al., 2006). Dopamine agonists are also known to have adverse effects on impulse control, which can lead to compulsive buying, pathological gambling and sexual addiction (Weintraub et al., 2006). A recent meta-analysis of dopamine agonists found that using them in early Parkinson's disease can reduce symptoms without the unwanted motor complications of levodopa (Baker et al., 2009). However, it also found that the adverse effects caused higher withdrawal rates from the these pharmaceutical studies (Baker, et al., 2009).

2.5 NEUROPROTECTION

Neuroprotection refers to something that would slow the rate of progression of Parkinson's disease. Currently, nothing has been developed that has proven successful (Hirsch, 2007). However, assessing neuroprotective mechanisms can be challenging due to difficulty in determining the rate of progression. Simply monitoring the rate of symptomatic development would be flawed considering that most Parkinson's patients are using pharmaceutical therapies. Tracking the amount of neuronal decline is the only valid way to measure neuroprotection. Several studies have been done to assess neuroprotective qualities of dopamine agonists using neuroimaging as surrogate markers. Clarke and Guttman (2002) found that the effects of pharmacological intervention on dopamine transport obscured neuroimaging measures. Therefore, it has been determined that there is not enough evidence to support the use of radiotracer imaging in Parkinson's disease. Currently, the only method of measuring the quantity of neurons, with a strong degree of validity, is by postmortem dissection. Hence there are currently no conclusive

methods to assess neuroprotective agents in a living patient, although this hasn't impeded the scientific community from researching methods of neuroprotection (Olanow & Jankovic, 2005b; Winkler, Sauer, Lee, & Bjorklund, 1996).

Selegiline was thought to provide neuroprotection by decreasing free radical production. This medication was designed as an antidepressant but in the 1980s was found to prevent Parkinsonism symptoms after MPTP injection. Clinical trials showed that selegiline delayed the need for levodopa (Myllyla, Sotaniemi, Vuorinem, & Heinonen, 1992). However, the American Academy of Neurology stated that there wasn't enough conclusive evidence to suggest that selegiline had neuroprotective properties (Suchowersky, Gronseth, et al., 2006). Pilot studies on creatine, minocycline and coenzyme 10, have all had promising results, but further study is needed (Martin, et al., 2010).

2.6 SURGICAL INTERVENTION

2.6.1 DEEP BRAIN STIMULATION

Deep brain stimulation has become a widely accepted treatment for Parkinson's disease. It is often used in conjunction with pharmaceutical treatment when the patient is suffering from fluctuating "off" phases, severe dyskinesia or advanced Parkinson's disease that is unresponsive to medication (Ostergaard, Sunde, & Dupont, 2002). Deep brain stimulation involves the implantation of electrodes within the ventralis intermedius nucleus of the thalamus (Vim), the posteroventral portion of the globus pallidus interna (GPi), or the subthalamic nucleus (STN). Deep brain stimulation to the GPi and the STN is effective for treating tremor, rigidity and dyskinesias, whereas deep brain stimulation to the Vim primarily affects tremor (Weaver, Stern, & Follett, 2006).

Candidates for this treatment tend to be younger and have less co-morbidity, with severe motor fluctuation despite drug treatment. A patient's prior positive responsiveness to levodopa, that over time has become less effective, is considered to be a good indication that this surgical intervention will be effective (Martin, et al., 2010).

Deep brain stimulation is one of the few treatments for Parkinson's disease that has shown improvement in postural instability. A recent study that evaluated the effects of deep brain stimulation on 26 patients with advanced Parkinson's disease found significant improvement in postural instability as measured by part III of the Unified Parkinson's Disease Rating Scale (Ostergaard, et al., 2002). Rocci et al. (2002) found impressive results when studying postural sway by measuring the centre of foot pressure in open eyed, quiet stance of Parkinson's subjects who had deep brain stimulation. The mean velocity of centre of foot trajectory was very similar to that of the control subjects ($p>0.05$). A five year follow-up study on bilateral subthalamic nucleus stimulation showed an improvement in postural stability as well as gait and freezing episodes during the "off" phase (Krack et al., 2003). However, balance and gait deteriorated during the "on" phase.

2.6.2 NEURAL TRANSPLANTATION

Neural implantation surgeries are currently being used in clinical trials however, they are still considered to be in the investigational stage. The most common and widely controversial neural transplantation surgery is human fetal mesencephalic cells.

Individual trials report varying results on the effects of controlling motor symptoms in Parkinson's patients. A review of 23 clinical studies concluded that there was insufficient evidence to confirm the efficacy of neural fetal transplantation for control of motor symptoms, prevention of motor symptoms and the prevention of disease

progression ("Surgical treatment for Parkinson's disease: Neural transplantation," 2002). However, this review did note that post mortem studies have shown that fetal cells survive after implantation.

Ethical concerns over the use of human fetal cells, have spurred researchers to experiment with alternate neural cells. The most common alternative was the implantation of the patient's adrenal medullary cells or cervical sympathetic ganglion cells in to the substantia nigra. However, poor results have largely caused researchers to abandon this surgery (Olanow & Jankovic, 2005a).

2.7 PHYSICAL THERAPY

Physical therapy is often used as an adjunct therapy to pharmacological treatment. Physical therapy intervention in Parkinson's addresses muscle strength, motor coordination, gait training, mobility, flexibility and balance. The American Physical Therapy Association outlined a model of physical therapy for Parkinson's disease. It proposes a progression of treatment beginning with relaxation, breathing exercises, passive muscle stretching, active range of motion, postural alignment, weight shifting, balance responses, gait activities and home-based exercises (Schenkman et al., 1989).

Auditory, visual and external cueing techniques are commonly used in physiotherapy intervention. This is thought to by-pass the basal ganglia by utilizing the frontal lobe to control sequential movement (Morris, 2000). One small study that substantiates the hypothesis of external cueing assessed 14 Parkinson's disease subjects with minimal to moderate balance instability. These subjects were asked to balance on a rubber inflated disc that was placed over a force plate. The subjects were assessed three times. Initially they were asked to *stand still* for a control baseline, then they were asked

to *focus on minimizing movements of their feet*, which assessed internal focus, and lastly the subjects were asked to *focus on minimizing movements of the disc,* which was meant to simulate an external focus condition. This study concluded that there was less postural sway in the external focus group compared to the other two groups (Wulf, Landers, Lewthwaite, & Tollner, 2009).

Although physiotherapy is part of a standard treatment of Parkinson's, there is varying evidence to support this protocol. A review of eight clinical studies concluded that physical therapy for Parkinson's patients is *possibly useful*. It stated that physiotherapy should result in motor gains but that the gains most probably will not continue after therapy has ceased ("Physical and occupational therapy in Parkinson's disease," 2002). A Cochrane review of randomized controlled trials found that there was insufficient evidence to support or refute the effect of physiotherapy (Deane, Jones, Clarke, & Playford, 2001). Another meta-analysis that critically reviewed 12 studies concluded that Parkinson's patients benefited from physical therapy when used as an adjunct to medication. This research specifically found benefits in activities of daily living, walking speed, stride length and quality of living. This review did not look at balance and it did not find statistically significant changes in neurological symptoms such as bradykinesia, tremor and rigidity (Goede, Keus, Kwakkel, & Wagenaar, 2001).

Overall, the effectiveness of physiotherapy in treating balance in Parkinson's disease has not been substantiated in reviews and meta-analysis. This may be due to methodological concerns with studies that resulted in exclusion from the reviews (Deane, et al., 2001; Goede, et al., 2001). Many of the studies involving physiotherapy and Parkinson's do show improvements in gait and quality of life.

A large study that assessed the effectiveness of an inpatient rehabilitation program for people with Parkinson`s disease showed significant improvements in the Functional Independence Measure that is comprised of motor and cognitive sections. However, the intervention within this study included not only physical therapy, but also a neurologist specializing in neuro-rehabilation and movement disorders, speech pathologist, occupational therapist, nurses and case managers. There was no long term follow-up to determine if the results continued when the subjects returned to their home setting (Ellis et al., 2008).

One study found significant improvement in bradykinesia ($p=0.009$), and rigidity ($p=0.005$) after a four-week exercise program. There was no change to resting or active tremor (Comella, Stebbins, Brown-Toms, & Goetz, 1994). In a controlled trial on home-based exercise and the reduction of falls in Parkinsonian subjects, there were lower rates of repeated falls after 8 weeks ($p=0.0004$) and 6 months ($p=0.007$) (Ashburn et al., 2007). However, there was no difference between the groups on the Berg Balance Scale.

A recent (small) study measuring the effect of physiotherapy on balance had positive results. This research compared two groups: the first completed a balance exercise program and the second completed the same balance exercise program in addition to strength training. Balance was assessed using dynamic posturography pre-intervention and post-intervention and at a four-week follow-up. The results showed that both balance training and strength training increased balance and latency to falls and that the effects lasted for a four-week period (Hirsh, Toole, Maitland, & Rider, 2003). A refutation of this study included the lack of control group, the small sample size and the validity of the posturography (Grimbergen, Munneke, & Bloem, 2004).

2.8 THEORIES ON POSTURAL INSTABILITY IN PARKINSON'S DISEASE

One of the clinical signs of advancing idiopathic Parkinson's is postural instability. This postural instability results in falls and, consequently, injury and loss of independence. Falling comes at great cost to the Parkinson's patient. A great deal of research has been done to identify the reasons for postural dyscontrol and therefore falling in people with Parkinson's (Horak, Nutt, & Nashner, 1992). However, despite this, the pathophysiology of postural instability still remains an inadequately understood process in Parkinson's disease.

Postural dyscontrol in Parkinson's disease may be due to a single factor or a combination of factors such as abnormal muscle firing, postural inflexibility, and/or freezing.

One of the roles of the basal ganglia is to maintain the neurons within the motor cortex in a state of readiness for action. This enables the postural muscles to engage in order to maintain the centre of gravity during movement. This readiness for action is a form of behavioural plasticity that is referred to as *set* and *changing set*. *Set* is the nervous systems immediate response to any given situation based on previous experiences. A *changing set* refers to the ability of the nervous system to respond to a change in condition or context. The ability to c*hange set* quickly is necessary for adaptation to changes in conditions. This allows for a more appropriate and efficient response necessary to provide balance (Chong & Horak, 1998).

In Parkinson's disease, the inability to maintain balance is thought to be rooted in these motor cortex neurons. The dysfunction of these neurons disrupts anticipatory postural muscles by decreasing the timing and size of muscle activation (Smithson, et al., 1998).

2.8.1 CHANGING SET

The hypothesis that changing set is difficult for Parkinson's patients has been tested in various recent studies as a possible cause for postural instability.

One study was designed to assess postural set changes in subjects with Parkinson's disease compared to Alzheimer's subjects as well as a third group of healthy age-matched controls. The first experiment compared tibialis anterior activation while rising up into dorsiflexion (toe stance) during standing without support compared to the same action with support. The control group and the Alzheimer's subjects reduced the activity of tibialis anterior while coming into a toe stance while holding onto a support. The Parkinson's subjects were unable to inhibit tibialis anterior initially; however after several trials set changes were made (Chong & Horak, 1998).

These same subjects were subjected to a second study designed to assess postural set changes using surface perturbation. The subjects sat on a stool, initially with their feet planted on the ground and secondarily with their feet dangling above the ground. A backwards translation was placed through the support surface. Normally, to maintain an upright position a forward sway of the trunk occurs and the soleus is activated when the feet are planted. In the Parkinson's subjects, the soleus activated pointlessly when the legs were dangling, providing further evidence that set changing is difficult in patients with Parkinson's disease. Medication did not have an effect on set change (Chong & Horak, 1998).

Another study designed to assess the hypothesis that basal ganglia dysfunction, such as Parkinson's, impairs the ability to change set quickly also supported this hypothesis. This study involved two experiments, a sensorimotor set experiment and a cognitive set experiment. Ten subjects with idiopathic Parkinson's disease were

compared with ten healthy young subjects and ten healthy older subjects. In the sensorimotor experiment the gastrocnemius response was measured during backward translation. Parkinson's subjects had difficulty suppressing gastrocnemius to the first translation but response improved as the same test continued. This suggested to the authors that Parkinson's disease subjects had slow set changing response that recovered with continued practice. These results were similar when the subjects experienced a change from translation to rotation (Chong, Horak, & Woollacott, 2000).

In the cognitive set experiment subjects were given instructions to give or resist perturbations, while the amplitude to their responses was measured. The results of this experiment showed that subjects had greater difficulty with the instruction to resist surface perturbations as opposed to the instruction to *give in.* The researchers hypothesized that this may be due to the opportunity for the subjects to prepare to let go to muscular contraction when asked to give in. This contrasts the instruction of resisting for the subject was unaware as to the direction of surface perturbations, and therefore did not have the opportunity to prepare. Both instructions showed greater delay in the Parkinson's subjects (Chong, et al., 2000).

Chong et al. (2000) concluded that normal postural response latency and continued postural instability are a result of difficulty changing set.

2.8.2 POSTURAL INFLEXIBILITY

Postural inflexibility is a widely used term to describe one of the causes for falling in Parkinson's disease. It denotes inter-segmental stiffening that occurs in Parkinson's patients. This stiffening decreases mobility and therefore provides some stability by decreasing sway. However, according to Gruneberg, Bloem, Honegger, and Allum

(2004) this stiffening also removes the ability to have flexible responses to changes in environment, which predisposes the Parkinson's patient to falls.

In contrast with the previous statement, Mitchell, Collins, De Luca, Burrows, and Lipsitz (1995) hypothesized that postural instability in Parkinson's disease is due to the increase in postural sway. Mediolateral sway is associated with falling and poor performance on balance assessment. A controlled trial measuring 22 Parkinson's subjects against 24 age-matched control subjects analyzed postural sway during open eyed quiet stance. It concluded that there is an increase in mediolateral (ML) postural sway in Parkinson's subjects. Interestingly, they also found a decrease in anteroposterior (AP) sway in Parkinson's subjects compared to healthy control subjects. From these findings the researches postulated that the lack of AP sway is a sign of postural inflexibility and may contribute to the increase of compensatory ML sway (Mitchell, et al., 1995).

The absence of protected arm motions was tested by assessing PD subjects on a sudden rotating platform. It was seen that instead of arm flexion the subjects adducted their arms. This doesn't explain postural instability but may provide insight as to why Parkinson's patients sustain more injury due to falls, including hip fractures (Carpenter, Allum, & Honegger, 2004).

2.8.3 FREEZING

Freezing of gait is associated with a high incidence of falling in patients with Parkinson's disease (Bloem, Hausdorff, Visser, & Giladi, 2004). Freezing is a peculiar phenomenon that refers to the sudden inability to move, which occurs most typically during gait. It is often associated with the action of turning during the "off" state of medication. One study that highlighted the difficulty in turning compared healthy elderly subjects to Parkinson's subjects. Control subjects performing a 360-degree turn took

fewer than six steps as opposed to Parkinson's subjects, who took 20 steps to turn (Yekutiel, Pinhasov, Shahar, & Sroka, 1991). Bloem et al (2004) found that the majority of falls in Parkinson's occur forwards. The authors postulated that this may be due to freezing of gait. The actual mechanism behind freezing is not well understood.

2.9 SUMMARY OF NON-OSTEOPATHIC LITERATURE REVIEW

Although much research has been done in the pathogenesis, etiology and treatment of Parkinson's disease, a great deal is still unknown. Parkinson's disease is a neurodegenerative disease with its pathological origins based within the basal ganglia of the encephalon. It is characterized by tremor, rigidity, bradykinesia and postural instability. Current theories on the etiology of Parkinson's disease include toxicity, environmental factors, oxidative stress model, and genetic predisposition. The current theories on the causes of Parkinson's disease provide an insight into how osteopathic treatment may be beneficial as a conjunct treatment in the care of Parkinson's patients.

The treatment of postural instability in Parkinson's disease is very limited. The majority of people living with Parkinson's are being treated with pharmaceuticals. The medications available for treatment of Parkinson's disease are effective in temporarily curbing symptoms of tremor, bradykinesia and rigidity, but have little effect on the treatment of postural instability. All the pharmaceuticals available in the treatment of Parkinson's disease have mild to major side effects and none have been proven to slow the progression of the disease.

Deep brain stimulation is a surgical procedure that has shown some improvement in the treatment of postural instability. However, this surgery is only available to younger candidates with severe motor fluctuations and like all surgeries it is invasive and comes with risks. Neural fetal transplantation is another treatment option, but similar to deep

brain stimulation, it is not available to all patients. Ethical concerns have arisen over this procedure and therefore it is not available everywhere.

Physical therapy is a common adjunct treatment to pharmaceutical intervention. Studies have indicated various results on the treatment of balance in Parkinson's disease. Postural instability in Parkinson's disease is thought to be rooted in one or a combination of factors - difficulty with changing set, postural inflexibility and freezing phenomenon. These all occur due to the neuronal loss of the basal ganglia.

2.10 OSTEOPATHIC LITERATURE ON PARKINSON'S DISEASE

A.T. Still discussed *shaking palsy,* which was described as a "shaking and tremor in muscles, accompanied by in-coordination" (1910,p.139). Still refuted all theories on the causes of shaking palsy at the time, and reasoned that there was a mechanical cause. A.T. Still states that "shaking palsy is an effect of a cause, producing atrophy of the whole system from the eighth rib to the atlas, by shutting off the blood, cerebrospinal and other fluids that nourish the nervous system" (1910,p.140).

2.11 OSTEOPATHIC STUDIES ON PARKINSON'S

Four studies were reviewed that specifically dealt with Parkinson's disease and manual osteopathy. None of these studies looked at balance.

One study was designed to determine if there was a commonality between cranial strain patterns in subjects with idiopathic Parkinson's disease against normal age-matched controls. The results found a higher frequency of bilateral occipitoatlantal compression ($p<.02$) and bilateral occipitomastoid compression ($p<.05$) among the subjects with Parkinson's disease (Rivera-Martineza, Wells, & Capobianco, 2002). This study suggested that osteopathy in the cranial field may contribute significantly to the management of patients with neurological disorders and that there is a basis for further

study to examine the effectiveness of cranial osteopathy. Rivera-Martineza et al. (2002) postulated that cranial osteopathic manipulation will not regenerate the affected areas of the brain, but may provide enough support to decrease the progression of the disease process.

A refute to this study focused its criticism on the small sample size and the inter-reliability of cranial examination (Boehm, Lawner, & McFee, 2003). It should be noted here that inter-reliability and intra-reliability are limitations of all cranial osteopathic studies. Hartman and Norton (2002) critically reviewed six published studies on inter-reliability in the cranial field and found only one had statistically significant results. A separate study found high intra-observer reliability of cranial strain patterns (k=0.67) (Halma et al., 2008). There is much difficulty in proving inter-reliability and intra-reliability and this challenge will continue to plague osteopathic cranial research until addressed.

The second study of the four aforementioned research studies measured gait in people with mild to moderate Parkinson's. This study showed an increase in stride length ($p<0.02$) and cadence ($p<0.005$) after a single standardized 30 minute osteopathic treatment (Wells et al., 1999). A power of n=28 was employed in this study. Twenty of the subjects had idiopathic Parkinson's disease and eight were healthy age-matched control subjects. The subjects with idiopathic Parkinson's disease were placed in equal groupings, ten in the experimental and ten in the sham group. The remaining eight healthy controls were treated with the same osteopathic protocol. The single osteopathic treatment was a set protocol consisting of 14 osteopathic techniques however, only one technique was a cranial osteopathic technique, which was an occipitoatlanto release.

A third study on the effectiveness of osteopathic manipulative treatment for Parkinson's disease was set up as a single-blind placebo controlled study. The sample size of 27 subjects placed 14 subjects in the treatment group while 13 received a sham osteopathic treatment. The outcome was measured using the Unified Parkinson's Disease Rating Scale and the Quality of Life Inventory (PDQ-39). This study revealed no statistically significant difference between the treatment and sham group, however the authors cited a trend towards improvement that was clinically significant (Snider et al., 2007). The treatment protocol was not included in the report obtained; therefore it is difficult to extract what the osteopathic manipulative treatment entailed.

An unpublished thesis from the Weiner Schule fur Osteopathie studied the effects of two osteopathic treatments on 19 patients with idiopathic Parkinson's disease. The osteopathic treatment was unspecified and individualized. The results showed a 10% increase in the speed of gait post-treatment (Petzl, 2004).

2.12 NEUROLOGICAL OSTEOPATHIC LITERATURE

With such little research on the effects of Osteopathy on Parkinson's disease, it is necessary to extrapolate from various osteopathic neurological studies.

Greenman and McPartland (1995) used cranial osteopathy to examine and treat 55 subjects that had experienced a traumatic brain injury. All but one patient had a decrease in the primary respiratory mechanism, and cranial strain patterns were found in 95% of the subjects. Although the study did not note the primary respiratory mechanism (PRM) rate that was used as a comparison, it did cite four other studies that had determined normal PRM rate between 12.47 and 11.9 cycles per minute. The subjects with traumatic brain injuries averaged 7.2 cycles per minute. This study concluded that cranial osteopathy in traumatic brain injuries can be useful, but unfortunately it did not discuss

the particular benefits for this population. Instead the paper focused on the 5% that had adverse reactions to the treatment (Greenman & McPartland, 1995).

Frymann has contributed a great deal to the study of cranial osteopathy and neurological changes. In one such study, Frymann extracted clinical data to determine if there was a traumatic pattern in the craniosacral mechanism in children with learning difficulties. The cranial strain patterns were noted in 103 children with learning difficulties compared to 106 children without learning difficulties. Lateral strain lesions were found in 86.4% of children with learning difficulties compared to 70.7% in the unaffected group. Likewise, it was noted that 46.5% of vertical strains were present in the affected group compared to 37.7% in the control group (Frymann, 1976).

Clinical experience motivated Frymann, Carney, and Springall (1992) to study the effects of osteopathic treatment on the neurological development in children. The children were assigned to either a medical group, which included medical or structural issues without neurological symptoms, or a neurological group, which consisted of children with known neurological issues. These included poor academic performance, behavioural issues, abnormal neuromotor function and delayed development or learning. All subjects received 6 to 12 treatments, although the waiting-list group was initially used as a baseline before treatment was administered. The measuring tool used was Houle's Profile of Development, which measures mobility, manual dexterity, speech, visual ability, auditory competence and perceptive tactility. In total, 43 of the original 186 subjects completed the study in its entirety and 13 subjects in the wait-list group completed the study. High attrition rates may be due to the length of study, the waiting period for some participants and relying on parents and caretakers. The results of the

experimental neurological group showed a significant increase in scores between the tests ($P<.01$) whereas the waiting-list neurological group's scores slightly decreased between testing. Interestingly, there was little difference between the pre-testing and post-testing of both the experimental medical group compared to the waiting-list medical group. Follow-up testing was administered several months after treatment was completed. The results were significant in that they showed a continuation of the positive effects of osteopathic treatment ($P<.001$) (Frymann, et al., 1992).

Lassovetskaia, who was inspired by Frymann's research, added to the body of knowledge by studying 96 children with delayed academic performance. They were treated with cranial osteopathy over a six to twelve week period. The results showed a significant increase in academic performance compared to students with similar academic delay that did not receive treatment (Lassovetskaia, 2002). This research has been cited in numerous works, however this author was unable to utilize this paper as it was published in Russian and a translation is not available.

A recent pilot study focused on the effectiveness of cranial osteopathy and myofascial release compared to acupuncture on children with spastic cerebral palsy. There were 55 subjects in total, with 15 in the osteopathic intervention and 18 in the acupuncture intervention. The remaining 22 subjects were placed in the wait-list control and participated in non-therapeutic play for the duration of the study. After the intervention groups had completed their study, the wait-list group was randomized and put through the same intervention protocol. These results were then added to the overall statistics for this study. Each subject in the osteopathic intervention was treated 10 times over a 24-week period, while the acupuncture subjects had 30 sessions over 24 weeks.

The results showed that osteopathic intervention was statistically significant (P<.05) in the Gross Motor Function Measurement and the Functional Independence Measure for Children. It did not, however, show significant difference in the other nine outcome measures. The author stated that three of the outcome measures used were unreliable or irreproducible. The acupuncture group showed no significant differences (Duncan, 2007).

2.13 CRANIAL VALIDITY

All of the previously cited studies had some component of cranial osteopathy in the intervention portion of the research, however none endeavored to use cranial osteopathy as the sole means of treatment. This research is using cranial osteopathy as complete treatment methodology to elicit changes in the balance of Parkinson's patients. It therefore seems prudent to review research on the validity of cranial osteopathy.

Cranial osteopathy is based on Sutherland's model of the five phenomena that make up the primary respiratory mechanism (PRM). For the purpose of this research we will look at the first four.

2.13.1 THE INHERENT MOBILITY OF THE BRAIN AND SPINAL CORD

There is a fair amount of supporting evidence to substantiate motion of the central nervous system. This is the least controversial of all the elements that make up the primary respiratory mechanism. However, prior to the scientific evidence, osteopaths postulated the mechanism of PRM.

In *Osteopathy in the Cranial Field*, Magoun noted that during the embryonic formation of the cerebral cortex the neural tube "curls up like ram's horns" (1976, p. 24). He postulated that the central nervous system inherently moves in by coiling and uncoiling similar to this embryonic formation.

In the 1960s, Frymann set up a series of experiments to record a third rhythm within the living cranium, slower than the respiratory or cardiac rhythm. Frymann (1971) saw the need for instrumentation and got an electronics engineer to design a device that was sensitive enough to pick up mechanical recordings of this rhythm. Recording of cranial motion ranged from 0.0005 to 0.001 inch. In one of the experiments, cranial rhythm was recorded in conjunction with respiratory rhythm, which was monitored using a pneumogram. The cranial rhythm was found to be slower chest motion, with the maximum cranial motion occurring during interrupted respiration.

More recent research substantiating this theory has been done through the medical community. The lack of osteopathic research in this field is possibly due to the lack of funding.

Maier, Hardy and Jolesz (1994), used magnetic resonance imagining (MRI) in real time to capture periodic motion of the brain. The imaging showed the hemispheres of the brain squeezing the ventricles at a velocity of 1mm per sec with a recoil that appeared slower. However, these researchers found the motion of the brain and cerebrospinal fluid moving in frequency with the heart rate, which is considerably faster than the primary respiratory rhythm.

In a separate study, cine echo-planar MR imaging was used to determine the intrinsic pulsations of the brain parenchyma. The movement was measured in conjunction with the cardiac cycle and the researchers noted that there was rapid brain motion during systole with a slow diastolic recovery. Although this provides some evidence of inherent motion of the CNS, it differs from PRM in that PRM has an equal length flexion and extension phase. This study also found the motion of the brain primarily occurred in

cephalocaudal and lateral directions. The velocity was recorded at 2mm/sec in the brainstem and 1.5mm/sec in the central thalami (Poncelet, Wedeen, Weisskoff, & Cohen, 1992).

Another study that utilized MR imaging technology found that the highest velocity of movement in the brain occurred during systole at the basal ganglia at a rate of 1.0 mm per sec and the brain stem at 1.5 mm per second. The researchers concluded that the encephalon moves in a *piston-like* action due to arterial expansion, which in turn causes expansion of the brain that compresses the ventricular system pushing cerebrospinal fluid into the spinal cord (Greitz et al., 1992).

2.13.2 THE FLUCTUATION OF CEREBROSPINAL FLUID

The movement of cerebrospinal fluid through the ventricles, brain and spinal cord is a well recognized mechanism. The concept of cerebrospinal fluid fluctuation has often been noted in associated research on the movement of the brain and spinal cord. In one such study, Maier et al. (1994) determined that the cerebrospinal fluid had periodic motion as seen in MR imaging. It was also noted during this imaging that the cerebrospinal fluid moved in a cephalic direction when the subjects were instructed to cough (Maier, Hardy, & Jolesz, 1994).

Moskalenko, a large contributor to the knowledge of intracranial hemodynamics, collaborated with Naumenko in the 1960s to provide us with specific research analyzing the connection between the fluctuation of cerebrospinal fluid and the dynamics of blood. Using a Plethysmography on canines, the researchers measured the blood volume and cerebrospinal fluid within the cranium, as well as the intracranial pressure. Results showed that 10% of the total cerebrospinal fluid shifts from the cerebrum to the spinal cavity with every exhalation, returning on inspiration. A much slower shift in location

was seen moving at a frequency of 30 to 40 times per hour. They conclude that there is a constant shift of cerebrospinal fluid that occurs between the cranial and spinal cavities (Moskalenko, 1980).

In a more recent study, intracranial hemodynamics in human subjects were assessed using the bioimpedance method to measure volume ratios within the cranial cavity and transcranial ultrasound to measure variation of blood flow within the middle cerebral artery. The results found slow oscillations that were related to cerebrospinal fluid and cerebral circulation (Moskalenko et al., 2001).

2.13.3 THE MOBILITY OF INTRACRANIAL AND INTRASPINAL MEMBRANES

The intracranial and intraspinal membranes consist of the dural membranes surrounding the spinal cord, the falx cerebri and the tentorium cerebelli. The continuity of the dural tissue is well documented; however, the motion as described in the concept of the primary respiratory rhythm has minimal supporting evidence. Practitioners of cranial osteopathy utilize this continuity of tissue and the reciprocal tension membrane it forms to treat cranial, cervical and sacral mal-alignments. Sutherland wrote that the structures attach to all bones of the neurocranium and are responsible for their movement (Sutherland).

Kostopoulos and Keramidas (1992) tested out the hypothesis that cranial techniques have an impact on dural membranes. They used an embalmed cadaver in which the brain tissue was removed but the intracranial membranes were left intact. Instrumentation was used to detect elongation of the falx during various cranial techniques. The results were significant, showing a 1.44mm elongation with a frontal lift, a 1.08mm due to a parietal lift, -.033mm during a sphenobasilar compression, and 0.28mm during a sphenobasilar decompression.

A radiology study on fixed spinal cords showed pulsating motions of the spinal cord during MR imaging. It concluded that cord motion was decreased in subjects with tethering or cord compression compared to normal control subjects. The pulsative velocity particularly slowed down at the level of cervical and upper thoracic vertebrae (Levy et al., 1988).

2.13.4 THE ARTICULAR MOBILITY OF THE CRANIAL BONES

It has been postulated that the role of mobility with the cranial bones is necessary as a protective mechanism for the brain during times of increased intracranial pressure due to volume fluidic changes.

A preliminary study of cranial bone mobility in six anesthetized adult squirrel monkeys had promising results. A hole was drilled into the parietal bone without disrupting the dura and an eye screw was placed in the bone. A transducer was attached to the screw to monitor parietal bone displacement. Blood pressure, heart rate and respiratory rate were also measured. The results showed a significant displacement of parietal bones when various pressures were applied to the monkeys. In addition, a cranial bone displacement pattern emerged that cycled between 5-7 per min and was not associated with the respiratory or heart rate (Micheal, 1975).

An artificial cerebrospinal fluid was injected into the lateral ventricles in anesthetized cats and an instrument was applied to either side of the sagittal suture. The instrumentation showed that the cranial bones moved as the intracranial volume increased. The authors postulated that cranial compliance is dependent on the mobility of blood and cerebrospinal fluid and the movement of cranial bones (Heisey & Adams, 1993).

NASA has done considerable research on intracranial pressures that are associated with nausea, headaches and projectile vomiting due to microgravity exposure. A non-invasive ultrasound was developed to measure intracranial pressures by detecting skull movements (Ueno et al., 1998). This device was used in a study with six healthy volunteers placed in various tilt positions and their cranial diameter pulsations were measured in conjunction with arterial blood pressure. The results showed that cranial diameter was significantly altered ($p < 0.001$) with a tilt angle and there was no correspondence to arterial blood pressure (Ueno, Ballard, Macias, Yost, & Hargens, 2003).

Moskalenko et al. studied the skulls of 23 patients using serial x-rays and nucleo-paramagnetic resonance (NMR) tomograms. A radiopaque solution was injected into the carotid followed by a series of imaging for 35-45 seconds at a rate of two pictures per second. The analysis of the x-ray data revealed a periodic "dislocation" of the cranial bones with an amplitude that ranged from 0.38 – 0.21 mm. The NMR images resulted in a similar amplitude, however it was an incomplete analysis due to the short duration of the imaging. A significant dislocation of the cranial bones (1mm) was seen after injection of the radioplaque solution (Moskalenko et al., 1999).

2.14 SUMMARY OF OSTEOPATHIC LITERATURE REVIEW

There is very little research on Parkinson's disease and osteopathy and even fewer studies with a focus on cranial osteopathy. This researcher was able to indentify four studies that specifically looked at Parkinson's and osteopathy. Although, some of the results looked promising, none evaluated balance or used cranial osteopathy as a sole treatment method. A variety of other studies were found that supported the use of cranial osteopathy in other neurological conditions, such as traumatic brain injuries, learning

disabilities and cerebral palsy. From these studies concepts were extrapolated that may apply to the use of cranial osteopathy for the treatment of balance in Parkinson's disease.

Research on the efficacy of cranial osteopathy is limited. The current literature review endeavored to examine cranial osteopathy as a viable treatment option. Research suggests that the main criticism of cranial osteopathy is existence of the primary respiratory mechanism. Examining literature that supports or refutes Sutherland's model of the phenomena that constitute the primary respiratory mechanism provides an avenue into this discussion. Many studies were reviewed that point to the existence of the osteopathic concept of the primary respiratory mechanism.

3 CHAPTER THREE: OSTEOPATHIC JUSTIFICATION

Balance can be defined as the ability to maintain the centre of gravity over the base of support. Postural equilibrium is the condition in which all the forces acting on the body are balanced such that the centre of mass is controlled relative to the base of support (Horak, Henry, & Shumway-Cook, 1997). However, the action of balance itself is multi-faceted.

Balance control was once thought of as a single system of a fixed set of reflexes that responded to sensory stimuli. Research on postural response to surface perturbations has provided a different view of balance control (Horak, et al., 1997). It is now seen as an adaptable, learned complex motor skill derived from the interaction of many sensorimotor processes. This evolution of knowledge has changed the understanding of postural instability in Parkinson's disease. There is now a strong alternative view that postural instability in Parkinson's disease is not a result of dysfunctional peripheral reflexes, but rather is caused by neuronal loss within the basal ganglia (Chong, et al., 2000; Horak, et al., 1997).

Postural instability in Parkinson's disease has been related to difficulty with changing set, freezing and axial stiffness. Difficulty changing set and freezing are impairments that occur within the central nervous system primarily within the basal ganglia. Hirsch (2009) suggested that intensive targeted training may slow or stop the progression of Parkinson's disease by promoting neurorestoration and reorganization.

Traditionally, postural instability in Parkinson's disease has been managed by physiotherapy. This treatment focus was primarily on proprioceptive training of hip, knee and ankle reflexes, postural alignment, and increasing strength and flexibility. Although the results were varied, there were no long-term gains ("Physical and occupational

therapy in Parkinson's disease," 2002). Our new understanding of balance has adjusted the physiotherapy approach to postural dyscontrol in Parkinson's. Current models use central nervous system compensatory strategies such as auditory and visual cueing, behavioral therapy as well as exercise-based therapy (Goodwin, Richards, Taylor, Taylor, & Campbell, 2008). There is a growing postulation that a multi-faceted approach is required to treat balance disorder with Parkinson's disease.

Cranial osteopathy is a unique treatment option in that it may have an influence on the central nervous system. Sutherland's principles of the primary respiratory mechanism or cranial mechanism demonstrate this connection with the central nervous system (Magoun, 1976). These principles include: fluctuation of cerebrospinal fluid, mobility of the reciprocal tension membrane, inherent motility of the brain and spinal cord, mobility of the cranial bones, and involuntary motion of the sacrum between the ilia. The relationship between the principles of the primary respiratory mechanism and their influence on the central nervous system will be discussed in detail within this chapter.

It follows that if cranial osteopathy has the potential to affect the central nervous system this treatment option would be of greater efficacy when focused on pathologies where the primary cause is rooted in the central nervous system. The focus of this research is to assess the hypothesis that cranial osteopathy will be effective in the treatment of balance in Parkinson's patients due to its targeted action at the level of the central nervous system. There are a number of reasons that would suggest that cranial osteopathy would be beneficial in this regard.

Researchers hypothesize that toxicity is a contributing factor in the etiology of Parkinson's disease. Cranial osteopathy could be beneficial in that it may soften cranial restrictions which may allow for an increase in circulation and venous drainage as well as assist in the normalization of cerebrospinal fluid. All of these components could promote the removal of toxins and reduce inflammation within the basal ganglia. In addition, an increase in cerebral circulation and normalization of cerebrospinal fluid may allow for an improved environment for neuroplasticity to take place. Neuroplasticity of the brain denotes the ability for the brain to self repair and is often discussed in Parkinson's literature (Hirsch & Farley, 2009). In theory, if the brain has the ability to regenerate or reorganize neural pathways, then the minimizing cranial osteopathic lesions should promote this restoration.

As noted in the literature review, neuroprotection is a topic of much interest in Parkinson's research. To date, pharmaceuticals have been the therapy of choice to promote neuroprotection. By increasing the suppleness of the cranium and increasing cerebral circulation, osteopathy, in theory may be able to contribute to the removal of free radicals by helping to decrease oxidative stress and promote neuroprotection.

The principles of osteopathy provide an avenue into the justification of this research and further expand on the rationale presented above.

3.1 THE PRINCIPLE OF THE ROLE OF THE ARTERY IS ABSOLUTE

Ever since MPTP was found to elicit Parkinson's symptoms in the late 1980s, much research has been done on environmental toxic exposure and the incidence of Parkinson's disease (Kontakos & Stokes, 2000). Research on additional noxious agents have shown that carbon monoxide poisoning affects the basal ganglia, while chronic

magnesium exposure, hydrocyanic acid, and copper cause focal changes in the brain (Martin, et al., 2010).

Although studies on toxic exposure have had varying results, there is a common understanding in the scientific community that environmental factors play a role in Parkinson's disease (Forno, 1996; Hirsch, 2007; Martin, et al., 2010).

The osteopathic principle of the role of the artery implies that fluidic flow is essential to a homeostatic state and therefore stasis or obstruction of circulation will lead to cell death. This principle is of utmost importance when applied to the brain. Because the brain requires more oxygen and glucose than any other organ, vascular insufficiency will result in necrosis, with devastating results (Nolte, 1981). Cranial osteopathy may have an effect on the arterial supply, venous drainage and the flow of cerebrospinal fluid within the cranium. This was highlighted by Magoun (1976) when he noted the importance of using cranial osteopathy to eliminate circulatory stasis in the brain. The removal of structural impedances allows for optimal circulatory flow as suggested by Moskalenko's (1980) research, which found that the movement of cranial bones had an effect on vascular circulation in the brain.

The use of cranial osteopathy in Parkinson's patients can potentially increase blood flow to the effected basal ganglia, contributing to the removal of toxins. This is of particular importance considering that research has shown continued degeneration long after initial exposure to MPTP, suggesting that toxic substances released by glial cells continues to occur and may contribute to the degeneration of the basal ganglia in Parkinson's disease (Hirsch, 2007).

In one study of post mortem dissections, the basal ganglia showed significant increase in the blood vessel branches of the substantia nigra pars compacta compared to control dissections. The researchers postulate that the increased number of blood vessels may be due to a neuroprotective mechanism that contributes to the increase of elimination of toxins (McGeer & McGeer, 2004). This further shows that optimal vascularization to the basal ganglia and surrounding tissues will contribute to the removal of potentially damaging toxins.

3.1.1 ANATOMY OF ARTERIAL SUPPLY TO THE BASAL GANGLIA

Magoun stated that the "basal ganglia in the center of the cerebral hemispheres, having to do with muscle rigidity and tremor, receive their blood from the arteries in intimate relation to the body of the sphenoid bone and hence are subject to the effects of shifts in the bone" (1968, p.41). Therefore, for the purpose of this study, the vasculature innervating the basal ganglia must be taken into consideration.

Arterial supply to the brain comes from the internal carotid arteries and the two vertebral arteries. The internal carotid arteries bifurcate into the middle and anterior cerebral arteries, but prior to this bifurcation it branches off to form the anterior chorodial artery and the posterior communicating artery. The anterior chorodial artery supplies some portions of the internal capsule, the globus pallidus, caudate nucleus and the amygdala of the basal ganglia as well as supplying the optic tract, the choroid plexus of the lateral ventricle and part of the cerebral peduncle (Fix, 2009; Nolte, 1981).

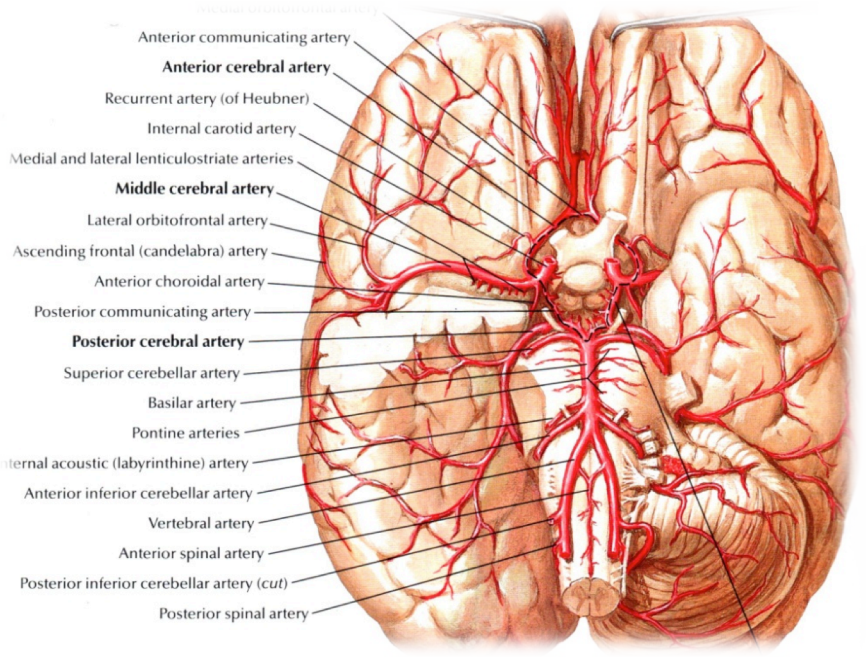

Figure: 1. Inferior view: Arterial supply to the brain (Netter, 1989).

Some of the blood supply to the basal ganglia comes directly off of the circle of Willis, which is located at the base of the cranium surrounding the sella turcia of the sphenoid. The circle of Willis is an arterial loop consisting of the anterior cerebral, the internal carotid and the posterior cerebral arteries. The posterior cerebral artery, which extends off of the basilar artery, provides blood to the mid-brain, including the substantia nigra (Fix, 2009).

The middle cerebral artery supplies blood to the sensory and motor regions around the central fissure (Gray, 2003). The middle cerebral artery passes along the lesser wing of the sphenoid and can be subjected to mechanical pressure with a torsion or sidebending rotation lesion of the sphenobasilar symphysis, causing an alteration of the circulation to the sensory and motor regions around the central fissure (Kimberly, 1954; Magoun, 1976). The anterior choroidal artery, which supplies most of the basal ganglia,

is usually a branch of the internal carotid, however in some cases it branches off of the middle cerebral artery (Nolte, 1981).

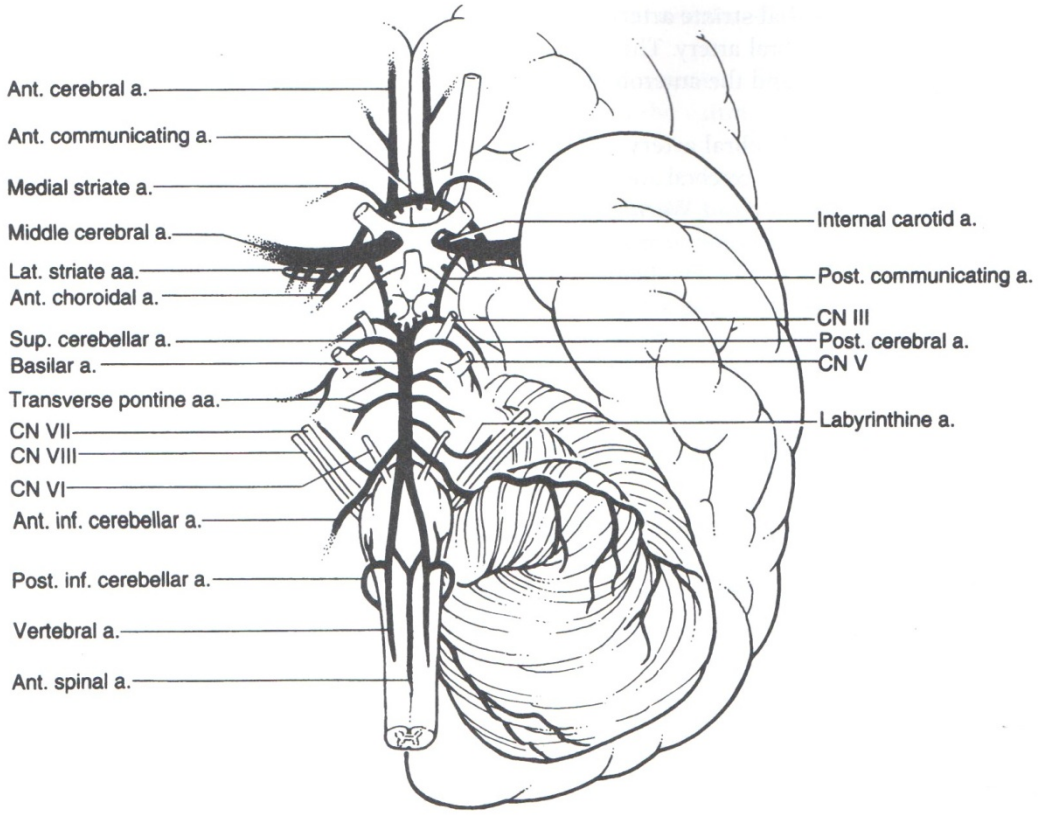

Figure: 2. Arteries of the base of the brain and brain stem (Fix, 2009, p.39).

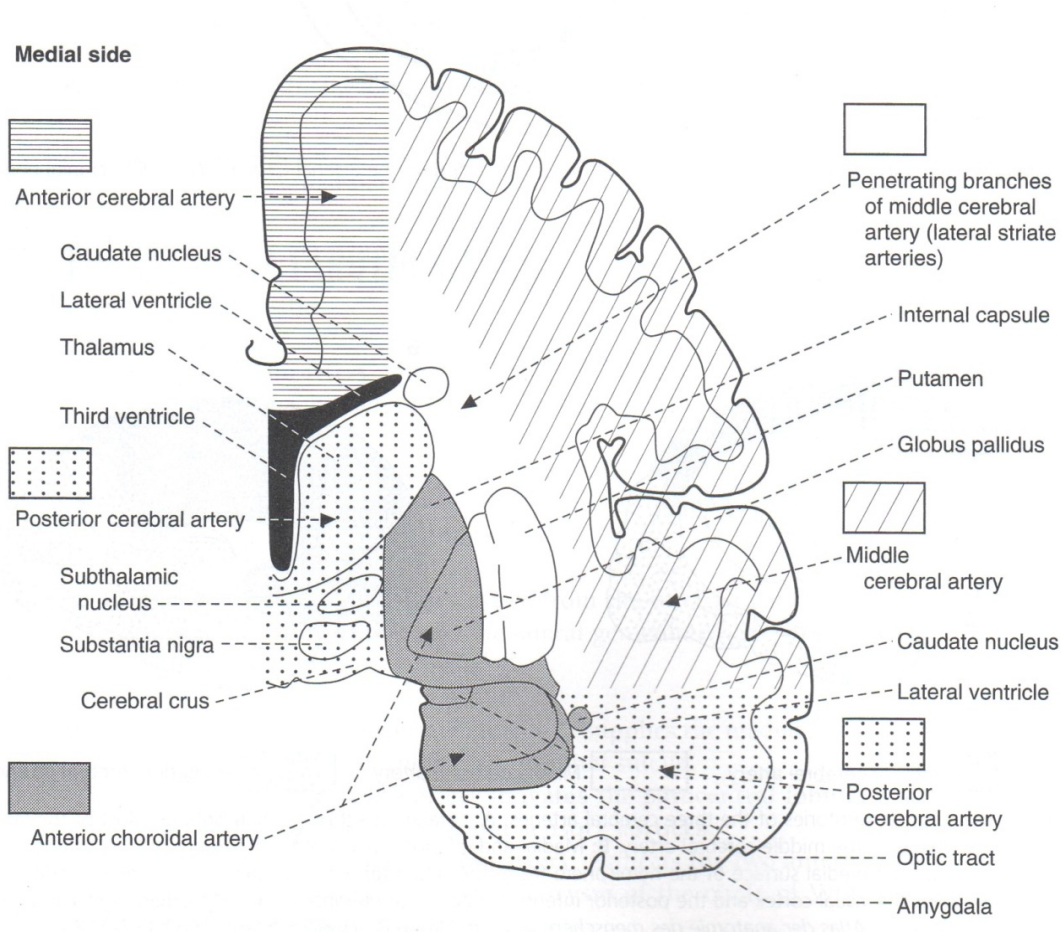

Figure: 3. Coronal section through cerebral hemisphere at internal capsule and thalamus, showing distribution of arterial supply (Fix, 2009, p.40).

3.1.2 VENOUS DRAINAGE

Magoun noted that the main cause of circulatory stasis within the brain is impeded vascular drainage due to the anatomical vulnerability of the venous vessels (1976). These vessels are particularly vulnerable to distortion because unlike other veins, they do not have a muscular wall to maintain their shape.

The venous drainage of the basal ganglia enters the straight sinus via the great cerebral vein (Gray, 2003). Therefore a lesion of the straight sinus could have an effect on the drainage of the basal ganglia. Ninety-five percent of the venous drainage occurs

through the jugular foramina, which can also be restricted due to muscular, dural, or fascial tensions (Frick, 1991). A venous impedance in this area can also affect cranial nerves IX, X and XI as they exit the jugular foramina (Magoun, 1976). This is of particular importance, for Rivera-Martineza et al. (2002) noted various cranial lesions in Parkinson's patients and found an increased incidence of occiptomastoid lesions, which can impede the venous drainage via the jugular vein.

Magoun stated that "any retardation of venous drainage can be a grave predisposing factor to pathology in the central nervous system" (1976, p. 96). Any impedance in venous drainage will cause a backflow, resulting in ischemia to the delicate neural tissue. For this reason it is essential to address the venous flow of the cranium and encephalon of patients with Parkinson's disease.

3.1.3 ANATOMY OF THE VENOUS SINUSES

The venous blood exits from the dorsal aspect of the brain from two interconnected systems of cerebral veins. The Galenic system, or great cerebral venous system, lies deep within the brain and receives venous drainage from the internal structures of the entire brain. The superficial veins, or meningeal veins, comprise the second system, which drains the superficial aspect of the cerebral hemispheres into the superior sagittal sinus (Kaplan & Ford, 1966; Nolte, 1981).

Although these systems join at the straight sinus, for the purpose of this justification the focus will be on the deep galenic system. The paired internal cerebral veins are the chief veins that are found deep in the brain. They are formed from the septal vein and the thalamostriate vein at the interventricular foramen. The thalamostriate vein is responsible for the drainage of most of the thalamus and the caudate nucleus. This vein also receives the choroidal vein, which drains the choroid plexus of the lateral ventricles.

The internal cerebral veins journey through the transverse cerebral fissure and fuse to form the great cerebral vein of the vein of Galen. The great cerebral vein travels a short distance superiorly to join the inferior sagittal sinus together to form the straight sinus. Prior to forming the straight sinus, the great cerebral vein is joined by the paired basilar veins. The middle cerebral vein, which is responsible for drainage of the insula and the inferior portion of the basal ganglia, drains into the basilar veins (Nolte, 1981). The middle cerebral vein follows the medial surface of the temporal lobe, travelling around the cerebral peduncle before joining the basilar veins and great vein. The majority of the venous drainage then exits at the straight sinus, which drains into the internal jugular veins (Frick, 1991; Nolte, 1981).

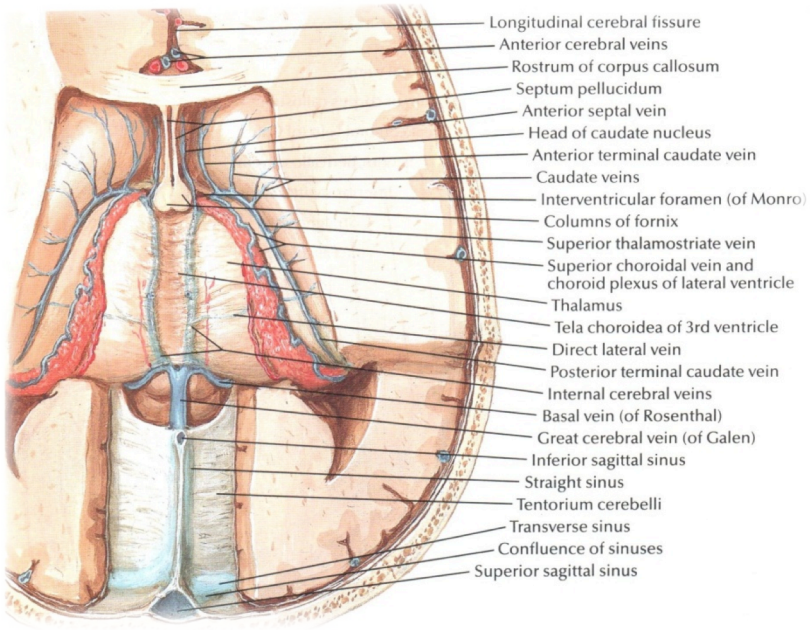

Figure: 4. Deep veins of the brain: Superior view (Netter, 1989)

3.1.4 CEREBROSPINAL FLUID

Still stated that " the cerebrospinal fluid is the highest known element that is contained in the human body and unless the brain furnishes this fluid in abundance a disabled condition on the body will remain" (Still, 1986, p. 39).

The role of cerebrospinal fluid in the pathogenesis or treatment of Parkinson's disease is not commonly discussed in the medical literature. However, the importance of cerebrospinal fluid on the health of the central nervous system is commonly discussed in osteopathic literature. Becker described the cerebrospinal fluid as a dielectric envelope that acts as an insulating substance through which electrical forces occur. When there is a change in chemical makeup or restriction of movement there is a loss of this protective character, and therefore a change in nerve impulses. Becker stated that "as long as cerebrospinal fluid is normal in physical characteristics and freely fluctuating, there will be normal discharge of nerve impulses from the brain and spinal cord" (Becker, 1948a, p.43).

Becker (1948a) hypothesized that pathology can develop due to the loss of the normal dielectric envelope in the brain. In conclusion, Becker stated that the use of cranial manipulative treatment could restore the normal dielectric envelope for the nerve impulses. Based on Becker's conclusions, one can hypothesis, that restoration of the normal dielectric envelope of the cerebrospinal fluid could promote neuroplasticity and neuroprotection within Parkinson's patients.

Although Becker's work is important to the osteopathic profession, one must not place too much credence on it for he was not an expert on cerebrospinal fluid.

In addition to the cerebrospinal fluid's effect on neural impulses, it also has an impact on the physiology of the brain. Magoun stated "should any part of the craniosacral

mechanism fail to function normally, the accompanying cerebrospinal fluid stasis leads to chemical change, accumulation of metabolites, local cell pathology and the perversion of physiology called disease" (1976, p. 96).

On post-mortem evaluation, researchers found cytokines in the cerebrospinal fluid of Parkinson's patients (McGeer & McGeer, 2004). This has led researchers to determine that an inflammatory process occurs within the encephalon of those afflicted with Parkinson's disease, but whether this inflammation is a cause or consequence of Parkinson's disease is undetermined (De Lau & Breteler, 2006; McGeer & McGeer, 2004). However, the presence of pro-inflammatory cytokines and active inflammation can be linked to neuronal loss (McGeer & McGeer, 1997). In fact, Jenner and Olanow (2006) raised the possibility that inflammation may be a primary cause in some cases of Parkinson's disease.

Research suggested that targeting the inflammatory process may have neuroprotective capabilities (De Lau & Breteler, 2006). Magoun (1976) stated that compression of the fourth ventricle can reduce edema and venous congestion, which can be compromised by an occipital malposition. Thus, cranial osteopathy may provide an avenue to address inflammation within the encephalon.

3.2 THE PRINCIPLE OF STRUCTURE GOVERNS FUNCTION

This osteopathic principle suggests that physiological health is dependent upon the anatomical structure being sound. Magoun (1968) stated "even a minimal lack of motion sooner or later will bring about dysfunction or even disease." The cranial bones are the *container* or the structure for the central nervous system. Based on this concept, an osteopathic lesion within the cranial bones may affect the function of the tissue that lies beneath it. This concept is of particular importance for this study, where cranial

osteopathy is employed to effect changes within the basal ganglia and therefore the balance of Parkinson's patients.

The principle of structure governs function within the cranium was outlined in a preliminary study in which magnetic resonance imaging was used to determine changes in the fornix and corpus collosum. The results showed that when pressure was applied externally to the maxilla and the bregma the shape of the fornix changed by 4mm and the corpus collosum changed by 5mm (Pick, 1994). This study highlights the effects that cranial osteopathy may have on the structure that lies beneath the cranial bones. Although these results are promising, it should be noted that a single cadaver was used for this study.

3.2.1 BASAL GANGLIA ANATOMY

The prosencephalon is the forebrain and consists of the hypothalamus, the thalamencephalon (in the diencephalon), the basal ganglia and the cerebral cortex (in the telecephalon). The basal ganglia are a paired structure consisting of two groups of cerebral nuclei that are situated in both the left and right cerebral hemispheres. The basal ganglia includes the amygdala, the caudate, the putmen and the globus pallidus. These nuclei are located laterally and slightly anterior to the thalamus and medial to the lateral ventricles (Frick, Leonhardt, & Starck, 1991).

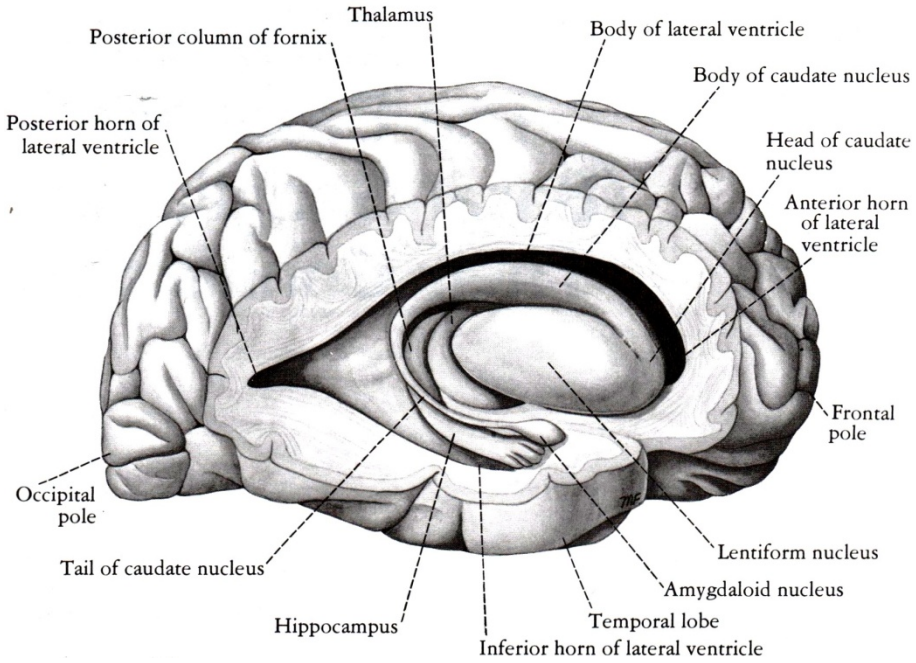

Figure: 5. Lateral view of right cerebral hemisphere dissected to show the position of the basal nuclei (Nolte, 1981).

The thalamus lies central to the basal ganglia. The caudate nucleus of the basal ganglia is a "C" shaped structure that encircles the other nuclei of the basal ganglia. The anterior portion of the caudate nucleus is connected to the more central structure, the putamen, by a series of bridges. Medial to the putamen lies the globus pallidus. The caudate is within close proximity to the lateral wall of the lateral ventricles. At the end of the tail-like caudate lies the almond-shaped amygdale. The amygdala lies within the temporal lobe just anterior to the inferior horn of the lateral ventricle. The amygdala is considered to be both a structure of the basal ganglia and the limbic system. Between the putamen and the thalamus lies the globus pallidus. Together, the caudate and the putamen are clinically known as the striatum. However, in international terminology the globus pallidus is also included in the striatum (Frick, 1991).

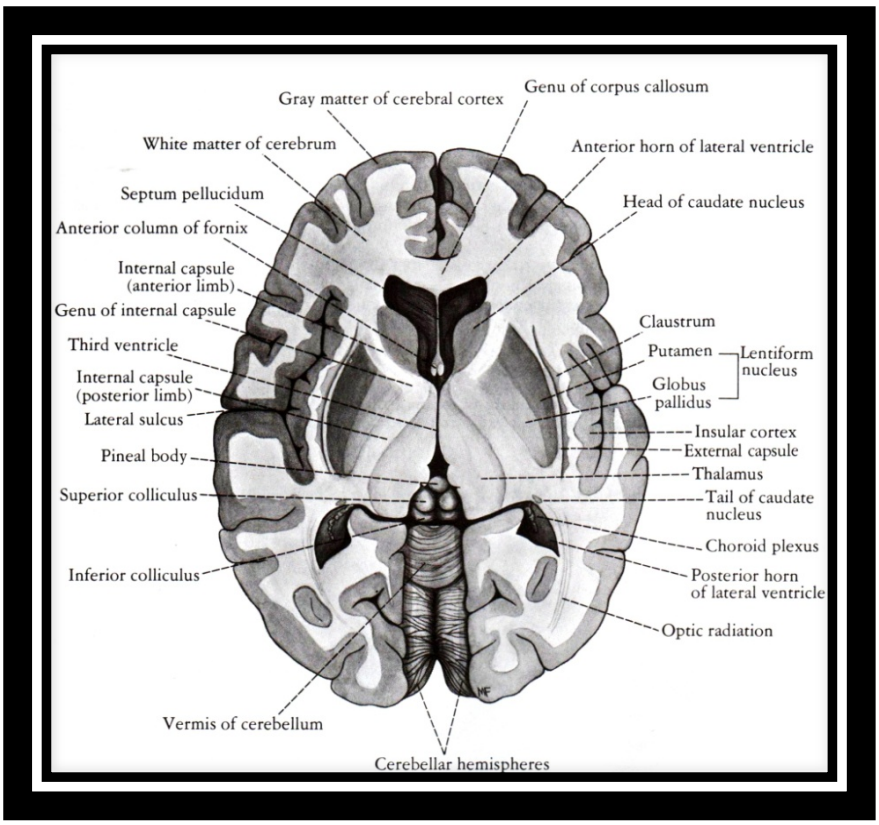

Figure: 6. Horizontal section of the cerebrum showing the relationship between the lentiform nucleus, the caudate nucleus, the thalamus, and the internal capsule (Nolte, 1981).

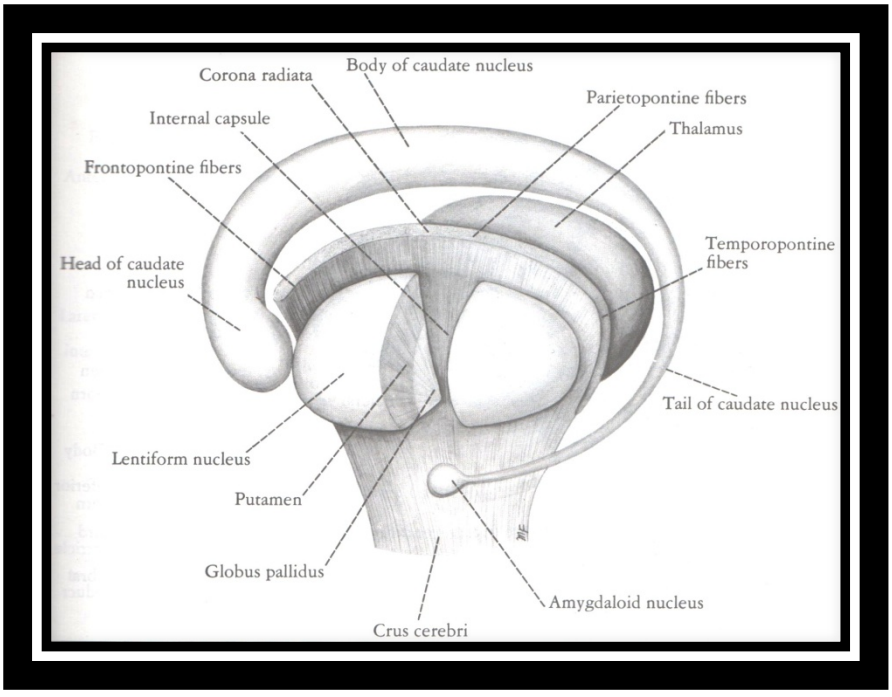

Figure: 7. Diagram showing the relationship between the lentiform nucleus, the caudate nucleus, the thalamus, and the internal capsule as seen from the left side (Nolte, 1981).

Although the basal ganglia lies deep in the grey matter of the telencephalon, it is in relation to the cranial base and therefore may be affected by cranial base dysfunctions. The hypothalamus, which is in anatomical relation to the basal ganglia, is subject to distortion due to occipital malpositions (Magoun, 1968).

3.2.2 EMBRYOLOGY

Although Parkinson's disease is a neurodegenerative disease that primarily occurs in the later years of life, it is significant to have an understanding of the embryonic origins of the basal ganglia. Firstly, knowledge of osteogenesis will provide an understanding of the connections between the structure and its function. Secondly, it may provide an understanding of pre-existing cranial lesions that have had an impact on the health of the central nervous system.

Magoun (1968) stated that pathology due to skull distortion may occur at the perinatal stage. This is due to the malleability of the fetal cranium, which causes cranial integrity to be maintained by the dural membranes and the external periosteum. For this reason, distortion of the tentorium cerebelli and the falx cerebri are quite common during the birth process. Deformation of the dural membranes may have a detrimental impact on the cerebrum and the cerebellum.

3.2.3 DEVELOPMENT OF THE HUMAN BRAIN

The brain develops from the cranial portion of the neural tube and separates into three primary brain vesicles that form the prosencephalon (forebrain), mesencephalon (mid-brain), and the rhombencephalon (hind-brain). The forebrain divides into the telecephalon and the diencephalon during the fifth week of gestation (Moore & Persaud, 2003).

The caudate nucleus and the putamen arise from a single embryological structure, the ganglionic hillock. The ganglionic hillock is divided in two by tracts coming from the internal capsule. Post maturation, the caudate nucleus and the putamen are referred to as the striatum and function as one unit (Moore & Persaud, 2003).

The globus pallidus derives from the diencephalon. The internal capsule pushes the globus pallidus against the putamen and although they are in close proximity their origin and function differ (Frick, 1991).

3.3 THE PRINCIPLE OF AUTOREGULATION

The osteopathic principle of autoregulation denotes that the body has an inherent ability to heal itself. The osteopath's role is to restore mobility in order to provide an optimal environment for the body to move towards homeostasis or balance.

Parkinson's disease is an imbalance of inhibitory and facilitatory neurons, caused by a deterioration of the dopaminergic neurons that project from the substantia nigra of the mid-brain to the striatum (Fix, 2009). This imbalance reduces the activation of the motor cortex, producing the cardinal symptoms of Parkinson's disease. Cranial osteopathic treatment may provide a treatment method to minimize lesions that impede homeostasis. The current research study postulates that providing cranial osteopathic treatment to people afflicted with Parkinson's disease will provide an optimal environment for the brain to autoregulate and move towards homeostasis.

3.3.1 NEUROPLASTICITY

Neuroplasticity, or cortico remapping, refers to the brain's ability to reorganize or repair in order to compensate for injury or pathology. Currently, Parkinson's research has steered towards neuroplasticity, specifically synaptic plasticity of the dopamine producing neurons of the basal ganglia (Calabresi, Picconi, Parnetti, & Di Filippo, 2006).

Picconi et al. (2003) demonstrated that levodopa was able to restore a form of synaptic plasticity referred to as long-term depression. There has been recent preliminary research that suggests exercise effects neuroplasticity in Parkinson's disease (Hirsch & Farley, 2009).

Similar to neuroplasticity, the osteopathic concept of autoregulation denotes the ability for the body to repair and reorganize to facilitate health. Current medical research on neuroplasticity in Parkinson's disease substantiates the osteopathic concept of autoregulation. This provides an avenue into the justification of the hypothesis that by creating a more balanced environment for the brain it will have a more optimal environment for neuroplasticity to occur.

This hypothesis is highlighted in Frymann's (1992) research on children with varying neurological impairments including poor academic performance, behavioural issues, abnormal neuromotor function and delayed development or learning. The results of this single-blinded, cross-over study showed a significant increase in scores on the Houle's Profile of Development test ($P<.01$). This study highlights that osteopathic treatment has an effect on the function of the central nervous system.

The concept of reorganization or cortico remapping may also be utilized by promoting an environment that would give the body a greater opportunity to heal itself by recruiting dopamine from other sites. Research has discovered that dopamine is also produced within the frontal lobe and the adrenal glands (Rakshi et al., 1999). Although adrenal gland transplantation into the brain has had varying results in curbing symptoms of Parkinson's, the recruitment of dopamine from the frontal lobe may provide different results.

3.3.2 NEUROPROTECTION

Neuroprotection is an essential component for consideration in a degenerative disease such as Parkinson's. Unfortunately, the clinical symptoms of Parkinson's disease often do not appear until 60%-80% loss of substantia nigra neurons (Adler & Ahlskog, 2000). This statistic demonstrates the importance of the need for neuroprotection of the remaining neurons within the basal ganglia. Loss of postural stability in Parkinson's patients is associated with an advancing disease process. By providing an environment for neuroprotection, cranial osteopathy may contribute to the slowing of disease progression and postural instability.

In theory, eliminating osteopathic cranial lesions may contribute to the removal of neuronal damaging free radicals.

3.4 THE BODY AS A FUNCTIONAL UNIT

The human body is a functional unit with the brain having a leading role as the center of control. Magoun stated that "the central nervous system and the pituitary gland control the rest of the body therefore structural deviations in the cranium are of the utmost importance" (1968, p. 186). From this statement it can be postulated that cranial osteopathic treatment has a global effect on the neural system of the body.

Osteopathy works under the premise that functional health of the central nervous system is influenced by the primary respiratory mechanism outlined by William Garner Sutherland. This is a recognized osteopathic concept considered to be responsible for the maintenance of homeostasis (Magoun, 1976). One of the phenomena of the primary respiratory mechanism is the mobility of the intracranial and intraspinal membranes or the corelink. Anatomically, the dura lines the interior of the cranium and attaches to the foramen magnum, the first to third cervical vertebra, and the second sacral segment

(Gray, 2003). Due to the continuity of the dural membrane a cranial lesion may have an effect at the sacrum (Druelle, 1985).

The anterior/posterior and the posterior/anterior lines of gravity begin at the foramen magnum of the occiput. The center of gravity exists at the level of the third lumbar vertebra, where the anterior/posterior and posterior/anterior lines cross. In order to maintain the center of gravity these two lines of gravity must be in balance. It is therefore essential that the occiput be in alignment with the rest of the body (Wernham, 1956).

Parkinsonian patients often present with a concavity to the less affected side, causing a scoliotic posture (Nutt, et al., 1992). Unilateral spasm or hypertonicity of SCM will draw the ipsilateral occipital condyle anterior.

Another typical presentation of Parkinsonian posture is the “stooped” or “simian” position, in which the body moves towards flexion (Nutt, et al., 1992). Cervical flexion is countered by the attempt to correct the horizontal line of vision causing the atlas to draw anterior on the posterior occipital condyles.

Sustained tension of the cervical musculature attaching to the occiput and temporal bones can cause dysfunction at the cranial base. Rectus capitus posterior minor and ligamentum flava and ligamentum nuchae has filmaments which attach to the dura (Mitchell, Humphreys, & O'Sullivan, 1998). This suggests that there is a relationship between the meninges and spinal dynamics.

The fascial chains of the body attach onto the cranium. Paoletti (2006) stated that the external fascia chains play a role in maintaining posture, while the central fascial chain plays a role in supporting functions. The cervical fascia attaches to the base of the

cranium and is continuous with the dura via the foramina (Page, 1952). A pull on the fascia from a cranial lesion of the base will change the reciprocal tension membrane causes changes to the position of the sacrum (Magoun, 1976).

3.4.1 OSTEOPATHIC TECHNIQUES

The cranial osteopathic techniques that were utilized in the current study were chosen for their possible influence on circulation. Increasing circulation may assist in decreasing inflammation and toxicity which could promote a healthier environment within the encephalon. This could provide an avenue for autoregulatory action, such as re-mapping and optimizing the health of basal ganglia. Although imperial evidence does not exist to substantiate the efficacy of these cranial techniques, they are frequently employed in osteopathic treatment to achieve circulatory effects. The following table depicts the techniques used within the current study, their indications and possible outcomes as taught by the Canadian College of Osteopathy (Druelle & Forget, 2000).

Table 1: Techniques and Proposed Effects

Techniques	Indications/Proposed Effects
Venous Sinus	Increases drainage Increase vitality
EV4, Posterior fossa	Opens membranes of the base of the cranium Restores straight sinus, increasing drainage
CV4	Increase fluid drive Decrease inflammatory process Influence the autonomic nervous system Balances membranes
Parietal lift	Balance membranes and fluids Release membranous sutural lesions of the vault Decompresses the encephalon against the base of the cranium To access the sensorimotor cortex
Bilateral temporal rocking	Stimulation of the thalamus and central nuclei Harmonization of membranes and fluids Increase vitality Increase venous drainage Increase resiliency of the encephalon

Lateral Ventricles	Increase vitality Increase systemic activity of the brain Increase production of cerebrospinal fluid Balance membranes and fluids Restore ventricular pumping function

3.5 CONCLUSION TO OSTEOPATHIC JUSTIFICATION

Cranial osteopathy is a unique therapy in that may have a direct influence on the central nervous system. Therefore, cranial osteopathy may have greater efficacy on a pathology that is rooted in the central nervous system such as Parkinson's disease.

Although the causes of Parkinson's disease are still unknown, one of the leading hypotheses in the etiology of Parkinson's disease is toxicity. Cranial osteopathy contributes to the elimination of cranial restrictions thus providing an optimal environment for homeostasis to occur. This optimal environment would include the increase in circulation and venous drainage which may promote the removal of toxins. In turn, this may lead to a reduction of inflammation within the basal ganglia. Reducing inflammation could promote an environment of neuroprotection for the remaining neurons.

The effect of cranial osteopathy on the circulatory flow and normalization of cerebrospinal fluid may allow for neuroplasticity to take place within the encephalon. Promoting normalization of the circulatory, venous and cerebrospinal fluid may provide an opportunity for the basal ganglia to function in an effective way, which could in turn increase postural stability in Parkinson's subjects.

4 CHAPTER FOUR: RESEARCH METHODOLOGY

4.1 TYPE OF RESEARCH

This study was a randomized single blinded between-group design which examined the effect of cranial osteopathy on the balance in Parkinson's subjects. It consisted of a control group that entailed exercise only and an experimental group that received exercise and four cranial osteopathic treatments. The tool utilized to measure balance was the Berg Balance Scale. A pre-intervention and post-intervention assessment using the Berg Balance Scale was administered to both groups. In addition an osteopathic evaluation of the cranial system was administered to both groups pre-intervention and post-intervention by the evaluating researcher.

4.2 TARGET POPULATION

The target population was comprised of eleven volunteers (five male and six female) between the ages of fifty-five and eighty-two that had a previous diagnosis of idiopathic Parkinson's disease. A power analysis performed for the research proposal of this study determined a power of sixteen (8 per group) was needed to show statistical power (80% and an alpha = 0.05). A number of studies employing the Berg Balance Scale were used to determine this power. A synthesis of these papers revealed that Parkinson's patients in earlier stage of Parkinson's, determined using the Hohen and Yahr scale or the Unified Parkinson's disease rating scale, scored around 50 on the Berg Balance Scale and that physical exercise produced a negligible improvement in the Berg Balance Scale. It was felt that an improvement of two points on the Berg Balance Scale would show significant change.

4.3 INCLUSION CRITERIA

Participants included in this study fit the following criteria:

- Primary diagnosis of idiopathic Parkinson's disease by a physician;
- Can ambulate with or without an assisting device;
- Between the ages of 55 and 90 years old;
- Signed consent form, See Appendix A: Consent Form
- Completed medical questionnaire; rating between stage II and stage IV on the Hoehn and Yahr Scale. See Appendix B: Hoehn and Yahr Scale.

4.4 EXCLUSION CRITERIA

If any of the following criteria were present in potential subject they would have been excluded from this study.

- Central nervous system problems other than Parkinson's: stroke, Multiple Sclerosis, Muscular Dystrophy;
- Traumatic brain injury;
- Previous osteopathic treatment;
- Receiving any kind of other manual therapy concurrently to avoid potential confounds;
- Diagnosed with balance disorder;
- History of encephalitis, cerebral vascular disease, neoplasm;
- Uncorrected visual impairment;
- Altered medication during study;
- Any condition that would contraindicate manual treatment or the ability to complete the outcome tasks.

4.5 INDEPENDENT VARIABLE

The independent variable in this research was the exercise protocol and cranial osteopathic treatment plus the exercise protocol. Although guidelines had been set out to ensure each subject in the experimental group received some uniformity within the treatment, each individual was different and therefore various techniques were employed to the subjects.

One of the main criticisms of cranial osteopathic research is the lack of inter-rater reliability and intra-rater reliability (Hartman & Norton, 2002). In order to curb some of this concern an independent Osteopath validated the hands of Stacey Hauserman and Thomas Hein, prior to commencement of this study. On September 8th 2009 at the Canadian College of Osteopathy, Toronto, the researchers were asked to evaluate four structures and write down the lesions they perceived. Brad McCutcheon, D.O.M.P, Principle of the Canadian College of Osteopathy, had previously evaluated the volunteer and recorded his findings. The findings were compared and Brad McCutcheon determined the researcher's hands to be validated.

4.6 DEPENDENT VARIABLES

The only dependent variable within this research was the Berg Balance Scale scores for the subjects' pre-assessment and post-assessment.

4.7 CONTROL AND EXERCISE

Each subject participated in four weekly, one hour exercise sessions that were published by the Parkinson's Society of Canada. (See Appendix C: Exercise Protocol) The exercise sessions were run by an independent physiotherapist, Hermina Vas. The exercise regimen was included primarily as an incentive to encourage participation and

was not the primary variable under study. As such the type of exercise was restricted to a basic and commonly recognized level for all subjects in the study.

The physiotherapist was instructed to assist each subject through the exercise protocol by reading directly from the instruction booklet. The first section included a brief description on good postural habits, as outlined on page three of the exercise package. Included in this description were three demonstrations of healthy posture in standing, sitting, and supine positions. Three postural alignment exercises were also preformed and included, spinal roll downs while sitting, rhomboid and middle trapezius strengthening with prone, forward head postural alignment.

The second portion included seven stretching exercises. The physiotherapist gave the exact instructions written in the guidelines. If the subject needed clarification the physiotherapist would stray from the specific wording to assist the subject. The stretching exercises targeted the following muscles; spinal rotators, latissimus dorsi, pectoralis major, anterior deltoids, iliopsoas and quadriceps, soleus and gastrocnemius. All stretching was maintained for 30 seconds with breath, within the subjects' limitations.

The third portion of the exercise protocol included the strengthening portion. A series of movements with isometric contraction targeting the upper body and quadriceps were preformed, followed by deltoid strengthening with a two pound weight. A series of movements to target the lower body were then preformed while holding onto a chair for support. Lastly, a side stepping motion and marching activity were performed

The goal was for all subjects to be able to complete this exercise protocol however the physiotherapist removed the side stepping component for two of the subjects due to concern over safety. This did not seem to affect their outcome measures.

4.8 MEASURING TOOL

The Berg Balance Scale (BBS) is an objective functional performance assessment which measures a person's ability to maintain stability while performing tasks. It is commonly used for the elderly population, and people with Parkinson's disease, stroke, and other various balance impairment. The Berg Balance Scale is an inexpensive assessment tool that can be performed in any clinical setting. It is often used to evaluate the risk of falling, and to monitor progression of physical therapy (Thorbahn & Newton, 1996).

4.8.1 DESCRIPTION

The BBS is a 14 item scale that can be performed within a 20 minute time interval. Each item on the scale is scored out of five points; zero being the lowest level of function and four being the highest. There is a total score of 56. All of the 14 items on the BBS are task orientated, and come with a small set of instructions that is relayed to the subjects performing the tasks. The tasks progress from sitting to standing, standing with feet together, to tandem standing and single-leg standing. Lower ratings are given if the subject needs assistance, specific supervision, cueing, and if the time or distance measurements are not met. A detailed description of the Berg Balance scale can be found in Appendix C: Berg Balance Scale

Two studies on the BBS on the predictability of falls in the elderly confirmed that those who scored lower than 45 were more likely to fall than those who scored above 45 (Kornetti, Fritz, Chiu, Light, & Velozo, 2004; Thorbahn & Newton, 1996).

4.8.2 BERG BALANCE SCALE VALIDITY

A review of literature on various functional balance assessment tools indicated the Berg Balance Scale is used as a *gold standard* to validate other balance measures

(Langley & Mackintosh, 2001). It was the only functional performance test that correlates with the United Parkinson's Disease Rating Scale, Forward Functional Reach Test, Backward Functional Reach Test, and the Timed Get Up and Go Test (Brusse, Zimdars, Zalewski, & Steppen, 2005). The Berg Balance Scale has both high construct validity (r = 0.62-0.94) and excellent inter-rater reliability (ICC = 0.98) (Berg, Maki, Williams, Holliday, & Wood-Dauphinee, 1992)

4.8.3 EQUIPMENT USED

The Berg Balance Scale requires a step stool that is 23cm from the floor, a chair with arms (the seat being 46cm from the ground), a stopwatch, a 40cm ruler, and a slipper. Each subject used the same equipment and all instructions were administered by the Assessor.

Figure: 8. PT Fitness stopwatch, product number 84-0794-0, 1/100th second accuracy. Used in a number of the tasks within the Berg Balance Scale.

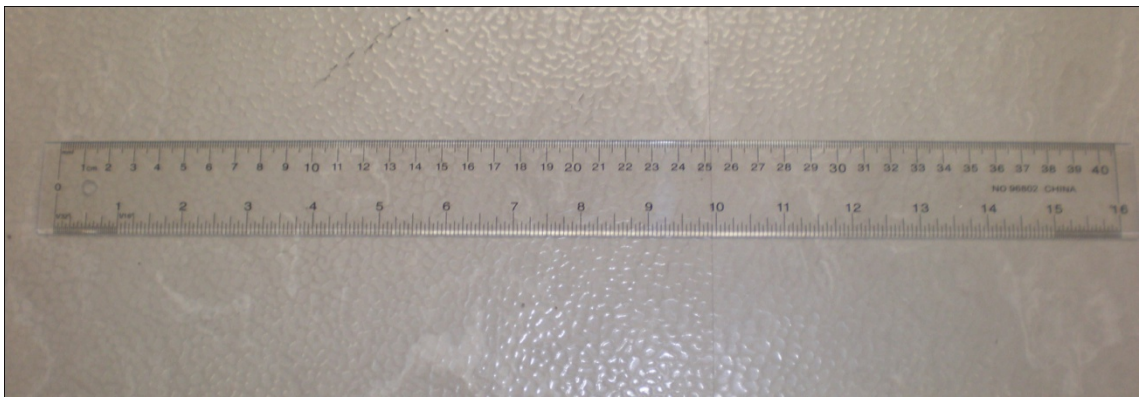

Figure: 9. 40 cm ruler used to measure length of arm reach measured in centimeters as listed in item eight in the Berg Balance Scale

Figure: 10. Ikea step stool 23 cm from ground as used in item twelve on the BBS. Seen with Ikea slipper used in item nine in the Berg Balance Scale

4.9 RECRUITMENT METHODOLOGY

Recruitment occurred within the greater Toronto area. Contacts were initially made with Dr. Guttmann of the Movement Disorder Center at Markham Stouffville

Hospital and Dr. Adams at Baycrest Hospital. Both neurologist's were initially enthusiastic regarding this study and offered to refer patients once the study was approved. Further contact was made after the research was approved for a study. Regretfully, Dr. Guttmann and Dr. Adams were unable to act on their earlier enthusiasm due to the lack of a formal ethics review board at the College of Osteopathy and a possible conflict with a rehabilitation study at the Movement Disorder Centre. Physician letters were sent out to various physicians in the north end of Toronto without any response. (See Appendix D: Physician Letter) Business cards with contact information and study details were placed with flyers in numerous health centers, assisted living homes and retirement homes within Toronto. (See Appendix E: Business Card, See Appendix F: Recruitment Notice) A few calls were received from this source but the potential subjects felt the distance was too far to travel. Personal contact was made with the organizer of Sutherland-Chan Massage School Parkinson outreach clinic. The organizer was concerned about breaching their privacy policies but she did agree to hand out business cards containing the study details on an individual basis. However, no subjects were attained through this source. The researchers attended the 2010 Parkinson's Walk in Toronto and left cards on the information tables.

Both researchers emailed their respective data base of patients to inform them about the study. The patients were asked to pass on the information to anyone that they believe might benefit from this research. Seven inquiries and four subjects were attained from this source.

Direct contact was made with the organizers of five separate Parkinson's support groups in the Greater Toronto Area. From these contacts an arrangement was made to

make a presentation to the Thornhill Parkinson's support group. Many of the participants in the group were interested in becoming subjects in the current study.

Overall, the majority of inquires and subsequent subjects were attained from direct referral from both former subjects and the researchers personal database. In total 21 people called to inquire about the study. Three of which were declined due to ambulatory issues because they would not be able to complete the outcome measures. One potential subject was declined due to a possible Parkinson's Plus syndrome. A diagnosis of idiopathic Parkinson's disease was specified for inclusion into the study. Another subject was accepted after the initial screening, but did not show up for her first appointment. The clinic receptionist reached this potential subject a week later and was told she was longer interested in participating. The remaining five potential subjects were either unable or unwilling to attend five consecutive weeks of the study.

4.10 TELEPHONE SCREENING

Interested subjects called the clinic number and received a semi-scripted interview by the clinic receptionist. (See Appendix G: Telephone Interview) This interviewed determined whether the potential subject met the initial criteria for the study. If the subject did not meet the criteria outlined in the inclusion and exclusion section, they were thanked for the call and rejected for the study. If the potential subject met the criteria they were scheduled for their first appointment.

4.11 RANDOMIZATION

During the initial appointment each patient was asked to randomly choose an envelope from a table top, where the envelopes had been placed an equal distance apart. Each envelope contained either a red or a black card. Eight of each color playing card

were placed facing down in envelopes and sealed at the commencement of this research. The black cards represented the experimental group and red cards the control group.

Two researchers participated in the study. A coin toss prior to commencement of this research determined that Stacey Hauserman was to be the treating Osteopath (Osteopath 2) and Thomas Hein was to be the Assessor (Osteopath 1).

After the subject selected the envelope they handed it to the clinic receptionist. The receptionist passed the envelope to Osteopath 2 who then removed the card from the envelope and recorded the participant's data into the data log. (See Appendix H: Data Log)

Figure: 11. Envelopes for subject randomization

4.12 PROCEDURE

Once the subjects had passed the initial telephone screening, the receptionist booked the subject in for the next available appointment in the study. The subject then

arrived at 9:45am on the allotted date. They were then asked to fill out a medical questionnaire and an informed consent form in the clinic waiting room. (See Appendix I: Medical Questionnaire, See Appendix A: Informed Consent) Osteopath 1 (the assessor) introduced himself and brought the potential subject into the physiotherapy clinic. Osteopath 1 reviewed the medical history to ensure there were no conflicts with the study. He then gave a brief description of the study and the expectations of both the patient and the researchers were outlined. The subject was informed of the privacy policy, their right to withdrawal from the study at any time and forfeit of participation if there were any changes that excluded them from the study. The subjects were also informed of the need for their medication to remain consistent throughout the duration of the study. If the subject's medication required alteration during the study they were asked to disclose this information to the researchers. A copy of the privacy policy statement compliant with PIPEDA guidelines was given to the subject. (See Appendix J: Privacy Policy)

Osteopath 1 preformed an evaluation to determine the subjects staging of the Hoehn and Yahr Scale. The Hoehn and Yahr Scale is a simple, well known rating scale that determines the stage of Parkinson's disease. It was used in this study to determine that some postural instability was present in the subjects. If the subject's staging was within II to IV they were accepted into the study.

Osteopath 1 then administered the 14 item Berg Balance Scale in the main gym of the PhysioActive clinic. The subject was then asked to choose an envelope from a table top and the results of the randomization were recorded in the data log as described in the above section. Osteopath 1 was not privy to this information. Osteopath 2 then

determined the dates and times for the remaining appointments. If the subject was assigned to the experimental group they were asked to come back for four consecutive weekly two hour sessions. If they were assigned to the control group they were asked to come back for four consecutive weekly sessions for one hour each week. The last session included the post-evaluation using the Berg Balance Scale and an osteopathic assessment preformed by Osteopath 1.

Osteopath 1 escorted the subject to the treatment room where an osteopathic evaluation was preformed. Upon completion the subject was brought back into the gym for their first exercise session with an independent physiotherapist Hermina Vas, Registered Physiotherapist.

The information of the subjects remaining appointments were recorded by Osteopath 2 on a patient appointment form and handed to the subject privately. (See Appendix K: Appointment Form)

If the subject was placed in the experimental group, on their next appointment they were greeted by the receptionist and escorted to the treatment room where Osteopath 2 was waiting to commence the subject's first 60 minute cranial osteopathic treatment. Upon completion of the first treatment, Osteopath 2 escorted the subject out to the gym for their second exercise session with the physiotherapist. This sequence was repeated for the subject's third and fourth visit to the clinic.

It should be noted that in order to ensure blinding Osteopath 1 was not on the premise during the subjects' second to fourth appointments. However, Osteopath 1 was on site during the first and fifth appointments. During the study hours a sign reading Parkinson's Study in Session was placed on the door of the treatment room regardless of

whether an osteopathic treatment was being preformed. Osteopath 1 was privy to the knowledge that the sign would always be on the door during study hours. This was to ensure that if Osteopath 1 walked by the treatment room he would not know whether a treatment was being preformed. Also prior to commencement of the study Osteopath 1 was informed that Osteopath 2 would be in the treatment room during the study hours. This ensured that if by chance Osteopath 1 were to accidently see Osteopath 2 at the clinic it would not confound blinding.

On the subject's final visit the subject was escorted to the treatment room to be met by Osteopath 2 for the fourth and final cranial osteopathic treatment. At the end of the treatment the subject was asked to go back to the waiting room and take a seat. Fifteen minutes after the treatment, Osteopath 1 met the subject in the waiting room and escorted him/her to the gym for a post-study assessment with the Berg Balance Scale. This was followed by an osteopathic assessment to determine the severity of lesions and vitality. The data was recorded on the osteopathic assessment form. (See Appendix L: Osteopathic Assessment Form)

If the subject was placed in the control group, they returned for their second appointment and were greeted by the receptionist. The receptionist then escorted the subject to the gym for their second exercise session with the physiotherapist. Upon completion of each session the subject escorted themselves out. On the final appointment the subject was greeted by the receptionist and asked to take a seat. Osteopath 1 then escorted the subject into the gym for the post-assessment of the Berg Balance Scale, followed by an osteopathic assessment.

The exercise protocol was administered on an individual basis in order to ensure subject blinding.

4.13 DESCRIPTION OF CLINICAL ENVIRONMENT AND TREATMENT ROOM

This research was held at PhysioActive Clinic, 1450 Clark Ave., Thornhill, Ontario. The clinic waiting room was bright and large with a reception area 15 feet from the front door. A long hallway leading to the gym and treatment rooms extends off of the reception area.

Figure: 12. Hallway extending to treatment room

The gym is a large mirrored room, equipped with two treatment tables and various exercise equipment. It was in this room that the subjects preformed the Berg Balance Scale as well as the weekly standardized exercise program.

Figure: 13. Gym where exercise protocol was administered

All osteopathic treatments were preformed in a treatment room at the end of the hallway. The room is large and equipped with a Kor Innovations Euro Lift 2000 hydraulic table, a small desk, a stool and a chair. The subjects often used the chair to remove their shoes before getting onto the table. The room had high windows for privacy and natural light.

Figure: 14. Treatment room

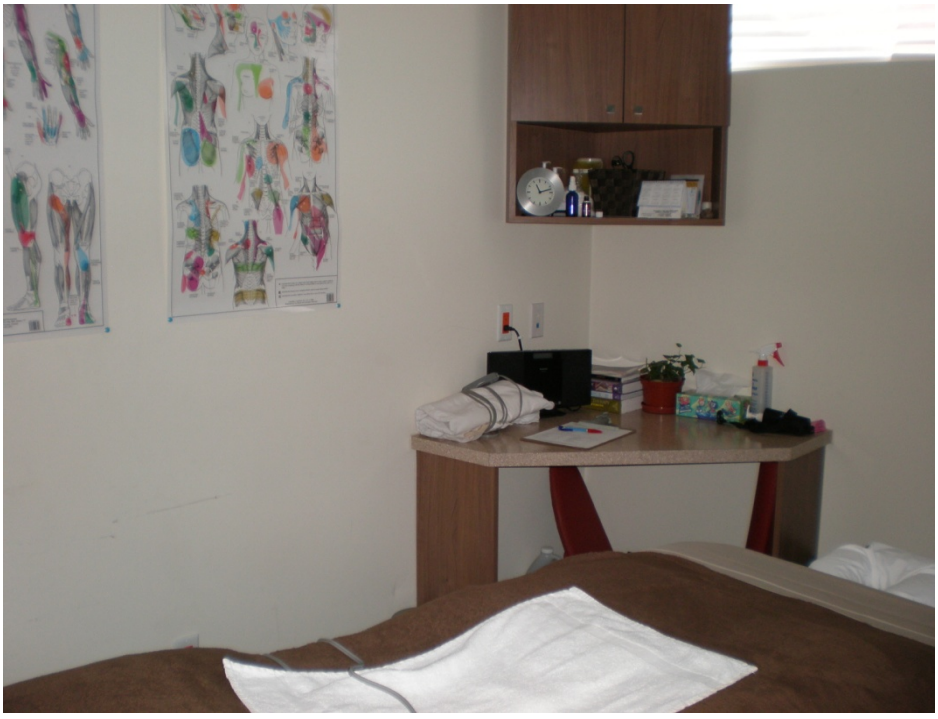

Figure: 15. Treatment room showing desk

4.14 OSTEOPATHIC TREATMENT

The subjects assigned to the experimental group received four, weekly, one hour exercise sessions supervised by an independent physiotherapist. In conjunction to the exercise protocol the experimental group also received four, one hour cranial osteopathic treatments. The first treatment was aimed at clearing non-physiological lesions that were impeding the craniosacral mechanism. This included, but was not limited to the pelvis and the spinal column. This was followed with a venous sinus technique to increase vitality of the primary respiratory mechanism and to increase drainage and encourage optimal circulation. All techniques listed in this section are written in further detail in Appendix N: Technique Descriptions. Strict adherence to the Canadian College of Osteopathy (CCO) methodology was followed. See Appendix O: Canadian College of Osteopathy Methodology.

The second treatment addressed the cranial sphere and was based on the individual's cranial lesions, prioritized using the CCO methodology. Therefore, non-physiological without respect to the axis lesions within the cranial base, C1 and C2 were addressed. In addition, a posterior fossa technique, an EV4 and CV4 were administered to increase overall fluidic drive.

The focus of the third treatment was to restore the cranial axis and increase vascular flow. A parietal lift was used during this treatment to balance membranes, access the sensorimotor cortex and to apply a pumping effect to the encephalon.

The final treatment addressed the endocranium in some capacity. A bilateral temporal rocking technique was used to stimulate the central nuclei and the thalamus. This was followed by lateral ventricle technique to increase the systemic activity of the

brain (Druelle & Forget, 2000). A local, regional and global integration was given following each treatment.

Upon completion of the final treatment, the subject was re-assessed by the Assessor using the Berg Balance Scale and an osteopathic evaluation.

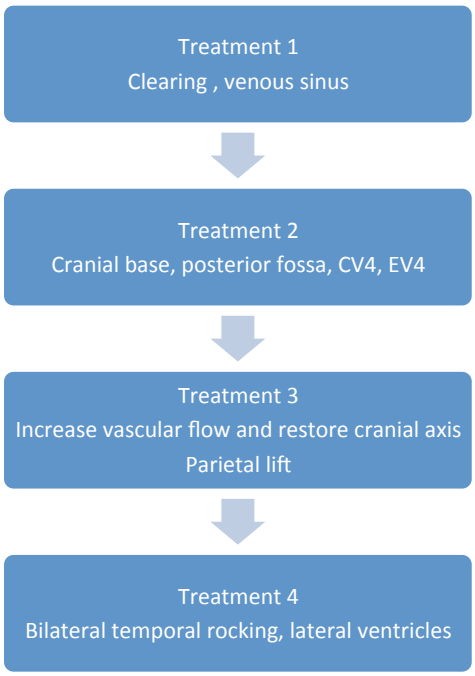

Figure: 16. Treatment outline

4.15 ETHICS

In this study, each subject was required to read and sign an informed consent form. This form indicated that the subject was able to terminate their participation at any time, for any reason, without consequence. The subject was informed that they were randomly placed into either a control or experimental group and that both groups received a form of treatment. Furthermore, subjects were also informed that at no time before, during or after their participation were they to be made aware of the treatment

group to which they had been assigned. All interventions were thought to be of minimum risk of harm.

All of the subjects' personal information is and will remain confidential. The subjects were asked to read a privacy statement that was compliant with the PIPEDA guidelines. The interventions used were low risk to health and the inclusion/exclusion criteria further was designed to reduced risk to subjects with significant co-morbidities. There was no monetary incentive used to attract subjects. The author declares that no conflict of interest exists.

5 CHAPTER FIVE: DATA ANALYSIS AND RESULTS

The data for the current study was analyzed by statistician Peter Lewycky. Mr. Lewycky also provided the pre-analysis for the proposal portion of this study. All analysis, tables and graphs were done using SPSS Release 16. A letter from Peter Lewycky, validating his contribution to this work can be found in Appendix P: Statistician Letter.

The data for the hypothesis that cranial osteopathy will improve balance in Parkinson's subjects were analyzed using the Wald Chi Square method of analysis. In the primary analysis, results were analyzed to determine the effect of cranial osteopathic treatment on Berg Balance scale (BBS) in subjects in the control group versus the experimental group. Both groups saw improvement in the BBS score (BBS pre to BBS post) whether treatment was exercise or exercise plus cranial osteopathic treatment. However, improvement was greater in the experimental group receiving osteopathic treatment in conjunction with exercise. This result was significant at the p=.028 level.

Table 2: Hoehn and Yahr Rating Per Group

			HoehnYahr			
			2		3	
			BBSpre	BBS post	BBSpre	BBS post
Treatment	Control	Valid N	2	2	2	2
		Mean	53.5	55.5	44.0	47.0
		Standard Deviation	.7	.7	9.9	7.1
		Minimum	53	55	37	42
		Maximum	54	56	51	52
	Experimental	Valid N	4	4	3	3
		Mean	53.8	56.0	49.0	53.0
		Standard Deviation	1.3	.0	6.2	4.4
		Minimum	52	56	42	48
		Maximum	55	56	54	56

Table 3: Generalized Linear Model Information

Dependent Variable	BBS post
Probability Distribution	Normal
Link Function	Identity

Table 4: Categorical Variable Information

			N	Percent
Factor	Treatment	Control	4	36.4%
		Experimental	7	63.6%
		Total	11	100.0%
	HoehnYahr	2	6	54.5%
		3	5	45.5%
		Total	11	100.0%

Table 5: Continuous Variable Information

		N	Minimum	Maximum	Mean	Std. Deviation
Dependent Variable	BBS post	11	42	56	53.45	4.547
Covariate	BBSpre	11	37	55	50.64	5.767

Table 6: Tests of Model Effects

	Type III		
Source	Wald Chi-Square	df	Sig.
(Intercept)	24.541	1	.000
BBSpre	85.519	1	.000
Treatment	4.825	1	.028
HoehnYahr	1.084	1	.298
Treatment * HoehnYahr	2.931	1	.087

Participants in the study were classified on the Hoehn and Yahr rating scale of Parkinson's severity. All participants were at either stage two or stage three, indicating some postural instability. The Hoehn and Yahr rating was analyzed to determine whether cranial osteopathic treatment may have a differential effect on BBS depending upon severity level of Parkinson's disease. The statistical test returned a probability value of p=0.87, suggesting no interaction between treatment and severity level of Parkinson's disease. This result indicates that cranial osteopathic treatment on BBS shows positive improvement regardless of the staging level of Hoehn and Yahr.

An additional analysis was performed to determine whether cranial osteopathic treatment within a given severity level of Parkinson's may result in greater improvement.

This test was almost statistically significant at p=.055. While one cannot conclude a true effect, the data would suggest that the difference in treatment effect is most pronounced at the greater Hoehn and Yahr severity level 3.

The following three tables are pairwise comparisons of estimated marginal means based on the original scale of the dependent variable BBS post scores.

Table 7: Pairwise Comparison 1

(I) Treatment	(J) Treatment	Mean Difference (I-J)	Std. Error	df	Sequential Bonferroni Sig.	95% Wald Confidence Interval for Difference	
						Lower	Upper
Control	Experimental	-1.45[a]	.659	1	.028	-2.74	-.16
Experimental	Control	1.45[a]	.659	1	.028	.16	2.74

a. The mean difference is significant at the .05 level.

Table 8: Pairwise Comparsion 2

(I) Hoehn Yahr	(J) Hoehn Yahr	Mean Difference (I-J)	Std. Error	df	Sequential Bonferroni Sig.	95% Wald Confidence Interval for Difference	
						Lower	Upper
2	3	.86	.822	1	.298	-.76	2.47
3	2	-.86	.822	1	.298	-2.47	.76

Table 9: Pairwise Comparison 3

(I) Treatment*HoehnYahr	(J) Treatment*HoehnYahr	Mean Difference (I-J)	Std. Error	df	Sequential Bonferroni Sig.	95% Wald Confidence Interval for Difference	
						Lower	Upper
[Treatment=1.00]* [HoehnYahr=2]	[Treatment=1.00]* [HoehnYahr=3]	1.97	1.224	1	.427	-.42	4.37
	[Treatment=2.00]* [HoehnYahr=2]	-.33	.866	1	1.000	-2.03	1.37
	[Treatment=2.00]* [HoehnYahr=3]	-.59	.972	1	1.000	-2.50	1.31
[Treatment=1.00]* [HoehnYahr=3]	[Treatment=1.00]* [HoehnYahr=2]	-1.97	1.224	1	.427	-4.37	.42
	[Treatment=2.00]* [HoehnYahr=2]	-2.30	1.129	1	.207	-4.52	-.09
	[Treatment=2.00]* [HoehnYahr=3]	-2.57	.986	1	.055	-4.50	-.63
[Treatment=2.00]* [HoehnYahr=2]	[Treatment=1.00]* [HoehnYahr=2]	.33	.866	1	1.000	-1.37	2.03
	[Treatment=1.00]* [HoehnYahr=3]	2.30	1.129	1	.207	.09	4.52
	[Treatment=2.00]* [HoehnYahr=3]	-.26	.841	1	1.000	-1.91	1.39
[Treatment=2.00]* [HoehnYahr=3]	[Treatment=1.00]* [HoehnYahr=2]	.59	.972	1	1.000	-1.31	2.50
	[Treatment=1.00]* [HoehnYahr=3]	2.57	.986	1	.055	.63	4.50
	[Treatment=2.00]* [HoehnYahr=2]	.26	.841	1	1.000	-1.39	1.91

5.1 OSTEOPATHIC EVALUATION ANALYSIS

An osteopathic assessment was also conducted in an effort to gain greater insight into the effects of cranial osteopathic treatment on the participants. The assessor was unaware of whether the subjects were in the experimental or control group. The tables below show the results of measures of both vitality and cranial lesion classifications.

This first table provides descriptive statistics on 38 evaluated anatomical structures, assessed at both pre-treatment and post-treatment stages for both experimental and control groups on cranial lesion severity. The rating scale is based on the methodology used and taught at the Canadian College of Osteopathy. Each structure was assigned a number from 0 to 4 representing the severity of the lesion. On this scale, 0 represents no lesion, 1 represents a physiological lesion, 2 represents a non-physiological lesion with respect to the axis, 3 represents a non-physiological lesion without respect to the axis, and 4 represents a compaction. In this table all structures that were assessed pre-intervention and post-intervention are listed. It is divided into control and experimental groups so a visual comparison can be made. The mean provides an arithmetic average of severity of lesion per structure within each group. The standard deviation is a measurement of variability which shows the amount of variation from the average. It is commonly used to measure confidence in the statistical conclusion.

Table 10: Lesion Variable Severity

		Treatment			
		Control		Exp	
		Time		Time	
		pre	post	pre	post
SBS	Valid N	4	3	7	7
	Mean	3.0	2.7	2.4	.0
	Standard Deviation	.8	.6	1.5	.0
	Minimum	2	2	0	0
	Maximum	4	3	4	0
TB OM - L	Valid N	4	3	7	7
	Mean	2.2	3.0	2.9	.9
	Standard Deviation	2.1	1.7	1.2	1.1
	Minimum	0	1	1	0
	Maximum	4	4	4	3
TB OM R	Valid N	4	3	7	7
	Mean	1.5	.7	1.9	.1
	Standard Deviation	1.7	.6	1.2	.4
	Minimum	0	0	1	0
	Maximum	4	1	4	1
Pet - Bas L	Valid N	4	3	7	7
	Mean	3.5	3.3	2.6	.7
	Standard Deviation	1.0	1.2	1.4	1.5
	Minimum	2	2	1	0
	Maximum	4	4	4	4
pet - Bas R	Valid N	4	3	7	7
	Mean	.8	.7	1.4	.0
	Standard Deviation	.5	.6	1.3	.0
	Minimum	0	0	0	0
	Maximum	1	1	4	0
Co-C1 L	Valid N	4	3	7	7
	Mean	3.0	3.3	2.4	.0
	Standard Deviation	1.4	1.2	1.4	.0
	Minimum	1	2	0	0
	Maximum	4	4	4	0
CO-C1R	Valid N	4	3	7	7
	Mean	1.5	2.7	.6	.0
	Standard Deviation	1.9	2.3	1.1	.0
	Minimum	0	0	0	0
	Maximum	4	4	3	0
C0-C1/C2	Valid N	4	3	7	7
	Mean	1.5	1.0	1.9	.1
	Standard Deviation	1.7	1.7	1.5	.4
	Minimum	0	0	0	0
	Maximum	3	3	3	1
Eth	Valid N	4	3	7	7
	Mean	1.0	2.3	2.6	.4
	Standard Deviation	2.0	2.1	1.4	1.1
	Minimum	0	0	0	0
	Maximum	4	4	4	3
Jug L	Valid N	4	3	7	7
	Mean	3.5	2.7	2.6	.0
	Standard Deviation	.6	1.5	1.9	.0
	Minimum	3	1	0	0
	Maximum	4	4	4	0

		Treatment			
		Control		Exp	
		Time		Time	
		pre	post	pre	post
Jug R	Valid N	4	3	7	7
	Mean	1.5	1.3	.3	.1
	Standard Deviation	.6	.6	.8	.4
	Minimum	1	1	0	0
	Maximum	2	2	2	1
For Mag	Valid N	4	3	7	7
	Mean	2.2	1.7	2.1	.1
	Standard Deviation	1.0	1.2	.9	.4
	Minimum	1	1	1	0
	Maximum	3	3	3	1
Par L	Valid N	4	3	7	7
	Mean	2.2	1.7	1.3	.1
	Standard Deviation	2.1	2.1	1.5	.4
	Minimum	0	0	0	0
	Maximum	4	4	4	1
Par T	Valid N	4	3	7	7
	Mean	.2	.7	1.0	.1
	Standard Deviation	.5	.6	1.5	.4
	Minimum	0	0	0	0
	Maximum	1	1	4	1
T L	Valid N	4	3	7	7
	Mean	2.0	1.7	1.9	1.1
	Standard Deviation	1.4	.6	1.2	.4
	Minimum	1	1	1	1
	Maximum	4	2	4	2
TB R	Valid N	4	3	7	7
	Mean	1.0	.7	1.3	.6
	Standard Deviation	.8	.6	.5	.5
	Minimum	0	0	1	0
	Maximum	2	1	2	1
FB L	Valid N	4	3	7	7
	Mean	1.5	1.0	1.6	.4
	Standard Deviation	1.7	.0	1.1	.5
	Minimum	0	1	1	0
	Maximum	4	1	4	1
FB R	Valid N	4	3	7	7
	Mean	.5	1.0	1.4	.1
	Standard Deviation	.6	1.0	1.3	.4
	Minimum	0	0	0	0
	Maximum	1	2	4	1
Occ Sq	Valid N	4	3	7	7
	Mean	2.5	2.0	2.0	.1
	Standard Deviation	1.3	1.0	1.0	.4
	Minimum	1	1	1	0
	Maximum	4	3	3	1
Sph > wing	Valid N	4	3	7	7
	Mean	1.2	1.3	1.7	1.1
	Standard Deviation	1.5	1.2	1.9	1.5
	Minimum	0	0	0	0
	Maximum	3	2	4	3

		Treatment			
		Control		Exp	
		Time		Time	
		pre	post	pre	post
Zyg L	Valid N	4	3	7	7
	Mean	.8	.7	1.7	.7
	Standard Deviation	.5	.6	1.6	1.1
	Minimum	0	0	0	0
	Maximum	1	1	4	3
Zyg R	Valid N	4	3	7	7
	Mean	.2	.0	.9	.0
	Standard Deviation	.5	.0	.9	.0
	Minimum	0	0	0	0
	Maximum	1	0	2	0
Max L	Valid N	4	3	7	7
	Mean	.8	2.0	1.0	.4
	Standard Deviation	1.0	1.0	1.2	.5
	Minimum	0	1	0	0
	Maximum	2	3	3	1
Max R	Valid N	4	3	7	7
	Mean	1.2	.3	1.7	.3
	Standard Deviation	1.0	.6	1.4	.8
	Minimum	0	0	0	0
	Maximum	2	1	4	2
Man L	Valid N	4	3	7	7
	Mean	2.0	1.0	1.4	1.3
	Standard Deviation	1.4	1.0	1.1	1.7
	Minimum	1	0	0	0
	Maximum	4	2	3	4
Mand R	Valid N	4	3	7	7
	Mean	1.2	.3	.9	.6
	Standard Deviation	1.9	.6	1.2	1.1
	Minimum	0	0	0	0
	Maximum	4	1	3	3
Pal L	Valid N	4	3	7	7
	Mean	.8	.0	.6	.7
	Standard Deviation	1.0	.0	1.1	1.5
	Minimum	0	0	0	0
	Maximum	2	0	3	4
Pal R	Valid N	4	3	7	7
	Mean	1.0	1.3	1.0	.0
	Standard Deviation	2.0	2.3	1.5	.0
	Minimum	0	0	0	0
	Maximum	4	4	4	0
Vomer	Valid N	4	3	7	7
	Mean	2.0	1.3	2.4	.6
	Standard Deviation	1.6	2.3	1.1	1.1
	Minimum	0	0	1	0
	Maximum	4	4	4	3
Lac L	Valid N	4	3	7	7
	Mean	1.8	3.0	1.9	1.1
	Standard Deviation	2.1	1.7	2.0	2.0
	Minimum	0	1	0	0
	Maximum	4	4	4	4

		Treatment			
		Control		Exp	
		Time		Time	
		pre	post	pre	post
Lac R	Valid N	4	3	7	7
	Mean	.2	.3	.6	.3
	Standard Deviation	.5	.6	1.1	.8
	Minimum	0	0	0	0
	Maximum	1	1	3	2
Nas L	Valid N	4	3	7	7
	Mean	2.5	4.0	2.7	1.3
	Standard Deviation	1.9	.0	1.9	1.9
	Minimum	0	4	0	0
	Maximum	4	4	4	4
Nas R	Valid N	4	3	7	7
	Mean	1.0	.0	1.4	.0
	Standard Deviation	2.0	.0	1.8	.0
	Minimum	0	0	0	0
	Maximum	4	0	4	0
Sacrum	Valid N	4	3	7	7
	Mean	1.2	1.7	2.1	.4
	Standard Deviation	1.3	1.2	1.1	.5
	Minimum	0	1	1	0
	Maximum	3	3	3	1
L5-S1	Valid N	4	3	7	7
	Mean	2.2	2.3	2.7	1.4
	Standard Deviation	1.7	2.1	1.5	1.8
	Minimum	0	0	0	0
	Maximum	4	4	4	4
INN L	Valid N	4	3	7	7
	Mean	.5	.3	1.1	.1
	Standard Deviation	.6	.6	1.7	.4
	Minimum	0	0	0	0
	Maximum	1	1	4	1
Inn R	Valid N	4	3	7	7
	Mean	1.2	1.7	2.1	.7
	Standard Deviation	1.0	1.5	1.2	.5
	Minimum	0	0	0	0
	Maximum	2	3	3	1
C3	Valid N	4	3	7	7
	Mean	.5	1.7	1.0	.0
	Standard Deviation	.6	1.5	1.0	.0
	Minimum	0	0	0	0
	Maximum	1	3	3	0

A detailed statistical analysis was performed to show the relationship between pre-intervention lesion classification scores and post-intervention lesion classification scores. This analysis was done using the Somers'd test to evaluate change in the pre-classification and post-classification scores of both the control and experimental groups.

The results showed that 17 out of 38 structures showed improvement after treatment in the experimental group. ($p<0.05$) In contrast, the control group had no statistically significant differences.

The following table shows the p-value for each structure in both the control and experimental group. A detailed analysis is included in the appendix. See Appendix Q: Lesion Classification Correlation Tables.

Table 11: Results of Lesion Classification.

Structure	P Value Control N=4	P Value Experimental N=7
SBS	P=0.771	P=0.005*
OM Left	P=0.821	P=0.010*
OM Right	P=0.771	P=0.001*
Petrobasilar Left	P=1.000	P=0.016*
Petrobasilar Right	P=1.000	P=0.005*
C0-C1 Left	P=0.829	P=0.005*
C0-C1 Right	P=0.743	P=0.462
C0-C1/C2	P=1.000	P=0.037*
Ethmoid	P=0.657	P=0.021*
Jugular Left	P=0.657	P=0.021*
Jugular Right	P=1.000	P=1.000
Foramen Magnum	P=0.743	P=0.002*
Parietal Left	P=1.000	P=0.119
Parietal Right	P=0.486	P=0.315
Temporal bone Left	P=1.000	P=0.315
Temporal bone Right	P=0.771	P=0.073

Frontal Bone Left	P=1.000	P=0.033*
Frontal Bone Right	P=0.657	P=0.021*
Occipital Squama	P=0.771	P=0.002*
Sphenoid G Wing	P=1.000	P=0.510
Zygoma Left	P=1.000	P=0.122
Zygoma Right	P=1.000	P=0.070
Maxilla Left	P=0.200	P=0.478
Maxilla Right	P=0.314	P=0.021*
Mandible Left	P=0.486	P=0.781
Mandible Right	P=0.829	P=0.780
Palantine Left	P=0.429	P=1.000
Palantine Right	P=1.000	P=0.192
Vomer	P=0.571	P=0.013*
Lacrimal Left	P=0.400	P=0.592
Lacrimal Right	P=1.000	P=0.731
Nasal Left	P=0.429	P=0.286
Nasal Right	P=1.000	P=0.192
Sacrum	P=0.771	P=0.012*
L5/S1	P=0.971	P=0.202
Ilium Left	P=1.000	P=0.315
Ilium Right	P=0.686	P=0.0318
C3	P=0.571	P=0.021*

*represents a statistically significant improvement

An additional assessment measure looked at each of the 38 anatomical structures pre and post treatment as measured on a scale of vitality. The vitality scale was measured

using scores ranging from 0-3, with 0 representing no vitality and 3 representing normal vitality. The table below shows descriptive statistics for each of the 38 variables on the vitality measure.

Table 12: Vitality Severity Variables

		Treatment			
		Control		Exp	
		Time		Time	
		pre	post	pre	post
SBS	Valid N	4	3	6	7
	Mean	.2	.3	.3	2.7
	Standard Deviation	.5	.6	.5	.5
	Minimum	0	0	0	2
	Maximum	1	1	1	3
TB OM - L	Valid N	4	3	6	7
	Mean	.5	1.0	.2	1.6
	Standard Deviation	.6	.0	.4	1.0
	Minimum	0	1	0	0
	Maximum	1	1	1	3
TB OM R	Valid N	4	3	6	7
	Mean	.8	1.0	.5	2.7
	Standard Deviation	1.0	.0	.5	.5
	Minimum	0	1	0	2
	Maximum	2	1	1	3
Pet - Bas L	Valid N	4	3	6	7
	Mean	.0	.7	.3	2.0
	Standard Deviation	.0	.6	.5	1.0
	Minimum	0	0	0	0
	Maximum	0	1	1	3
pet - Bas R	Valid N	4	3	6	7
	Mean	1.8	1.3	.8	2.7
	Standard Deviation	.5	.6	.8	.5
	Minimum	1	1	0	2
	Maximum	2	2	2	3
Co-C1 L	Valid N	4	3	6	7
	Mean	.5	.7	.5	2.4
	Standard Deviation	1.0	.6	.8	.5
	Minimum	0	0	0	2
	Maximum	2	1	2	3
CO-C1R	Valid N	4	3	6	7
	Mean	1.2	.7	1.5	2.7
	Standard Deviation	1.3	1.2	1.0	.5
	Minimum	0	0	0	2
	Maximum	3	2	3	3
C0-C1/C2	Valid N	4	3	6	7
	Mean	1.2	1.7	.8	2.3
	Standard Deviation	1.0	1.5	1.3	.5
	Minimum	0	0	0	2
	Maximum	2	3	3	3
Eth	Valid N	4	3	6	7
	Mean	1.8	.7	.3	2.0
	Standard Deviation	1.3	1.2	.5	1.0
	Minimum	0	0	0	0
	Maximum	3	2	1	3
Jug L	Valid N	4	3	6	7
	Mean	.0	.7	.5	2.1
	Standard Deviation	.0	1.2	.8	.7
	Minimum	0	0	0	1
	Maximum	0	2	2	3

		Treatment			
		Control		Exp	
		Time		Time	
		pre	post	pre	post
Jug R	Valid N	4	3	6	7
	Mean	1.5	1.7	1.3	2.4
	Standard Deviation	.6	.6	.5	.8
	Minimum	1	1	1	1
	Maximum	2	2	2	3
For Mag	Valid N	4	3	6	7
	Mean	.2	1.3	.8	2.1
	Standard Deviation	.5	1.2	.4	.4
	Minimum	0	0	0	2
	Maximum	1	2	1	3
Par L	Valid N	4	3	6	7
	Mean	1.2	.7	1.0	2.4
	Standard Deviation	1.3	1.2	1.1	.8
	Minimum	0	0	0	1
	Maximum	3	2	2	3
Par T	Valid N	4	3	6	7
	Mean	2.0	1.3	1.3	2.4
	Standard Deviation	.8	1.2	1.0	1.1
	Minimum	1	0	0	0
	Maximum	3	2	2	3
T L	Valid N	4	3	6	7
	Mean	.2	1.0	.5	1.1
	Standard Deviation	.5	.0	.5	.9
	Minimum	0	1	0	0
	Maximum	1	1	1	2
TB R	Valid N	4	3	6	7
	Mean	1.8	1.3	.8	2.6
	Standard Deviation	.5	.6	.4	.8
	Minimum	1	1	0	1
	Maximum	2	2	1	3
FB L	Valid N	4	3	6	7
	Mean	.8	1.3	.5	2.3
	Standard Deviation	1.0	1.5	.5	1.0
	Minimum	0	0	0	1
	Maximum	2	3	1	3
FB R	Valid N	4	3	6	7
	Mean	1.5	1.3	.8	2.9
	Standard Deviation	1.0	1.5	.8	.4
	Minimum	0	0	0	2
	Maximum	2	3	2	3
Occ Sq	Valid N	4	3	6	7
	Mean	.8	1.3	.8	2.1
	Standard Deviation	.5	.6	1.0	.4
	Minimum	0	1	0	2
	Maximum	1	2	2	3
Sph > wing	Valid N	4	3	6	7
	Mean	2.0	1.3	.8	2.6
	Standard Deviation	1.2	.6	1.0	.5
	Minimum	1	1	0	2
	Maximum	3	2	2	3

		Treatment			
		Control		Exp	
		Time		Time	
		pre	post	pre	post
Zyg L	Valid N	4	3	6	7
	Mean	1.2	1.3	.7	1.7
	Standard Deviation	1.0	.6	.8	1.1
	Minimum	0	1	0	0
	Maximum	2	2	2	3
Zyg R	Valid N	4	3	6	7
	Mean	2.0	2.0	1.5	2.7
	Standard Deviation	.0	.0	.5	.5
	Minimum	2	2	1	2
	Maximum	2	2	2	3
Max L	Valid N	4	3	6	7
	Mean	1.5	1.3	.7	1.9
	Standard Deviation	.6	1.2	.5	.4
	Minimum	1	0	0	1
	Maximum	2	2	1	2
Max R	Valid N	4	3	6	7
	Mean	1.5	1.7	.7	2.1
	Standard Deviation	.6	.6	.5	.7
	Minimum	1	1	0	1
	Maximum	2	2	1	3
Man L	Valid N	4	3	6	7
	Mean	.8	1.0	1.0	1.4
	Standard Deviation	.5	1.0	1.3	.8
	Minimum	0	0	0	0
	Maximum	1	2	3	2
Mand R	Valid N	4	3	6	7
	Mean	1.5	2.0	2.0	2.1
	Standard Deviation	1.0	.0	1.1	1.1
	Minimum	0	2	0	0
	Maximum	2	2	3	3
Pal L	Valid N	4	3	6	7
	Mean	2.0	2.3	1.2	1.9
	Standard Deviation	.8	.6	1.0	.9
	Minimum	1	2	0	0
	Maximum	3	3	2	3
Pal R	Valid N	4	3	6	7
	Mean	1.8	1.3	1.8	2.6
	Standard Deviation	1.3	1.2	.4	.5
	Minimum	0	0	1	2
	Maximum	3	2	2	3
Vomer	Valid N	4	3	6	7
	Mean	1.2	1.7	.5	1.9
	Standard Deviation	1.0	1.5	.8	.9
	Minimum	0	0	0	0
	Maximum	2	3	2	3
Lac L	Valid N	4	3	6	7
	Mean	1.2	.3	.8	1.9
	Standard Deviation	1.5	.6	1.0	1.1
	Minimum	0	0	0	0
	Maximum	3	1	2	3

		Treatment			
		Control		Exp	
		Time		Time	
		pre	post	pre	post
Lac R	Valid N	4	3	6	7
	Mean	2.0	2.0	1.5	2.9
	Standard Deviation	.8	1.0	.8	.4
	Minimum	1	1	0	2
	Maximum	3	3	2	3
Nas L	Valid N	4	3	6	7
	Mean	.8	.3	.7	1.7
	Standard Deviation	1.5	.6	1.0	1.3
	Minimum	0	0	0	0
	Maximum	3	1	2	3
Nas R	Valid N	4	3	6	7
	Mean	1.5	2.3	1.3	2.7
	Standard Deviation	1.0	.6	.8	.5
	Minimum	0	2	0	2
	Maximum	2	3	2	3
Sacrum	Valid N	4	3	6	7
	Mean	1.2	1.3	.8	1.6
	Standard Deviation	1.0	1.2	.8	.5
	Minimum	0	0	0	1
	Maximum	2	2	2	2
L5-S1	Valid N	4	3	6	7
	Mean	.5	1.0	.7	1.6
	Standard Deviation	.6	1.0	.5	.8
	Minimum	0	0	0	0
	Maximum	1	2	1	2
INN L	Valid N	4	3	6	7
	Mean	1.2	2.0	1.5	2.7
	Standard Deviation	.5	1.0	.8	.5
	Minimum	1	1	1	2
	Maximum	2	3	3	3
Inn R	Valid N	4	3	6	7
	Mean	.8	1.0	1.0	2.3
	Standard Deviation	.5	1.0	.6	.5
	Minimum	0	0	0	2
	Maximum	1	2	2	3
C3	Valid N	4	3	6	7
	Mean	2.0	1.7	1.7	2.3
	Standard Deviation	.8	1.2	.5	.5
	Minimum	1	1	1	2
	Maximum	3	3	2	3

Again, a Somers'd test was used to determine the effect of treatment on vitality before and after treatment in both the experimental and control groups on all 38 variables.

Out of the 38 structures evaluated 29 showed statistically significant improvements between the pre-vitality and post-vitality scores in the experimental group.

There was no statistical significance in any structure between the pre-vitality and post-vitality scores in the control group.

The following table shows the p-value for the vitality measure for each structure in both the control and experimental group. This table depicts the essential results of a more detail analysis that can be seen in Appendix R: Vitality Correlation Tables

Table 13: Results of Vitality Severity

Structure/Variable	P-Value Control N=4	P-Value Experimental N=7
SBS	P=1.000	P=0.001*
OM Left	P=0.429	P=0.015*
OM Right	P=0.771	P=0.001*
Petrobasilar Left	P=0.143	P=0.012*
Petrobasilar Right	P=0.486	P=0.003*
C0-C1 Left	P=0.829	P=0.003*
C0-C1 Right	P=0.486	P=0.040*
C0-C1/C2	P=0.686	P=0.031*
Ethmoid	P=0.400	P=0.012*
Jugular Left	P=0.429	P=0.009*
Jugular Right	P=1.000	P=0.035*
Foramen Magnum	P=0.257	P=0.001*
Parietal Left	P=0.486	P=0.020*
Parietal Right	P=0.486	P=0.020*
Temporal bone Left	P=0.143	P=0.269
Temporal bone Right	P=0.486	P=0.004*
Frontal Bone Left	P=0.657	P=0.009*

Frontal Bone Right	P=1.000	P=0.002*
Occipital Squama	P=0.571	P=0.016*
Sphenoid Greater wing	P=0.657	P=0.012*
Zygoma Left	P=1.000	P=0.125
Zygoma Right	Missing value	P=0.009*
Maxilla Left	P=1.000	P=0.003*
Maxilla Right	P=1.000	P=0.003*
Mandible Left	P=1.000	P=0.464
Mandible Right	P=1.000	P=0.965
Palantine Left	P=0.771	P=0.241
Palantine Right	P=0.771	P=0.049*
Vomer	P=0.686	P=0.034*
Lacrimal Left	P=0.543	P=0.134
Lacrimal Right	P=1.000	P=0.003*
Nasal Left	P=1.000	P=0.186
Nasal Right	P=0.571	P=0.009*
Sacrum	P=1.000	P=0.122
L5/S1	P=0.657	P=0.048*
Ilium Left	P=0.371	P=0.026*
Ilium Right	P=1.000	P=0.003*
C3	P=0.571	P=0.147

*represents a statistical significant result $p<0.05$

6 CHAPTER SIX: DISCUSSION

The hypothesis for this study was that cranial osteopathic treatment in conjunction with exercise will produce a greater improvement in the balance of subjects with Parkinson's disease, as compared to a control group receiving only exercise. The resulting post-treatment Berg Balance Scale scores showed a greater improvement for the experimental group than for the control group (p=.028)

While the study results were favorable with respect to the positive effects of cranial osteopathic treatment on balance in Parkinson's subjects, and would suggest that the null hypothesis can be rejected, the researcher calls for caution in interpretation due to the small sample size (n=11) and to the uneven groups. The experimental group, with seven participants, had greater power to detect an effect than the control group, with four participants. This skewed distribution of subjects to experimental and control groups was an outcome of the randomization procedure which was based on a planned sample size of 16 subjects. The randomization procedure is discussed in greater detail in section 4.11.

With a larger sample size it is possible that additional subjects in the control group may have shown improvements similar to that seen in the experimental group, thus eradicating any differential effect postulated to be due to cranial osteopathic treatment in the experimental group participants.

It is therefore recommended that this research be interpreted as a pilot study only, and that further research be conducted with a larger sample size.

While this study should be regarded as a pilot, the discussion section addresses possible reasons for the positive effect under an assumption that the result was real rather than a function of chance. The reader is encouraged to consider this rationale, in the hope that future researchers may find it provocative and may draw further hypotheses worth

exploring.

6.1 DISCUSSION OF RESULTS BASED ON OSTEOPATHIC JUSTIFICATION

Cranial osteopathic treatment has been critically scrutinized both within and outside of the osteopathic profession. It is nonetheless widely used in clinical practice and taught to student osteopaths throughout Canada. This research endeavored to demonstrate the efficacy of cranial osteopathy in improving balance in Parkinson's patients. Parkinson's disease is a progressive neurological disease with its neuronal destruction based within the encephalon. Cranial osteopathy may have beneficial effects in treatment of a pathology that is based within the central nervous system.

The cranial osteopathic treatment may have produced the resulting balance improvement in the experimental subjects due primarily to its effect in increasing circulation to the basal ganglia. In cerebral pathology, arterial insufficiency causes an ischemic state which can lead to necrosis of the neural cells (Nolte, 1981). Moskalenko's research found that the removal of structural impedances, using cranial osteopathic treatment, had an effect in improving circulation in the brain. The cranial osteopathic treatment may have had the effect of removing structural impedances thus increasing circulation in and to the basal ganglia. This increased blood flow to the basal ganglia may allow a more optimal environment for neuronal activity and a resulting increase in postural stability.

Researchers believe that toxicity is one of the leading contributing factors to Parkinson's disease. Although the actual effects of techniques such as venous sinuses are not well documented it has been suggested within the teachings at the Canadian College of Osteopathy that this technique has an effect on the venous drainage of the cranium (Druelle & Forget, 2000). It would therefore follow that increasing vascular flow and

consequently contributing to the removal of toxins could have an impact on balance in Parkinson's subjects

Cerebrospinal fluid acts as an insulating property in which electrical impulses occur. It has been suggested that a lack of normal characteristics within the cerebrospinal fluid may cause pathology to occur (Becker, 1948b). Magoun (1976) believed that if any part of the craniosacral mechanism was compromised it would affect the cerebrospinal fluid leading to stasis and eventually pathology. Cranial osteopathic treatment may have contributed to the restoration of the cerebrospinal fluid which could aid in the process of neuroprotection and neuroplasticity.

McGeer and McGeer (2004) found cytokines in the cerebrospinal fluid of Parkinson's subjects leading them to determine that an inflammatory process occurs in the encephalon of these patients. The theory of an inflammatory process contributing to Parkinson's disease is well known and can be seen in the oxidative stress model (Jenner, 2003; Jenner & Olanow, 2006). Cranial osteopathy may have an effect on the reduction of edema providing an avenue to address inflammation within the encephalon (Magoun, 1968). This may have contributed to the positive results of cranial treatment on the balance in Parkinson's subjects.

6.2 DISCUSSION ABOUT OSTEOPATHIC ASSESSMENT

The results of the osteopathic assessment showed marked positive change in the experimental subjects in the majority of categories and no positive change in any of the categories in the control group subjects. In the lesion classification data of the experimental group 17 out of 38 structures showed improvement after analysis of the pre-intervention and post-intervention scores. Analysis of the vitality scores of the experimental group showed 29 out of 38 structures with positive improvement. In the

analysis of the lesion and vitality classifications there was no case where the control group showed improvement from pre to post scores.

Although these results look promising one must exercise caution in interpretation as sample size was small. The control sample was very small with only four subjects. If this group had been larger it is possible that improvement in some of the variables would have appeared.

The osteopathic evaluation also provided some data on the most commonly affected cranial structures within this subject pool. These structures include: the left nasal bone, sphenobasilar symphysis, the left petro-basilar suture and C0/C1 on the left. Excluding the nasal bone, the remaining structures are within the base of the cranium which is in direct relation to the vascular vessels that innervate the basal ganglia. The most common structures affected were primarily on the left side, however the researcher is unable to provide a basis for this result. Although these results are interesting they are anecdotal at best and should be viewed as such.

The osteopathic assessor performed assessments both before and after treatment on all subjects and was consistent in the application of his measurements. By virtue of the methodology, the osteopathic assessment results are subjective in nature, and are not considered as part of the overall hypothesis. These assessment results are offered rather as further information on the treatment results.

It should also be noted that the assessment results demonstrated a change which provides a basis for a conclusion that there was an effect from the treatment. The detectable effect suggests that the outcome changes are not a simple placebo.

6.3 DISCUSSION OF HOEHN AND YAHR RESULTS

The eleven subjects were assessed on the Hoehn and Yahr scale of Parkinson's severity. Out of the 11 subjects 6 scored at stage II on the Hoehn and Yahr Scale while 5 subjects scored at stage III on the Hoehn and Yahr scale. Four of the stage II's were in the experimental group while the remaining two were in the control group. Three of those that scored III were n the experimental group while the remaining two were in the control group. Thus, severity of Parkinson's was relatively evenly assigned to the groups.

Hoehn and Yahr Scale	Experimental Group	Control Group
Rating of II	N=4	N=2
Rating of III	N=3	N=2

Figure: 17. Randomization based on the Hoehn and Yahr Rating Scale

The Wald chi-square analysis to determine whether cranial osteopathic treatment had differential effects on those with greater or lesser severity of Parkinson's failed to reach significance (p=.055), suggesting that the effect of treatment does not depend on the severity of Parkinson's. However, while the result did not reach the technical criterion for statistical significance, it does suggest that the difference in treatment effect is most pronounced at the greater Hoehn and Yahr (HY) severity level III.

The researcher had hypothesized that cranial osteopathic treatment would have greater effect on a Parkinson's subject in an early stage of progression. Symptoms of Parkinson's disease often appear after approximately 70% of neuronal death within the substantia nigra, giving rise to the postulation that early osteopathic cranial intervention would have greater neuroprotective effect (Martin, et al., 2010). Cranial osteopathy may provide an avenue into increasing circulation and venous drainage, therefore providing

the removal of toxins from the basal ganglia. However, the data from this current study suggests that earlier cranial osteopathic intervention does not have a greater effect on balance in Parkinson's subjects. In contrast, the data suggested a possible correlation between treatment effect and a higher level of Parkinson's severity.

Hirsch (2007) suggested that toxins released by the glial cells may occur long after intial exposure to MPTP and may contribute to the ongoing destruction of the basal ganglia in Parkinson's disease. This may provide an explanation for the results of the current research which suggests that cranial osteopathy has a greater effect on balance in more severe Parkinson's cases. However, a larger study must be pursued for this data to be considered conclusive.

Another interpretation is possible. This result however, even had it reached significance, may in fact be an anomaly of the Berg Balance Scale. A subject with a Hoehn and Yahr rating of III would commence the study with a lower pre-BBS. The Berg Balance Scale has a ceiling value of 56 and subjects with Hoehn and Yahr ratings of II often had a pre-BBS score of 54 or 55. Therefore, those with a higher BBS score had less total opportunity for improvement, which may skew the result artificially to show a greater total improvement for those with a lower pre-BBS score.

6.3.1 DISCUSSION ON THE HOEHN AND YAHR RATING SCALE

The Hoehn and Yahr is the most commonly used scale to describe the severity of Parkinson's disease (Goetz, et al., 2004). Many Parkinson's disease and balance related studies employ the Hoehn and Yahr (Ellis, et al., 2008; Hirsh, et al., 2003; Mitchell, et al., 1995). Although all the subjects fell within stages II and III on the Hoehn and Yahr rating scale there was a wide variety of presentations within the subject pool.

The Hoehn and Yahr is a simple classification that may miss the subtleties of severity. The original design of the Hoehn and Yahr scale was a five point scale, but in recent years 0.5 increments have been added in some clinical trials. In the current research the original version of the Hoehn and Yahr scale was used. The assessing osteopath found this original version limiting and had some difficultly with placement of the subjects. This same concern was expressed in a study conducted by the Movement Disorder Society in which 69% of the 236 members polled thought the staging categories to be too broad (Goetz et al., 2004).

Stage I	Unilateral involvement with minimal or no functional impairment
Stage II	Bilateral or midline impairment with little impairment to balance
Stage III	Bilateral disease: mild to moderate disability with impaired postural reflexes
Stage IV	Severely disabling; still able to walk or stand unassisted
Stage V	Confined to bed or wheelchair

Figure: 18. Hoehn and Yahr Original Scale (Goetz, et al., 2004, p. 1021)

A more recent version of Hoehn and Yahr has added more rating from I to III to reflect the spectrum of the disease process.

Stage 1.0	Unilateral involvement only
Stage 1.5	Unilateral and axial involvement
Stage 2.0	Bilateral involvement without impairment of balance
Stage 2.5	Mild bilateral disease with recover on pull test
Stage 3.0	Mild to Moderate bilateral disease; some postural instability; physically independent
Stage 4.0	Severe disability; still able to walk or stand unassisted
Stage 5.0	Wheelchair bound or bedridden unless aided

Figure: 19. Modified Hoehn and Yahr scale (Goetz, et al., 2004, p. 1021)

Although this new version is not officially published it is being used by physicians globally. It is recommended that for future research the modified version of the Hoehn and Yahr be employed and the new criteria include staging between 2.5 and 3.0 to ensure enough postural instability that the subjects have room for significant change.

6.4 DISCUSSION OF THE BERG BALANCE SCALE

There are two categories of tools that are used to measure balance. First there is computerized instrumentation such as force plates, however this option has considerable expense associated with it as well as a need for specific training to effectively apply the instrumentation. The second option for evaluating balance is clinical standardized assessments, which was the method chosen for the current study. The benefit of these assessments is their simplicity in administering the tests, the ability to use this assessment tests in any clinical setting, and the low cost associated with them. In addition, clinical standardized assessments mimic real-life function and are therefore functional relevant to the patient.

The Berg Balance scale was specifically designed to assess balance, to screen patients for rehabilitative therapy, to predict falls, and to monitor changes in postural instability. Those that score 45 or lower out of 56 are at higher risk for falling. Those that score greater than 45 are less likely to experience falls associated with balance issues (Kornetti, et al., 2004). Within this current research there were only two subjects that scored below the 45 on the Berg Balance scale and therefore that had an increase risk of falling.

The Berg Balance Scale is an excellent instrument for identifying the risk of falling, but may not be sensitive enough to determine change of balance in those without

an increased risk of falling. As noted earlier, some of the subjects within this current study started with a 54 on the Berg Balance Scale which only allowed for an increase of two points. Earlier statistical analysis determined that a change of two was significant to show statistical change. However with a starting score of 54, any true improvement in balance for subjects in the study in either control or experimental group may be masked due to the ceiling effect.

An analysis of the Berg Balance Scale determined that the scale was not sensitive enough to detect changes in balance at the upper end of the scale (Kornetti, et al., 2004). Kornetti et al. (2004) produced a revised scale that makes the testing more sensitive however, it has not yet been adopted as the new *gold standard.* This revised scale provides a more accurate representation of balance in subjects with Parkinson`s disease.

6.5 DISCUSSION OF INTERVENTION

The osteopathic literature review found only a limited number of cranial osteopathic studies relevant to this topic. The lack of research made it difficult to determine the amount of treatment required to make a difference within this population. After much discussion four treatments were decided on. This was based on the essential structures that the author felt needed to be addressed as well as what seemed like a reasonable amount of treatments for patient compliance. In retrospect more than one clearing treatment would have been helpful to increase the effects of the cranial treatment. This protocol of multiple clearing treatments was used in David Bergstein's, CCO thesis (Bergstein, 2008). In this study, pre-study treatments were used to clear all major lesions as per Canadian College of Osteopathy's methodology. The pre-study treatments were included as an adjunct to the study to ensure greatest effects from the endocranial treatment.

6.6 DISCUSSION OF THE EXERCISE PROTOCOL

The exercise protocol was placed in this study both as a reasonable control and as an incentive for subject recruitment. A methodology was derived that allowed for all subjects to have some intervention without confounding the results of cranial osteopathic treatment. By providing an exercise protocol, supervised by an independent physiotherapist, all potential subjects received some intervention.

The inclusion of the exercise protocol provided a large incentive to the recruitment process, for all subjects knew they would be receiving treatment, but were unaware of the nature of the treatment. The subjects' knowledge that they were receiving some form of treatment also provided a blind for the subjects. By providing exercise the subjects were unaware of which group they were assigned to.

The exercise protocol may have also contributed to the null attrition that was experienced within this study. All participants verbally shared their appreciation and felt they benefited from the study.

6.7 DISCUSSION RELATED TO LITERATURE REVIEW

The medical literature is vast and numerous, making it difficult to address all aspects of Parkinson's research. The intention was to provide an overview of Parkinson's disease and treatment options specifically for postural instability.

The osteopathic literature review did not reveal any studies that focused on cranial osteopathy and balance in Parkinson's disease. This unfortunately limited the researcher when developing a methodological plan based on successful past studies. The few studies that were found on Parkinson's disease and osteopathic treatment employed either a single osteopathic global treatment or did not list what the osteopathic treatment entailed. This lack of literature prompted the researcher to review studies involving other

neurological disorders with the intention of extrapolating pertinent information for the current study. This was somewhat helpful in providing inspiration for many of these studies were statistically significant.

The remainder of the osteopathic literature review focused on the validity of cranial osteopathy. By choosing to use cranial osteopathy rather than a global approach it was felt that a review of the validity of cranial osteopathy was a necessary addition. In conclusion this review revealed the need for more research within the field of osteopathy and neurological disorders.

6.8 DISCUSSION OF OBSERVATIONS

There were several significant findings in the health history questionnaire that may have indicated several of the subjects would be less responsive to overall cranial osteopathic treatment. One subject in the experimental group was diagnosed with tuberculosis of the spine when she was eight years old. She spent two years bed-ridden in a hospital and was separated from her family. She was told she would never walk again, but went on to prove the physicians wrong. Two difficult pregnancies that ended in infant mortality were followed by a complete hysterectomy. This subject also presented with liver hemangiomas and macular degeneration. Restrictions in this patient's lumbar spine were very severe and had an overall effect on the expression of the primary respiratory mechanism.

Two other subjects in the experimental group presented with osteoarthritis of the hip. One had also experienced a bilateral knee replacement seven years prior to the current study. This may have affected the ability for lasting effect of treatment on the pelvis which may have contributed to restriction in the craniosacral system.

Another subject in the experimental group had her thyroid removed and experienced chronic stiffness and pain in her cervical spine. Some amount of treatment of the cervical spine was necessary in each session in order to ensure enough vitality to treat the cranium.

One of the subjects within the experimental group experienced severe tremor at rest. This made it difficult for the subject to remain on the table for a 60 minute session without agitation. It is questionable whether the severity of this tremor affected the Berg Balance Scale scores.

The treating osteopath became aware of these possible confounding physical issues throughout the current study. The researcher questioned whether these health history concerns affected the efficacy of the osteopathic cranial treatment, or the Berg Balance Scale assessment. However, the subjects represented real life case scenario's and therefore may depict an accurate representation of the clinical challenges found in the Parkinson's population.

A review of the medical heath history forms determined that subjects within the control group also noted co-morbidities. One subject noted that they had degenerative disc disease in the lumbar spine that is associated pain and stiffness. Another subject within the control group had a previous pelvic fracture due to a fall. The pelvic fracture did not affect this subject's ability to ambulate without assistance and therefore was accepted into the current study. However, she did mention that she experienced recurrent pelvic pain, which may have affected the Berg Balance Scale score.

One of the subjects within the control group was fearful of having anyone touch his neck and at the end of the study he refused to have the post osteopathic assessment.

All subjects had some degree of difficulty with digestion, which is most likely due to adverse side effects of the Parkinson's medications.

All subjects in both the control and experimental groups noted some form of co-morbidity. However, it remains unknown as to what degree this would have affected the cranial osteopathic treatment or the Berg Balance Scale score.

6.9 DISCUSSION RELATED TO PILOT STUDY

This study intended on being a fully powered study, however due to recruitment issues and time restraint this study came under the minimum amount of subjects. Upon approval of the thesis committee the title was changed to make it a pilot study. See Appendix S: Pilot Study Approval Letter. It was felt by the researcher, the thesis advisor, and the thesis committee that this study had the appropriate attributes to be a pilot study.

A pilot study tests the logistics of the research in order to improve the quality and efficiency of a larger study. Pilot studies are prominent in the Osteopathic profession. A quick search on the JAOA data base with pilot study as the search word turned up 181 published studies. The prevalence of pilot studies in the osteopathic profession may be due to a lack of funding compared to allopathic medical research, and difficultly of recruitment.

Pilot studies are used to identify deficiencies in the research design which can be addressed before a larger study takes place. In the current study, the research design was basic and largely effective. However, this study revealed a number of issues, critical to future effective research, discussion of which should prove to be a valuable contribution to the literature at this stage. These issues include, limitations of the Berg Balance Scale, challenges with participants with less severe Hoehn and Yahr stages, and efficacy of the osteopathic assessment form, which is covered in the self critique section.

Pilot studies are used to assess a floor or ceiling effect, meaning if the task is too difficult or too easy the results will be skewed. In this study, the basic exercise protocol was sufficient for some improvement. This improvement, and using the BBS rating scale with a ceiling effect, may have masked greater results of exercise and cranial osteopathy together.

A larger study based on the same protocol is needed to determine whether the results are statistically significant however, there is still a concern that the Berg Balance scale is not sensitive enough to assess the balance of some potential subjects. If there was access to increased funding, the use of instrumentation such as a force plate would be an excellent adjunct to the Berg Balance scale.

Pilot studies can be appropriate for inexperienced researchers. There is a learning curve involved in research, and regardless of the amount of effort spent on the proposal and planning of the research, methodological errors frequently occur in student research. In a letter published in the JAOA, McCombs (2006) stated that the use of student practitioners to administer osteopathic manual medicine is not as effective as seasoned osteopaths providing treatment. He continues to say that "judging the efficacy of osteopathic manual medicine by the results achieved by students is a deeply flawed concept" (McCombs, 2006, p. 380).

Perhaps most compelling in this study is the fact that results in the experimental group were quite positive, based on the descriptive statistics. This statement is also true when examining the results of the osteopathic assessments with positive results in a majority of categories for the experimental group and virtually no improvement in the control group. For these reasons, this researcher feels strongly that the current pilot study

produced promising results, and warrants a full study in the effects of cranial osteopathy on balance in Parkinson's subjects.

7 SELF –CRITIQUE

7.1 RECRUITMENT

The researchers were aware that recruitment for this study was going to be a challenge. However, it was thought a power of n=16 would be attainable. As seen in the methodology section of this paper, much time and effort went into recruitment.

Recruitment notices that were posted throughout the city proved to be ineffective. Attending Parkinson's fundraisers was also ineffective for recruitment. In an additional effort to obtain subjects, letters were sent out to local physicians and physiotherapists. Two movement disorder clinics expressed interest in our study and had originally offered to refer subjects, however the offer was revoked when they became aware of a lack of a formal ethics review board at the Canadian College of Osteopathy.

The most effective source of potential subjects came from direct referral from the researchers own patient database as well as a Parkinson's support group in the north end of Toronto. The members of this support group were pro-active in their medical care and were thankful for the opportunity to be involved in the current study.

One of the main challenges for recruitment of potential subjects was the five-week commitment required to complete this study. Many of the subjects were retired and had travel plans or medical appointments that interfered with the study dates. Although the clinic location allowed for public transport, some of the subjects required an assistant to bring them to and from their appointments, which proved challenging.

The lack of a formal ethics review board at the Canadian College of Osteopathy limited the ability to obtain subjects, and regretfully may have been the largest contributing factor to the small power of the current study. In Ontario, all academic-based research involving human subjects is subjected to an ethics review board. The Canadian

College of Osteopathy has recently provided an option of applying for review with an external ethics board at the Canadian Memorial Chiropractor College. Unfortunately, this was suggested halfway through the study, and after careful review the researchers determined it wasn't a plausible option due to time restrictions.

In the author's opinion, the lack of a formal ethics review board discredited the current study. A strong suggestion for future research would be to attain approval from a formal review board prior to commencement of the study.

7.1.1 CRITIQUE OF SAMPLE SIZE

Although much effort went into the recruitment of subjects, the largest regret of the current study was the inability to attain enough subjects for a fully powered study. The results of the data analysis demonstrated improvement of the Berg Balance Scale scores in the experimental group of $p< 0.28$. Although these results are promising, a true statistical difference cannot be determined without a power of n=16.

7.2 CRITIQUE OF RANDOMIZATION

Ideally, the randomization of subjects would produce equal numbers in both the control and experimental group; however, the intended fully powered study of n=16 was instead completed with n=11. Due to the randomization procedure chosen, the groups were uneven – there were four subjects in the control group and seven subjects in the experimental group. The randomizing method used would have produced equal groups had the study obtained the suggested power of n=16, as the subjects chose from 16 envelopes containing cards that represented the groups. Unfortunately, the subjects began the study as they became available and therefore the randomization occurred on a subject to subject basis. Had all the subjects been obtained prior to commencement of the study, a computer-generated randomization could have been employed to assure equal grouping.

However, due to the recruitment difficulties, it was necessary to accept subjects as they became available.

7.3 CRITIQUE OF INCLUSION/EXCLUSION CRITERIA

This study was designed to assess the efficacy of cranial osteopathic treatment on balance in Parkinson's subjects. Within the methodology, consideration was taken to ensure both that the subjects had idiopathic Parkinson's disease and that they were between stage II to IV on the Hoehn and Yahr scale, in order to guarantee some postural instability was present. Of the subjects that were assessed, all rated between a II and III on the Hoehn and Yahr Scale. However, the subjects presented with varying degrees of postural instability. A revised version of the Hoehn and Yahr Scale that has an increased sensitivity to the subtleties of Parkinson's disease progression is strongly suggested for future research.

This researcher would also suggest adding an inclusion criterion requiring potential subjects to have significant cranial lesions. Regardless of their common pathology, individuals present differently since each has lived a life without osteopathic intervention prior to this study. All of the subjects within this study presented with varying degrees of cranial lesions, some being much more severe than others. Sandhouse et al. (2010) added an inclusion criterion of sphenobasilar cranial strain pattern in their research on the effects of cranial osteopathic treatment on visual function. A strong recommendation for future research would be to include a substantial cranial restriction within the inclusion criteria. It would be beneficial to identify potential subjects with Parkinson's disease that have primary cranial lesions.

Removal of the age requirement of 55-90 from the inclusion criteria is suggested for future research. Although this is a common addition to rehabilitative and

physical therapy research studies, an age requirement was unnecessary within the current study because the onset of Parkinson's disease can occur in the early twenties. The symptoms and progression of early onset Parkinson's disease do not differ from that of later onset Parkinson's disease (Martin, 2010). Therefore, it follows that the age restriction would have no overall effect on balance and treatment in the current study. Removing the age requirement may have provided more potential subjects.

A diagnosis of idiopathic Parkinson's disease was required to be considered as a potential subject for the current study. Unfortunately, the study thus excluded one potential subject with a diagnosis of Parkinson's plus syndrome. Although the symptoms of Parkinson's plus differ slightly from idiopathic Parkinson's disease, their pathogenesis is similar. Therefore, the justification and postulations as to why cranial osteopathy may be effective would not have changed. A revision of this inclusion is suggested for further research.

7.4 CRITIQUE OF OUTCOME MEASURES

As mentioned in the discussion of the Berg Balance Scale, this measuring tool of balance may not be sensitive enough. A revised version of the rating scale is now available and is strongly recommended for use in future research.

7.5 CRITIQUE OF THE OSTEOPATHIC ASSESSMENT FORM

A revised assessment form is suggested for future continuation of this research. Different rating scales were used for the lesion classifications and the vitality ratings. Lesions were rated using the Canadian College of Osteopathy methodology on a scale from 0-4: 0 representing no lesion and 4 representing compaction, or the most severe lesion. The vitality was scored from 0-3: 0 representing an absent vitality and 3

representing a healthy, powerful vitality. The inconsistency of these two scales proved to be somewhat challenging when formulating the data.

A secondary source of concern with the osteopathic assessment form used was that many of the variables were problematic. There were several duplications of structures such as the cervical vertebrae, the temporal bones, and the sphenoid. The sacral-iliac joint and the sacrum were listed as separate variables. The assessor determined that the sacral-iliac joint denoted the iliac bones, and they were therefore evaluated as such. In addition, the removal of the foramen magnum and jugular foramen variables is suggested for future research as there was some ambiguity in evaluation of these structures. For the purpose of this preliminary research, these structures were evaluated for their motility and vitality.

Another suggestion would be to include a category that names specific lesions. Although this research allowed us to see which structures were most affected, it did not give us specific details. This type of lesion category would have provided some interesting data into the most common lesions in Parkinson's disease subjects.

7.6 RECOMMENDATIONS FOR FUTURE RESEARCH

There are several suggestions for future research based on the current pilot study.

1. Obtaining an approval from a formal ethics review board so that future researchers can pursue assistance with recruitment by contacting neurologists associated with local movement centres. Prior to commencing future research, obtaining this approval is strongly recommended.
2. Revising the inclusion and exclusion criteria, in order to increase recruitment and improve overall design of the study. These suggestions include using the revised version of the Hohen and Yahr Scale, removing age requirements, and including

all forms of Parkinson's disease as opposed to only including the idiopathic presentation.

3. Using a revised osteopathic assessment form including, but not limited to, the addition of classification of specific osteopathic lesions.
4. Using the revised rating scale for the Berg Balance Scale, which is more sensitive than the original. If the resources are available, adding a second computerized measuring tool such as a force plate would strengthen the results on balance.
5. Adding a follow-up assessment of one month to determine the long-term effects of cranial osteopathic treatment on balance in Parkinson's subjects.
6. Adding a quality of life questionnaire such as an SF39 to gather information regarding the effects of treatment on depression, sleep and the general well-being of the subjects.
7. Changing the treatment protocol to increase the number of cranial treatments to six or eight to determine whether this would increase the effect of treatment.

8 CONCLUSIONS

Parkinson's disease is the second leading neurodegenerative disease in Canada, affecting one in 100 people over the age of 60. It is a progressive disorder that causes the destruction of dopamine neurons within the substantia nigra of the basal ganglia. The destruction of these neurons leads to motor symptoms such as tremor, bradykinesia, rigidity and postural instability. Postural instability contributes to falls, which are associated with injury, reduction of quality of life, and expense to the individual and the healthcare system. Current demographics suggest that incidence, and therefore the expense, of Parkinson's disease will increase with the aging of the baby boomer generation.

Treatment of postural instability is limited to medications that have been shown to be relatively ineffective for instability, and physiotherapy, which has been shown to provide only short-term improvement. There is a need for alternate forms of treatment that target balance in Parkinson's patients.

Cranial osteopathy provides a unique, non-invasive therapy that focuses on the mobility of the cranial bones, which may have an influence on the central nervous system. However, the lack of cranial osteopathic research within the field of neurological disorders makes it difficult to support this premise.

The current research was designed to assess the effects of cranial osteopathic treatment on balance in subjects diagnosed with idiopathic Parkinson's disease. Eleven subjects with Parkinson's disease volunteered to participate in this five-week study. The subjects were randomly assigned to an experimental group, consisting of exercise and cranial osteopathic treatment, and a control group, consisting of exercise alone. Balance was evaluated pre-intervention and post-intervention using the Berg Balance Scale. The

Berg Balance Scale is a *gold standard* clinical assessment, consisting of 14 functional tasks that are rated from zero to five, for a total of 56. Statistical analysis revealed that an improvement of two on the Berg Balance Scale would be considered significant.

A power analysis based on four equally spaced cranial osteopathic treatments determined that a power of n=16 was needed to show statistical significance ($p<0.05$). Unfortunately, recruitment efforts produced only 11 subjects and therefore this study was re-titled a pilot study. Seven subjects were randomly assigned to the experimental group and four subjects were randomly assigned to the control group.

The results of this pilot study showed a greater improvement in the Berg Balance Scale scores for the group receiving cranial osteopathic treatment compared to the control group ($p=.028$). Analysis of the osteopathic evaluation determined that 17 of 38 structural variables that were measured pre-treatment and post-treatment on the lesion severity scale and 29 of 38 structural variables that were measured pre-treatment and post-treatment on the vitality scale showed improvement in the experimental group. There was no change in the control group in either measure. It should be noted that the osteopathic evaluation was a subjective measure and therefore the data should be interpreted as such.

Although the results show a positive change in both the pre-treatment to post-treatment Berg Balance Scale scores and the descriptive analysis from the osteopathic evaluation, they are to be interpreted with caution due to the small sample size and the uneven groups. A larger sample size is required to show a statistical significance.

Although cranial osteopathy is a widely used modality within osteopathy, there is a dearth of research that provides evidence into the efficacy of osteopathic cranial

treatment. More research is needed to demonstrate the clinical efficacy of cranial osteopathic treatment with specific focus in neurological disorders.

The researcher believes that this pilot study provides some compelling information regarding the use of cranial osteopathy in the treatment of balance in Parkinson's patients that may add to the osteopathic body of knowledge.

It is the researcher's hope that this study be replicated on a larger scale so that it may provide some much-needed data into the efficacy of cranial osteopathy in the treatment of Parkinson's disease.

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APPENDIX A: CONSENT FORM

Informed Consent

Thank you for your interest in this research project.

Parkinson's Disease Study

To participate in this study I _____ (subject) agree that:

1. I understand that this study is part of a thesis requirement for the Canadian College of Osteopathy.
2. The health questionnaire was filled out truthfully to the best of my knowledge. I will inform Mr. Thomas Hein (905) 695-0371 should this information change while participating in this study.
3. I am aware that subjects may have to remove some external clothing for assessment or treatment purposes (shorts or bathing suits acceptable). I also understand that, although I am participating in a study treatment for Parkinson's Disease, other areas of my body (head, face, neck, shoulders, arms, abdomen, rib cage, hips, pelvis, legs, knees, feet, back) may be treated.
4. I understand I will not have to receive any aspect of treatment that I am uncomfortable with.
5. I will make myself available for 5 clinic appointments over 5 weeks
6. I am aware that participants are randomly placed in a control group and an experimental group. Only the experimental group will receive osteopathic treatment and this is randomly determined. The participant will not be informed which group they are in.
7. I understand that I may withdraw from the study at any time without consequence.

Participant Name _____

Participant Signature _____ Date _____

Witness's signature:_____

The coordinators of this study agree to:

1. To keep all personal and medical information confidential.
2. To destroy all private information upon completion of this study.
3. To give reasonable notice to all participants regarding appointment times.
4. To respect a subject's desire to withdraw from participation for any circumstances without consequence.

Coordinator Signature _____

Date_____

Investigators:

Stacey Hauserman

Osteopathic thesis candidate at the Canadian College of Osteopathy

Phone: 416-839-4652

Thomas Hein

Osteopathic thesis candidate at the Canadian College of Osteopathy

Phone 905-695-0371

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Phone: 416-597-0367

APPENDIX B: HOEHN AND YAHR SCALE

Hoehn and Yahr staging of Parkinson's disease:

Stage I: Unilateral involvement with minimal or no functional disability

- Signs and symptoms which include tremor, muscle stiffness appear unilateral
- Slowness
- Symptoms mild

Stage II: Bilateral or midline involvement without impairment of balance

- Symptoms are bilateral
- Minimal disability
- Swallowing and talking may be difficult
- Facial masking
- Posture and gait affected

Stage III: Bilateral mild to moderate disability with impaired postural reflexes

- Significant slowing of body movements
- Impairment of righting reflexes
- Equilibrium, balance and postural instability
- Generalized dysfunction that is moderately severe

Stage IV: Severely disabled still able to walk or stand unassisted

- Symptoms severe
- Walking limited
- Rigidity and bradykinesia
- May need assistance with activities of daily living
- Tremor may have decreased

Stage V: Bed-ridden or wheelchair bound

- Cannot stand or walk
- Requires constant assistance
- Convalenscent stage

APPENDIX C: EXERCISE PROTOCAL

Parkinson Society Canada
Société Parkinson Canada

Exercises for People with Parkinson's

Having Parkinson's does not mean you should sit down and stop being active. Actually the opposite is true. Exercise, which includes being active, stretching, practicing good posture and doing specific exercises, should be a key component of your daily life.

WHY IS EXERCISE IMPORTANT FOR PEOPLE WITH PARKINSON'S?

Being active is one of the most important things you can do to maintain your physical and mental well-being. Exercise will not alter the progression of Parkinson's but it is essential for maintaining your quality of life. Studies clearly show that people with Parkinson's who exercise fare better in the long run than people with Parkinson's who do not exercise.

People with Parkinson's need to exercise to prevent the negative effects of inactivity. Moving, stretching and exercising as much as you can will also help prevent secondary effects that may develop such as:

- Poor posture
- Decreasing range of movement (losing flexibility)
- Decreased strength particularly in the muscles that hold you upright, resulting in a tendency to stoop forward
- Decreasing endurance (being out of breath or fatigued)
- Poor balance

frightened, sad, Scared, lonely, upset, depressed, excited,

CHOOSE A VARIETY OF ACTIVITIES – AND MINUTES COUNT!

Most people will say they gain strength, flexibility and balance by being active. How can you be active? There are many ways:

Aerobic activities such as:

- Using a treadmill
- Using a stationary bike or rowing machine
- Walking
- Swimming
- Dancing

Other activities such as:

- Yoga
- Tai Chi
- Pilates
- Golf
- Gardening
- Exercises – sitting or standing
- And many more…

The greatest benefit comes from doing things that YOU enjoy. Some activities will give you more benefit than others. However, consistency is more important than the specific activity you choose to do. Perhaps do more of what you are already doing. Begin with activities you can do comfortably – listen to your body! Choose the time of day that is best for you to do your exercises.

Gradually add minutes of activity to your program. Minutes count and your goal is to build up your activity level to a total of 30 to 60 minutes a day.

It is never too late to become active. Here are some specific ideas you can try to increase your activity level:

- Take a walk
  - Walk 20 steps in your normal way
  - Then take 20 long steps
  - Then 20 normal steps
  - Then swing your arms for 20 steps
  - Repeat for the duration of your walk
- Get off the bus one stop early or park the car one block away
- Use the stairs instead of the elevator
- Lift cans of soup, or any small weight, to exercise your arms (see strengthening exercises section)
- Do leg exercises while watching television.
- Join an exercise class. One of the best ways to stay motivated is to exercise with others. See our listing of regional contacts on page 12. Call them for suggestions about programs that may be available in your community.
- Play your favorite music and dance or move to the beat!

Remember, be sure to only choose activities that you feel safe doing. It is always wise to check with your doctor before starting an exercise program.

A physiotherapist may be an excellent resource for creating an exercise program to suit you personally. Consider consulting one.

POSTURE

Some of the first noticeable changes with Parkinson's are in your posture. There is a tendency for the shoulders to slump, the chin to stick out, and the elbows and knees to bend slightly. This makes the following more difficult:

- Breathing deeply
- Swallowing
- Speaking clearly and loudly
- Moving, balancing, and walking

The following photo illustrates common Parkinson's Posture:

You can help to prevent these changes.

MAKE GOOD POSTURE A HABIT

You can change your posture. When trying to develop good posture, repetition is very important. These suggestions need to be practiced frequently throughout your day – do not think of them as exercises to be done once a day and then forgotten.

Try to find a cue that will remind you to do these activities frequently. For example, if you are watching TV, you could do one activity each time a show breaks for a commercial.

1) Each day (as often as you can) check your posture. Stand against a wall and be sure your lower back and shoulder BLADES are touching the wall. Try to pull the back of your head towards the wall as well. Do not TIP your head back. As you walk away try to maintain this posture. Recheck at the next available wall. Or pick a spot in your home, ie. on the way into the bathroom or kitchen. Each time you walk past it, stop and do this posture check.

✔ correct

✘ incorrect

2) When you wake each morning, lie flat on your back, with just enough support to keep your head and neck from tipping back for 5 minutes. Do not press your shoulders or head back into the bed. RELAX! Allow gravity to stretch you as straight as possible. You may do this on the floor or other firm surface if you prefer.

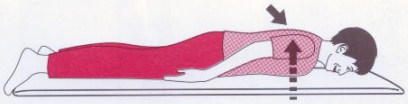
3) Every time you sit in a chair, make your shoulder blades touch the back of the chair. Hold for a few seconds. Do this three times, each time you sit down.

✔ correct ✘ incorrect

Parkinson Society Canada • EXERCISES FOR PEOPLE WITH PARKINSON'S

MAKE GOOD POSTURE A HABIT

4) Sitting in a chair, relax forward and let your arms and head hang down toward the floor. Then slowly roll back up starting low in your spine and letting your head come up last. Sit tall for several seconds. (If low blood pressure is a problem, skip this exercise.)



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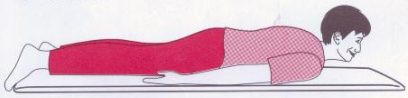
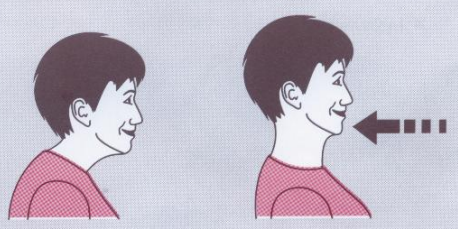


✘ incorrect

5) Lie face down on the bed or on a mat on the floor with your arms beside you. Gently pull your shoulder blades together. Keeping your head and neck in a straight line lift slightly. Hold for a few seconds. DO NOT LIFT WITH YOUR LOWER BACK.

6) Anytime you are sitting or standing, gently pull your chin straight in and straighten your neck. BE SURE NOT TO TIP YOUR HEAD BACK. Hold this position for five seconds and relax. Try not to let your head drop all the way forward again when you relax.



Begin End

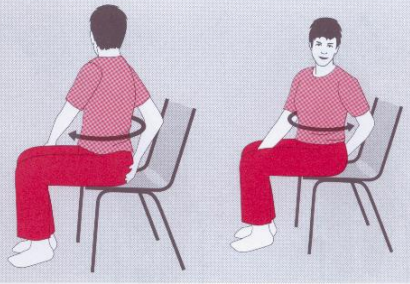
FLEXIBILITY OR STRETCHING

If you are less active and allow poor posture to develop, gradually over time your muscles and joints will tighten. Flexibility or stretching activities help you to maintain good range of movement in all of your joints and muscles. They are different than strengthening exercises because you hold positions and relax, allowing your muscles to gently stretch. Not everyone needs to do all of the following stretches. Choose any that seem right for you. Choose one or two to do before the strengthening exercises in the next section, then complete the rest of your chosen stretches after the strengthening exercises.

Safety Tips for Stretching Activities

a. Stretch slowly without bouncing or jerking the movements.
b. Hold the stretch in a comfortable position long enough for the muscles to relax
c. Aim for a stretched, relaxed feeling – avoid pain.
d. Breathe naturally – don't hold your breath.

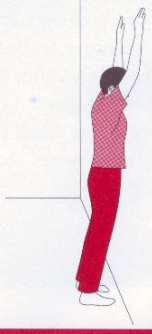
1) Sit tall on the edge of your seat. Turn your shoulders to the right. Reach your right hand behind you and stretch it towards your left hip. Turn your head and body as well. Relax your muscles and hold that position for at least ten seconds. STAY TALL! Repeat the other way.



2a) If you are tall enough, stand in a doorway and rest your hands on the frame overhead. Keep your arms straight. Gently lean forward. Feel a gentle stretch in your shoulders and chest. DO NOT OVER STRETCH! Hold for at least ten seconds.

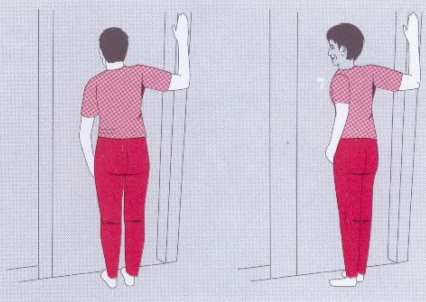
OR

2b) Stand facing a wall with your feet about a foot from the wall. Place your hands as high up on the wall as possible. Keep your head in line with your back. Gently lean forward so that your nose touches the wall. Don't arch your back. Feel the stretch in your shoulders and chest. Hold for at least ten seconds. If this is too easy, take one step back and try from this position.



FLEXIBILITY OR STRETCHING

3) Stand in a doorway. Bend your right arm. Rest your hand and forearm on the doorframe beside you. Gently turn your whole body to the left. Feel a stretch in your right shoulder. DO NOT OVER STRETCH! Relax the muscles in that shoulder. Hold for at least ten seconds. Repeat the other way.



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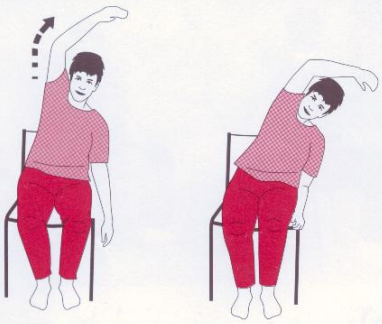
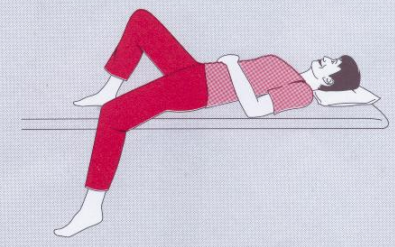


✔ correct ✘ incorrect

4) Sit tall in a chair. With your right arm curved over your head, slowly reach for the opposite wall. Do not tip your body. DO NOT BEND FORWARD. Feel a stretch in your right side muscles. Hold for at least ten seconds. Repeat the other way.

5) Lie on your back on a firm surface. Allow one leg to hang over the side. Press your back flat on the firm surface. Relax your leg that is hanging over, from the knee down. Feel a gentle stretch in the front of your hip. Allow your hip muscles to relax. Hold for at least ten seconds. Repeat with your other leg.



FLEXIBILITY OR STRETCHING

6a) Stand facing a wall (or hold a chair back for support). Rest hands on wall for balance. Place your left foot a comfortable distance behind your right foot. Keep your left leg straight, and gently bend your right knee leaning towards the wall. Feel a stretch in your left heel and calf. Hold for at least ten seconds. Repeat with the other leg.

OR

6b) Sit on the edge of a chair. Move right foot back under the chair so that your heel is slightly off the floor. Place your hands on your right knee and press down until your heel touches the floor. Allow your calf muscles to relax. Feel a stretch in your heel and calf. Hold for at least ten seconds. Repeat with left foot.

STRENGTHENING EXERCISES

Strengthening exercises challenge your muscles to remain healthy and strong. They require you to use your muscles repeatedly in a specific, controlled way. They can include activities such as golf or gardening or specific exercises. When you do regular activity and exercise you are:

- able to maintain and improve muscle strength
- able to improve balance and posture
- less likely to fall
- able to get around more safely
- able to carry out more daily activities.

If you do not have access to an exercise facility or a physiotherapist, here are some exercises that are particularly useful for people with Parkinson's. Adjust these exercises to suit your needs. If balance is a problem, stand behind a sturdy chair that will not tip easily and use the chair back for support. You may enjoy doing these exercises to music.

As you gain confidence in doing these exercises, use the chair back less for support. If you can eventually do these exercises without the support of the chair, you will be developing your balance even more.

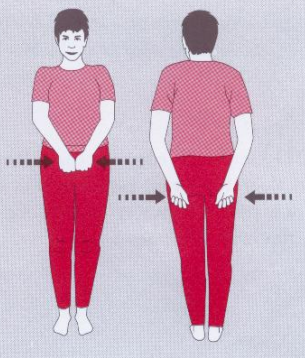
All of the following exercises may not be right for you. Choose the ones you feel safe doing.

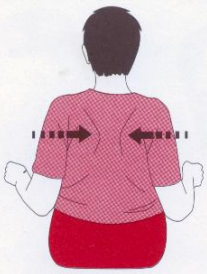
Some people like to do all of their exercises at one time while others prefer doing their exercises for short periods at different times during the day. Either way is correct.

DO EACH EXERCISE TEN TIMES IF YOU CAN.

DO EACH EXERCISE SLOWLY AND IN A CONTROLLED WAY.

1) Sit or stand tall. Keeping arms straight, slowly touch fists together in front and then behind your back. Pull your shoulder blades together as you touch in back.

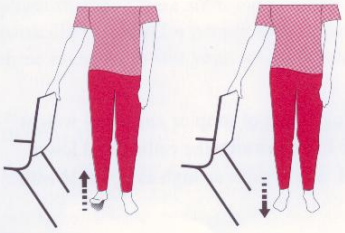




2) Sit or stand tall. Keep elbows at ninety degrees. Pull shoulder blades together in back.

STRENGTHENING EXERCISES

7) Stand behind a chair. If necessary, rest your hands on the back for support. Go up on your toes and come down flat.



8) Stand or sit. Pull up the toes of one foot and replace. Repeat with other foot. You can do this one foot at a time or alternating feet.

9) Stand tall with feet apart. Rest hands on a chair back for support if necessary. Slowly do small knee bends. Concentrate on squeezing the muscles in your buttocks when you straighten.

10) Stand tall. Hold the back of a chair for support if necessary. Keep your right leg straight. Slide your right leg back as far as you comfortably can. Repeat with other leg.

STRENGTHENING EXERCISES

11) Stand tall with feet wide apart. Hold the back of a chair for support if necessary. Lean on one bent knee and then the other. When you feel steady, try this without holding on.

12) March (around the room or outside). Ten steps.
March with long steps. Ten steps.
March. Ten steps.
March with knees high. Ten steps.
March. Ten steps.
March and swing arms. Ten steps.
Note: This can be done while sitting, just march in your seat with no long steps. It is more fun with music.

13) Exercises for the facial muscle groups can help to retain muscle integrity and the range of motion in the face and mouth – helping the range of facial expressions. Begin with a gentle rubbing of the face, like putting on cream. Repeat each of the following exercises a couple of times:

- Sour Lemon – tighten your facial muscles as if sucking on a lemon, then relax
- Eyebrow raising/frowning – lift your eyebrows to look surprised. Frown, creasing your forehead towards your eyes.
- Yawning – yawn dramatically
- Smiling – try smiling with your lips open, and then try again with your lips closed.

THE MORE ACTIVE YOU ARE, THE BETTER YOU FEEL!!!

More active people prolong their independence whether or not they have Parkinson's.

Even if you have not been very active, once you get started your body will adjust. Just try something … a little bit every day will make a difference. When planning your exercise program, it is best to choose a variety of activities to do on different days. For example, you might do exercises on one day, go for a walk the next and go swimming or do tai chi the next. Your schedule should suit your personal circumstances. Choosing a variety of activities is not only better for your overall health and fitness; it is also less likely to lead to boredom with your exercise program.

APPENDIX D: BERG BALANCE SCALE

Subjects are instructed that they must maintain their balance throughout the performance of the tasks. Points are deducted if the time or distance requirements are not met, if the subject needs assistance or uses an external support, or if supervision is required by the Assessor.

1. SITTING TO STANDING: The subject is instructed to stand without using their hand for support.
( ) 4 able to stand without using hands and stabilize independently
( ) 3 able to stand independently using hands
( ) 2 able to stand using hands after several tries
( ) 1 needs minimal aid to stand or stabilize
( ) 0 needs moderate or maximal support to stand

2. STANDING UNSUPPORTED: The subject is instructed to stand for two minutes without holding on. If the subject is able to stand without support for two minutes score full points for #3 and move to #4.
( ) 4 able to stand safely for two minutes
( ) 3 able to stand two minutes with supervision
( ) 2 able to stand 30 seconds unsupported
( ) 1 needs several tries to stand 30 seconds unsupported
( ) 0 unable to stand 30 seconds unsupported

3. SITTING WITH BACK UNSUPPORTED BUT FEET SUPPORTED ON THE FLOOR: The subject is instructed to sit with arms crossed for two minutes.
( ) 4 able to sit securely for two minutes
( ) 3 able to sit for two minutes under supervision
( ) 2 able to sit for 30 seconds
( ) 1 able to sit for 10 seconds
( ) 0 unable to sit unsupported for 10 seconds

4. STANDING TO SITTING: Instruct to subject to sit down.
( ) 4 sits safely with minimal use of hands
( ) 3 controls decent by using hands
( ) 2 uses back of legs against chair to control decent
( ) 1 sits independently but has uncontrolled decent
( ) 0 needs assistance to sit

5. TRANSFERS: Instruct subject to transfer towards a seat with armrests than towards a seat without armrests.
( ) 4 able to transfer safely with minimal use of hands
( ) 3 able to transfer safely with definite use of hands

( ) 2 able to transfer safely with verbal cuing and/or supervision
( ) 1 needs one person to assist
( ) 0 needs two people to assist or supervise to be safe

6. **STANDING UNSUPPORTED WITH EYES CLOSED**: Instruct the subject close their eyes and stand still for 10 seconds.

( ) 4 able to stand for 10 seconds safely
( ) 3 able to stand 10 seconds with supervision
( ) 2 able to strand 3 seconds
( ) 1 unable to keep eyes closed for 3 seconds but stays safely
( ) 0 needs help to keep from falling

7. **STANDING UNSUPPORTED WITH FEET TOGETHER**: Instruct subject to place feet together and stand without holding on.

( ) 4 able to place feet together independently and stand safely for one minute
( ) 3 able to place feet together independently and stand one minute with supervision
( ) 2 able to place feet together independently but unable to hold for 30 seconds
( ) 1 needs help to obtain position but able to stand 15 seconds feet together
( ) 0 needs help to attain position and unable to hold to 15 seconds

8. **REACHING FORWARD WITH OUTSTRETCHED ARM WHILE STANDING:** Instruct the subject to lift an arm to 90 degrees and to stretch out their fingers and reach forward as far as they can. The Assessor places a ruler at the end of the fingertips, but not touching. It is up to the individual as to which arm they chose to use. The recorder measure is the distance forward that the fingers reach while the subject is in the most forward lean position. When possible ask the subject to use both arms to minimize trunk rotation.

( ) 4 can reach forward confidently 25cm
( ) 3 can reach forward 12 cm
( ) 2 can reach forward 5 cm
( ) 1 reaches forward but needs supervision
( ) 0 loses balance while trying/requires external support

9. **PICK UP OBJECT FROM THE FLOOR FROM A STANDING POSITION**: Instruct subject to pick up the shoe/slipper, which is placed in front of their feet.

( ) 4 able to pick up slipper safely and easily
( ) 3 able to pick up slipper but needs supervision
( ) 2 unable to pick up but reaches 2-5cm from slipper and keeps balance independently
( ) 1 unable to pick up and needs supervision while trying
( ) 0 unable to try/needs assistance to keep from losing balance or falling

10. **TURNING TO LOOK BEHIND OVER LEFT AND RIGHT SHOULDERS WHILE STANDING:** Instruct subject to turn and look behind their left shoulder. Repeat to the right.
( ) 4 Looks behind from both sides and weight shifts well
( ) 3 Looks behind one side only other side shows less weight shift
( ) 2 Turns sideways only but maintains balance
( ) 1 Needs supervision when turning
( ) 0 Needs assistance to keep from losing balance or falling

11. **TURN 360 DEGREES:** Ask subject to turn around in a full circle. Pause. Then turn in a full circle in the other direction.
( ) 4 able to turn 360 degrees safely in 4 seconds or less
( ) 3 able to turn 360 degrees safely one side in 4 seconds or less
( ) 2 to turn 360 degrees safely but slowly
( ) 1 needs close supervision or verbal cuing
( ) 0 needs assistance while turning

12. **PLACE ALTERNATE FOOT ON STEP OR STOOL WHILE STANDING UNSUPPORTED:** Instruct subject to place each foot alternately on the step or stool. Continue until each foot has touched the stool four times.
( ) 4 able to stand independently and safely and complete 8 steps in 20 seconds
( ) 3 able to stand independently and complete 8 steps in >20 seconds
( ) 2 able to complete 4 steps without aid with supervision
( ) 1 able to complete >2 steps needing minimal supervision
( ) 0 needs assistance to keep from falling/unable to try

13. **STANDING UNSUPPORTED ONE FOOT IN FRONT:** This needs to be demonstrated to subject. Instruct subject to place one foot directly in front on the other. If they feel that cannot place one foot directly in front, instruct them to step far enough ahead so that the heel of the forward foot is ahead of the toes of the other foot. (In order to score three points, the length of the step should exceed the length of the other foot and the width of the stance should be the subjects normal stride length.)
( ) 4 able to place the foot stride length ahead independently and hold for 30 seconds
( ) 3 able to place foot ahead independently and hold for 30 seconds
( ) 2 able to take small step independently and hold 30 seconds
( ) 1 needs help to step but can hold 15 seconds
( ) 0 loses balance while stepping or standing

14. **STANDING ON ONE LEG:** Instruct subject to stand on one leg as long as they can without holding on.
( ) 4 able to lift leg independently and hold **>** 10 seconds
( ) 3 able to lift leg independently and hold 5-10 seconds
( ) 2 able to lift leg independently and hold > or equal to 3 seconds

( ) 1 tries to lift leg unable to hold 3 seconds but remains standing independently
( ) 0 unable to try or needs assistance to prevent fall

( ) Total Score (maximum 56)

APPENDIX E: PHYSICIAN LETTER

October 1, 2009.

Dear Dr. _____

In order to complete the program at the Canadian College of Osteopathy a research thesis is required. I am studying the effect of osteopathic treatment on the balance/mobility of patients with Parkinson's. This will be in conjunction with a standardized exercise program. The outcome measures used are the TIMED UP-AND-GO Test and the Berg Balance Scale. This study hopes to improve mobility, prove that gentle and non-invasive osteopathic manual treatment will increase function and improve health related quality life. This study utilizes four free treatments over approximately 4 weeks beginning August 2009 at a clinic located at 22-1450 Clark Avenue West in Thornhill.

I would greatly appreciate if you could ask patients who fulfill the criteria below if they would be interested in participating.

Inclusion Criteria:
See attached

Exclusion Criteria:

Please forward study contact information to interested patients who qualify.

Thanks very much for your support and help in our research.

Sincerely,
Thomas Hein
Registered Physiotherapist

Stacey Hauserman
Registered Massage Therapist

APPENDIX F: BUSINESS CARD

APPENDIX G: RECRUITMENT NOTICE

Are you interested in *FREE* treatment?

Stacey Hauserman and **Thomas Hein** are currently working on their graduate thesis study from the Canadian College of Osteopathy on:

The Effect of Osteopathic Treatment and Exercise for Mobility and Balance in those with PARKINSON'S DISEASE.

The purpose of the study is to determine whether osteopathic treatment can improve walking and balance.

Participants will undergo 4 osteopathic treatments and 5 individual and small group exercise classes **FREE of charge.**

To be included in this research participants are required to:

- Attend 5 sessions of exercise &/or osteopathy (a form of hands on manual therapy)
- Have a primary diagnoses of Parkinson's disease
- Be able to walk with or without an assisted device
- Be between the ages of 55 and 90

If you are interested in participating, please call 905-695-0371

Or email: parkinsons.study.osteopathy@gmail.com

APPENDIX H: TELEPHONE INTERVIEW

Preliminary Telephone Subject Screening Interview (First Contract)

Subject Name _____ Age _____

Contact Number(s)_____

Thank you for responding. This study is for a thesis at the Canadian College of Osteopathy and it is studying Parkinson's Disease.

1. Can you understand spoken and written English?
2. You will be required to visit a clinic 5 times (over 5 weeks) at 22-1450 Clark Avenue West in Thornhill. It is called Physioactive. Your first and last appointments may be up to 2 hours long and the others one hour or less. You must be able to attend all these appointments to participate in this study.
3. Parking is free or the clinic is accessible by public transit.
4. To qualify you need to be diagnosed with Parkinson's Disease by a physician. You need to have a doctor's note stating that diagnosis. It can be faxed to 905 695 0833 with ATTN: Parkinson's disease Study.
5. Osteopathy is a gentle, non-invasive, manual therapy but you still need to be in reasonable physical health. When you are at the clinic you will need to sign a consent sheet, fill out a health history (once) and several questionnaires.
6. You will be required to perform a functional test
7. Your Parkinson's disease medications will be monitored for the duration of the study.
8. All personal information is confidential and will be destroyed upon completion of the study.

To see if you qualify I need to ask you a few questions:

1. How old are you?
2. Do you have a physician diagnosis of Parkinson's disease?
3. Are you able to walk safely across a room?
4. Is there any reason that you might not be able to stand from sitting and walk three meters and then sit back down again?
5. Do you have significant back or leg pain that would inhibit you from getting up out of a chair?

6. Do you have any other diagnosed central nervous system disorders, such as stroke, multiple sclerosis, muscular dystrophy?
7. Have you ever had a traumatic brain injury?
8. Have you ever had osteopathic treatment?
9. Are you currently involved in a physiotherapy program? Would you be willing to cease other physical therapy throughout the duration of the study?
10. Do you have a diagnosed balance disorder?
11. Do you have any condition that would not allow you to complete functional activities of daily living?
12. Do you have hyper/hypotension that is not under control?
13. Have you had a heart attack?

Included: We will contact you when the study will begin and then you will make your appointments. Physioactive Clinic number 905-695-0371. If something comes up and you are unable to participate please let us know at this number (905) 695-0371. Please wear comfortable clothing/shoes to your first appointment.

Excluded: Thank you for your call and interest but we are sorry you are excluded from this study because of _____. If you would like to pursue osteopathic treatment please contact: www.osteopathyontario.com/ or the Canadian College of Osteopathy student clinic at 416-591-1123 (at Duncan and Richmond).

APPENDIX I: DATA LOG

Patient Number	Control	Experimental
1.		X
2.	X	
3.	X	
4.		X
5.		X
6.		X
7.		X
8.	X	
9.	X	X
10.		X
11.		X
12.		
13.		
14.		
15.		
16.		

APPENDIX J: MEDICAL QUESTIONNAIRE

Name:_____ **ID#:**_____
Address:_____ **D.O.B:**_____
_____ **AGE:**_____
Phone#:_____
Emergency Contact:_____**Phone:**_____
Physican's Name:_____ **Phone#:**_____
Current Medication:_____

Past Medical History

1. Trauma's/injuries (car accidents, fractures ect): please list year and type

2. Hospitalizations (year, and reason)

3. Surgeries (year and type)

4. Medical history

System Overview: Please circle any that apply and elaborate if necessary

1. **Musculoskeletal**: (back pain, shoulder, bursitis, tendonitis, arthritis, myelitis)

2. **Circulatory:** (phlebitis, varicosities, cramps, high/low blood pressure, cholesterol, cardiac problems, numbness, cold extremities)

3. **Respiratory**: (allergies, cold, cough, asthma, bronchitis, emphysema)

4. **Digestive:** (gastric reflux, heartburn, ulcer, bowel movements, gas, bloating, nausea, appetite, dysphasia, slow digestion, hemorrhoid, cirrhosis, hepatitis, gallstone, diarrhea, constipation)

5. **Urinary**: (cystitis, dysuria, polyuria, burning, kidney stone, kidney insufficiency, incontinence, bladder ptosis)

6. **Gynecology/Urogenital** : (prostatitis, sexual dysfunction, pregnancy, endometrosis)

7. **Ear/Nose/Throat**: (tinnitus, rhinitis, loss of hearing, glaucoma, transitory loss of vision)

8. **Nervous System**: (headaches, migraine)

9. **Skin:** (psoriasis, parasites, infection, eczema, dermatitis)

10. **Endocrine**: (thyroid, adrenals, pancreas, gonads)

11. **Sleeping habits**:

12. **Anything else that you feel is relevant:**

APPENDIX K: PRIVACY POLICY

Privacy Policy

Personal information is regulated federally and may be defined as any information of a personal nature including personal characteristics, health, activities and views. In Ontario, health specific information is regulated provincially and may be collected either orally or recorded. All information collected and used is for the primary purpose of compiling research to complete a thesis with the Canadian College of Osteopathy.

Paper information and electronic hardware is either under supervision or secured in a locked area at all times. Paper files containing personal information will be destroyed by shredding when they are no longer needed for the purpose of this study. Electronic data will be destroyed by deleting when the information is no longer required for the purpose of this study. In the unlikely event of privacy breech you will be informed immediately.

If you have any questions regarding these privacy practices please feel free to ask for clarification from either Stacey Hauserman or Thomas Hein.

If we are not able to satisfy your concerns please feel free to contact the Canadian College of Osteopathy at 416-

Thank you,

Stacey Hauserman, RMT
Thomas Hein, PT
(osteopathic thesis writers)

For general inquires regarding privacy information in Canada:
Information and Privacy Commissioner of Canada
12 Kent St, Ottawa, ON K1A 1H3
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APPENDIX L: APPOINTMENT FORM

Parkinson's Study

The following are your scheduled appointments for participation in the Parkinson's and Osteopathy study located at PhysioActive: 22-1450 Clark Ave. If for any reason you cannot make one of your appointments please call Stacey Hauserman at 416-839-4652.

1._____ Initial appointment 2 hours long

2._____

3._____

4._____

5. _____ Final appointment 2 hours long

In order to maintain blinding we ask that you do not discuss the study with other participants until completion of the study. If you have any questions pertaining to this study please feel free to contact Stacey Hauserman at 416-839-4652 or staceyhauserman@rogers.com.

Thank you for your participation.

APPENDIX M: OSTEOPATHIC ASSESSMENT FORM

Subject #_____
Date_____

Observations			
Line of Barre	Ascending	Descending	Neutral
Typology	Anterior	Posterior	Neutral
Compensation	Compensated	Decompensated	
Symmetry	Eyes		
	Ears		
	Shoulders		
	Scapula		
	Iliacs		

Vitality
0- absent
1-poor
2-fair
3-normal

Classification of Lesions According to CCO Methodology
4- Compaction
3- Non Physiological Without Respect to the Axis
2- Non Physiological With Respect to the Axis
1- Physiological
0-Normal

Cranial Base

Structure	Specific Structure	Position	Classification	Vitality
Sphenobasilar Symphysis				
Temporal	**OM** **Right** **Left**			
	Petrobasilar **Left** **Right**			
Occiput				

CO/C1	**Right**			
	Left			
	Bilateral			
CO/C1 on C2				
Ethmoid				
Jugular Foramen				
Foramen Magnum				

Cranial Vault

Structure	Position	Classification	Vitality
Parietals **Left** **Right**			
Temporals **Left** **Right**			
Frontal **Left** **Right**			
Occipital **Squama**			
Sphenoid **Greater** **Wings**			

Facial Bones

Structure	Position	Classification	Vitality
Zygoma **Left** **Right**			
Maxilla **Left** **Right**			
Mandible			
Palatine **Left** **Right**			
Vomer			
Lacrimal **Left** **Right**			
Nasal **Left** **Right**			

Sacrum

Structure	Position	Classification	Vitality
Sacrum			
L5 – S1			
Sacroiliac **Left** **Right**			

Cervical Spine

Segment	Position	Classification	Vitality
C1			
C2			
C3			

APPENDIX N: TECHNIQUE DESCRIPTIONS

The Venous Sinus Technique

This technique is designed to encourage vascular drainage within the encephalon. It is a seven part technique beginning at the base of the cranium at the jugular foramen. The intention of this technique is to provide optimal passage for the internal jugular vein via the sigmoid sinus. Throughout all seven steps of this technique the patient remains supine while the osteopath remains seated at the head of the patient.

1. Cranial base and jugular foramen: The patient is supine with their chin up while the treating osteopath sits at the head of the table. The osteopaths' forearms are resting to leave the hand free of tension for optimal palpatory sense. With the hands in contact, the 4th and 5th fingers are flexed and the tips of the 2nd and 3rd are extended. The middle fingers are in contact with each other and in close contact with the occipital condyles. The osteopath increases presence by leaning forward and dropping hands into table. A longitudinal traction and lateral spreading is performed by the osteopath moving the thorax posteriorly while the elbows are drawn towards one another. During this technique a balance or neutral point is felt, followed by expansion, release of heat, and increase in local vitality.
2. Transverse Sinus: With the hypothenar of both hands together, all fingers are flexed and are placed on either side of the inion in a horizontal line. The osteopath abducts their fingers creating a horizontal traction of the transverse sinus. This technique comes to completion with a release of heat, a palpatory sense of expansion and increased local vitality.
3. Lambda: The osteopath's thumbs are crossed and placed the parietals on either side of sagittal suture. The rest of the cranium is gently support by the

osteopath's fingers. The weight of the cranium on the thumbs will create the action needed to open the suture.

4. Straight Sinus: The medial portion of the osteopath's hands are in contact with the tips of the 5th metacarpals at the inion and the thumbs towards the vertex. The osteopath leans forward to create a reciprocal tension between the fingers. This is held until a balance point and an increase in expansion and retraction.
5. Obelion: The osteopath's thumbs are crossed and placed on either side of the saggital suture on the parietal bones, in relation to the obelion. The osteopath leans forward to increase the tension between their thumbs. This tension is held until a balance point, warmth and an increase in expansion and retraction are felt.
6. Metopic Suture: With elbows resting the osteopath places her fingers on either side of the metopic suture with her nails facing each other. The osteopath induces a longitudinal and lateral spreading of the fingers. This is held until a balance point, warmth and an increase expansion and retraction are felt.
7. Ethmoid: The osteopath places the middle finger intra-oral on the cruciform and the thumb on externally at the base of the nasal bones. The other hand is placed in relation to the greater wings of the sphenoid. The osteopath stabilizes the sphenoid and performs a slight traction in an oblique direction with the intra-oral hand. This action will disengage the ethmoid from the sphenoid. The second portion of this technique is to disengage the ethmoid from the frontal bones. This is similar to the last technique in that the intra-oral hand is in the same position, however the external hand is on the wings of the frontal bone. A frontal lift is performed in conjunction with a slight traction of the intra-oral hand. The

technique is completed when there is a neutral point, heat and an increase in expansion and retraction.

Normalization of the Posterior Fossa (EV4)

The EV4 is a reciprocal balance technique that is used to normalize the posterior fossa.

Patient is positioned supine with his chin slightly elevated. The osteopath sits at the head of the patient and supports her arms on the table. She places her 5th fingers at the level of the inion while the rest of her fingers are in relation to the falx cerebella insertion. The osteopath's thumbs are on the tips of the mastoids. The osteopath than spreads the posterior fossa into two compartments by providing a longitudinal and transverse between the fingers. This reciprocal balance is maintained around a calm reference point until a balance point, still point, and return of PRM are obtained. Dorsiflexion can be added to increase overall tension.

Compression of the Fourth Ventricle (CV4)

Patient is positioned in supine with her chin slightly elevated. The osteopath sits at the head of the patient with his arms supported. The osteopath hypothenar's and ulnar border of his hands are connected in a cup like position. The hands are placed below the tentorium with the thenar eminence of both hands on the external bevels of the occiput. The osteopath then applies a light medial compression followed by a reciprocal tension posteriorly with a light cephalic traction. A reciprocal balance is established at the osseous level followed by a second reciprocal balance corresponding to the ventricular volume. The osteopathy waits for a balance point, still point and release. After release an expansion and retraction of the ventricular system should be felt.

Parietal Lift

The patient is supine with the osteopath sitting at the head of the patient. The osteopaths fingers are placed along the external bevels of the parietal bones, with the thumbs joined above the head. The osteopath disengages the external bevels by creating a medial compression and then leans back to traction within the spring of the tissue, to disengage the parietals. This is held until a balance point, still point and return of the primary respiratory mechanism are achieved.

The second portion of the parietal lift is used to decompress the encephalon from the base of the cranium. The osteopath maintains the same position but allows palpation to sink to the second spring. A reciprocal tension is placed on the membranes on the inspiration phase and held for three to four primary respiratory cycles. This tension is released at 2/3 of the expiration cycle. This is repeated a few times until more motion is perceived.

Bilateral Rocking of the Temporal Bones

(W.G Sutherland, adapted by Phillipe Druelle, D.O.)

This technique is used to stimulate the central thalamic region and the basal ganglia.

The patient lies supine with a pillow under their knees and the chin slightly elevated. The treating osteopath sits at the head of the patient with their forearms supported. The osteopath's hands are cupped under the occiput with the tips of her thumbs at the mastoid processes. The osteopath leans forward flexing her trunk to bring the mastoids into a flexion state. This is done at the speed and intensity of the patient's

tissue. This rocking motion is continued until the initial inertia that was perceived has softened.

Lateral Ventricles

The osteopath sits at the head of the patient and places with both thumbs along the external bevels of the parietals facing the ceiling. The 4th and 5th fingers are place at the tips of the mastoid processes with the index fingers along the mandibular condyles and the fingers along the mandible. The osteopath achieves a reciprocal tension by straighten their thorax which causes a medial compression of the parietals and a anterior-inferior disengagement of the mandible.

Often a still point is required in the first spring in order to reach the lateral ventricles. Once the second spring level is achieved the osteopath establishes a reciprocal balance of the endocranium in its entirety. Following a still point and release the osteopath will land onto the lateral ventricles after perceiving an anterior tilt and a deeper sinking sensation. Once on the lateral ventricles the osteopath will balance each ventricle around its calm reference point. A third reference point is then established in the middle. After a still point and release are achieved there should be an increase in the ventricular motion.

APPENDIX O: CANADIAN COLLEGE OF OSTEOPATHY METHODOLOGY

Classification of Lesions in order of priority based on the clinical methodology of the Canadian College of Osteopathy

Classification of Lesions

1. **Scars**
2. **Compactions**
3. **Non-physiological without respect to the axis lesions**
4. **Non-physiological with respect to the axis lesions**
5. **Physiology lesions**

The priority can be further determined by the vitality of each tissue assessed.

APPENDIX P: STATISTICIAN LETTER

June 5, 2011

To Whom It May Concern,

I was the Statistician for the research thesis: 'Osteopathic Treatment for Balance in Parkinson's Disease Patients' authored by Stacey Hauserman.

The analysis, tables and graphs were done with SPSS Release 16. All work was done in a professional manner using appropriate statistical techniques.

Regards,

Peter Lewycky

B.Sc., M.Eng., P.Eng.

APPENDIX Q: LESION CLASSIFICATION CORRELATION TABLES

Crosstab

Count

Treatment			SBS					
			0	1	2	3	4	Total
Control	Time	pre			1	2	1	4
		post			1	2	0	3
		Total			2	4	1	7
Exp	Time	pre	1	1	1	2	2	7
		post	7	0	0	0	0	7
		Total	8	1	1	2	2	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.231	.324	-.691	.490	.771
			Time Dependent	-.214	.296	-.691	.490	.771
			SBS Dependent	-.250	.361	-.691	.490	.771
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.764	.103	-6.481	.000	.005
			Time Dependent	-.689	.122	-6.481	.000	.005
			SBS Dependent	-.857	.132	-6.481	.000	.005

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of SBS scores; Exact significance, p= 0.771. For Experimental, there is a statistically significant difference in the distribution of SBS scores between pre and post; Exact significance, p= 0.005.

Crosstab

Count

Treatment			TB OM - L					
			0	1	2	3	4	Total
Control	Time	pre	1	1			2	4
		post	0	1			2	3
		Total	1	2			4	7
Exp	Time	pre	0	1	2	1	3	7
		post	3	3	0	1	0	7
		Total	3	4	2	2	3	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.231	.324	.691	.490	.829
			Time Dependent	.214	.296	.691	.490	.829
			TB OM - L Dependent	.250	.361	.691	.490	.829
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.619	.123	-5.146	.000	.010
			Time Dependent	-.506	.104	-5.146	.000	.010
			TB OM - L Dependent	-.796	.155	-5.146	.000	.010

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of TB OM-L scores; Exact significance, p= 0.821. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.010.

Crosstab

Count

Treatment			TB OM R					
			0	1	2	3	4	Total
Control	Time	pre	1	2			1	4
		post	1	2			0	3
		Total	2	4			1	7
Exp	Time	pre	0	4	1	1	1	7
		post	6	1	0	0	0	7
		Total	6	5	1	1	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.231	.324	-.691	.490	.771
			Time Dependent	-.214	.296	-.691	.490	.771
			TB OM R Dependent	-.250	.361	-.691	.490	.771
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.783	.076	-11.456	.000	.003
			Time Dependent	-.682	.089	-11.456	.000	.003
			TB OM R Dependent	-.918	.080	-11.456	.000	.003

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of TB OM-R scores; Exact significance p= 0.771. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.001.

Crosstab

Count

Treatment			Pet - Bas L				
			0	1	2	4	Total
Control	Time	pre			1	3	4
		post			1	2	3
		Total			2	5	7
Exp	Time	pre	0	2	2	3	7
		post	5	1	0	1	7
		Total	5	3	2	4	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.091	.378	-.240	.811	1.000
			Time Dependent	-.100	.416	-.240	.811	1.000
			Pet - Bas L Dependent	-.083	.348	-.240	.811	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.600	.177	-3.501	.000	.016
			Time Dependent	-.507	.156	-3.501	.000	.016
			Pet - Bas L Dependent	-.735	.210	-3.501	.000	.016

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Pet-Bas L scores; Exact significance, p= 1.000. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.016.

Crosstab

Count

Treatment			pet - Bas R				
			0	1	2	4	Total
Control	Time	pre	1	3			4
		post	1	2			3
		Total	2	5			7
Exp	Time	pre	1	4	1	1	7
		post	7	0	0	0	7
		Total	8	4	1	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.091	.378	-.240	.811	1.000
			Time Dependent	-.100	.416	-.240	.811	1.000
			pet - Bas R Dependent	-.083	.348	-.240	.811	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.792	.109	-6.481	.000	.005
			Time Dependent	-.737	.125	-6.481	.000	.005
			pet - Bas R Dependent	-.857	.132	-6.481	.000	.005

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Pet-Bas R scores, p= 1.000, For Experimental, there is a

statistically significant difference in the distribution of scores between pre and post; p= 0.005.

Crosstab

Count

Treatment			Co-C1 L					
			0	1	2	3	4	Total
Control	Time	pre		1	0	1	2	4
		post		0	1	0	2	3
		Total		1	1	1	4	7
Exp	Time	pre	1	1		4	1	7
		post	7	0		0	0	7
		Total	8	1		4	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.148	.344	.429	.668	.829
			Time Dependent	.133	.311	.429	.668	.829
			Co-C1 L Dependent	.167	.388	.429	.668	.829
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.792	.109	-6.481	.000	.005
			Time Dependent	-.737	.125	-6.481	.000	.005
			Co-C1 L Dependent	-.857	.132	-6.481	.000	.005

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of C0-C1 L scores; Exact significance, p= 0.829, For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.005.

Crosstab

Count

Treatment			CO-C1R					
			0	1	2	3	4	Total
Control	Time	pre	2		1		1	4
		post	1		0		2	3
		Total	3		1		3	7
Exp	Time	pre	5	1		1		7
		post	7	0		0		7
		Total	12	1		1		14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.296	.354	.837	.403	.743
			Time Dependent	.267	.321	.837	.403	.743
			CO-C1R Dependent	.333	.397	.837	.403	.743
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.378	.134	-1.673	.094	.462
			Time Dependent	-.560	.138	-1.673	.094	.462
			CO-C1R Dependent	-.286	.171	-1.673	.094	.462

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of C0-C1 R scores; Exact significance p= 0.743. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.462.

Crosstab

Count

Treatment			C0-C1/C2			
			0	1	3	Total
Control	Time	pre	2		2	4
		post	2		1	3
		Total	4		3	7
Exp	Time	pre	2	1	4	7
		post	6	1	0	7
		Total	8	2	4	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.167	.369	-.450	.652	1.000
			Time Dependent	-.167	.370	-.450	.652	1.000
			C0-C1/C2 Dependent	-.167	.370	-.450	.652	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.610	.174	-3.276	.001	.037
			Time Dependent	-.571	.165	-3.276	.001	.037
			C0-C1/C2 Dependent	-.653	.199	-3.276	.001	.037

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of C0-C1/C2 scores; Exact significance, p= 1.000. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.037.

Crosstab

Count

Treatment			Eth				
			0	2	3	4	Total
Control	Time	pre	3		0	1	4
		post	1		1	1	3
		Total	4		1	2	7
Exp	Time	pre	1	2	2	2	7
		post	6	0	1	0	7
		Total	7	2	3	2	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.308	.353	.876	.381	.657
			Time Dependent	.286	.333	.876	.381	.657
			Eth Dependent	.333	.379	.876	.381	.657
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.614	.168	-3.731	.000	.021
			Time Dependent	-.538	.158	-3.731	.000	.021
			Eth Dependent	-.714	.191	-3.731	.000	.021

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Eth scores; Exact significance, p= 0.657. For Experimental, there

is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.021.

Crosstab

Count

Treatment			Jug L					
			0	1	2	3	4	Total
Control	Time	pre		0		2	2	4
		post		1		1	1	3
		Total		1		3	3	7
Exp	Time	pre	2		1		4	7
		post	7		0		0	7
		Total	9		1		4	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.296	.342	-.837	.403	.657
			Time Dependent	-.267	.299	-.837	.403	.657
			Jug L Dependent	-.333	.397	-.837	.403	.657
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.714	.135	-4.183	.000	.021
			Time Dependent	-.714	.137	-4.183	.000	.021
			Jug L Dependent	-.714	.171	-4.183	.000	.021

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Jug L scores; Exact significance, p= 0.657. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.021.

Crosstab

Count

Treatment			Jug R			
			0	1	2	Total
Control	Time	pre		2	2	4
		post		2	1	3
		Total		4	3	7
Exp	Time	pre	6	0	1	7
		post	6	1	0	7
		Total	12	1	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.167	.369	-.450	.652	1.000
			Time Dependent	-.167	.370	-.450	.652	1.000
			Jug R Dependent	-.167	.370	-.450	.652	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.027	.249	-.108	.914	1.000
			Time Dependent	-.040	.368	-.108	.914	1.000
			Jug R Dependent	-.020	.189	-.108	.914	1.000

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Jug R scores; Exact significance, p= 1.000. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 1.000.

Crosstab

Count

Treatment			For Mag				
			0	1	2	3	Total
Control	Time	pre		1	1	2	4
		post		2	0	1	3
		Total		3	1	3	7
Exp	Time	pre	0	2	2	3	7
		post	6	1	0	0	7
		Total	6	3	2	3	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.296	.354	-.837	.403	.743
			Time Dependent	-.267	.321	-.837	.403	.743
			For Mag Dependent	-.333	.397	-.837	.403	.743
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.797	.060	-21.326	.000	.002
			Time Dependent	-.681	.077	-21.326	.000	.002
			For Mag Dependent	-.959	.045	-21.326	.000	.002

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of For Mag scores; Exact significance, p= 0.743. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.002.

Crosstab

Count

Treatment			Par L				
			0	1	2	4	Total
Control	Time	pre	1	1		2	4
		post	1	1		1	3
		Total	2	2		3	7
Exp	Time	pre	3	1	2	1	7
		post	6	1	0	0	7
		Total	9	2	2	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.143	.348	-.411	.681	1.000
			Time Dependent	-.125	.305	-.411	.681	1.000
			Par L Dependent	-.167	.405	-.411	.681	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.471	.189	-2.215	.027	.119
			Time Dependent	-.453	.175	-2.215	.027	.119
			Par L Dependent	-.490	.221	-2.215	.027	.119

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Par L scores; Exact significance, p= 1.000. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.119.

Crosstab

Count

Treatment			Par T				
			0	1	2	4	Total
Control	Time	pre	3	1			4
		post	1	2			3
		Total	4	3			7
Exp	Time	pre	4	1	1	1	7
		post	6	1	0	0	7
		Total	10	2	1	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.417	.347	1.188	.235	.486
			Time Dependent	.417	.348	1.188	.235	.486
			Par T Dependent	.417	.348	1.188	.235	.486
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.340	.214	-1.444	.149	.315
			Time Dependent	-.356	.213	-1.444	.149	.315
			Par T Dependent	-.327	.226	-1.444	.149	.315

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Par T scores; Exact significance, p= 0.486. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.315.

Crosstab

Count

Treatment			TL				
			1	2	3	4	Total
Control	Time	pre	2	1		1	4
		post	1	2		0	3
		Total	3	3		1	7
Exp	Time	pre	4	1	1	1	7
		post	6	1	0	0	7
		Total	10	2	1	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.000	.363	.000	1.000	1.000
			Time Dependent	.000	.327	.000	1.000	1.000
			TL Dependent	.000	.408	.000	1.000	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.340	.214	-1.444	.149	.315
			Time Dependent	-.356	.213	-1.444	.149	.315
			TL Dependent	-.327	.226	-1.444	.149	.315

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of TL scores; Exact significance, p= 1.000. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.315.

Crosstab

Count

Treatment			TBR			
			0	1	2	Total
Control	Time	pre	1	2	1	4
		post	1	2	0	3
		Total	2	4	1	7
Exp	Time	pre	0	5	2	7
		post	3	4	0	7
		Total	3	9	2	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.231	.324	-.691	.490	.771
			Time Dependent	-.214	.296	-.691	.490	.771
			TB R Dependent	-.250	.361	-.691	.490	.771
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.580	.099	-3.577	.000	.073
			Time Dependent	-.569	.042	-3.577	.000	.073
			TB R Dependent	-.592	.165	-3.577	.000	.073

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of TB R scores; Exact significance, p= 0.771. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.073.

Crosstab

Count

Treatment			FB L				
			0	1	2	4	Total
Control	Time	pre	1	2		1	4
		post	0	3		0	3
		Total	1	5		1	7
Exp	Time	pre	0	5	1	1	7
		post	4	3	0	0	7
		Total	4	8	1	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.000	.369	.000	1.000	1.000
			Time Dependent	.000	.386	.000	1.000	1.000
			FB L Dependent	.000	.354	.000	1.000	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.642	.096	-4.555	.000	.033
			Time Dependent	-.596	.065	-4.555	.000	.033
			FB L Dependent	-.694	.152	-4.555	.000	.033

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of FB L scores; Exact significance, p= 1.000. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.033.

Crosstab

Count

Treatment			FB R				
			0	1	2	4	Total
Control	Time	pre	2	2	0		4
		post	1	1	1		3
		Total	3	3	1		7
Exp	Time	pre	1	4	1	1	7
		post	6	1	0	0	7
		Total	7	5	1	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.296	.342	.837	.403	.657
			Time Dependent	.267	.299	.837	.403	.657
			FB R Dependent	.333	.397	.837	.403	.657
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.679	.146	-4.459	.000	.021
			Time Dependent	-.617	.138	-4.459	.000	.021
			FB R Dependent	-.755	.169	-4.459	.000	.021

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of FB R scores; Exact significance, p= 0.657. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.021.

Crosstab

Count

Treatment			Occ Sq					
			0	1	2	3	4	Total
Control	Time	pre		1	1	1	1	4
		post		1	1	1	0	3
		Total		2	2	2	1	7
Exp	Time	pre	0	3	1	3		7
		post	6	1	0	0		7
		Total	6	4	1	3		14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.200	.316	-.627	.530	.771
			Time Dependent	-.167	.262	-.627	.530	.771
			Occ Sq Dependent	-.250	.397	-.627	.530	.771
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.793	.067	-14.981	.000	.002
			Time Dependent	-.687	.080	-14.981	.000	.002
			Occ Sq Dependent	-.939	.063	-14.981	.000	.002

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Occ Sq scores; Exact significance, p= 0.771. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.002.

Crosstab

Count

Treatment			Sph > wing					
			0	1	2	3	4	Total
Control	Time	pre	2		1	1		4
		post	1		2	0		3
		Total	3		3	1		7
Exp	Time	pre	3	1	0	1	2	7
		post	4	0	1	2	0	7
		Total	7	1	1	3	2	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.000	.363	.000	1.000	1.000
			Time Dependent	.000	.327	.000	1.000	1.000
			Sph > wing Dependent	.000	.408	.000	1.000	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.191	.238	-.799	.424	.510
			Time Dependent	-.167	.206	-.799	.424	.510
			Sph > wing Dependent	-.224	.281	-.799	.424	.510

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Sph > wing scores; Exact significance, p= 1.000. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.510.

Crosstab

Count

Treatment			Zyg L 0	1	3	4	Total
Control	Time	pre	1	3			4
		post	1	2			3
		Total	2	5			7
Exp	Time	pre	1	4	0	2	7
		post	4	2	1	0	7
		Total	5	6	1	2	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.091	.378	-.240	.811	1.000
			Time Dependent	-.100	.416	-.240	.811	1.000
			Zyg L Dependent	-.083	.348	-.240	.811	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.404	.213	-1.898	.058	.122
			Time Dependent	-.354	.188	-1.898	.058	.122
			Zyg L Dependent	-.469	.247	-1.898	.058	.122

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Zyg L scores; Exact significance, p= 1.000. For Experimental,

there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.122.

Crosstab

Count

Treatment			Zyg R			
			0	1	2	Total
Control	Time	pre	3	1		4
		post	3	0		3
		Total	6	1		7
Exp	Time	pre	3	2	2	7
		post	7	0	0	7
		Total	10	2	2	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.333	.180	-1.146	.252	1.000
			Time Dependent	-.500	.204	-1.146	.252	1.000
			Zyg R Dependent	-.250	.217	-1.146	.252	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.602	.138	-3.055	.002	.070
			Time Dependent	-.636	.136	-3.055	.002	.070
			Zyg R Dependent	-.571	.187	-3.055	.002	.070

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Zyg R scores; Exact significance, p= 1.000. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.070.

Crosstab

Count

Treatment			Max L				
			0	1	2	3	Total
Control	Time	pre	2	1	1	0	4
		post	0	1	1	1	3
		Total	2	2	2	1	7
Exp	Time	pre	3	2	1	1	7
		post	4	3	0	0	7
		Total	7	5	1	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.533	.213	2.366	.018	.200
			Time Dependent	.444	.176	2.366	.018	.200
			Max L Dependent	.667	.272	2.366	.018	.200
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.239	.236	-.981	.327	.478
			Time Dependent	-.217	.208	-.981	.327	.478
			Max L Dependent	-.265	.271	-.981	.327	.478

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Max L scores; Exact significance, p= 0.200. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.478.

Crosstab

Count

Treatment			Max R					
			0	1	2	3	4	Total
Control	Time	pre	1	1	2			4
		post	2	1	0			3
		Total	3	2	2			7
Exp	Time	pre	1	3	1	1	1	7
		post	6	0	1	0	0	7
		Total	7	3	2	1	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.500	.259	-1.871	.061	.314
			Time Dependent	-.438	.229	-1.871	.061	.314
			Max R Dependent	-.583	.305	-1.871	.061	.314
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.591	.179	-3.405	.001	.021
			Time Dependent	-.515	.169	-3.405	.001	.021
			Max R Dependent	-.694	.204	-3.405	.001	.021

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Max R scores; Exact significance, p= 0.314. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.021.

Crosstab

Count

Treatment			Man L					
			0	1	2	3	4	Total
Control	Time	pre	0	2	1		1	4
		post	1	1	1		0	3
		Total	1	3	2		1	7
Exp	Time	pre	2	1	3	1	0	7
		post	4	0	1	1	1	7
		Total	6	1	4	2	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.345	.290	-1.126	.260	.486
			Time Dependent	-.294	.238	-1.126	.260	.486
			Man L Dependent	-.417	.367	-1.126	.260	.486
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.085	.258	-.331	.741	.781
			Time Dependent	-.072	.223	-.331	.741	.781
			Man L Dependent	-.102	.309	-.331	.741	.781

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Man L scores; Exact significance, p= 0.486. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.781.

Crosstab

Count

Treatment			Mand R					
			0	1	2	3	4	Total
Control	Time	pre	2	1			1	4
		post	2	1			0	3
		Total	4	2			1	7
Exp	Time	pre	4	1	1	1		7
		post	5	1	0	1		7
		Total	9	2	1	2		14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.231	.324	-.691	.490	.829
			Time Dependent	-.214	.296	-.691	.490	.829
			Mand R Dependent	-.250	.361	-.691	.490	.829
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.137	.250	-.547	.585	.780
			Time Dependent	-.132	.240	-.547	.585	.780
			Mand R Dependent	-.143	.261	-.547	.585	.780

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Man R scores; Exact significance, p= 0.829. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.780.

Crosstab

Count

Treatment			Pal L					
			0	1	2	3	4	Total
Control	Time	pre	2	1	1			4
		post	3	0	0			3
		Total	5	1	1			7
Exp	Time	pre	5	1		1	0	7
		post	5	1		0	1	7
		Total	10	2		1	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.522	.189	-1.954	.051	.429
			Time Dependent	-.545	.203	-1.954	.051	.429
			Pal L Dependent	-.500	.250	-1.954	.051	.429
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.021	.257	.083	.934	1.000
			Time Dependent	.022	.268	.083	.934	1.000
			Pal L Dependent	.020	.246	.083	.934	1.000

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Pal L scores; Exact significance, p= 0.429. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 1.000.

Crosstab

Count

Treatment			Pal R				
			0	1	2	4	Total
Control	Time	pre	3			1	4
		post	2			1	3
		Total	5			2	7
Exp	Time	pre	4	1	1	1	7
		post	7	0	0	0	7
		Total	11	1	1	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.091	.378	.240	.811	1.000
			Time Dependent	.100	.416	.240	.811	1.000
			Pal R Dependent	.083	.348	.240	.811	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.494	.137	-2.291	.022	.192
			Time Dependent	-.583	.137	-2.291	.022	.192
			Pal R Dependent	-.429	.187	-2.291	.022	.192

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Pal R scores; Exact significance, p= 1.000. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.192.

Crosstab

Count

Treatment			Vomer					
			0	1	2	3	4	Total
Control	Time	pre	1		2		1	4
		post	2		0		1	3
		Total	3		2		2	7
Exp	Time	pre	0	2	1	3	1	7
		post	5	1	0	1	0	7
		Total	5	3	1	4	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.214	.386	-.558	.577	.571
			Time Dependent	-.188	.341	-.558	.577	.571
			Vomer Dependent	-.250	.447	-.558	.577	.571
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.628	.149	-4.375	.000	.013
			Time Dependent	-.528	.132	-4.375	.000	.013
			Vomer Dependent	-.776	.177	-4.375	.000	.013

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Vomer scores; Exact significance, p= 0.571. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.013.

Crosstab

Count

Treatment			Lac L				
			0	1	3	4	Total
Control	Time	pre	2	0	1	1	4
		post	0	1	0	2	3
		Total	2	1	1	3	7
Exp	Time	pre	3	1		3	7
		post	5	0		2	7
		Total	8	1		5	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.414	.293	1.437	.151	.400
			Time Dependent	.353	.261	1.437	.151	.400
			Lac L Dependent	.500	.344	1.437	.151	.400
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.235	.253	-.929	.353	.592
			Time Dependent	-.226	.245	-.929	.353	.592
			Lac L Dependent	-.245	.264	-.929	.353	.592

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Lac L scores; Exact significance, p= 0.400. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.592.

Crosstab

Count

Treatment			Lac R				
			0	1	2	3	Total
Control	Time	pre	3	1			4
		post	2	1			3
		Total	5	2			7
Exp	Time	pre	5	1	0	1	7
		post	6	0	1	0	7
		Total	11	1	1	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.091	.378	.240	.811	1.000
			Time Dependent	.100	.416	.240	.811	1.000
			Lac R Dependent	.083	.348	.240	.811	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.165	.245	-.656	.512	.731
			Time Dependent	-.194	.288	-.656	.512	.731
			Lac R Dependent	-.143	.218	-.656	.512	.731

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Lac R scores; Exact significance, p= 1.000. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.731.

Crosstab

Count

Treatment			Nas L					
			0	1	2	3	4	Total
Control	Time	pre	1		1		2	4
		post	0		0		3	3
		Total	1		1		5	7
Exp	Time	pre	2	0		1	4	7
		post	4	1		0	2	7
		Total	6	1		1	6	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.522	.189	1.954	.051	.429
			Time Dependent	.545	.203	1.954	.051	.429
			Nas L Dependent	.500	.250	1.954	.051	.429
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.309	.238	-1.298	.194	.286
			Time Dependent	-.279	.215	-1.298	.194	.286
			Nas L Dependent	-.347	.267	-1.298	.194	.286

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Nas L scores; Exact significance, p= 0.429. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.286.

Crosstab

Count

Treatment			Nas R			
			0	3	4	Total
Control	Time	pre	3		1	4
		post	3		0	3
		Total	6		1	7
Exp	Time	pre	4	2	1	7
		post	7	0	0	7
		Total	11	2	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.333	.180	-1.146	.252	1.000
			Time Dependent	-.500	.204	-1.146	.252	1.000
			Nas R Dependent	-.250	.217	-1.146	.252	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.500	.142	-2.291	.022	.192
			Time Dependent	-.600	.139	-2.291	.022	.192
			Nas R Dependent	-.429	.187	-2.291	.022	.192

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Nas R scores; Exact significance, p= 1.000. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.192.

Crosstab

Count

Treatment			Sacrum 0	Sacrum 1	Sacrum 3	Total
Control	Time	pre	1	2	1	4
		post	0	2	1	3
		Total	1	4	2	7
Exp	Time	pre	0	3	4	7
		post	4	3	0	7
		Total	4	6	4	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.231	.324	.691	.490	.771
			Time Dependent	.214	.296	.691	.490	.771
			Sacrum Dependent	.250	.361	.691	.490	.771
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.708	.078	-7.201	.000	.012
			Time Dependent	-.625	.051	-7.201	.000	.012
			Sacrum Dependent	-.816	.113	-7.201	.000	.012

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Sacrum scores; Exact significance, p= 0.771. For Experimental,

there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.012.

Crosstab

Count

Treatment			L5-S1					
			0	1	2	3	4	Total
Control	Time	pre	1		1	1	1	4
		post	1		0	1	1	3
		Total	2		1	2	2	7
Exp	Time	pre	1	0	2	1	3	7
		post	3	2	0	0	2	7
		Total	4	2	2	1	5	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.067	.349	.191	.849	.971
			Time Dependent	.056	.291	.191	.849	.971
			L5-S1 Dependent	.083	.436	.191	.849	.971
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.328	.226	-1.437	.151	.202
			Time Dependent	-.274	.187	-1.437	.151	.202
			L5-S1 Dependent	-.408	.284	-1.437	.151	.202

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of L5-S1 scores; Exact significance, p= 0.971. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.202.

Crosstab

Count

Treatment			INN L				
			0	1	3	4	Total
Control	Time	pre	2	2			4
		post	2	1			3
		Total	4	3			7
Exp	Time	pre	4	1	1	1	7
		post	6	1	0	0	7
		Total	10	2	1	1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.167	.369	-.450	.652	1.000
			Time Dependent	-.167	.370	-.450	.652	1.000
			INN L Dependent	-.167	.370	-.450	.652	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.340	.214	-1.444	.149	.315
			Time Dependent	-.356	.213	-1.444	.149	.315
			INN L Dependent	-.327	.226	-1.444	.149	.315

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of INN L scores; Exact significance, p= 1.000. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.315.

Crosstab

Count

Treatment			Inn R				
			0	1	2	3	Total
Control	Time	pre	1	1	2	0	4
		post	1	0	1	1	3
		Total	2	1	3	1	7
Exp	Time	pre	1	1	1	4	7
		post	2	5	0	0	7
		Total	3	6	1	4	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.207	.366	.558	.577	.686
			Time Dependent	.176	.309	.558	.577	.686
			Inn R Dependent	.250	.447	.558	.577	.686
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.552	.196	-2.770	.006	.031
			Time Dependent	-.478	.170	-2.770	.006	.031
			Inn R Dependent	-.653	.236	-2.770	.006	.031

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of INN R scores; Exact significance, p= 0.686. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.031.

Crosstab

Count

Treatment			C3				
			0	1	2	3	Total
Control	Time	pre	2	2	0	0	4
		post	1	0	1	1	3
		Total	3	2	1	1	7
Exp	Time	pre	2	4		1	7
		post	7	0		0	7
		Total	9	4		1	14

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.414	.328	1.190	.234	.400
			Time Dependent	.353	.267	1.190	.234	.400
			C3 Dependent	.500	.417	1.190	.234	.400
Exp	Ordinal by Ordinal	Somers' d	Symmetric	-.714	.135	-4.183	.000	.021
			Time Dependent	-.714	.137	-4.183	.000	.021
			C3 Dependent	-.714	.171	-4.183	.000	.021

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of C3 scores; Exact significance, p= 0.571. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.021.

APPENDIX R: VITALITY CORRELATION TABLES

Time * SBS * Treatment Crosstabulation

Count

Treatment			SBS				
			0	1	2	3	Total
Control	Time	pre	3	1			4
		post	2	1			3
		Total	5	2			7
Exp	Time	pre	4	2	0	0	6
		post	0	0	2	5	7
		Total	4	2	2	5	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.091	.378	.240	.811	1.000
			Time Dependent	.100	.416	.240	.811	1.000
			SBS Dependent	.083	.348	.240	.811	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.824	.030	23.367	.000	.001
			Time Dependent	.700	.051	23.367	.000	.001
			SBS Dependent	1.000	.000	23.367	.000	.001

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of SBS scores; Exact significance, p= 1.000. For Experimental, there is a statistically significant difference in the distribution of SBS scores between pre and post; Exact significance, p= 0.001.

Crosstab

Count

Treatment			TB OM - L				
			0	1	2	3	Total
Control	Time	pre	2	2			4
		post	0	3			3
		Total	2	5			7
Exp	Time	pre	5	1	0	0	6
		post	1	2	3	1	7
		Total	6	3	3	1	13

Symmetric Measures

Treatment			Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Kendall's tau-b	.548	.209	1.954	.051	.429
		Kendall's tau-c	.490	.251	1.954	.051	.429
		Gamma	1.000	.000	1.954	.051	.429
		N of Valid Cases	7				
Exp	Ordinal by Ordinal	Kendall's tau-b	.674	.138	4.718	.000	.015
		Kendall's tau-c	.781	.166	4.718	.000	.015
		Gamma	.943	.080	4.718	.000	.013
		N of Valid Cases	13				

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of TB OM-L scores; Exact significance, p= 0.429. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.015.

Crosstab

Count

Treatment			TB OM R 0	1	2	3	Total
Control	Time	pre	2	1	1		4
		post	0	3	0		3
		Total	2	4	1		7
Exp	Time	pre	3	3	0	0	6
		post	0	0	2	5	7
		Total	3	3	2	5	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.231	.388	.602	.547	.771
			Time Dependent	.214	.368	.602	.547	.771
			TB OM R Dependent	.250	.415	.602	.547	.771
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.816	.024	23.367	.000	.001
			Time Dependent	.689	.041	23.367	.000	.001
			TB OM R Dependent	1.000	.000	23.367	.000	.001

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of TB OM-R scores; Exact significance p= 0.771. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.001.

Crosstab

Count

Treatment			Pet - Bas L				
			0	1	2	3	Total
Control	Time	pre	4	0			4
		post	1	2			3
		Total	5	2			7
Exp	Time	pre	4	2	0	0	6
		post	1	0	4	2	7
		Total	5	2	4	2	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.727	.215	2.366	.018	.143
			Time Dependent	.800	.179	2.366	.018	.143
			Pet - Bas L Dependent	.667	.272	2.366	.018	.143
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.667	.145	4.453	.000	.012
			Time Dependent	.567	.127	4.453	.000	.012
			Pet - Bas L Dependent	.810	.178	4.453	.000	.012

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Pet-Bas L scores; Exact significance, p= 0.143. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.012.

Crosstab

Count

Treatment			pet - Bas R				
			0	1	2	3	Total
Control	Time	pre		1	3		4
		post		2	1		3
		Total		3	4		7
Exp	Time	pre	2	3	1	0	6
		post	0	0	2	5	7
		Total	2	3	3	5	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.417	.347	-1.188	.235	.486
			Time Dependent	-.417	.348	-1.188	.235	.486
			pet - Bas R Dependent	-.417	.348	-1.188	.235	.486
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.777	.051	14.422	.000	.003
			Time Dependent	.656	.054	14.422	.000	.003
			pet - Bas R Dependent	.952	.052	14.422	.000	.003

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Pet-Bas R scores, p= 0.486, For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; p= 0.003.

Crosstab

Count

Treatment			Co-C1 L				
			0	1	2	3	Total
Control	Time	pre	3	0	1		4
		post	1	2	0		3
		Total	4	2	1		7
Exp	Time	pre	4	1	1	0	6
		post	0	0	4	3	7
		Total	4	1	5	3	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.231	.395	.602	.547	.829
			Time Dependent	.214	.380	.602	.547	.829
			Co-C1 L Dependent	.250	.415	.602	.547	.829
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.752	.067	9.034	.000	.003
			Time Dependent	.644	.058	9.034	.000	.003
			Co-C1 L Dependent	.905	.092	9.034	.000	.003

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of C0-C1 L scores; Exact significance, p= 0.829, For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.003.

Crosstab

Count

Treatment			CO-C1R				
			0	1	2	3	Total
Control	Time	pre	1	2	0	1	4
		post	2	0	1	0	3
		Total	3	2	1	1	7
Exp	Time	pre	1	2	2	1	6
		post	0	0	2	5	7
		Total	1	2	4	6	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.276	.348	-.802	.422	.486
			Time Dependent	-.235	.303	-.802	.422	.486
			CO-C1R Dependent	-.333	.414	-.802	.422	.486
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.592	.164	3.343	.001	.040
			Time Dependent	.518	.138	3.343	.001	.040
			CO-C1R Dependent	.690	.204	3.343	.001	.040

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of C0-C1 R scores; Exact significance p= 0.486. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.040.

Crosstab

Count

Treatment			C0-C1/C2				
			0	1	2	3	Total
Control	Time	pre	1	1	2	0	4
		post	1	0	1	1	3
		Total	2	1	3	1	7
Exp	Time	pre	4		1	1	6
		post	0		5	2	7
		Total	4		6	3	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.207	.366	.558	.577	.686
			Time Dependent	.176	.309	.558	.577	.686
			C0-C1/C2 Dependent	.250	.447	.558	.577	.686
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.521	.234	2.212	.027	.031
			Time Dependent	.463	.209	2.212	.027	.031
			C0-C1/C2 Dependent	.595	.268	2.212	.027	.031

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of C0-C1/C2 scores; Exact significance, p= 0.686. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.031.

Crosstab

Count

Treatment			Eth				
			0	1	2	3	Total
Control	Time	pre	1		2	1	4
		post	2		1	0	3
		Total	3		3	1	7
Exp	Time	pre	4	2	0	0	6
		post	1	0	4	2	7
		Total	5	2	4	2	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.444	.278	-1.528	.127	.400
			Time Dependent	-.400	.249	-1.528	.127	.400
			Eth Dependent	-.500	.323	-1.528	.127	.400
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.667	.145	4.453	.000	.012
			Time Dependent	.567	.127	4.453	.000	.012
			Eth Dependent	.810	.178	4.453	.000	.012

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Eth scores; Exact significance, p= 0.400. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.012.

Crosstab

Count

Treatment			Jug L				
			0	1	2	3	Total
Control	Time	pre	4		0		4
		post	2		1		3
		Total	6		1		7
Exp	Time	pre	4	1	1	0	6
		post	0	1	4	2	7
		Total	4	2	5	2	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.444	.219	1.214	.225	.429
			Time Dependent	.667	.192	1.214	.225	.429
			Jug L Dependent	.333	.272	1.214	.225	.429
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.686	.115	5.748	.000	.009
			Time Dependent	.583	.103	5.748	.000	.009
			Jug L Dependent	.833	.141	5.748	.000	.009

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Jug L scores; Exact significance, p= 0.429. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.009. Post has higher scores.

Crosstab

Count

Treatment			Jug R 1	2	3	Total
Control	Time	pre	2	2		4
		post	1	2		3
		Total	3	4		7
Exp	Time	pre	4	2	0	6
		post	1	2	4	7
		Total	5	4	4	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.167	.369	.450	.652	1.000
			Time Dependent	.167	.370	.450	.652	1.000
			Jug R Dependent	.167	.370	.450	.652	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.612	.162	3.719	.000	.035
			Time Dependent	.536	.144	3.719	.000	.035
			Jug R Dependent	.714	.190	3.719	.000	.035

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Jug R scores; Exact significance, p= 1.000. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.035.

Crosstab

Count

Treatment			For Mag 0	1	2	3	Total
Control	Time	pre	3	1	0		4
		post	1	0	2		3
		Total	4	1	2		7
Exp	Time	pre	1	5	0	0	6
		post	0	0	6	1	7
		Total	1	5	6	1	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.538	.306	1.650	.099	.257
			Time Dependent	.500	.281	1.650	.099	.257
			For Mag Dependent	.583	.348	1.650	.099	.257
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.884	.049	23.367	.000	.001
			Time Dependent	.792	.089	23.367	.000	.001
			For Mag Dependent	1.000	.000	23.367	.000	.001

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of For Mag scores; Exact significance, p= 0.257. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.001.

Crosstab

Count

Treatment			Par L				
			0	1	2	3	Total
Control	Time	pre	1	2	0	1	4
		post	2	0	1	0	3
		Total	3	2	1	1	7
Exp	Time	pre	3	0	3	0	6
		post	0	1	2	4	7
		Total	3	1	5	4	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.276	.348	-.802	.422	.486
			Time Dependent	-.235	.303	-.802	.422	.486
			Par L Dependent	-.333	.414	-.802	.422	.486
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.594	.151	3.903	.000	.020
			Time Dependent	.508	.134	3.903	.000	.020
			Par L Dependent	.714	.180	3.903	.000	.020

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Par L scores; Exact significance, p= 0.486. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.020.

Crosstab

Count

Treatment			Par T				
			0	1	2	3	Total
Control	Time	pre	0	1	2	1	4
		post	1	0	2	0	3
		Total	1	1	4	1	7
Exp	Time	pre	2		4	0	6
		post	1		1	5	7
		Total	3		5	5	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.296	.304	-.921	.357	.486
			Time Dependent	-.267	.265	-.921	.357	.486
			Par T Dependent	-.333	.360	-.921	.357	.486
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.577	.206	2.876	.004	.048
			Time Dependent	.509	.188	2.876	.004	.048
			Par T Dependent	.667	.230	2.876	.004	.048

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Par T scores; Exact significance, p= 0.486. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.020.

Crosstab

Count

Treatment			TL			
			0	1	2	Total
Control	Time	pre	3	1		4
		post	0	3		3
		Total	3	4		7
Exp	Time	pre	3	3	0	6
		post	2	2	3	7
		Total	5	5	3	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.750	.203	3.240	.001	.143
			Time Dependent	.750	.217	3.240	.001	.143
			TL Dependent	.750	.217	3.240	.001	.143
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.371	.222	1.628	.104	.269
			Time Dependent	.327	.191	1.628	.104	.269
			TL Dependent	.429	.263	1.628	.104	.269

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of TL scores; Exact significance, p= 0.143. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.269.

Crosstab

Count

Treatment			TB R				
			0	1	2	3	Total
Control	Time	pre		1	3		4
		post		2	1		3
		Total		3	4		7
Exp	Time	pre	1	5	0	0	6
		post	0	1	1	5	7
		Total	1	6	1	5	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.417	.347	-1.188	.235	.486
			Time Dependent	-.417	.348	-1.188	.235	.486
			TB R Dependent	-.417	.348	-1.188	.235	.486
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.779	.095	7.434	.000	.004
			Time Dependent	.698	.103	7.434	.000	.004
			TB R Dependent	.881	.112	7.434	.000	.004

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of TB R scores; Exact significance, p= 0.486. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.004.

Crosstab

Count

Treatment			FB L				
			0	1	2	3	Total
Control	Time	pre	2	1	1	0	4
		post	1	1	0	1	3
		Total	3	2	1	1	7
Exp	Time	pre	3	3	0	0	6
		post	0	2	1	4	7
		Total	3	5	1	4	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.207	.341	.602	.547	.657
			Time Dependent	.176	.290	.602	.547	.657
			FB L Dependent	.250	.415	.602	.547	.657
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.713	.072	7.813	.000	.009
			Time Dependent	.610	.060	7.813	.000	.009
			FB L Dependent	.857	.103	7.813	.000	.009

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of FB L scores; Exact significance, p= 0.657. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.009.

Crosstab

Count

Treatment			FB R 0	1	2	3	Total
Control	Time	pre	1	0	3	0	4
		post	1	1	0	1	3
		Total	2	1	3	1	7
Exp	Time	pre	2	3	1	0	6
		post	0	0	1	6	7
		Total	2	3	2	6	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.069	.412	-.168	.867	1.000
			Time Dependent	-.059	.353	-.168	.867	1.000
			FB R Dependent	-.083	.496	-.168	.867	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.820	.054	18.774	.000	.002
			Time Dependent	.707	.075	18.774	.000	.002
			FB R Dependent	.976	.031	18.774	.000	.002

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of FB R scores; Exact significance, p= 1.000. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.002.

Crosstab

Count

Treatment			Occ Sq				
			0	1	2	3	Total
Control	Time	pre	1	3	0		4
		post	0	2	1		3
		Total	1	5	1		7
Exp	Time	pre	3	1	2	0	6
		post	0	0	6	1	7
		Total	3	1	8	1	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.522	.153	1.954	.051	.571
			Time Dependent	.545	.073	1.954	.051	.571
			Occ Sq Dependent	.500	.250	1.954	.051	.571
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.674	.113	4.118	.000	.016
			Time Dependent	.638	.090	4.118	.000	.016
			Occ Sq Dependent	.714	.171	4.118	.000	.016

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Occ Sq scores; Exact significance, p= 0.571. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.016.

Crosstab

Count

Treatment			Sph > wing				
			0	1	2	3	Total
Control	Time	pre		2	0	2	4
		post		2	1	0	3
		Total		4	1	2	7
Exp	Time	pre	3	1	2	0	6
		post	0	0	3	4	7
		Total	3	1	5	4	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.308	.324	-.921	.357	.657
			Time Dependent	-.286	.298	-.921	.357	.657
			Sph > wing Dependent	-.333	.360	-.921	.357	.657
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.713	.068	7.813	.000	.012
			Time Dependent	.610	.046	7.813	.000	.012
			Sph > wing Dependent	.857	.103	7.813	.000	.012

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Sph > wing scores; Exact significance, p= 0.657. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.012.

Crosstab

Count

Treatment			Zyg L				
			0	1	2	3	Total
Control	Time	pre	1	1	2		4
		post	0	2	1		3
		Total	1	3	3		7
Exp	Time	pre	3	2	1	0	6
		post	1	2	2	2	7
		Total	4	4	3	2	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.000	.363	.000	1.000	1.000
			Time Dependent	.000	.327	.000	1.000	1.000
			Zyg L Dependent	.000	.408	.000	1.000	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.442	.192	2.264	.024	.125
			Time Dependent	.371	.160	2.264	.024	.125
			Zyg L Dependent	.548	.241	2.264	.024	.125

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Zyg L scores; Exact significance, p= 1.000. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.125.

Crosstab

Count

Treatment			Zyg R			
			1	2	3	Total
Control	Time	pre		4		4
		post		3		3
		Total		7		7
Exp	Time	pre	3	3	0	6
		post	0	2	5	7
		Total	3	5	5	13

Directional Measures

Treatment				Value	Asymp. Std. Error[b]	Approx. T[c]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.[a]				
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.742	.081	7.813	.000	.009
			Time Dependent	.655	.067	7.813	.000	.009
			Zyg R Dependent	.857	.103	7.813	.000	.009

a. No statistics are computed because Zyg R is a constant.

b. Not assuming the null hypothesis.

c. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically testable relationship between pre and post and the distribution of Zyg R scores. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.009.

Crosstab

Count

Treatment			Max L			
			0	1	2	Total
Control	Time	pre	0	2	2	4
		post	1	0	2	3
		Total	1	2	4	7
Exp	Time	pre	2	4	0	6
		post	0	1	6	7
		Total	2	5	6	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.000	.407	.000	1.000	1.000
			Time Dependent	.000	.378	.000	1.000	1.000
			Max L Dependent	.000	.441	.000	1.000	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.809	.086	9.034	.000	.003
			Time Dependent	.731	.090	9.034	.000	.003
			Max L Dependent	.905	.092	9.034	.000	.003

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Max L scores; Exact significance, p= 1.000. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.003.

Crosstab

Count

Treatment			Max R				
			0	1	2	3	Total
Control	Time	pre		2	2		4
		post		1	2		3
		Total		3	4		7
Exp	Time	pre	2	4	0	0	6
		post	0	1	4	2	7
		Total	2	5	4	2	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.167	.369	.450	.652	1.000
			Time Dependent	.167	.370	.450	.652	1.000
			Max R Dependent	.167	.370	.450	.652	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.745	.068	9.034	.000	.003
			Time Dependent	.633	.065	9.034	.000	.003
			Max R Dependent	.905	.092	9.034	.000	.003

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Max R scores; Exact significance, p= 1.000. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.003.

Crosstab

Count

Treatment			Man L 0	1	2	3	Total
Control	Time	pre	1	3	0		4
		post	1	1	1		3
		Total	2	4	1		7
Exp	Time	pre	3	1	1	1	6
		post	1	2	4	0	7
		Total	4	3	5	1	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.154	.399	.380	.704	1.000
			Time Dependent	.143	.366	.380	.704	1.000
			Man L Dependent	.167	.438	.380	.704	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.218	.273	.809	.418	.464
			Time Dependent	.186	.237	.809	.418	.464
			Man L Dependent	.262	.323	.809	.418	.464

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Man L scores; Exact significance, p= 1.000. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.464.

Crosstab

Count

Treatment			Mand R			
			0	2	3	Total
Control	Time	pre	1	3		4
		post	0	3		3
		Total	1	6		7
Exp	Time	pre	1	3	2	6
		post	1	3	3	7
		Total	2	6	5	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.333	.180	1.146	.252	1.000
			Time Dependent	.500	.204	1.146	.252	1.000
			Mand R Dependent	.250	.217	1.146	.252	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.085	.262	.325	.745	.965
			Time Dependent	.077	.236	.325	.745	.965
			Mand R Dependent	.095	.293	.325	.745	.965

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Man R scores; Exact significance, p= 1.000. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.965.

Crosstab

Count

Treatment			Pal L				
			0	1	2	3	Total
Control	Time	pre		1	2	1	4
		post		0	2	1	3
		Total		1	4	2	7
Exp	Time	pre	2	1	3	0	6
		post	1	0	5	1	7
		Total	3	1	8	1	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.231	.324	.691	.490	.771
			Time Dependent	.214	.296	.691	.490	.771
			Pal L Dependent	.250	.361	.691	.490	.771
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.382	.217	1.658	.097	.241
			Time Dependent	.362	.201	1.658	.097	.241
			Pal L Dependent	.405	.243	1.658	.097	.241

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Pal L scores; Exact significance, p= 0.771. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.241.

Crosstab

Count

Treatment			Pal R				
			0	1	2	3	Total
Control	Time	pre	1		2	1	4
		post	1		2	0	3
		Total	2		4	1	7
Exp	Time	pre		1	5	0	6
		post		0	3	4	7
		Total		1	8	4	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.231	.324	-.691	.490	.771
			Time Dependent	-.214	.296	-.691	.490	.771
			Pal R Dependent	-.250	.361	-.691	.490	.771
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.628	.120	3.755	.000	.049
			Time Dependent	.614	.097	3.755	.000	.049
			Pal R Dependent	.643	.169	3.755	.000	.049

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Pal R scores; Exact significance, p= 0.771. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.049.

Crosstab

Count

Treatment			Vomer				
			0	1	2	3	Total
Control	Time	pre	1	1	2	0	4
		post	1	0	1	1	3
		Total	2	1	3	1	7
Exp	Time	pre	4	1	1	0	6
		post	1	0	5	1	7
		Total	5	1	6	1	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.207	.366	.558	.577	.686
			Time Dependent	.176	.309	.558	.577	.686
			Vomer Dependent	.250	.447	.558	.577	.686
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.611	.178	3.343	.001	.034
			Time Dependent	.547	.164	3.343	.001	.034
			Vomer Dependent	.690	.204	3.343	.001	.034

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Vomer scores; Exact significance, p= 0.686. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.034.

Crosstab

Count

Treatment			Lac L				
			0	1	2	3	Total
Control	Time	pre	2	0	1	1	4
		post	2	1	0	0	3
		Total	4	1	1	1	7
Exp	Time	pre	3	1	2	0	6
		post	1	1	3	2	7
		Total	4	2	5	2	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.296	.304	-.921	.357	.543
			Time Dependent	-.267	.265	-.921	.357	.543
			Lac L Dependent	-.333	.360	-.921	.357	.543
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.431	.197	2.146	.032	.134
			Time Dependent	.367	.166	2.146	.032	.134
			Lac L Dependent	.524	.243	2.146	.032	.134

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Lac L scores; Exact significance, p= 0.543. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.134.

Crosstab

Count

Treatment			Lac R				
			0	1	2	3	Total
Control	Time	pre		1	2	1	4
		post		1	1	1	3
		Total		2	3	2	7
Exp	Time	pre	1	1	4	0	6
		post	0	0	1	6	7
		Total	1	1	5	6	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.000	.364	.000	1.000	1.000
			Time Dependent	.000	.319	.000	1.000	1.000
			Lac R Dependent	.000	.425	.000	1.000	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.800	.087	9.034	.000	.003
			Time Dependent	.717	.095	9.034	.000	.003
			Lac R Dependent	.905	.092	9.034	.000	.003

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Lac R scores; Exact significance, p= 1.000. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.003.

Crosstab

Count

Treatment			Nas L				
			0	1	2	3	Total
Control	Time	pre	3	0		1	4
		post	2	1		0	3
		Total	5	1		1	7
Exp	Time	pre	4		2	0	6
		post	2		3	2	7
		Total	6		5	2	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.000	.369	.000	1.000	1.000
			Time Dependent	.000	.386	.000	1.000	1.000
			Nas L Dependent	.000	.354	.000	1.000	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.426	.210	1.953	.051	.186
			Time Dependent	.385	.186	1.953	.051	.186
			Nas L Dependent	.476	.243	1.953	.051	.186

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Nas L scores; Exact significance, p= 1.000. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.186.

Crosstab

Count

Treatment			Nas R				
			0	1	2	3	Total
Control	Time	pre	1		3	0	4
		post	0		2	1	3
		Total	1		5	1	7
Exp	Time	pre	1	2	3	0	6
		post	0	0	2	5	7
		Total	1	2	5	5	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.522	.153	1.954	.051	.571
			Time Dependent	.545	.073	1.954	.051	.571
			Nas R Dependent	.500	.250	1.954	.051	.571
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.727	.076	7.813	.000	.009
			Time Dependent	.632	.064	7.813	.000	.009
			Nas R Dependent	.857	.103	7.813	.000	.009

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Nas R scores; Exact significance, p= 0.571. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.009.

Crosstab

Count

Treatment			Sacrum			
			0	1	2	Total
Control	Time	pre	1	1	2	4
		post	1	0	2	3
		Total	2	1	4	7
Exp	Time	pre	2	3	1	6
		post	0	3	4	7
		Total	2	6	5	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.077	.373	.207	.836	1.000
			Time Dependent	.071	.347	.207	.836	1.000
			Sacrum Dependent	.083	.403	.207	.836	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.489	.195	2.357	.018	.122
			Time Dependent	.442	.168	2.357	.018	.122
			Sacrum Dependent	.548	.231	2.357	.018	.122

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Sacrum scores; Exact significance, p= 1.000. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.122.

Crosstab

Count

Treatment			L5-S1			
			0	1	2	Total
Control	Time	pre	2	2	0	4
		post	1	1	1	3
		Total	3	3	1	7
Exp	Time	pre	2	4	0	6
		post	1	1	5	7
		Total	3	5	5	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.296	.342	.837	.403	.657
			Time Dependent	.267	.299	.837	.403	.657
			L5-S1 Dependent	.333	.397	.837	.403	.657
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.577	.206	2.876	.004	.048
			Time Dependent	.509	.188	2.876	.004	.048
			L5-S1 Dependent	.667	.230	2.876	.004	.048

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of Sacrum scores; Exact significance, p= 0.657. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.048.

Crosstab

Count

Treatment			INN L			
			1	2	3	Total
Control	Time	pre	3	1	0	4
		post	1	1	1	3
		Total	4	2	1	7
Exp	Time	pre	4	1	1	6
		post	0	2	5	7
		Total	4	3	6	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.462	.300	1.437	.151	.371
			Time Dependent	.429	.268	1.437	.151	.371
			INN L Dependent	.500	.344	1.437	.151	.371
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.646	.173	3.646	.000	.026
			Time Dependent	.574	.155	3.646	.000	.026
			INN L Dependent	.738	.200	3.646	.000	.026

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of INN L scores; Exact significance, p= 0.371. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.026.

Crosstab

Count

Treatment			Inn R				
			0	1	2	3	Total
Control	Time	pre	1	3	0		4
		post	1	1	1		3
		Total	2	4	1		7
Exp	Time	pre	1	4	1	0	6
		post	0	0	5	2	7
		Total	1	4	6	2	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	.154	.399	.380	.704	1.000
			Time Dependent	.143	.366	.380	.704	1.000
			Inn R Dependent	.167	.438	.380	.704	1.000
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.755	.080	7.434	.000	.003
			Time Dependent	.661	.072	7.434	.000	.003
			Inn R Dependent	.881	.112	7.434	.000	.003

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of INN R scores; Exact significance, p= 1.000. For Experimental, there is a statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.003.

Crosstab

Count

Treatment			C3			
			1	2	3	Total
Control	Time	pre	1	2	1	4
		post	2	0	1	3
		Total	3	2	2	7
Exp	Time	pre	2	4	0	6
		post	0	5	2	7
		Total	2	9	2	13

Directional Measures

Treatment				Value	Asymp. Std. Error[a]	Approx. T[b]	Approx. Sig.	Exact Sig.
Control	Ordinal by Ordinal	Somers' d	Symmetric	-.214	.386	-.558	.577	.571
			Time Dependent	-.188	.341	-.558	.577	.571
			C3 Dependent	-.250	.447	-.558	.577	.571
Exp	Ordinal by Ordinal	Somers' d	Symmetric	.537	.106	2.912	.004	.147
			Time Dependent	.550	.037	2.912	.004	.147
			C3 Dependent	.524	.178	2.912	.004	.147

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

For Control, there is no statistically significant relationship between pre and post and the distribution of C3 scores; Exact significance, p= 0.571. For Experimental, there is no statistically significant difference in the distribution of scores between pre and post; Exact significance, p= 0.147.

APPENDIX S: PILOT STUDY APPROVAL LETTER

CANADIAN COLLEGE OF
OSTEOPATHY
150 Bridgeland Ave. Suite 102, North York ON M6A 1Z5
1-416-597-0367 Fax 1-416-597-9919
www.osteopathy-canada.com

March 31st, 2011

Dear Stacey and Tom,

I have read over your request, dated March 20th 2011, to have your current thesis study reduced to a Pilot Study. I find that you have provided sufficient proof of a more than satisfactory effort to recruit subjects for your study. I'm sure that you would have liked to use the full complement of subjects but I realize that you are quickly running out of time.

I am satisfied that you are both more than ready to receive your D.O.M.P. in 2011, so you can be sure that we will get you to a jury this year. However, you've come so far it would be a shame to stop the study prematurely.

I am pleased to offer you an extension to July 6th, 2011 (an additional six weeks) to hand in your thesis Pre-Read, in order for you to get a few more subjects. This opportunity is based partly on the fact that I know that your advisor will ensure that your hand-in is already in top shape before you submit it.

If you take this offer, and don't find the 16 subjects that you require, you just might find enough to give your study the power it needs so that it can be completed as a full study.
If it turns out that you fall short of subjects then you will be required to provide some compelling scientific reasons (within the body of your thesis) as to why a pilot study is warranted. This requirement is non-negotiable.

As I can only offer you an extension on the pre-read deadline (not the final deadline) it is imperative that you both work closely with your advisor and especially your statistician (as statisticians have proven to be the least reliable member of research teams at the CCO) to ensure that you can complete the project in this new time-line without stressing out your advisor at the last minute.

This note should be included in your thesis appendix, which will permit you to officially change your title.

Good Luck!

Sincerely,

Jane Stark

Jane Stark

cc. Brad McCutcheon
cc. Ryan Marciniak

APPENDIX T: PROPOSAL